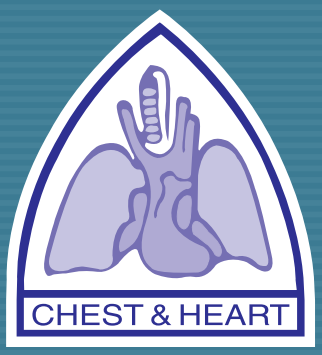
ISSN 1562 - 5044



Chest & Heart Journal

Volume 39 Number 02 July 2015

A Journal
and
Official Organ
of the Chest &
Heart Association
of Bangladesh

THE CHEST & HEART ASSOCIATION OF BANGLADESH

EXECUTIVE COMMITTEE FOR 2013-2015

President	:	Prof. Mirza Mohammad Hiron
Vice-President	:	Dr. Biswas Akhtar Hossain Dr. Bashir Ahmed Dr. Md. Rafiqul Islam
Secretary General	:	Dr. Md. Naimul Hoque (Shammi)
Treasurer	:	Dr. Krishna Chandra Ganguly
Joint Secretary	:	Dr. Md. Abu Raihan Dr. Golam Sarwar L.H. Bhuiyan
Organizing Secretary	:	Dr. Md. Mofizur Rahman Mia
Office Secretary	:	Dr. S.M. Abdur Razzaque
Members	:	Prof. Md. Rashidul Hassan Dr. Md. Abdur Rouf Dr. Mohammed Shahedur Rahman Khan Dr. Mahmud Masum Attar Dr. Md. Khairul Anam Dr. Barkat Ullah Dr. Md. Zahirul Islam Shakil Dr. S.M. Mostafa Zaman Dr. Nihar Ranjan Saha Dr. Abdullah Al Mujahid Dr. Md. Serajul Islam

THE CHEST & HEART JOURNAL

(An official organ of the Chest & Heart Association of Bangladesh)

Volume 39, Number 2, July 2015

EDITORIAL BOARD

Published by : Dr. Md. Abdur Rouf, on behalf of Chest and Heart Association of Bangladesh

Printed at : Asian Colour Printing, 130, DIT Extension Road, Fakirerpool, Dhaka-1000, Bangladesh
Phone : 9357726, 58313186, E-mail: asianclr@gmail.com

Address of Correspondence : The Editor, Chest and Heart Journal.
Association Secretariat, Administrative Block, Institute of Diseases of the Chest & Hospital.
Mohakhali, Dhaka-1212, Phone/Fax: +88-02-55067145
E-mail: chestheart@gmail.com Website: www.chestheart.org

THE CHEST & HEART ASSOCIATION OF BANGLADESH

ACADEMIC CELL

Chairman	:	Prof. Dr. Md. Shamiul Islam
Co-Chairman	:	Dr. Md. Abdur Rouf
Co-Ordinator	:	Dr. Bipul Kanti Biswas

RESEARCH & INFORMATION CELL

Chairman	:	Prof. Md. Ali Hossain
Co-Chairman	:	Dr. Md. Khairul Hassan Jessy
Co-Ordinator	:	Dr. Syed Rezaul Huq

CONTENTS

INSTRUCTION TO AUTHORS ABOUT UNIFORM MANUSCRIPT WRITING

The Chest and Heart Journal is published twice in a year in the months of January and July. The journal publishes original papers, reviews concerned with recent practice and case report of exceptional merits. Papers are accepted for publication with an understanding that they are subject to editorial revision. A covering letter signed by all authors must state that the data have not been published elsewhere in whole or in part and all authors agree their publication in Chest and Heart Journal. All submitted manuscripts are reviewed by the editors and rejected manuscripts will not be returned. Ethical aspects will be considered in the assessment of the paper. Three typed copies of the article and one soft copy in CD or Pen Drive processed all MS Word 6.0 should be submitted to the editor.

Preparation of Manuscripts

Manuscripts should be typed on one side of good quality paper, with margins of at least 25mm and using double space throughout. Each component of the manuscript should begin on a new page in the sequence of title page, abstract, text, references, tables, and legend for illustrations. The title page should include the title of the paper, name of the author(s), name of the departments) to which work should be attributed. The text should be presented in the form of Introduction, Materials and Methods, Results, and Discussion. The text should not exceed 2500 words and a word count should be supplied.

Abstracts/Summary

Provide on a separate page an abstract of not more than 250 words. This abstract should consist of four paragraphs, labeled Background, Methods, Results and Conclusions. They should briefly describe the problem being addressed in the study, how the study was performed, the salient results, and what the authors conclude from the results.

Table

Each table should be typed in on separate sheet. Table should have brief title for each, should be numbered consecutively using Roman numbers and be cited in the consecutive order, internal horizontal and vertical rules should not be used.

Results should be presented in logical sequence in the text, tables or illustration. Do not repeat in the text all data in the tables or illustrations; emphasize or summarize only important observations.

Drug Names

Genetic names should generally be used. When proprietary brands are used in research, include the brand name in parentheses in the Methods section.

Illustrations

Figure should be professionally designed symbols, lettering, and numbering should be clear and large. The back of each figure should include the sequence number and the proper orientation (e.g. “top”). Photographs and photomicrographs should be supplied as glossy black and white prints unmounted. Legend for each illustration should be submitted in separate sheets. All photographs, graphs and diagrams should be referred to as figures numbered consecutively in the text in Roman numerals.

Discussion

Emphasize the new and important aspects of the study and the conclusions that follow from them. The detail data or other material given in the Introduction or the Results section should not be repeated. The implications of the findings and their limitations, including implication for future research should be included in the Discussion section. The observations should be compared and related to other relevant studies, new hypothesis is appreciated, and however they should be clearly labeled as such. Recommendations may be included only when appropriate.

References

References should be numbered consecutively in the order in which they are first mentioned in the text. Identify references in text, tables, and legend by Roman numerals in parenthesis. Use the styles of the example below, which are based on the formats used by the US National Library of Medicine (NLM) in the Index Medicus.

Avoid using abstracts as references. References to paper accepted but not yet published should be designated as "in press" or "forthcoming"; authors should obtain written permission to cite such papers as well as verification that they have been accepted for publication. Information from manuscripts submitted but not accepted should be cited as "unpublished observations" with written permission from the source. Avoid using a "personal communication" unless it provides essential information not available from a public source. For scientific articles, authors should obtain written permission and confirmation of accuracy from the source of a personal communication.

The references must be verified by the authors(s) against the original documents.

1. Articles in Journal

a) List all six authors when six or less;
Connors JP, Roper CL, Ferguson TB. Transbronchial Catheterisation of Pulmonary Abscess. Ann Thorac Surg 1975; 19 : 254-7.

b) When seven or more, list the first three and then add et al;
Karalus NC, Cursons RT, Leng RA, et al. Community acquired pneumonia: aetiology and prognostic Index evaluation. Thorax 1991; 46 : 413-12.

c) No author given;
Cancer in South Africa (editorial). S Afr Med J 1994; 84-15.

d) Organization as author
The Cardiac Society of Australia and New Zealand. Clinical exercise stress training. Safety and performance guideline. Med J Aust 1996; 164 : 282-4.

2. Books and Other Manuscripts

a) Personal author
Tierney LM, -McPhee SJ, Papakadis MA. Current Medical Diagnosis and Treatment. Lange Medical books/Mcgrow Hill 2000.

b) Editor(s), complier(s) as author
Baum GL, Wolinsky E, editor. Text Book of Pulmonary diseases. 5th ed. New York: Little Brown Co. 1994.

c) Organization as author and publisher
World Health Organization, Ethical Criteria for Medical Drug Promotion. Geneva: World Health Organization; 1988.

d) Chapter in a book
Macnee W. Chronic bronchitis and emphysema. Seaton A, Seaton D, editors. Crofton and Douglas's Respiratory Diseases. 5th ed. UK. The Blackwell Science; 2000; p.616-95.

e) Dissertation
Kaplan SJ. Post-hospital home health care: the elderly's access and utilization (dissertation). St. Louis (MO). Washington Univ; 1995.

3. Other published material

a) Newspaper article
Lee G. Hospitalizations tied to ozone pollution: study estimates 50,000 admissions annually. The Washington Post 1996, June 21; Sect. A : 3(col. 5).

b) Dictionary and similar references
Student's medical dictionary. 26th ed. Baltimore: Williams & Wilkins; 1995. Apraxia; p.119-20.

4. Unpublished Material

a) In press
Leshner AI. Molecular mechanisms of cocaine addition. N Engl J Med In Press 1997.

5. Electronic Material

a) Journal articles in electronic format
Morse SS. Factors in the emergence of infectious diseases. Emerg Infect Dis Serial online I 1995 Jan-Mar I cited 1996 June 5 I; 1(1): 24 screens I

Available from: URL: http://www.cdc.gov/ncidod/E[D/eid.htm

Nomenclature and Abbreviation

1. Abbreviations and symbols must be standard and SI units should be used thoughtout.
2. Terms such as electrocardiogram, ultrasonogram etc. should when mentioned first, be written in full followed by accepted abbreviations (ECG, USG etc.)

Permissions

A written statement must accompany materials taken from other sources from both author and publisher giving permission to the Journal for reproduction. Obtain permission in writing from at least on a author of papers still in press, unpublished data, and personal communications.

Review and Action

Manuscripts are examined by the editorial staff and are usually sent to reviewers, but we reserve the right of final selection.

Proof

Two marked copies of the proofs may be sent to the principal author, which should be read carefully for error. One corrected copy must be returned to the editor within the next three days. Major alteration in the text can not be accepted.

Editorial Mail

Manuscripts and other communication for the editors should be addressed to

The Editor
Chest and Heart Journal
Association Secretariat, Administrative Block, Institute of Diseases of the Chest & Hospital.
Mohakhali, Dhaka-1212, Phone/Fax: 8851668

Chest & Heart Journal
Vol. 39, No. 2, July 2015

ORIGINAL ARTICLE

Role of Genexpert MTB/RIF in the Diagnosis of Tubercular Pleural Effusion

Md. Meer Mahbubul Alam[1], K C Ganguly[2], S M Mostafa Kamal[3], Shah Muhammad Saifur Rahman[4], Md. Khairul Anam[4], Md. Hasanur Rashid, [4] Mst. Shamima Akter[5], Mir Iftekhar Mostafiz[6], Bijoy Pada Gope[7], Pulok Kumar Dey[5]

Abstract

To determine the role of Gene Xpert MTB/RIF in the diagnosis of tubercular pleural effusion and to compare GeneXpert MTB/RIF with other procedures, such as, ADA level and protein in pleural effusion and pleural tissue biopsy a prospective observational analytical study was conducted in the National Institute of Diseases of the Chest and Hospital (NIDCH), Mohakhali, Dhaka. Sixty adult patient of either sex clinically highly suspicious of Tubercular pleural effusion were enrolled finally for the study.

The mean age of the patients was 36.7 ± 15.284 years. Most of the patients were below 44 years of age. Their age ranged from 18 to 72 years.

Four modalities of tests were employed to reach a diagnosis. Pleural tissue biopsy detected 38 tubercular cases(63.3%). Pleural fluid ADA was able to identify 45 patients (75%). However, the performances of AFB culture (8.3%) and GeneXpert MTB/RIF (5%) were poor.

Xpert MTB/RIF was only 5.66% sensitive to diagnose TPE correctly it showed 100% specificity and positive predictive value. The high specificity and positive predictive value of Xpert MTB/RIF test on pleural effusion gives a clinician confidence to make a diagnosis of pleural TB when Xpert MTB/RIF test on pleural fluid is positive.

The present study shows that a negative Xpert MTB/RIF test does not completely exclude the diagnosis of pleural TB given the fact that the test was unable to identify 94.3% of confirmed pleural TB cases (low sensitivity) and when Xpert MTB/RIF test was negative, 87.7% of patients still had pleural TB. The findings imply that clinical decision in combination with routine pleural fluid analysis is still crucial in the diagnosis of pleural TB in clinically and radiologically suspected patients

In conclusion, it was found that use of the Xpert MTB/RIF assay on pleural fluid samples is feasible but has low sensitivity. New, rapid and accurate tests for the diagnosis of pleural TB are still warranted.

[Chest & Heart Journal 2015; 39(2) : 69-74]

1. Junior Consultant, Chest Disease Clinic, Madaripur
2. Associate Professor, Respiratory Medicine, NIDCH, Dhaka
3. Associate Professor, Microbiology, NIDCH, Dhaka
4. Assistant Professor, Respiratory Medicine, NIDCH, Dhaka
5. Medical Officer, NIDCH, Dhaka
6. Junior Consultant, Chest Disease Clinic, Feni
7. Junior Consultant, Chest Disease Clinic, Moulvibazar

Correspondence to: Dr. Md. Meer Mahbubul Alam, Junior Consultant, Chest Disease Clinic, Madaripur

Introduction

Pleural effusion is an abnormal accumulation of fluid within pleural space. A pleural effusion is not a disease entity, rather a clinical sign of systemic or pleural disease. Tubercular pleural effusion is one of the commonest presentations of extra pulmonary tuberculosis in (25%). Other causes of pleural effusion include neoplasia (22.9%), congestive cardiac failure (17.9%), and pneumonia (14%).[1]

Tuberculosis is a disease of great antiquity. Tubercular lesion has been found in the vertebrae of Neolithic man in Europe and in Egyptian mummies dating from as early as 3700 B.C.[2]

Today TB has become the most important communicable disease in the world. In 2010, WHO estimated that there were 8.8 million cases of TB and 1.1 million deaths from the disease globally. Regionally, South-East Asia carries 36% of the TB cases, 30% in the African region, 20% in the Western Pacific region, 7% in the Eastern Mediterranean region, 4% in the Europe, and 3% in the American region. [3]

Bangladesh ranked 6th in the list of 22 highest TB burden countries in the world. According to WHO, in 2010, there were approximately 411 TB cases (all forms)/ 100 thousand population. It was estimated that, 225 new cases occur/100 thousand population/ year. Of these approximately 100/100 thousand were infectious (smear positive). It was further estimated that, about 51/100 thousand people died of TB /year. Drug resistance among the new cases was 2.5% and among the retreatment cases was 15%. Annual risk of infection was 2.14%. The HIV prevalence in adult TB patients was about less than 0.2%).[4]

A variety of diagnostic tools are used to establish the diagnosis of different causes of pleural effusion, such as, pleural fluid concentration of Adenosine deaminase (ADA), C-reactive protein (CRP), Interferon gamma (IFN-g) and GeneXpert MTB/ RIF assay. Among these, GeneXpert MTB/RIF assay of pleural fluid is highly specific for TB with a turnover time of only two(2) hours. In addition it also can provide evidence of resistance to Rifampicin, thereby facilitating MDR-TB diagnosis.

The World Health Organization (WHO) has endorsed Xpert MTB/RIF in 2010. It combines sample processing and real time PCR in a fully automated platform and detects *M. tuberculosis complex* and rifampicin resistance in less than 2 hours.[5-7] Xpert MTB/RIF has been used successfully on various extra pulmonary specimens including pleural fluid, CSF, ascitic fluid, urine, pus and stool .

Materials and Methods

Study design:

Prospective observational analytical study

Study setting:

Time: This study period was two years from January 2013 to December 2014.

Place: The study was carried out in the National Institute of Diseases of the Chest and Hospital (NIDCH), Mohakhali,

Study population:

Adult 18years & above of either sex clinically highly suspicious of Tubercular pleural effusion .

Sample selection:

Composite reference standard(CRS)

On the basis of sputum smear for AFB,sputum for GeneXpert and pleural fluid analysis for AFB, GeneXpert, protein and ADA level and in combination of clinical sign-symptoms, radiological findings and histolpathological reports a composite references standard was developed which is shown below in the table (ref: viral vadwai et al,2011)

Table-I

Composite reference standard (CRS).

	AFB Smear	AFB Culture	CRS Category	Results	Histology / Cytology	Follow Up at 3 months (with Anti Tubercular drugs)
Confirmed TB	+/ -	+	+	+/-	+/-	+
Probable TB	+/ -+/-+/-	—-	+++	++-	+-+	+++
Possible TB	+/ -	-	+	-	-	+
Not TB	-	-	+	-	-	-

NB: Confirmed TB means: AFB culture +VE
Probable TB means: Clinically, radiologically &/or histologicaly.
Possible TB means: Clinically positive & responded to anti TB therapy
Not TB means: No evidence of TB Pleural fluid exudative means: Pleural fluid protein >3gm/dl Pleural fluid transudative means : Pleural fluid protein <3gm/dl Pleural fluid ADA: e"40 U/L is taken as the cut off value.

Results

This prospective observational analytical study was done to determine the role of GeneXpert MTB/RIF in the diagnosis of tubercular pleural effusion in the National Institute of Diseases of the Chest and Hospital (NIDCH), Mohakhali, Dhaka.

Initially 96 patients were enrolled in the study but later on, diagnosis changed in 11 patients (Bronchial carcinoma with pleural effusion, lymphoma with pleural effusion),10 patients died in hospital during investigation period, 15patients failed to report for follow-up. So finally 60 patients of age 18 years & above of either sex clinically having highly suspicious Tubercular pleural effusion were included in the study.

The findings derived from the data analysis are presented below:

Some investigation findings

A total of 60 patients were finally included in the study. GeneXpert MTB/RIF was able to detect 3 patients(5%) and AFB culture was positive in 5 patients(8.3%). Pleural tissue biopsy detected 38 (63.3%) tubercular patients and pleural fluid ADA was able to identify 45(75%) patients . The results are shown below in the table II.

Table III

Results of pleural fluid GeneXpert MTB/RIF, AFB C/S, ADA, pleural biopsy among the collected samples

Tests/investigation	Positive	Negative
Xpert MTB/RIF	3 (5.0)	57 (95.0)
AFB culture	5 (8.3)	55 (91.7)
Pleural tissue histopathology	38 (63.3)	22 (36.7)
Pleural fluid ADA	45 (75.0)	15 (25.0)

Composite reference standard (CRS):

Data were placed in the CRS table and the result was confirmed TB 5(8.33%), probable TB 33(55%), possible TB 15(25%) and non TB 7(11.66%).

Table-IV

Results of different procedures according to CRS

CRS Category	Final diagnosis (total cases)
Confirmed TB	5 (8.33%)
Probable TB	33 (55%)
Possible TB	15 (25%)
Not TB	7 (11.66%)
Total	60

NB:

Confirmed TB means: AFB culture +VE

Probable TB means: clinically, radiologically &/or histologicaly.

Possible TB means: clinically positive & responded to anti TB therapy

Not TB means: no evidence of TB

Diagnostic characteristics of GeneXpert MTB/RIF

A total of 60 patients were finally selected for the study. The effectiveness of Gene Xpert MTB/RIF to rule in or rule out the tubercular pleural effusion (TPE) was assessed against the composite reference standard (CRS). It was found that Gene Xpert MTB/RIF was only 5.66% sensitive to diagnose TPE correctly while it showed 100% specificity to rule out the condition. The diagnostic accuracy of GeneXpert MTB/RIF was found to be 11.7% (Table IV).

Table-V

Sensitivity, specificity, positive and negative predictive values, and diagnostic accuracy of GeneXpert MTB/RIF compared to composite reference standard

Gene Xpert MTB/RIF	CRS +ve	CRS -ve	Sn[a] (%)	Sp[b] (%)	PPV[c] (%)	NPV[d] (%)	DA[e] (%)
+ve	3	0	5.66	100.0	100.0	12.28	11.66
-ve	5	0					

TP true positive, *TN* true negative, *FP* false positive, *FN* false negative

[a] Sn=Sensitivity = [TP/(TP + FN)] X 100

[b] Sp=Specificity = [TN/(TN + FP)] X 100

[c] Positive predictive value (PPV) = [TP/(TP + FP)] X 100

[d] Negative predictive value (NPV) = [TN/(TN + FN)] X 100

[e] Diagnostic accuracy (DA) = [(TP+TN)/(TP+TN+FP+FN)] X1

Diagnostic characteristics of AFB culture

A total of 60 patients were finally selected for the study. The effectiveness of AFB culture to rule in or rule out the tubercular pleural effusion (TPE) was assessed against the composite reference standard (CRS). It was found that AFB culture was only 9.43% sensitive to diagnose TPE correctly while it showed 100% specificity to rule out the condition. The diagnostic accuracy of AFB culture was found to be 20.0% (Table-V).

Table-VI

Sensitivity, specificity, positive and negative predictive values, and diagnostic accuracy of AFB culture compared to composite reference standard

AFB	CRS		Sn[a]	Sp[b]	PPV[c]	NPV[d]	DA[e]
culture	+ve	-ve	(%)	(%)	(%)	(%)	(%)
+ve	5	0	9.43	100.0	100.0	12.72	20.00
-ve	48	7					

TP true positive, *TN* true negative, *FP* false positive, *FN* false negative

[a]Sn=Sensitivity = [TP/(TP + FN)] X 100

[b]Sp=Specificity = [TN/(TN + FP)] X 100

[c] Positive predictive value (PPV) = [TP/(TP + FP)] X 100

[d] Negative predictive value (NPV) = [TN/(TN + FN)] X 100

[e]Diagnostic accuracy (DA) = [(TP+TN)/(TP+TN+FP+FN)] X100

Diagnostic characteristics of pleural tissue biopsy

A total of 60 patients were finally selected for the study. The effectiveness of pleural tissue histopathology to rule in or rule out the tubercular pleural effusion (TPE) was assessed against the composite reference standard (CRS). It was found that histopathology was 71.69% sensitive to diagnose TPE correctly while it showed 85.7% specificity to rule out the condition. The diagnostic accuracy of pleural tissue histopathology was found to be 73.3% (Table VI).

Diagnostic characteristics of pleural fluid ADA level

A total of 60 patients were finally selected for the study. The effectiveness of pleural fluid ADA level (cut off point e"40 IU) to rule in or rule out the tubercular pleural effusion (TPE) was assessed against the composite reference standard (CRS). It was found that pleural fluid ADA level was 84.9% sensitive to diagnose TPE correctly while it showed 57.14% specificity to rule out the condition. The diagnostic accuracy of pleural fluid ADA level was 81.7% (Table VII).

Diagnostic characteristics of some tests

A total of 60 patients were finally selected for the study. The effectiveness of pleural fluid ADA level (cut off point e"40 IU), AFB culture, pleural tissue biopsy and Gene Xpert MTB/RIF to rule in or rule out the tubercular pleural effusion (TPE) were assessed against the composite reference standard (CRS). It was found that pleural fluid ADA level had highest sensitivity (84.9%) and highest diagnostic accuracy (81.7%). Pleural tissue biopsy showed good sensitivity (71.7%) and diagnostic accuracy (73.3%). GeneXpert MTB/RIF performed poorly in these regard (sensitivity 5.7%; diagnostic accuracy 11.7% Table VIII.

Table-VII

Sensitivity, specificity, positive and negative predictive values, and diagnostic accuracy of pleural tissue biopsy compared to composite reference standard

Histopathology	CRS		Sn[a] (%)	Sp[b] (%)	PPV[c] (%)	NPV[d] (%)	DA[e] (%)
	+ve	-ve					
+ve	38	1	71.69	85.7	97.4	28.6	73.3
-ve	15	6					

TP true positive, *TN* true negative, *FP* false positive, *FN* false negative

[a] Sn=Sensitivity = [TP/(TP + FN)] X 100

[b] Sp=Specificity = [TN/(TN + FP)] X 100

[c] Positive predictive value (PPV) = [TP/(TP + FP)] X 100

[d] Negative predictive value (NPV) = [TN/(TN + FN)] X 100

[e] Diagnostic accuracy (DA) = [(TP+TN)/(TP+TN+FP+FN)] X100

Table-VIII

Sensitivity, specificity, positive and negative predictive values, and diagnostic accuracy of pleural fluid ADA level compared to composite reference standard

Pleural fluid ADA	CRS		Sn[a] (%)	Sp[b] (%)	PPV[c] (%)	NPV[d] (%)	DA[e] (%)
	+ve	-ve					
+ve	45	3	84.90	57.14	93.7	33.3	81.7
-ve	8	4					

TP true positive, *TN* true negative, *FP* false positive, *FN* false negative
[a] Sn=Sensitivity = [TP/(TP + FN)] X 100
[b] Sp=Specificity = [TN/(TN + FP)] X 100
[c] Positive predictive value (PPV) = [TP/(TP + FP)] X 100
[d] Negative predictive value (NPV) = [TN/(TN + FN)] X 100
[e] Diagnostic accuracy (DA) = [(TP+TN)/(TP+TN+FP+FN)] X100

Table-IX

Sensitivity, specificity and diagnostic accuracy of several tests

Tests	Sn[a] (%)	Sp[b] (%)	DA[c] (%)
Pleural fluid ADA	84.90	57.14	81.7
Histopathology	71.69	85.7	73.3
AFB culture	9.43	100.0	20.0
GeneXpert MTB/RIF	5.66	100.0	11.66

[a] Sn=Sensitivity [b] Sp=Specificity [c] Diagnostic accuracy (DA)

Discussion:

To determine the role of GeneXpert MTB/RIF in the diagnosis of tubercular pleural effusion a prospective observational analytical study was conducted on 60 patients of either sex clinically highly suspicious of Tubercular Pleural Effusion (TPE) in the National Institute of Diseases of the Chest and Hospital (NIDCH), Mohakhali, Dhaka.

A total of 60 patients were finally included in the study. The mean age of the patients was 36.7 ± 15.284 years. Most of the patients were below 44 years of age. Their age ranged from 18 to 72 years. Regarding sex distribution three-fourth of the patients 45 (75%) were male. Four modalities of tests were employed to reach a diagnosis. Pleural tissue biopsy was better in detecting tubercular cases (38, 63.3%). By taking e"40 IU/L as cut off point pleural fluid ADA was able to identify 45 patients (75%). However, the performances of AFB culture 5 patients (8.3%) and GeneXpert MTB/RIF 3 patients (5%) were poor.

Usually pleural TB occurred in two thirds of patients presenting with exudative pleural effusions . [8] Xpert MTB/RIF test on pleural effusion showed high specificity but low sensitivity in the diagnosis of pleural TB when compared to composite reference standard.

The high proportion of pleural TB cases among patients presenting with exudative pleural effusion obtained in our study is similar to an earlier report from the same setting where 91% of patients with exudative pleural effusion had pleural TB. [9]

While Gene Xpert MTB/RIF was only 5.66% sensitive to diagnose TPE correctly it showed 100% specificity and positive predictive value. The high specificity and positive predictive value of Xpert MTB/RIF test on pleural effusion gives a clinician confidence to make a diagnosis of pleural TB when Xpert MTB/RIF test on pleural fluid is positive. However, in this study shows that a negative Xpert MTB/RIF test does not completely exclude the diagnosis of pleural TB given the fact that the test was unable to identify 94.3% of confirmed pleural TB cases (low sensitivity) and when Xpert MTB/RIF test was negative, 87.7% of patients still had pleural TB. The findings imply that clinical decision in combination with routine pleural fluid analysis is still crucial in the diagnosis of pleural TB in clinically and radiologically suspected patients.

This study has important strengths. In order to classify the patients correctly as having pleural TB, I performed both pleural fluid AFB culture and pleural tissue histopathology that were used as the reference standard, making my findings reliable and accurate.

References:

1. Valdes L, A.D, Valley JM, Pose A, San jose E, The etiology of pleural effusion in an area with high incidence of tuberculosis. *Chest,* 1996. 109: p-158-62.
2. Seaton. A, Seaton D, Leitch AG 2008, Crofton and Douglas's respiratory diseases, 5th edition, Blackewell science Ltd, India.
3. WHO global TB control: a short update to the 2008 report, Geneva: World Health Organization; 2009 WHO coated by the global burden of TB in 2009.
4. WHO, 12 August2010,accession date.WHO endorses new rapid tuberculosis test.2010.WHO,Geneva,Switzerland.http://www.who.int/tb/features_archive/new_rapid_test/en.
5. Alvarez-uria, G, Azcona, JM & Midde, M 2012, 'Rapid Diagnosis of Pulmonary and Extrapulmonary Tuberculosis in HIV-Infected patients. Comparison of LED Fluorescent Microscopy & the Gene Xpert MTB/ RIF Assay in a District Hospital in India', *Tuberculosis Research & Treatment*, vol.3. doi: 10.1155/2012/932862
6. Boehme. c.c, et al. 2010 Rapid molecular detection of tuberculosis and rifampicin resistance. N. Eng. J. Med. 363:1005-1015.
7. Boehme.C.C,et al.2011.Feasibility,diagnostic accuracy, and effectiveness of decentralized use of the Xpert MTB/RIF test for diagnosis of tuberculosis and multidrug resistance: a multicentre implementation study.Lancet 377;1495-1505.
8. Friedrich, SO, von Groote-Bidlingmaier, F & Diacon, AH 2011, 'Xpert MTB/RIF Assay for Diagnosis of Pleural Tuberculosis', *J. Clin. Microbiol*., vol. 49, no. 12, pp. . 4341–4342.
9. Luzze, H, Elliott, AM, Joloba, ML, Odida, M, Oweka-Onyee, J, et al. 2001, 'Evaluation of suspected tuberculous pleurisy: clinical and diagnostic findings in HIV-1-positive and HIV-negative adults in Uganda', *Int J Tuberc Lung Dis*, vol. 5, pp.746–753.

Chest & Heart Journal
Vol. 39, No. 2, July 2015

ORIGINAL ARTICLE

Pericardial Effusion in Children and outcome: Experience from Tertiary Care Hospital, Noakhali, Bangladesh

Lutfan Nessa[1],Md.Abdur Rouf[2], Sukhendu Shekhar Sen[3],Md. Yeakub Ali Munshi[4], Abdullah Shahriar[2]

Abstract

Background: *Pericardial effusion requires attention because of its underlying pathology and its potential for hemodynamic compromise. Awareness of this disease has increased because of the introduction of noninvasive diagnostic techniques, such as echocardiography, computed tomography(CT), and cardiac magnetic resonance (CMR). The disease can be severe and even lethal, especially in children with immunosuppression.*

Objective: *To describe profile and outcome in children with significant pericardial effusion.*

Methods: *Hospital records of 30 children admitted with significant pericardial effusion during January 2011 to December 2015 were analyzed.*

Results: *Viral pericardial effusion was detected in eight (27%) children, tubercular seven (23.33%) and Idiopathic seven (23.33%) In equal frequencies, five(17%) bacterial followed by hypothyroid, uraemic and systemic lupus erythematosus in equal frequencies.*

Conclusion: *In our resource limited settings Echocardiography is an accurate and sensitive, noninvasive diagnostic tool. It is recognized that early recognition and diagnosis as well as timely intervention can improve the outcome of pericardial effusion in children.*

Keywords: *Echocardiography, Pericardial Effusion, Outcome*

[Chest & Heart Journal 2015; 39(2) : 75-78]

Introduction:

A pericardial effusion is an abnormal amount of fluid between the heart and the pericardium, which is the sac surrounding the heart. Pericardial effusions are associated with many different medical conditions. Most pericardial effusions are not harmful, but large pericardial effusions can cause problems by impairing heart function[1,2].

The pericardium is a tough, layered sac that wraps around the heart. When the heart beats, it slides easily within the sac. Normally, only 15-35ml of clear-yellow pericardial fluid are present between two layers, which lubricates the heart's movements within the sac.

In pericardial effusions, significantly larger amounts of pericardial fluid accumulate. Small

1. Assistant Professor, Department of Paediatrics, Abdul Malek Ukil Medical College and Jano-Neta Nurul Haque Adhunic Hospital, Noakhali, Bangladesh.
2. Associate Professor, Department of Respiratory Medicine, NIDCH, Mohakhali, Dhaka
3. Junior Consultant, Department of Paediatrics, District Sadar Hospital, Cox's Bazar
4. Junior Consultant, Department of Paediatrics, Jano-Neta Nurul Haque Adhunic Hospital, Noakhali, Bangladesh.
5. Associate Professor, Department of Paediatric Cardiology, NICVD, Sher-e-Bangla Nagor, Dhaka.

Correspondence to: Assistant Professor, Department of Paediatrics, Abdul Malek Ukil Medical College and Jano-Neta Nurul Haque Adhunic Hospital, Noakhali, Bangladesh, E-mail: lutfan.nessa@yahoo.com, M- 01718174751

pericardial effusions may contain 100 milliliters of fluid. Very large pericardial effusions may involve more than two liters of fluid.

Most pericardial effusions are caused by inflammation of the pericardium, a condition called pericarditis. As the pericardium becomes inflamed, extra fluid is produced, leading to a pericardial effusion. Pericardial effusion in children is caused by bacterial and viral infections, connective tissue disease, endocrine, metabolic disorders and malignancies[2,3]. .The first challenge to the clinician is to try to establish an etiologic diagnosis[4].Viral infections are one of the main causes of pericarditis and pericardial effusions.

In a large number of people with pericardial effusion, no cause can be identified. These are called idiopathic pericardial effusions.

When a pericardial effusion is caused by pericarditis, the main symptom is chest pain[3,4,5]. The chest pain may be made worse by deep breathing and lessened by leaning forward. When pericarditis is causing a pericardial effusion, other symptoms may include:

- Fever
- Fatigue
- Muscle aches
- Shortness of breath
- Nausea, vomiting, and diarrhea (if viral illness is present)

In people with a pericardial effusion that's not due to pericarditis, there are often no symptoms.

Large, serious pericardial effusions may cause symptoms including:

- Shortness of breath
- Palpitations (sensation that the heart is pounding or beating fast)
- Light-headedness or passing out
- Cool, clammy skin

A pericardial effusion causing these symptoms is a medical emergency and may be life threatening. Children with pericardial effusion seek medical attention because of dyspnea or nonspecific chest discomfort and the thoracic X-ray shows the presence of an enlarged cardiac silhouette with clear lungs. In any case, the finding of cardiomegaly with clear lungs should raise the suspicion of a pericardial effusion. The echocardiogram is the most available and reliable technique in order to verify the presence and the amount of a pericardial effusion; in addition, the echocardiogram offers valuable data for evaluation of hemodynamic status

The aim of this study is to give a comprehensive review of the etiology, hemodynamic Status and management of moderate and severe pericardial effusion[4].

Methods:

Records of children admitted in pediatric department of Jano-Neta Nurul Haque Adhunic Hospital, Noakhali, Bangladesh with significant pericardial effusion from January 2011 to December 2015 were retrospectively analyzed. Clinical findings and investigations, including electrocardiogram, chest *X*-ray and echocardiography findings were recorded as well as other lab studies- CBC, blood culture, serological tests, ANA, anti double stranded

DNA, C3,C4, Thyroid function and renal function were done to know the etiology Cardiac tamponade was diagnosed by either : (*a*) paradoxical pulse of more than 12mm Hg, (*b*) poor peripheral perfusion and peripheral pulses with heart rate greater than 95^{th} percentile for age, or *c*) systolic blood pressure $<5^{th}$ percentile for age with increase of systolic pressures and decrease of heart rate after pericardiocentesis[4].

Echocardiography was performed. 'Significant pericardial effusion' was defined as echo-free space more than 1cm in front of the right/left ventricle, and pericardial tamponade was confirmed in the presence of right ventricular diastolic collapse[5].

Management plan was designed according to etiology of pericardial effusion.Some patients required emergency pericardiocentesis and reffered to National Institute of Cardiovascular Disease, Shere Bangla Nagar, Dhaka where two-dimensional echocardiography guided pericardial puncture was performed in patients with tamponade. A 7 F cordis introducer sheath (Johnson and Johnson) was placed in the pericardial space over a soft tipped 0.038 Terumo (Terumo; Tokyo, Japan) guidewire.A multiholed

soft 7 F pigtail catheter was advanced into the pericardial sac and the pericardial fluid was suctioned from the catheter tip into a reservoir. The catheter and sheaths were removed when echo showed almost complete emptying of the pericardial fluid with drainage volume decreased to less than 100 mL per day for more than 48 hrs. Surgical intervention was considered in thick organized pus not amenable to catheter drainage.Pericardial fluid was sent for Gram staining, Ziehl-Neelson (Z-N) staining, cytology, biochemistry, culture and sensitivity. Outcome was measured as hospital stay,mortality, constrictive pericarditis and recurrence. A descriptive analysis was done.

Results:

Thirty children (20 boys and 10 girls) were diagnosed having significant pericardial effusion during study period. Mean age was 6.5 years (range 1 mo-13 yr). All children had tachycardia and tachypnea; fever, cough and chest pain was present in 30, 28 and 12 subjects, respectively. Jugular venous pressure was raised in nine children: two had pedal edema and nine had hepatomegaly. Distant heart sounds could be appreciated in 12 children; only one had pericardial rub. Chest roentgenography revealed cardiomegaly in 22 (73%) and bronchopneumonia in five children. Electrocardiography showed sinus tachycardia and low-voltage QRS complex in all the study subjects, and ST segment

Changes in 3 cases:

Echocardiography guided pericardiocentesis was done in 7 patients but pigtail catheter was placed only in three.

There were no pericardiocentesis-related complications. Aspirate was exudative (pericardial fluid: serum protein ratio >0.5) in all the cases. Pericardiocentesis was not done in three children; one had echocardiographic evidence of myocarditis (dilated left ventricular cavity, global hypokinesia and ejection fraction of 48%) and the other

two had disseminated tuberculosis.Seven effusions were considered tubercular based on clinical findings, positive Mantoux test , ADA, PCR or ZN stain. Five (17%) had pyogenic pericardial effusion; blood culture was positive in 4 cases [*Staphylococcus aureus :* 2 ,Streptococcus Pneumoniae:2. No organism could be isolated in one case with bronchopneumonia. Eight had viral etiology after excluding other cause

Eight patients were labeled as having idiopathic pericardial effusion. . In these patients with no apparent cause of pericardial effusion at the time of diagnosis (23%) we found that the presence of inflammatory signs (characteristic chest pain, pericardial friction rub, fever or typical electrocardiographic changes) was predictive for acute idiopathic pericarditis ,irrespective of the size of the effusion and the presence or absence of tamponade. Pericardial Effusion in 3 patients were caused by hypothyroidism, uremia and systemic lupus erythematosus respectively.

Of the 7 children with tamponade, 2 were tubercular, 2 pyogenic, 1 hypothyroid and 2 had idiopathic pericardial effusion. Appropriate treatment was administered in patients with tubercular and pyogenic effusions along with supportive treatment of congestive heart failure. Follow-up of these patients showed complete resolution of effusion in all. None of them developed constrictive pericarditis over a median period of follow-up of 12 months (range: 3 to 21 months).

Discussion:

Viral (27%) was the most common etiological diagnosis in our series of pericardial effusion followed by tubercular (23.33%), idiopathic (23.33%), bacterial (17%) and hypothyroid, systemic lupus erythematosus and uremic pericarditis were in equal frequencies which contribute (10%) . All cases had congestive heart failure while 7 had cardiac tamponade. ECHO-guided percutaneous catheter drainage and pigtail catheter insertion was an effective and safe procedure for decompressing tamponade.

The study is limited by the fact that it reflects the profile in a tertiary care referral center and hence the results can not be generalized. Moreover diagnostic workup for all viruses, and detailed immunological work-up was not done. Most children had received pre-referral antibiotics, possibly affecting bacteriological results.

Idiopathic effusions account for 20% to 40% of the cases in adults whereas they are common in children[4-6]. With the advent of antibiotic therapy, there is decline in bacterial etiology and most frequent causes are presumed to be viruses in developed, and tuberculosis in developing countries[5,7-9] did not find any case of tubercular effusion in children. Reported tubercular etiology is 30% of their cases whereas we found it to be one of the important etiology[10].

Purulent pericardial effusion is most often associated with infection at another site, with hematogeneous or direct spread to the pericardium[4,5]. The most common

concomitant site involved is usually the lung. However, in our series bronchopneumonia was present in five cases (17%) and isolated. S.*aureus* and *S.pneumoniae* were isolated. S.*aureus, H. influenzae,* and *S. pneumoniae* are the usual causative agents. We found *S. aureus* to be the most common agent for purulent pericardial effusion[10,11]. Viruses commonly causing acute pericarditis are Coxackie group B and Echovirus type 8[9]. It is difficult to distinguish active viral pericarditis from idiopathic pericarditis; many cases of community acquired idiopathic pericarditis may be due to unrecognized viral infections[12]. Idiopathic effusions constituted 23.33% of our patients, which is Not similar to that reported cases[4]. In adults it constitutes around 20 to 40% of pericardial effusions[5,6,13,15].

Etiology from developed countries is quite different from the developing world where chest trauma, postpericardiotomy syndrome, infections, immunological and idiopathic pericarditis predominate; tuberculosis was not reported in these series[4,8].Immunological, traumatic and postsurgical pericardial effusions were not found in our study.

Echocardiography-guided pericardiocentesis has a well-established diagnostic and therapeutic role[14]. We also obtained reassuring results without any significant

procedural complications. Constrictive pericarditis, a common complication of tubercular etiology, can be prevented by early diagnosis and institution of antitubercular treatment and steroids as in this study[8,15].

References:

1. Pericardial Effusion: Causes, Symptoms and Treatment. www.webmed.com/.../
2. NK Bagri, DK Yadav, S Agarwal, T Aier, V Gupta.Pericardial Effusion in Children:Experience from Tertiary Care Center in Northen India. Indian Pediatr.2014 Mar ; 51(3):211-3
3. Pediatric Infective Pericarditis: Background, Anatomy….emedicine.medscape.com/ …/89
4. Jaume Sagrista-sauleda, Axel Sarrias Merce and Jordi Soler Soler . Diagnosis and Management of pericardial effusion. World J Cardiol. 2011 May 26; 3(5): 135-143
5. Kuhn B[1], Peters J, Marx GR, Breitbart RE. Etiology, management and outcome Pediatric pericardial effusions. Pediatr Cardiol. 2008 Jan;29(1):90-4. Epub 2007 Aug 3.
6. Rheuban KS. Pericardial Diseases. *In*: Allen HD, Gutgesell HP, editors. Moss and Adams' Heart Disease in Infants, Children, and Adolescents: Including the Fetus and Young Adult. 7th *ed*. Philadelphia:Lippincott Williams & Wilkins; 2008. p.1290-9.
7. Levine MJ, Lorell BH, Diver DJ, Come PC. Implications of echocardiographically assisted diagnosis of pericardial tamponade in contemporary medical patients: detection before hemodynamic embarrassment. J Am Coll Cardiol.1991;17:59-65.
8. Ziskind AA, Palacios IF. Percutaneous balloon pericardiotomy for patients with pericardial effusions and tamponade. *In*: Topol EJ, editor. Textbook of Interventional Cardiology. 3rd *ed*. Philadelphia: WB Saunders; 1999. p.869-77.
9. Zreik H, Li J, Garson AT. Etiology and danger of pericardial effusion in infants and children. Cardiol Young. 1996;6:162-5.
10. Guven H, Bakiler AR, Ulger Z, Iseri B, Kozan M, Dorak C. Evaluation of children with a large pericardial effusion
11. Lorell BH. Pericardial Disease. *In*: Braunwald E ,editors. Heart Disease. A Textbook of Cardiovascular Medicine. 14th *ed*. Philadelphia*:* Saunders; 1997.p.1505-34.
12. Jaiyesimi F, Abioye AA, Antia AU. Infective pericarditis in Nigerian children. Arch Dis Child. 1979;54:384-90.
13. Eisenberg MJ, Oken K. Prognostic value of echocardiography in hospitalized patients with pericardial effusion. Am J Cardiol. 1994:70:934-93.
14. Mueller XM, Tevaearai HT, Hurni M, Ruchat P, Fischer AP, Stumpe F, *et al*. Long-term results of surgical subxiphoid pericardial drainage. Thorac Cardiovasc Surg. 1997;45: 65-9.
15. Thebaud B, Sidi D, Kachaner J. Purulent pericarditis in children: a 15 year experience. Arch Pediatr. 1996;3:1084-90.

Chest & Heart Journal
Vol. 39, No. 2, July 2015

ORIGINAL ARTICLE

Comparison between the Role of 6-Minute Walk Test and Spirometry to Measure the Treatment Outcome of Stable Chronic Obstructive Pulmonary Disease Patients of Different Stages

Mir Iftekhar Mostafiz[1], Md. Rashidul Hassan[2], Biswas Akhtar Hossain[3], Md. Abdul Qayyum[3], Md. Khairul Anam[4], Md Meer Mahbubul Alam[5], Bijoy Pada Gope[6], Rezaul Haque[7], Mst. Shamima Akter[8]

Abstract:

Background: *Chronic obstructive pulmonary disease (COPD) is a heterogeneous, multi-component disease associated with significant clinical burden. Current methods for assessing COPD progression mainly rely on lung function tests with a particular focus on forced expiratory volume in 1 second (FEV_1). However, exercise capacity and physical activity have been applied as an essential part of the clinical assessment of COPD beyond FEV_1 measurements. The aim of this study was to evaluate the Six minute walk distance (6MWD) in assessing the response to treatment in patients with COPD and to compare it with spirometric measurements.*

Materials and Methods: *To find out the effectiveness of 6 Minute Walk Test (6MWT) to measure the treatment outcome over time in COPD patients as an alternative to spirometry, a prospective observational comparative study was done in the Department of Respiratory Medicine in National Institute of Diseases of the Chest and Hospital (NIDCH), Mohakhali, Dhaka. The study compared 6MWT, spirometry and CAT score to assess treatment outcome after 2 weeks and 12 weeks of treatment. Ninety six patients were included in the final analysis.*

Results: *The mean age of the 96 patients was 59.76 ± 8.845 years [male (91%)]. 48% patients had stage 3 COPD; and the rest had stage 2 & 4 COPD. COPD assessment test (CAT) score, 6-Minute Walk Distance (6MWD), percentage predicted of FEV_1 and FVC were measured and compared between baseline and follow up visits. When the patients of all stages added up together, the decrease in CAT score at 1st follow-up was 3.02 ±2.576 (11.78%) and that of the 2nd follow up was 6.97 ±6.393 (27.19%). Both these decreases were statistically highly significant (<0.001). Increase in 6MWD at 1st follow-up was 144.18 ±170.44 feet that of the 2nd follow up was 262.62 ±197.00 feet. Both these increases were statistically highly significant. The significant change between baseline and follow up visits spirometric parameters were also found. In all the patients change in*

1. Junior Consultant, Chest Diseases Clinic (CDC), Feni.
2. Director cum Professor, Respiratory Medicine, NIDCH, Dhaka.
3. Associate Professor, Respiratory Medicine, NIDCH, Dhaka.
4. Assistant Professor, Respiratory Medicine, NIDCH, Dhaka.
5. Junior Consultant, Chest Diseases Clinic (CDC), Madaripur.
6. Junior Consultant, Chest Diseases Clinic (CDC), Moulvibazar.
7. Residential Medical Officer (RMO), NIDCH, Dhaka.
8. Medical Officer, NIDCH, Dhaka.

Correspondence to: Mir Iftekhar Mostafiz, Junior Consultant, Chest Diseases Clinic (CDC), Feni. *Email: drmmifti34@gmail.com*

FEV_1 at 1st follow up, was 2.49±6.45 (6.21%) which was more than the change in FVC 2.11±6.03 (3.18%). At 2nd follow up, change in FEV_1 was 3.93± 6.03 (9.80%) and change in FVC was 3.42± 9.738 (5.15%).

Conclusion: 6MWT can be used as an alternative tool in monitoring of treatment outcome of COPD patients in areas where spirometry is not available because of its safety, simplicity, inexpensiveness, better reflectivity of changes in quality of life and it signify visible improvement by patient.

Keywords: *COPD, 6-Minute Walk Test, Spirometry, FEV_1, FVC.*

[Chest & Heart Journal 2015; 39(2) : 79-86]

Introduction:

Chronic obstructive pulmonary disease (COPD), a common preventable and treatable disease, is characterized by persistent air flow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles and gases. Exacerbations and co morbidities contribute to the overall severity in individual patients. It is the 4th leading cause of death worldwide and will become the 3rd leading cause of death by 2020[1]. In Bangladesh, total burden of COPD patient is about 6 million[2].

Spirometry is the gold standard for accurate and repeatable measurement of lung function. FEV_1 is used to define the severity of the disease. But it correlates weakly with dyspnoea. Exercise test like 6-minute walk test (6MWT), can be considered to substitute the spirometry. If spirometry is not available then both staging of the disease and follow up of patient can be done on the basis of severity of symptoms, PEFR and 6-MWT[3].

COPD leads to a reduction in exercise capacity. Assessment of exercise tolerance has gained importance in tracking functional capacity changes resulting from the disease progression or therapeutic intervention[4,5]. 6-MWT was shown to be reliable and valid as an assessment for exercise tolerance for COPD[6]. It is an objective method, to measure the ability to perform daily living activities. It is more often performed to evaluate the functional status, monitor therapy or assess the prognosis in patients with COPD. The main advantages of the 6MWT are its simplicity, inexpensiveness, minimal technological requirements, good reliability and validity[7].

Previous studies have demonstrated that the 6MWT is a better predictor of mortality than FEV_1[8], result of the 6MWT has been found more reproducible than the measurement of FEV_1[9] and it was more sensitive in differentiating patients with low or high work capacity[10]. In this study, a humble effort was taken to evaluate the improvement in COPD patients following the standard treatment. The degree of improvement is translated in the increment in the 6MWD which was recorded in the follow up visits of standardized treatment and it was compared with spirometric parameters.

Materials and methods:

This prospective observational comparative study was carried out in the Department of Respiratory medicine in National Institute of Diseases of the Chest and Hospital (NIDCH), Mohakhali, Dhaka, from January 2013 to December 2014. Samples were collected from both inpatient and outpatient department (OPD). 6-minute walk test and spirometry were performed in respiratory laboratory and spirometry room of NIDCH. Prior to the commencement of the study, the research protocol was approved by the ethical committee of the NIDCH. A total numbers of 130 samples were enrolled in the study by purposive sampling technique following the inclusion and exclusion criteria. A standard questionnaire was designed with a view to collect data. Informed written consent was obtained from each and every patient after elaborative explanation regarding the undergoing study. Initial evaluation of the patient by history and clinical examination was performed and recorded in the preformed data sheet. Pulse, blood pressure, base line laboratory investigations like CBC, CXR, blood sugar, ECG, Echocardiography, liver and renal function test were done. Patients were managed as per GOLD criteria according to stage. All COPD patients after

getting optimum management when attaining at stabilize condition; spiromtry, 6 minute walk tests (6MWT) and COPD assessment test (CAT) scoring were done. It was considered as baseline parameter of this study. Thereafter spirometry, 6MWT and CAT scoring were done after two weeks and after twelve weeks of getting standard management. Spirometry was performed by spirobank G (MIR, ITALY; version winspiroPRO) and severity was defined according to the GOLD guidelines. At first patient was explained the purpose of the test and demonstrated it clearly. Patient's age, sex, race, weight, height and time of last bronchodilator use were recorded. Procedures as per American Thoracic Society (ATS) guidelines were followed. The 6MWT was also conducted according to the ATS guideline. Pulse rate, oxygen saturation (SaO_2) by pulse oxymetry and respiratory rate were assessed at the start and at the end of the 6-min walk test.

Collected data were compiled and tabulated on a master sheet. Statistical analysis was performed by using SPSS for windows version 19.0. Data were expressed as mean + SD. A value of $P < 0.05$ was considered statistically significant. Paired't' test was applied to find the significance of difference. The spirometric data and CAT score are compared to the result of the six-minute walk distance (6MWD).

Results and observations:

Initially 130 cases were enrolled in the study. Of them 96 cases (male 87, female 9) completed two follow-ups and included in the final analysis. The mean age of the patients was 59.76 ± 8.845 years. 24% patients were of stage 2 but most of the patients (48%) had stage 3 COPD. 27% were in the stage 4. No statistical analysis could be done for stage 1 COPD patient due to single representation.

Table–I shows as the severity of COPD increased the 6-Minute Walk Distance (6MWD) distance decreased. After the treatment, 6MWD increased considerably.

Table-I

Comparison of 6-Minute Walk Distance (6MWD) between baseline and follow-up visits in different stages of COPD

Stage of COPD	6MWD* Baseline (mean ± SD)	At 1st follow up (mean ± SD)	At 2nd follow up (mean ± SD)
Stage 1	1040	1090	1270
Stage 2	1122.38 ±170.443	1286.04 ±214.263	1378.68 ±203.938
Stage 3	950.96 ±319.385	1098.39 ±281.423	1212.36 ±290.717
Stage 4	774.76 ±302.763	900.00 ±313.050	1067.63 ±276.309

* Unit of measurement = feet

Table–II shows maximum increase in 6MWD at 1st follow-up 163.667 ±158.819 feet was found in stage 2; followed by 147.426 ±203.753 feet in stage 3.

Table-II

Increases in 6-Minute Walk Distance (6MWD) at 1st follow-up in different stages of COPD patients

Stage of COPD	Increase in 6MWD* (mean ± SD)	% of Increase in 6MWD	*p*-value
Stage 1	50	4.81	-
Stage 2	163.67 ±158.82	14.58	<0.001
Stage 3	147.43 ±203.75	15.50	<0.001
Stage 4	125.24 ±103.31	16.16	<0.001

* Unit of measurement = feet

Table–III shows maximum increases in 6MWD at 2nd follow-up in 37.80% (292.87 ±135.620 feet) increase was found in stage 4; followed by 261.4 ±227.58 feet in stage 3.

Table-III

Increases in 6-Minute Walk Distance (6MWD) at 2nd follow-up in different stages of COPD patients

Stage of COPD	Increase in 6MWD* (mean ± SD)	% of Increase in 6MWD	*p*-value
Stage 1	230	22.12	-
Stage 2	256.3 ±196.90	22.83	<0.001
Stage 3	261.4 ±227.58	27.49	<0.001
Stage 4	292.87 ±135.62	37.80	<0.001

* Unit of measurement = feet

Table–IV shows as the severity of COPD increased the FEV_1 (% of predicted) values also decreased. After the treatment, FEV_1 increased notably.

Table-IV

Comparison of FEV_1 between baseline and follow-up visits in different stages of COPD

Stage of COPD	FEV_1*		
	Baseline (mean ± SD)	At 1st follow up (mean ± SD)	At 2nd follow up (mean ± SD)
Stage 1	86	82	87
Stage 2	59.55 ±7.793	62.27 ±9.269	64.09 ±10.424
Stage 3	39.20 ±6.260	42.12 ±9.642	43.66 ±9.137
Stage 4	24.00 ±3.090	26.22 ±5.239	28.35 ±7.290

* Unit of measurement = % of predicted

Table–V shows maximum increase in FEV_1 (% of predicted) at 1st follow-up 2.92 ±6.670 was found in stage 3; followed by 2.72 ±7.833 in stage 2.

Table-V

Increases in FEV_1 at 1st follow-up in different stages of COPD patients

Stage of COPD	Increase in FEV_1* (mean ± SD)	% of Increase in FEV_1	*p*-value
Stage 1	-4	-4.65	-
Stage 2	2.72 ±7.833	4.57	0.017
Stage 3	2.92 ±6.670	7.45	0.003
Stage 4	2.22 ±3.437	9.25	0.005

* Unit of measurement = % of predicted

Table–VI shows maximum increase in FEV_1 (% of predicted) at 2nd follow-up 4.54 ±7.360 was found in stage 2; followed by 4.46 ±6.038 in stage 3.

Table -VI

Increases in FEV_1 at 2nd follow-up in different stages of COPD patients

Stage of COPD	Increase in FEV_1 * (mean ± SD)	% of Increase in FEV_1	*p*-value
Stage 1	1.00	1.16	-
Stage 2	4.54 ±7.360	7.62	0.009
Stage 3	4.46 ±6.038	11.38	<0.001
Stage 4	4.35 ±5.201	18.13	0.001

* Unit of measurement = % of predicted

Table-VII shows as the severity of COPD increased the FVC values also decreased. After the treatment, FVC increased notably.

Table-VII

Comparison of FVC between baseline and follow-up visits in different stages of COPD

Stage of COPD	FVC *		
	Baseline (mean ± SD)	At 1st follow up (mean ± SD)	At 2nd follow up (mean ± SD)
Stage 1	107.0	101.0	109.0
Stage 2	86.27 ±11.857	87.68 ±11.968	88.55 ±12.389
Stage 3	67.820 ±15.390	70.84 ±18.642	71.70 ±16.506
Stage 4	46.09 ±6.973	48.78 ±10.193	51.57 ±12.873

* Unit of measurement = % of predicted

Table-VIII shows maximum increase in FVC (% of predicted) at 1st follow-up 3.02 ±7.784 was found in stage 3; followed by 2.69 ±5.988 in stage 4.

Table-III

Increases in FVC at 1st follow-up in different stages of COPD patients

Stage of COPD	Increase in FVC * (mean ± SD)	% of Increase in FVC	*p*-value
Stage 1	-6	-5.6	-
Stage 2	1.41 ±11.048	1.63	0.046
Stage 3	3.02 ±7.784	4.45	0.008
Stage 4	2.69 ±5.988	5.84	0.042

* Unit of measurement = % of predicted

Table-IX shows maximum increase in FVC (% of predicted) at 2nd follow-up 5.48 ±8.949 was found in stage 4; followed by 3.88 ±10.363 in stage 3.

Table-IX

Increases in FVC at 2nd follow-up in different stages of COPD patients

Stage of COPD	Increase in FVC * (mean ± SD)	% of Increase in FVC	*p*-value
Stage 1	2.0	1.87	-
Stage 2	2.28 ±9.346	2.64	0.047
Stage 3	3.88 ±10.263	5.72	0.043
Stage 4	5.48 ±8.949	11.89	0.008

* Unit of measurement = % of predicted

Table-X shows as the severity of COPD increased the CAT score also increased. After the treatment, CAT score values decreased considerably.

Table-X

Comparison of CAT scores between baseline and follow-up visits in different stages of COPD

Stage of COPD	CAT scores		
	Baseline (mean ± SD)	At 1st follow up (mean ± SD)	At 2nd follow up (mean ± SD)
Stage 1	24.0	22.0	17.0
Stage 2	23.77 ±3.337	19.64 ±4.260	15.27 ±4.744
Stage 3	25.40 ±4.819	21.93 ±5.047	18.54 ±5.136
Stage 4	29.35 ±4.376	26.87 ±4.576	23.83 ±4.569

Table-XI shows maximum decrease in CAT score at 1st follow-up 4.13 ±2.274 was found in stage 2; followed by 3.47 ±2.871 in stage 3.

Table-XI

Decreases in CAT scores at 1st follow-up in different stages of COPD patients

Stage of COPD	Decrease in CAT scores (mean ± SD)	% of Decrease in CAT scores	*p*-value
Stage 1	2.00	8.33	-
Stage 2	4.13 ±2.274	17.37	<0.001
Stage 3	3.47 ±2.871	13.66	<0.001
Stage 4	2.48 ±1.473	8.45	<0.001

Table-XII shows maximum decrease in CAT score at 2nd follow-up 8.50 ±4.478 was found in stage 2; followed by 6.86 ±4.159 in stage 3.

Table-XII

Decreases in CAT scores at 2nd follow-up in different stages of COPD patients

Stage of COPD	Decrease in CAT scores	% of Decrease in CAT scores	*p*-value
Stage 1	7.0	29.17	-
Stage 2	8.50 ±4.478	35.76	<0.001
Stage 3	6.86 ±4.159	25.01	<0.001
Stage 4	5.52 ±2.352	18.81	<0.001

Table XIII shows comparison of changes between 6MWD, FEV_1 (% of predicted), FVC (% of predicted) and CAT score in all patients.

Table-XIII

Comparison of changes between 6MWD, FEV_1, FVC and CAT score in all patients

Parameter score	6MWD* (mean ± SD)	FEV_1 (% of predicted)	FVC (% of predicted)	CAT
Baseline	942.56 ±309.46	40.11 ±14.48	66.35 ±19.16	25.63 ±4.917
At 1st follow-up	1086.736 ±294.06	42.60 ±15.84	68.46 ±20.59	22.61 ±5.431
At 2nd follow-up	1205.18 ±286.31	44.04 ±15.90	69.77 ±19.81	18.66 ±5.383
Changes at 1st follow-up	144.176 ±170.43	2.49 ±6.45	2.11 ±6.027	3.02 ±2.576
% of increase(1st follow up)	15.3	6.21	3.18	11.78†
Changes at 2nd follow-up	262.620 ±197.00	3.93 ±6.03	3.42 ±9.738	6.97 ±6.393
% of increase(2nd follow up)	27.9	9.80	5.15	27.19†

* Unit of measurement = feet † in case of CAT score- % of decrease

Discussion:

To find out the effectiveness of 6-Minute Walk Test (6MWT) to measure the treatment outcome over time in COPD patients as an alternative to spirometry a prospective observational comparative study was done in the Department of Respiratory Medicine in NIDCH, Dhaka.

Initially 130 patients were enrolled in the study. Out of them 96 patients completed two follow ups; first one after 2 weeks of standard optimum treatment and the last one after 12 weeks of standard optimum treatment. Nine patients only completed first follow up but were missing in the second follow up. Twenty five patients did not come for any follow-up. The causes of drop out were death, later diagnosis of associated Bronchial Carcinoma, associated Ischemic Heart Diseases, other diseases and refused to come due to economic constrain. They were excluded from final analysis. Patients were analyzed at baseline and during follow ups. After getting standard optimum treatment changes in 6MWD in different stages of COPD were evaluated. Significant increase was observed in the stage 2, 3, and 4. Maximum increase (163.667±158.819 feet) was found in stage 2; followed by 147.426±203.753 feet in stage 3. But in terms of percentage, highest increase observed in stage 4 (16.2%), while in 2nd follow up maximum 37.80% (292.87 ±135.62 feet) increase was found in stage 4; followed by 261.4 ±227.58 feet in stage 3. When the patients of all stages added up together the increase in 6MWD at 1st follow-up was 144.18 ±170.44 feet and that of the 2nd follow up was 262.62 ±197.00 feet. This increase was almost double in 2nd follow up (28%) in contrast to 1st follow up (15%). Both these increases were statistically highly significant (<0.001). These finding are well comparable with the findings of Gautam et al. (2014) which found increase in 6MWD of all stages patients was 42.83±5.98m (140.52±19.62feet) (11.99±1.66%).

COPD Assessment Test (CAT) scores were decrease significantly in the stage 2, 3, and 4. At 1st follow up, maximum decrease 4.13 ±2.278 was found in stage 2; followed by 3.47 ±2.871 in stage 3. In terms of percentage, highest decrease observed in stage 2 also (17.37%), while in 2nd follow up maximum decrease 8.50 ±4.478 was found in stage 2; followed by 6.86 ±4.159 in stage 3. In terms of percentage, highest decrease observed in stage 2 also (35.76%). When the patients of all stages added up together the decrease in CAT score at 1st follow-up was 3.02 ±2.576 (11.78%) and that of the 2nd follow up was 6.97 ±6.393 (27.19%). Both these decreases were statistically highly significant (<0.001). In case of $FEV_{1,}$ significant increase was observed in the stage 2, 3, and 4. At 1st follow up, maximum increase in FEV_1 2.92 ±6.67 was found in stage 3; followed by 2.72 ±7.833 in stage 2. But in terms of percentage, highest increase observed in stage 4 (9.25%), while in 2nd follow up maximum increase 4.54 ±7.360 was found in stage 2; followed by 4.46 ±6.038 in stage 3. But in terms of percentage, highest increase observed in stage 4 (18.13%). In case of FVC, significant increase was observed in the stage 2, 3, and 4. At 1st follow up, maximum increase 3.02 ±7.784 was found in stage 3; followed by 2.69 ±5.988 in stage 4. But in terms of percentage, highest increase observed in stage 4 (5.84%), while in 2nd follow up maximum increase 5.48 ±8.949 was found in stage 4; followed by 3.88 ±10.363 in stage 3. In terms of percentage, highest increase observed in stage 4 as well (11.89%).

When the patients of all stages added up together, at 1st follow up, change in FEV_1 was 2.49 ±6.45 (6.21%) which was more than the change in FVC 2.11 ±6.03 (3.18%). At 2nd follow up, change in FEV_1 was 3.93 ± 6.03 (9.80%) and change in FVC was 3.42 ± 9.738 (5.15%). These changes were statistically significant (p<0.001). Similar findings were reported by Gautum et al. (2014)[11] and Maji et al. (2013)[12].

In this study, it was found that standard treatment is associated with significant improvement in exercise capacity and pulmonary function tests such as 6-minute walk distance, FEV_1 and FVC. In a study Puhan et al. (2008) included 460 COPD patients with a mean ± SD forced expiratory volume in one second (FEV_1) of 39.2±14.1 % predicted and 6MWD of 361 ± 112 m at baseline[13]. In the present study the mean ± SD of FEV_1 was 52.19 ±14.48% and 6MWD was 942.56±309.46 feet at baseline. 6-min walk distance should change by <"115 feet (10% change of baseline) for patients with moderate to severe COPD in order to represent an important effect of treatment. In the current study, the change in 6MWD at 1st follow up was 144.18 feet (15.3%) from baseline which is

practically similar. But at 2nd follow up the increase much higher (27.9%).

Conclusio:

This study showed that there is significant improvement in 6MWD in response to treatment in COPD patients. This improvement was more than the improvement in FEV_1 and FVC and almost similar to CAT score. So, 6MWT can be considered as an alternative tool in monitoring of treatment outcome of stable COPD patients of different stages in areas where spirometry is not available because of its safety, simplicity, inexpensiveness, better reflectivity of changes in quality of life and it signify visible improvement by patient as patient can't understand improvement in FEV_1. Its validity to assess the treatment outcome of COPD patients can be further justified by large, multi-centered studies.

References:

1. Barbera JA, Buist AS, Calverley P, Celi B, Eliott MW et al., *Global Initiative for Chronic Obstructive Lung Disease*: Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease (rev.) 2014. http://www.goldcopd.org
2. Hassan MR, Hossain A, Mahmud AM, Asraf M, *Bangladesh Lung Health Manual*. Dhaka: Bangladesh Lung Foundation, 2010; 2.
3. Maheshwari VD, Gupta R, Khandelwal S, *International J. of Healthcare & Biomedical Research,* 2013; 1(3): 217-226
4. Butland RJ, Pang J, Gross ER, Woodcock AA & Geddes DM, Two, six and 12-minute walk tests in respiratory disease, *BMJ,* 1982; 284: 1607-1608.
5. Poualin M, Durand F, Palomba B, Ceugniet F, Desplan J & Varray A, Six-minute walk testing is more sensitive than maximal incremental cycle testing for detecting oxygen desaturation in patients with COPD, *Chest*, 2003; 123: 1401-1407.
6. Casanova C, Cote C, Marin JM, Pinto-Plata V, de Torres JP, Aguirre-Jaime A, et al., Distance and oxygen desaturation during the 6-min walk test as predictors of long-term mortality in patients with COPD. Chest 2008; 134:746-52.
7. ATS Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories. ATS statement: guidelines for the six-minute walk test. *Am J Respir Crit Care Med*. 2002; 166(1):111-7.
8. Pinto-Plata VM, Cote C, Cabral H, Taylor J & Celli BR, The 6-minute walk distance: change over time and value as a predictor of survival in severe COPD, *Eur Respir J*, 2004; 23: 28-33.
9. Knox AJ, Morrison JF & Muers MF, Reproducibility of walking test results in chronic obstructive airways disease, *Thorax*, 1988; 43: 388-392.
10. American Family Physician. 2004 Jan 1; 69(1):172-174.
11. Gautam AK, Chaudhri S, Kumar A, Bhagoliwal A, Gupta AK & Kumar A, Role of six minute walk test in comparison with spirometry to assess treatment in Chronic Obstructive Pulmonary Disease, *Indian Streams Research Journal,* 2014; 4:(4) ISSN 2230-7850. Retrieved October 28, 2014 from www.isrj.net
12. Maji A, Kundu A & Sarkar S, Correlation study between post-bronchodialator FEV1 and six minute walk distance in chronic obstructive pulmonary disease patients, conference paper, Conference of Association of Chest Physicians, Kolkata, India, 2013.
13. Puhan MA, Mador MJ, Held U, Goldstein R, Guyatt GH and Schunemann HJ, Interpretation of treatment changes in 6-minute walk distance in patients with COPD, *Eur Respir J,* 2008; 32: 637–643 .

Chest & Heart Journal
Vol. 39, No. 2, July 2015

ORIGINAL ARTICLE

Atrial Septal Defect Closure: Comparison of Right Vertical Infra-Axillary Thoracotomy(RVIAT) and Standard Median Sternotomy

M Anisuzzaman[1], MG Kibria[2]

Abstract:

Background: This study aims to evaluate whether or not the method of right vertical infraaxillary minithoracotomy (RVIAT) is preferable to and as reliable as conventional sternotomy surgery, and also assesses its cosmetic results.

Method: Twenty five patients (7 males, 18 females) with atrial septal defect were admitted to the Cardiovascular Surgery Clinic of Urobangla Heart Hospital and National Institute of Cardiovascular Diseases & Hospital, Dhaka from December 2007 until January 2012. The patients' ages ranged from 7 to 25. Patients who underwent right vertical infra axillary minithracotomy were assigned to group I, and those undergoing conventional sternotomy, to group II. Group I and group II were compared with regard to the pre- operative, perioperative and postoperative variables. Group I included 7 females and 4 males with an average age of 16.9±8.6. Group II comprised 11female and 3 male patients with an average age of 18.7±9.7 showing similar features and pathologies. The cases were in Class I–II according to the New York Heart Association (NYHA) Classification, and patients with other cardiac and systemic problems were not included in the study. The ratio of the systemic blood flow to the pulmonary blood flow (Qp/Qs) was 1.8±0.3. The average pulmonary artery pressure was 35±10 mmHg. Following the diagnosis, elective surgery was planned.

Results: No significant difference was detected in the average time of the patients' extraportal circulation, cross-clamp and surgery ($p>0.05$). In the early postoperative period of the cases, the duration of mechanical ventilator support, the drain- age volume in the first 24 hours, and the hospitalization time in the intensive care unit were similar ($p>0.05$). Postoperative pains were evaluated together with narcotic analgesics taken intravenously or orally. While 7 cases (43.7%) in group I needed postoperative analgesics, 12 cases (70.6%) in group II needed them. No mortality or major morbidity has occurred in the patients. The incision style and sizes in all of the patients undergoing RVIAT were preserved as they were at the beginning. Furthermore, the patients of group I were mobilized more quickly than the patients of group II. The patients of group I were quite pleased with the psychological and cosmetic results. No residual defects have been found in the early postoperative period and after the end of the follow-up periods. All of the patients achieved functional capacity per NYHA. No deformation of breast growth has been detected during 18 months of follow-up for the group I patients, who underwent RVIAT.

Conclusion: To conclude, the repair of atrial septal defect by RVIAT , apart from the limited working zone for the surgeon in these pathologies as compared to sternotomy may be considered in terms of the outcomes, and early and late complications. And this has accounted for less need of analgesics and better cosmetic results in recent years.

[Chest & Heart Journal 2015; 39(2) : 87-92]

1. Department of Cardiac Surgery, NICVD, Dhaka
2. Associate Professor, Department of Cardiac Surgery, NICVD, Dhaka

Correspondence to: Dr. M Anisuzzaman, Department of Cardiac Surgery, NICVD, Dhaka

Introduction:

Atrial septal defect (ASD) closure presents a normal life expectancy and quality of life to children and young adults [1]. Median sternotomy is the surgery technique applied in the treatment of congenital lesions. However, this method causes trouble due to poor cosmetic results and sternot- omy-related complications. Minimally invasive interventions have been preferred in recent years [2-6] in children and young adults, particularly females. In the postoperative period of minimally invasive interventions, less pain is observed and the hospitalization period is shorter [3,7]. The purpose of our study is to evaluate whether or not the right vertical infraaxillary minithoracotomy (RVIAT) technique is preferable to conven- tional sternotomy surgery in 25 patients who presented to our institution with secundum ASD. We also evaluated the con- sistency of results and the quality of cosmetic outcomes.

Methods:

Twenty five patients (7 males, 18females) with ASD were admitted to the Cardiovascular Surgery Clinic of National Institute of Cardiovascular Diseases and Urobangla Heart Hospital,Dhaka, Bangladesh from December 2008 until January 2013. The pa- tients' ages ranged from 7to 25 years. Surgery was performed with right vertical infraaxillary minithracotomy for the patients in group I and it was performed with conventional standard median sternotomy for those in group II. Undergoing different operations with different approaches, the group I and group II patients were compared with regard to preoperative, peroperative, and postoperative variables. Group I included 7females and 4 males with an average age of 16.9±8.6. Group II comprised 11 female and 3 male patients at an average age of 18.7±9.7 showing similar features and pathologies. The cases were in Class I–II ac- cording to the New York Heart Association (NYHA) Classification and patients with other cardiac and systemic problems were not included in the study. The ratio of the systemic blood flow to the pulmonary blood flow was (Qp/Qs) 1.8±0.2. The average rate of pulmonary artery pres- sure was 35±10 mmHg. Following the diagnosis, surgey was planned under elective conditions. All of the patients and their relatives were informed about the probability of median sternotomy as needed during the operation, and they signed Atrial Septal Defect Closure via Minithoracotomy and give their consent prior to the surgery. All of the patients of both of groups were prepared for the surgery with the same anaesthetic evaluation and application (such as electrocardiography and monitoring of arterial blood pressure, and urinary follow-up with a Foley catheter). Factors such as age, gender, and the presence of other diseases not only constituted the preoperative variables (Table 1) but also affected the decision of the surgeon about whether or not to apply a minimally invasive intervention through a limited incision. In selecting the patients, the hematologic values, the presence of other diseases, and the eligibility of the physical structure for an easy RVIAT application were concerned. Patients with the Fossa ovalis type of ASD were included in group I. Within our study, the patients of the group I underwent the surgery via RVIAT and direct aortic canulation while the group II patients underwent surgery by conventional sternotomy. Extracorporeal circulation (ECC) was started once activated clotting time exceeded 400 seconds after the canula- tion for all the patients. Arterial canulation through the as- cending aorta, bicaval venous canulation, were applied to all of the patients. Aortic arterial canulation, bicaval venous canulation were performed with stand- ard manipulations through the limited minithoracotomy. Except for the thoracic retractor for RVIAT, the same materi- als were used in the ECC and surgery for all of the cases. For the myocardial protection of the patients of both groups, systemic mild hypothermia at 32oC, cold blood cardioplegia, and outer cold saline administration were applied. After more or less equalizing all of the preoperative parameters, the patients of both groups had surgery through the sternal incision or minithoracotomy with suitable

Table-I

Demographic features of the patients undergoing surgery for atrial septal defect

Group I Group II

Group	Average age	Female/Male
Group-I	16.9±8.6	7/4
Group-II	18.7±9.7	11/3

surgery techniques for their existing cardiac lesions. A limited length RVIAT (range, 7 to 12cm) was applied in group I and median sternotomy 12 to 15cm incision were used for all of the patients of group II.

Table-II
Features of the patients undergoing surgery for ASD

Variable	Value
ASD type:	
Fossa ovalis	14
posterior inferior	10
sinuous venosus	01
Qp/Qs	1.8±0.3
Surgical repair:	1
Direct closure	02
Patch closure	23

Qp/Qs: the ratio of the systemic blood flow to the pulmonary blood flow.
ASD, atrial septal defect; PAP, pulmonary artery pressure.

The RVIAT incision was placed at the fourth or fifth intercostal spaces (Fig. 1). In both of the groups, standard canulation methods were applied for the ECC. Within this study, ECC time, cross clamp (CC) time and duration of surgery were considered the perioperative variables (Table 2). The post- operative variables were the mechanical ventilation times, hospitalization time in the intensive care unit (ICU), overall hospitalization time, mediastinal drainage volume in the first 24 hours, and volumes of transfused blood and blood prod- ucts (Table 3). The ECC time, CC time, duration of surgery, and mechanical ventilation time weren't measured in minutes, the postoperative ICU hospitalization time was measured in hours, the inpatient time was measured in days, the mediastinal drainage volume in mL/m2 of BSA (in the first 24 hours). In the statistical analysis, for the comparison of variables complying with the normal distribution, the Student t-test and chi-square test were applied, and to compare variables without a normal distribution, the Mann-Whitney U and Fisher's exact tests were used. Values± were given as the average standard error. Values with $p<0.05$ were accepted as statistically significant.

Results:

In comparing the average duration of the patients' ECC, their CC of time, and the duration of the surgery ($p>0.05$), no significant difference was

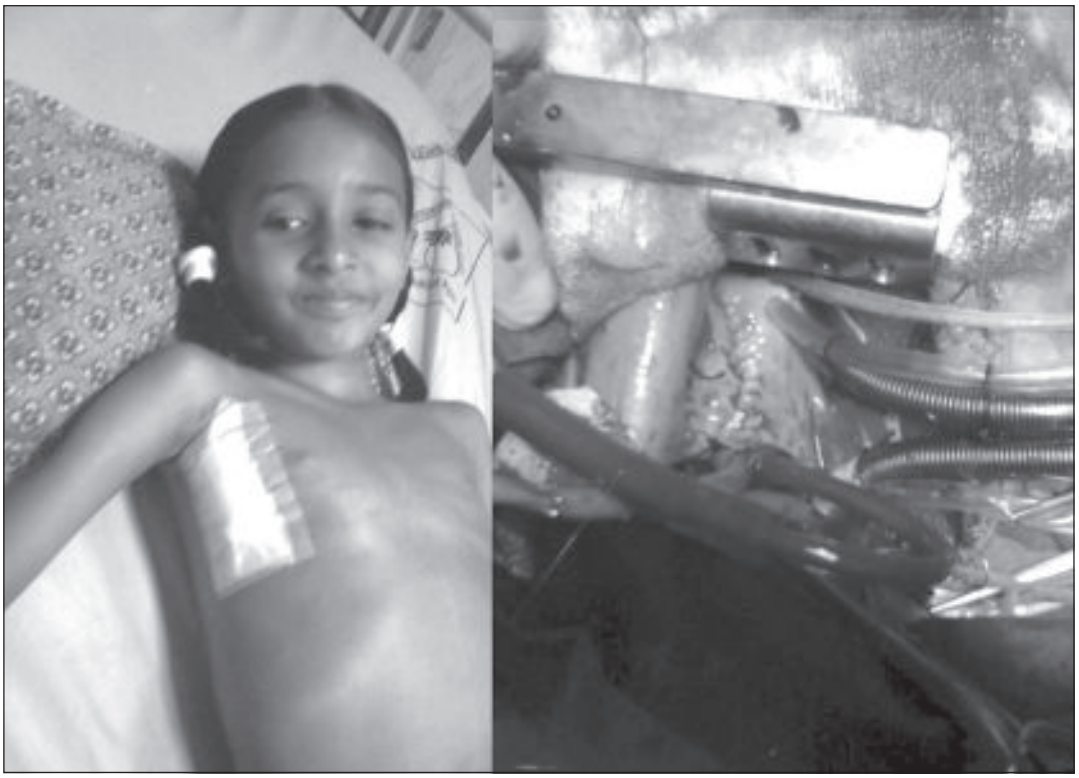


Fig.-1: *Right ventrical infraaxillary minithoracotomy technique*

Table-III

Peroperative and postoperative variables

Variable	Group-I	Group-II	Pvalue
MeanCC time(min)	11.011.3±1.3	10.8±1.2	NS
Mean CPB time (min)	17.3±2.8	16.9±2.6	NS
Mean operation time(min)	165±15	149±11.3	0.063
Mean ICU stay(hr)	22±3.6	23±2.6	NS
Mean hospital duration (day)	5.2±1.3	7.5±1.6	NS
Mean drainage/24hrs(ml/m2)	134±56	144±46	NS
Analgesic required(%)	43.6	78.4	<0.05

CC, cross clamp; CPB, cardiopulmonary bypass; ICU, intensive care unit; NS, non-spesific.

detected between the two groups. In the early postoperative period of the cases, the duration of mechanical ventilator support, the drainage amount in the first 24 hours, and the hospitalization time in the in- tensive care unit were similar in the two groups (p>0.05). The dosages of analgesics used were determined according to the need of the patients. Postoperative pain was evaluated together with narcotic analgesics taken intravenously or orally. While 7 cases (43.6%) in group I needed postoperative narcotic analgesics, 12 cases (78.4%) in group II needed them. The first 24 hours of postoperative follow-up of the cases is shown in Table 2. No mortality or major morbidity was found in the patients. The incision style and sizes in all of the patients undergoing RVIAT were preserved as they were at the beginning. No residual defects were found in the early post- operative period and at the end of the follow-up period.

All of the patients achieved a functional capacity as per NYHA. No deformation of breast growth was detected at 18 months' follow-up for the group I patients, who underwent RVIAT

Discussion:

Median sternotomy has been the gold standard in the repair of congential cardiac defects since Gibon closed an ostium secundum type ASD in 1953 [8]. However, the use of mini- mally invasive operations in the treatment of children and non-complicated young adults has been growing, and this technique has become the preferred method for the pediatric population and young adults.

In terms of cosmetic results, RVIAT is superior to median sternotomy and right posterolateral thoracotomy [9]. In the repair of small cardiac congenital defects like ASD, drawbacks of median sternotomy is include the length of the incision in the median sternotomy, postoperative pain, non-ideal cosmetic results, and possible complications of the sternotomy (medias- tinitis, osteomyelitis, etc.) [10]. The major advantage of minimally invasive cardiac surgery is avoiding sternotomy. Lan- caster et al. [11] repoted that the surgical scar was larger than the patient or surgeon expected in 58% of median sternotomy patients who were following at 1 and 5 months postoperatively. Experience with minimally invasive thoracic and cardiac surgery has shown that the surgical method has more reliable outcomes, minimizes surgical complications, provides rapid and functional healing, shortens the hospitalization time, and accordingly reduces the cost. Within these series, morbidity and mortality have not been observed. Outcomes have been almost perfect [11].

Most surgeons have preferred anterolateral thoracotomy in the closure of ASD [12]. The advantage of this approach is the field of vision. Its disadvantage is the dissection of large muscle zones and soft tissues, and accordingly, it may cause the deformation of muscles, decrease in the sensitivity of papilla, poor development of the breast and pectoral muscles and cosmetic problems [13].

There are certain advantages and disadvantages of the use of RVIAT as the incision for a minimally invasive approach as compared to the application of ministernotomy or subxiphoid approaches within our study. Either the ministernotomy or

subxiphoid approach provides direct access to the heart from front, which is the angle from which surgeons most often approach the heart. RVIAT approaches the heart from a different point of view, and all of the anatomic struc- tures in the mediastinum are perceived in a different orientation. However, the difference in orientation can be coped with after having only a bit of experience. As com- pared to the incisions that allow for front access to the mediastinum, one of the other advantages is the lower possibility of adhesion between the reverse side of the sternum and mediastinal structures in subsequent mediastinal surgeries. ECC times and CC times showed no detectable differences be- tween the two groups that we included in our study because the same techniques were applied in the repair of existing pathology except for location of the incision. Because of the fact that these cardiac defects were applied with minithoracotomy from a restricted space, surgery may last longer in the minithoractomy [14]. The study by Liu et al. [14] has demonstrated that the duration of surgery of the patients whose ASD is closed with minithoracotomy is longer, but their hospitalization time is shorter. Even though it was not a statistically significant difference in the minithoracotomy cases when we compared with conventional sternotomy, the duration of surgery was found to be longer time and no need Huseyin Hakan Poyrazoglu, et al arose for additional interventions. Similarly, in comparing the two operation types, no difference in the intensive care unit hospitalization time, drainage volume, or blood transfusion volume was detected. If and when the appropriate intercostal space is used during the RVIAT (typically intercostal space 4 or 5), the access to the aorta and the canulation of the aorta is easily ensured. In case a higher or lower intercostal space were needed, the angle of vision can be enlarged by separating the rib in the middle from the costacondral joint. The canulation of the aorta from the minithoracotomy incision can prevent the need for another incision and additional complications that may stem from the femoral artery. By applying aorta-bicaval canulation, we have avoided femoral artery and vein canulation in such cases. This has not caused any restrictions in the working field or visual field. Mishra et al. [15] reported that, in their experience, this method provides maximum security and requires less drainage,a lower transfusion volume, and less re-exploration and stated that it shortens the intensive care unit stay and offers early recovery as well. In case of before having a thoracic surgery operation, in which the right hemithorax adhesion has developed, the ac- cess to the mediastinum and the cannulation from this point through mini thoracotomy are quite challenging. Therefore, in selecting the cases, patients should be examined thoroughly for these anatomic changes and a plan for surgery should be arranged in accordance with these changes.

One of the most feared and serious complications in con- ventional median sternotomy is an infection of the sternum. A particularly deep sternum infection produces mediastinitis, which causes a high rate of morbidity and mortality [16]. On the other hand, in a minimally invasive intervention, the incidence of postoperative mediastinitis is quite low and scar site pain is minimized. No incisional or pleural infections were found in the patients undergoing sternotomy or thoracotomy in our study. In spite of the fact that greater pleural pain is expected with thoracotomy, Salzer et al. [17] noted that post-thoracotomy costal fractures do not cause the de- formation of costovertebral joints or the exacerbation of chest drainage. In our study, resection and division of the rib were not needed. Due to the small incision, 1-2 pericostal sutures have been adequate to stabilize the thoracic wall. In the 70.6% of the cases with a RVIAT , analgesics were not needed, and as compared to sternotomy cases, the use of analgesics was significantly lower($p<0.05$). Because we preserved the latissimus dorsi and serratus anterior muscles, our patients did not experience any pain or limitation in arm movement. Furthermore, the thoracotomy patients did not face any restrictions in their position when lying down or in their daily activites (such as riding in a car or lifting a weight). In comparison with right posterolateral thoracotomy, Baeza and Foster [18], who applied the right vertical infraaxillary thoracotomy for the first time, have revealed that this technique has functional and cosmetic advantages.

To conclude, repair of ASD with RVIAT technique, apart from the limited working zone for the surgeon as compared to sternotomy, has a number

of advantages including the reliability of surgical outcomes, similar early and late complications, less need for analgesics, and better cosmetic results years later. Additionally, when thinking an early recovery and avoiding sternum immobilization and sternum infections, we are of the opinion that RVIAT is a reliable alternative to sternotomy.

References:

1. Park PW, Roh JR, Kim CW, Suh KP, Lee Y. Secundum type atrial septal defect. Korean J Thorac Cardiovasc Surg 1981;14:241-6.
2. Burke RP. Minimally invasive techniques for congenital heart surgery. Semin Thorac Cardiovasc Surg 1997;9:337-44. 3. Massetti M, Babatasi G, Rossi A, et al. Operation for atrial septal defect through a right anterolateral thoracotalomy: current outcome. Ann Thorac Surg 1996;62:1100-3.
4. Rosengart TK, Stark JF. Repair of atrial septal defect throu- gh a right thoracotomy. Ann Thorac Surg 1993;55:1138-40. 5. Cremer JT, Boning A, Anssar MB, et al. Different approaches for minimally invasive closure of atrial septal defects. Ann Thorac Surg 1999;67:1648-52.
6. Black MD, Freedom RM. Minimally invasive repair of atrial septal defects. Ann Thorac Surg 1998;65:765-7.
7. Chang CH, Lin PJ, Chu JJ, et al. Surgical closure of atrial septal defect: minimally invasive cardiac surgersternotomy? Surg Endosc 1998;12:820-4.
8. Abdel-Rahman U, Wimmer-Greinecker G, Matheis G, et al. Correction of simple congenital heart defects in infants and children through a minithoracotomy. Ann Thorac Surg 2001;72:1645-9.
9. Yang X, Wang D, Wu Q. Repair of atrial septal defect through a minimal right vertical infra-axillary thoracotomy in a beating heart. Ann Thorac Surg 2001;71:2053-4.
10. Nicholson IA, Bichell DP, Bacha EA, del Nido PJ. Minimal sternotomy approach for congenital heart operations. Ann Thorac Surg 2001;71:469-72.
11. Lancaster LL, Mavroudis C, Rees AH, Slater AD, Ganzel BL, Gray LA Jr. Surgical approach to atrial septal defect in the female. Right thoracotomy versus sternotomy. Am Surg 1990;56:218-21.
12. Grinda JM, Folliguet TA, Dervanian P, Mace L, Legault B, Neveux JY. Right anterolateral thoracotomy for repair of at- rial septal defect. Ann Thorac Surg 1996;62:175-8.
13. Cherup LL, Siewers RD, Futrell JW. Breast and pectoral Atrial Septal Defect Closure via Minithoracotomy muscle maldevelopment after anterolateral and posterolateral thoracotomies in children. Ann Thorac Surg 1986;41:492-7.
14. Liu YL, Zhang HJ, Sun HS, Li SJ, Su JW, Yu CT. Correc- tion of cardiac defects through a right thoracotomy in child- ren. J Thorac Cardiovasc Surg 1998;116:359-61.
15. Mishra YK, Malhotra R, Mehta Y, Sharma KK, Kasliwal RR, Trehan N. Minimally invasive mitral valve surgery thro- ugh right anterolateral minithoracotomy. Ann Thorac Surg 1999;68:1520-4.
16. Kluytmans JA, Mouton JW, Ijzerman EP, et al. Nasal car- riage of Staphylococcus aureus as a major risk factor for wound infections after cardiac surgery. J Infect Dis 1995; 171:216-9.
17. Salzer GM, Klingler P, Klingler A, Unger A. Pain treatment after thoracotomy: is it a special problem? Ann Thorac Surg 1997;63:1411-4.
18. HongLuo,Jing Wang,Chenhui Qiao, Xin Zhang, Weihua Zhang,Laichun Song. Evaluation of different minimally invasive techniques in the surgical treatment of atrial septal defect. The Journal of Thoracic and Cardiovascular Surgery2014;148:;188-90.

Chest & Heart Journal
Vol. 39, No. 2, July 2015

ORIGINAL ARTICLE

Childhood Obesity and Its Association with Asthma in School Children - A Cross Sectional Study

Md. Saifuddin Khaled[1], Jahangir Khan [2], Firoza Akter[3], Khadija Rahman[4], Biswas Akhtar Hossain[4]

Abstract:

Background:*Obesity and asthma are diseases of high prevalence in childhood with significant increases in the last two decades. Obesity has been identified as a major risk factor for higher prevalence of asthma in children.*

Objective: *To investigate the association between the prevalence of childhoodasthma and obesity among school going children.*

Methods: *This cross-sectional study was conducted among 453 school children of 7-10 years of age through cluster sampling. The International Study of Asthma and Allergies in Childhood (ISSAC) questionnaire was used to identify the children having asthma and it's categorization. Body mass index (BMI) was calculated from the measured weight and height of the children as weight in kilogram (Kg) divided by square of height in meters (kg/m---^{2}). Centre for Disease Control and Prevention (CDC) BMI-for-age growth charts were used. Over weight was defined as a BMI between the 85th and 94th centiles and obesity was defined as a BMI equal to or greater than the 95th centile. The relationship between asthma and obesity and over-weight were determined by using chi-square tests.*

Results: *The prevalence of ever wheezing, current wheezing, obesity and overweight was 19.67%, 6.8%, 17.6% and 12.9% respectively.*

Conclusion: *An association was found between childhood asthma and obesity and overweight status among school-age children of both sex.*

Key words: *asthma, obesity, children, wheezing.*

[Chest & Heart Journal 2015; 39(2) : 93-96]

Introduction:

Asthma is a chronic inflammatory airway disorder with variable airflow obstruction causing recurrent episodes of wheezing, breathlessness and tightness of chest. Asthma is a major public health problem worldwide. An increasing prevalence and severity of asthma have been reported globally over the past few decades in certain geographical region [1]. Obesity has been identified as a risk factor for higher prevalence of asthma and asthma related symptoms in children[2]. Association between asthma and obesity have been observed in different cross-sectional studies in adults and children[3,4]. The substantial parallel increases in the prevalence of obesity and asthma observed over recent decades have led to the suggestion that obesity may be causally implicated in the risk of developing asthma [5]. The obesity is a potentially modifiable risk factor in which its relationship to asthma should be clarified [6]. So far knowledge, No such

1. Asst. Professor, Pediatric pulmonology, National Institute of Diseases of the Chest and Hospital (NIDCH).
2. Lecturer (Biochemistry), Chittagong Medical college.
3. OSD, DGHS, Mohakhali, Dhaka.
4. Asst. Professor (Pediatrics), Institute of Child Health & SSF hospital, Mirpur, Dhaka.
5. Associate Professor of Respiratory Medicine, National Institute of Diseases of the Chest and Hospital (NIDCH).

Correspondence to: Dr. Md. Saifuddin Khaled, Asst. Professor, Pediatric pulmonology, National Institute of Diseases of the Chest and Hospital (NIDCH), E-mail: drkhaledrmc@gmail.com Mobile: 01711-900665.

study has been conducted yet in this country. So, the present study was conducted to determine whether obesity, as measured by BMI was associated with a higher prevalence of asthma in children.

Methods:

This was a cross-sectional study carried out in a primary school located in Dhaka city in the month of October 2012. All the students of primary section, age belonging to 7-10 years were included in the study. Official permission was obtained from Head teacher of the participating school and parents gave their informed consent to their child's participation. Age was determined by consulting school records. The International Study of Asthma and Allergies in Childhood (ISSAC) questionnaire [7] was used to identify the children currently suffering from asthma. Standard guidelines for translation of the questionnaire from English to Bengali were used.The questionnaire was pre-tested on a sample of 30 students in the same school. Minor modifications in the language were done before final survey. With the help of school teacher the questionnaire was distributed among the guardians of all children between 7-10 years who were present on that particular day. Detailed explanations were given about theusefulness of the study, contents of the questionnaire and how to answer them. They were requested to return the questionnaire after filling it within next two working days and were collected by the research stuffs. In this study wheeze ever wasdefined as any past history of wheezing or whistling in the chest; current wheezing was a history of at least one attack of wheezing or whistling of the chest during last 12 months; diagnosed asthma was wheeze ever plus a doctors diagnosis of asthma. Diagnosis of allergic rhinitis included any history of sneezing or blocked nose during the past 12 months when he/she didn't have a cold or flu attack. Eczema was diagnosed by enquiring about any recurrent itchy rash for at least 6 months or by asking 'has your child ever had eczema?' Height and weight of all children were measured and recorded by trained investigators. Body mass index (BMI) was calculated as weight in kilogram (kg) divided by square of height in meters (kg/m^2)[8]. Age and gender specific BMI percentiles were used to categorize obesity and over weight. Obesity was defined as a BMI equal to or greater than 95^{th} centiles. Overweight was defined as a BMI between 85^{th} and 94^{th} centiles.[9] Statistical analysis were done by using Statistical Package for Social Service (SPSS) for windows version 19. Students t test and chi-square test were done. A p value of <.05 was considered significant.

Results:

Total 480 questionnaires were distributed among the parents of defined study population (7-12 yrs). Parents of 453 children responded by completing the questionnaires properly and returning timely. Thus the response rate was 94.37%. Out of total 453 students who responded, 240 (52.98%) were male and 213 (47.01%) were female. The median age was 8.16 years. The highest number of children were of 7-8 year age group (192, 42.38%) and the least number of children were of 10-12 year age group (80, 17.66%).

Presentation of various asthma symptoms with their prevalence in school children are summarized in table-1.Of the total, 15.01% children gave history of wheezing at one or the other time in their life (wheeze ever) and 7.06% children suffered from wheezing in the past 12 months of life (current wheezing).

Table-I

Presentations of various asthma symptoms in school children

Symptoms	No.	%
Wheeze ever	68	15.01
Current Wheezing	32	7.06
Doctor-diagnosed asthma ever	10	2.20
Allergic rhinitis	96	21.16
Exercise-induced wheeze	38	8.38

Table-2 shows the prevalence of current wheezing in different age groups. This was higher in children belonging to 7-8 years and least among 11-12 years. Current prevalence of wheezing was slightly higher among boys than girls (7.91% vs 6.10% respectively) but was not significant statistically (P >0.05) (Table-3).

Table-II

Prevalence of current wheezing in different age groups

Age (yrs)	No.	Wheeze in the Past Year	
		No.	%
7-8	192	15	7.81
9-10	181	12	6.62
11-12	80	5	6.25

Table-III

Prevalence of current wheezing in the both sexes

	Boy	Girl	Total
	N=240	N=213	N=453
	No.(%)	No.(%)	No.(%)
Current wheezing	19 (7.91)	13 (6.10)	32 (7.06)

The prevalence of obesity and overweight among the school children and its differences in boys and girls has been shown in table- 4.

Table-IV

Prevalence of obesity and overweight in school-age children

Sex	Prevalence Obesity		% Overweight	
	No.	%	No.	%
Both	78	17.21	62	13.68
Boys	46	19.6	32	15.02
Girls	32	13.3	30	14.08

In comparison to normal weight children, the prevalence of current wheezing and exercise induced wheezing was statistically significantly ($p < 0.001$) higher among the children with obesity and overweight. This has been shown in table- 5.

Table-V

Prevalence of current wheezing and exercise-induced wheezing among obese, overweight and normal weight children

Symptoms	Obesity N=78		Overweight N=62		Normal weight N=313		p value
	No.	%	No.	%	No.	%	
Current wheezing	48	58.97	26	41.93	5	1.59	<0.001
Exercise-induced wheeze	14	17.94	11	17.74	3	0.95	<0.001

Discussion:

The present study demonstrated that, current wheezing was more prevalent in obese children than normal weight children (58.97% vs 1.59%). Similarly the prevalence of current wheezing among overweight children was higher than normal weight children (41.43% vs 1.59%). These associations support the findings of other studies [10]. The present study also demonstrated that, the prevalence of exercise induced wheezing was higher among obese and overweight children than normal weight children (17.94% vs .95% and 17.74% vs .95% respectively). In comparison to normal weight children, exercise induced asthma was about 19 times more prevalent to be developed in obese and overweight children respectively. This finding is also consistent with other studies.[11]

In this study sex was identified as an independent variable in relating current wheezing with obesity and overweight. This finding was consistent with some studies [12] but some other studies showed the association was only in women [13]. This gender based inconsistency in results might be attributed to the differences in study populations, the age distribution of the participants and different definitions of asthma used.

Although the present study demonstrated a strong association between obesity, overweight, current wheezing and exercise induced wheezing, no significant association was found between obesity/ overweight and allergic rhinitis or eczema. This contradicts results from some studies [14] but support the results from other studies [15].

Based on the findings of present study and also findings from other studies it could be concluded that obesity and overweight are important risk factors for the development of current wheezing and exercise induced wheezing in children. As these are modifiable risk factors, children with obesity and over weight should be encouraged to do physical exercise and special precautions should be adopted to lose the weight and thus reduce the risk of development of asthma.

Limitations:

Regarding limitations of this study- It was a cross-sectional study in which there was an inability to establish a relationship between the onset of obesity and the subsequent development of asthma. Moreover, the identification of cases was based on responses to the ISSAC questionnaire and this may

have been affected by parental behaviors and diagnostic bias.

Conclusion:

The results of this study suggests that a high BMI in children might be a major risk factor for childhood asthma. As the development of asthma is probably multifactorial, further large scale studies are needed to confirm these results.

References:

1. Behl RK, Kashyap S, Sarkar M. Prevalence of bronchial asthma in school children of 6-13 years of age in Shimla city. Indian J Chest Dis Allied Sci 2010; 52: 145-148.
2. Carroll CL, Stoltz P, Raykov N, Smith SR, Zucker AR. Childhood overweight increases hospital admission rates in children. Pediatrics 2007; 120 (4): 734-740.
3. Figueroa-Munoz J, Chinn S, Rona R. Association between obesity and asthma in 4-11year old children in the UK. Thorax 2001; 56: 133-137.
4. Moudgil H. Prevalence of obesity in asthmatic adults. BMJ 2000; 321: (7258): 448.
5. Bustops P, Amigo H, Oyarzun M, Rona RJ. Is there a causual relation between obesity and asthma ? Evidence from Chile Int J Obes 2005; 29: 804-809.
6. Beuther DA, Sutherland ER. Overweight, obesity and incident asthma: a meta analysis of prospective epidemiologic studies. Am J Respir Crit Care Med 2007; 175 (7): 661-666.
7. The International study of asthma and allergies in childhood (ISSAC) steering committee. Worldwide prevalence of symptoms of asthma, allergic rhino-conjunctivitis and atopic asthma. Lancet 1998; 351: 1225-1235.
8. Daniels SR, Khoury PR, Morrison JA. The utility of body mass index as a measure of body fatness in children and adolescents: differences by race and gender. Pediatrics 1997; 99: 804-807.
9. Aggarwal T, Bhatia RC, Sing D, Sobti PC. Prevalence of overweight and obesity in affluent adolescents from Ludhiana, Punjab. Indian Pediatrics 2008; 45: 500-502.
10. Gilliand FD, Berhane K, Islam T, McConnel R, Gauderman WJ, Gilliland SS, Avol E, Peters JM. Obesity and the risk of newly diagnosed asthma in school-age children. Am J Epidemiol 2003; 158 (5): 406-415.
11. Del Rio-Navarro B et al. Exercise induced bronchospasm in asthmatic and non asthmatic obese children. Allergo Immunopathol (Madr) 2000; 28 (1): 5-11.
12. Nystad W, Meyer HE, Nafstad P, Tverdal A, Englend A. Body mass index in relation adult asthma among 135,000 Norwegian men and women. Am J Epidemiol 2004; 160 (10): 969-976.
13. C hen Y, Dales R, Krewski D, Breithaupt K. Increased effects of smoking and obesity on asthma among female Canadians. The National Population health Survey 1994-1995. Am J Epidemiol 1995; 15: 255-162.
14. Huang SL, Shiao G, Chou P. Association between body mass index and allergy in teen age girls in Taiwan. Clin Exp Allergy 1999; 29: 323-329.
15. Chen Y, Dales R, Jiany Y. The association between obesity and asthma is stronger in non allergic than allergic adults. Chest 2006; 130: 890-895.

Chest & Heart Journal
Vol. 39, No. 2, July 2015

ORIGINAL ARTICLE

Management of Severe Acute Asthma - Comparison between Inhaled vs Systemic Corticosteroid

Dewan Azmal Hussain[1], Md. Ali Hossain[2], Md. Abdur Rouf[3], Md. [1]Naimul Hoq[3], Mahmud Rahim[4], Md. Rustom Ali[5], Md. Bashir Ahmed[3]

Abstract:

Bronchial asthma is a chronic inflammatory disorder of airways. Around 300 million people of the world currently have asthma.[4] In Bangladesh, about 7 million people (5.2% of population) are suffering from Bronchial asthma.[17] Different drugs are used at emergency room for management of severe acute asthma. This study was done to formulate a new drug at Emergency Room for Severe Acute Asthma patients. Along with O_2 inhalation and salbutamol nebulization one group was given 2 puff of Baclomethason dipropionate (200µgm) 10 minutes interval for 2 hours – other group received Inj. Hydrocortisone (200m) I/V along with O_2 inhalation and salbutamol nebulization. Significant improvement was found at Baclomethasone group. Total 100 patients were enrolled in this study. 52 patients in Baclomethasone group and 48 patients in Hydrocortisone group. Parameter measured were FEV_1, FVC, Heart rate, respiratory rate, O_2, saturation at 30 minutes interval. This was a randomized, single blind control trial done at NIDCH from January 2007 to December 2007. Though a large number of children in our country suffer from bronchial asthma – it was not possible to include them in this study, sample size was also not so large; still it was shown that the study had its importance.

Key Word: Severe acute asthma, systemic corticosteroid, metered dose inhaler (MDI) steroid, comparison

[Chest & Heart Journal 2015; 39(2) : 97-102]

Introduction:

Bronchial asthma is a chronic inflammatory diseases. It is a major public health problem and important cause of morbidity and mortality. About 300 million people in the world currently have asthma.[4] It is estimated that there may be an additional 100 million people with asthma by 2025 (Masoli, et al. 2004).[13] The diseases cause physical, emotional and financial suffering for patient leading to its effects on socioeconomic status of the country. Asthma accounts for about 1 in every 250 deaths worldwide.[5] Although modern medication can prevent 80% of such death. Over 18 million working days are lost due to asthma each year (Masoli et al. 2004).[14] The aim of treatment are to abolish symptom, to restore normal or best possible long term air way obstruction, to reduce the risk of sever attack, to enable normal growth of children, to minimize absence from school, or employment. Inhaled corticosteroids are the most

1. Junior Consultant, National Institute of Diseases of the Chest & Hospital (NIDCH), Dhaka
2. Professor of Respiratory Medicine, NIDCH, Dhaka
3. Associate Professor of Respiratory Medicine, NIDCH, Dhaka
4. Assistant Professor of Respiratory Medicine, NIDCH, Dhaka
5. Junior Consultant, NIDCH, Dhaka

Correspondence to: Dewan Azmal Hussain, Junior Consultant, National Institute of Diseases of the Chest & Hospital (NIDCH), Dhaka

potent and effective long term medication for asthma.

The idea of treating of severe acute asthma with inhaled corticosteroid is a relatively newer one (RodRigo 2006).[18] Acute asthma reprersent 4% of all emergency room visit, which account about 2 million people.[7] Currently the corner store of the therapy for severe acute asthma is O_2 inhalation, salbutamol nebulizattion, i/v hydrocorticosone. Hydrocortisone takes 5 hours to have its therapeutic effect and 8 hours for prednisolone. At emergency room i/v theophyline is not recommended. Theophyline is reserved for those patients who failed to respond to salbutamol.[5] Theophylline has low therapeutic index and also there is chance of seizure.

These facts stress the need for the innovation of a emergency department based newer drug which will be an adjunct along with salbutamol nebulization and O_2 inhalation.

Objectives:

General Objective

- To formulate an alternative effective first line therapy for adult patients with severe acute asthma.

Specific Objective

- To compare effectiveness of multi-dose inhaled Beclomethasone and Hydrocortisone in patients with severe acute asthma.[6]
- To find out a cheap, available and easily introduceable treatment for patients with severe acute asthma.

Methodology

Place of Study : Emergency Room, National Institute of Diseases of the Chest and Hospital (NIDCH), Dhaka.

Period of Study : January to December, 2007

Type of Study : This is a single blind, randomized controlled, clinical trial.

Study Population : Adult patients with severe acute asthma attending the emergency department at NIDCH during the above mentioned period.

Sample Size : A total of 100 patients were enrolled in the study – 52 in Beclomethasone group and 48 in Hydrocortisone group.

Selection of Patients

Adult patients of either sex attended the emergency room, NIDCH were selected for this study. The diagnosis of severe acute asthma was done as per the National Asthma Guideline and history and physical examination and previous record examination.

Inclusion Criteria

1. Established cases of bronchial asthma
2. FEV_1 30%-50% predicted value.
3. Age – 18 years and above upto 50 years.
4. Had not received any systemic steroid or aminophyline drip.
5. An expressed willingness to participate in the study, with written, informed consent obtained.

Exclusion Criteria:

1. Long history of smoking, chronic cough, chronic obstructive pulmonary diseases, old pulmonary TB, fever (>30^0C).
2. Co-morbidity malignancy, congestive cardiac failure, chronic renal failure, chronic liver diseases.
3. Age <18 years.
4. Pregnancy, bladder dysfunction, prostatitis.

Study Procedure:

Subject fulfilling the study criteria were divides into two groups. One group titled as Beclomethasone group receives Baclomethasone HFA 200µgm of 10 minutes interval delivered by a respo chamber for 120 minutes. Along with O_2 suppliment and salbutamol nebulization. The second group (titled as Hydrocortisone group) received Inj. Hydrocortisone (200mg) i/v along with O_2 inhalation and salbutamol nebulization. Patient did not get any other modalities of treatment at emergency room. The protocol involved 2 hours duration.

Following variables were measured in each patient immediately before starting treatment and 30 minutes interval for 2 hours.

1. Forced Expiratory Volume in 1st second (FEV_1)
2. Peak Expiratory Flow (PEF)
3. Saturation of oxygen (SaO_2)
4. Respiratory Rate (RR)
5. Heart Rate (HR)
6. Accessory Muscle Uses
7. Dyspnoea
8. Wheeze

PEF was measured with mini-wright peak flow meter

FEV_1 was recorded with a vitalograph compact spimometer.

At the end of therapy each patient was asked for Nausea, palpitation, tremor anxiety, headache.

At the end of protocol, every patient was assessed clinically and PFT. Those not fulfilling the criteria for discharge were admitted and treated accordingly. Criteria for discharge - FEV_1 or PEF >60% of predicted, minimal wheeze, accessory muscle uses abated.

Results and Observation:

Initially a total number of 125 consecutive patients were enrolled in the study. In a randomized fashion the total number of patients were divides into 2 groups. One was Baclomethasone group and another was Hydrocortisone group. Out of them 25 patient failed to complete the study. 15 in the Beclomethasone group and 10 in the Hydrocortisone group. Finally 100 patients remained in the study and data were available from 100 subjects. 52 in Beclomethasone group and 48 in Hydrocortisone group.

Regarding age and sex distribution, nutritional status, occupation, socio-economic condition, and education, family history of asthma and duration of illness. No statistically significant difference was found between the two group.

Peak Expiratory Flow Rate

Mean pretreatment PEFR was 158.27 (SD±7.33) in Beclomethasone group and was 155.62 (SD±7.11) in Hydrocortisone group. After intervention Mean PEFR were 178.27 (SD±8.79), 198.65 (SD±9.29), 218.46 (SD±8.94), 240.77 (SD±7.36) in Baclomethasone group and 166.65 (SD±6.89), 179.38 (SD±6.96), 191.67 (SD±6.63) and 201.88 (SD±6.41) in Hydrocortisone group of 30 minutes, 60 minutes, 90 minutes and 120 minutes respectively. The difference in PEFR improvement was significant (P<0.05) at all the four stages.

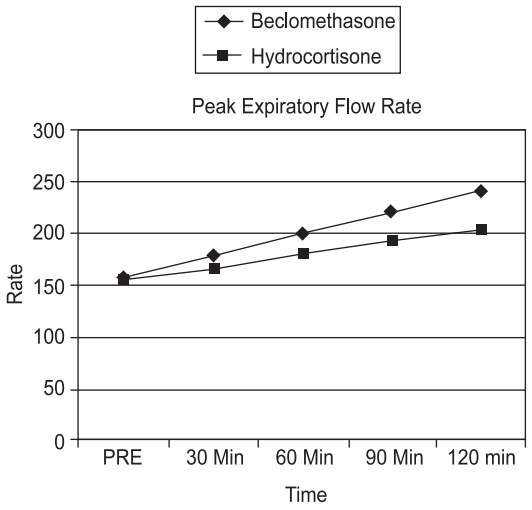


Fig.-1: *Peak expiratory flow rate*

Forced Expiratory Volume in 1ST SECOND (FEV_1%)

Mean pretreatment % of predicted FEV_1 was 37.69 (SD±4.32) in Baclomethasone group and was 39.71 (SD±3.579) in Hydrocortisone group. The difference of pretreatment FEV_1 value was not statistically significant (P=0.13). After intervention, mean FEV_1 was 47.40±4.71, 57.40±4.7, 67.40±4.71, 77.62±3.16 in Beclomethasone group and 46.17±3.39, 52.58±3.23, 58.96±3.14, 65.21±2.10 in

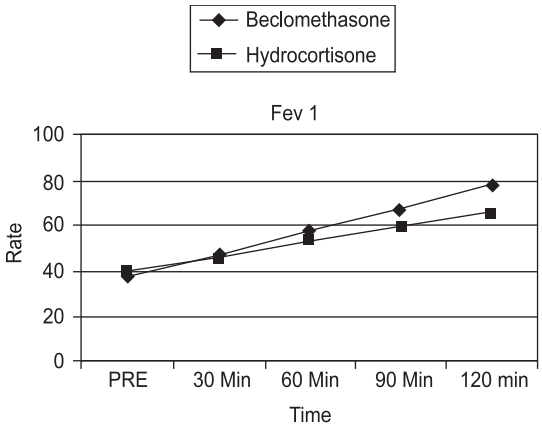


Fig.-2: *Forced expiratory volume in 1st second*

Hydrocortison group of 30, 60, 90 and 120 minutes respectively. The difference in FEV_1 improvement is significant (P<0.05) at 60, 90, 120 minutes.

Saturation of Oxygen

Mean pretreatment SAO_2 was 90.46±0.85 in Baclomethasone group and was 90.50±0.87 in Hydrocortisone group. The difference of pretreatment SAO_2 values was not statistically significant (P=0.82). After intervention mean SAO_2 were 94.31±1.50, 95.96±1.01 and 97.50±0.87 in Beclomethasone group and 92.88±1.00, 94.04±0.96, 95.33±0.95 and 95.79±0.61 at 30, 60, 90 and 120 minutes respectively. The difference in SAO_2 improvement is significant (P<0.05) of all four stages.

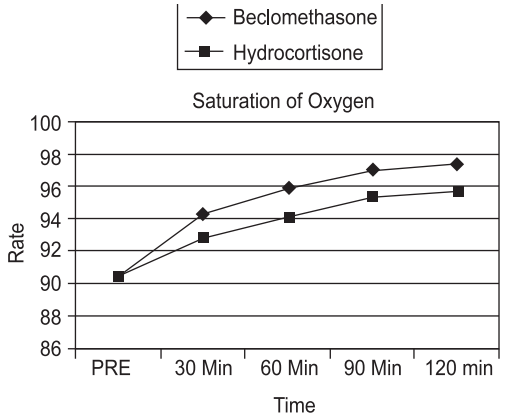


Fig.-3: *Saturaton of oxygen*

% decrease in heart rate	Also shows significantdifference in Baclomethasone group comparison to Hydrocortisone group at 60, 90 and 120 minutes respectively.
% decrease in respiratory rate	

Discussion:

This randomized, single blind, control clinical trial was done to formulate an alternative effective therapy for adult patients with severe acute asthma. The study was carried out at the emergency department of the National Institute of Diseases of the Chest and Hospital (NIDCH), Mohakhali, Dhaka; during the period of January to December, 2007. Total 100 patients fulfilling criteria of inclusion were included in this study and two group were done by randomization. One group (Beclomethasone) had 52 patients who received Beclomethasone HFA 200µgm at 10 minutes interval up to 2 hours. The second group (Hydrocortisone) had 48 patients who received Inj. Hydrocortisone (200mg) intravenous route. Both the groups received O_2 inhalation and nebulization with salbutamol solution. The objective of this randomized control trial was to compare the effect of repeated multi-dose inhaled Beclomethasone with the standard treatment of i/v hydrocortisone in adult patients with severe acute asthma. Data showed a significant advantage in the use of Beclomethasone HFA. This improvement was reflected in higher bronchodilator response, lower clinical ratings and minimal side effects.

Gustavo J. Rodrigo (2005)[10] designed a randomized double blind controlled trial on the hypothesis that patients with acute asthma (FEV_1 <50% of predicted) has better outcome with repeated use of multi-dose inhaled steroid rather then system steroid 106 patients were randomly selected. Among them 52 belongs to inhaled group and 54 in the systemic group. Inhaled group were given 500µgm of fluticasone through a metered-dose inhaler and spacer at 10 minutes interval (3000µgm per hour) for 3 hours. The other systemic group was given 500mg. Inj. Hydrocortisone intravenously both group received O_2 inhalation and salbutamol nebulization simultaneously. Subjects treated with fluticasone showed 30.5% and 46.4% more improvement in PEFR and FEV_1 respectively, compared with hydrocortisone group. The fluticasone group had better PEFR and FEV_1 at 120 minutes interval (P<0.05). At the end of protocol 7.7% of the patients (n=4) in the fluticasone group and 11.1% (n=6) of the patients in hydrocortisone group were admitted. Respiratory rate, heart rate, SAO_2 significantly improved in fluticasone group comparison to hydrocortisone group. In my study I used beclomethasone HFA instead of fluticasone. PEFR increase 39.81% and FEV_1 increase 65.90% from baseline. SAO_2, respiratory rate, heart rate, dysponea also improved in both groups but more in beclomethasone group then hydrocortisone group. The cause of better improvement of PEFR, FEV_1, may be that in my study – patients attended at emergency room took fewer medicine then that of the above study. The second cause may be racial. So this study correlates with and supports my study.

It is important to emphasize that this therapeutic early effect of inhaled corticosteroids was evident as soon as thirty minutes after emergency room arrival. Although the percentage of patients requiring hospitalization in the two groups was close in number. A more rapid improvement was seen in the beclomethasone group. Because systemic corticosteroids are believed to exert their effects over hours rather then minutes, one would expect a greater increase in the number of patients who obtained the discharge criteria with longer emergency department treatment times. Furthermore the hydrocortisone group had a sudden increase in meeting the discharge criteria at 120 minutes compared with 60 minutes. This finding agrees with previous data from a systematic review on the effect of systemic corticosteroids in patients with acute asthma, which show a significant reduction of hospital admission at 2 to 3 hours after the administration of systemic corticosteroids but not before that time.

The study had some limitations such as studied populations were small. Only 100 patients were included in this study. So, results might be re-evaluated by further large scale study. Pediatric age group of patients those were most frequently affected by asthma were not considered here. Very severe acute asthmatics (FEV_1 <30% of predicted) were excluded from this study for ethical issue. So, the study would not reflect effectiveness in this patient group. Baseline theophylline and steroid level were not measured here due to lack of logistic. So, these may influence study results. This was a single blind study. So, there was a chance of biasness in spite of taking all types of measure to prevent it.

All these evidences point to the fact that along with Oxygen inhalation and salbutamol nebulization repeated use of multi-dose steroid inhaler through spacer is very effective in bronchodilation and reduction of hospital admission of severe acute asthmatics in adult, thereby reduce the overall cost of treatment and reduce loss of working period of the productive age group patients which is very important for a developing country like Bangladesh.

Summary & Conclusions:

Different modalities of treatment are practiced for management of acute asthma worldwide. The main objective is to relieve of symptoms rapidly, improvement of pulmonary function and to reduce the rate of hospital admissions. This study was designed to observe rapid effect of multi-dose inhaled corticosteroid in acute asthma.

A total of hundred patients were selected according to the inclusion criteria.

They were divided into two groups by randomization.

One group received beclomethsone HFA metered dose inhaler 200µgm at 10 minutes interval delivered by a respo-chamber for 120 minutes along with O_2 supplement and salbutamol nebulization.

The second group (control) received Inj. Hydrocortisone (200mg IV) along with O_2 inhalation and Salbutamol nebulization. The study involves 2 hours duration.

Following variables were measured in each patient immediately before starting treatment and at 30 minutes interval for 2 hours. Forced Expiratory Volume in 1st second (FEV_1), Peak Expiratory Flow (PEF), Saturation of oxygen (SAO_2), Respiratory rate (RR), Heart rate (HR). Accessory muscles used, dyspnoea, wheeze were recorded in a pre-designed questionnaire. Clinical parameters, pulmonary functions were recorded and results were analyzed statistically.

The study demonstrated significant improvement in symptoms (dyspnoea, wheeze) pulmonary function (FEV_1 , PEFR, SAO_2 , Respiratory Rate, Heart Rate) and hospital admission and a few side effects with inhaled or systemic steroids.

The multi-dose regime of beclomethasone dipropionate by a metered dose inhaler through a spacer is very user-friendly, cheap and can be used at home. Thus patients will be very comfortable with the regimen.

As the young, productive age group patients are mostly affected by acute asthma, this regime will be very helpful in reducing their sufferings, save their earnings and working periods which are very important for a developing country like Bangladesh.

References:

1. Adam Wanner, Gabor Horvath, Jorge L. Brieva, Sunil D. Kumar, Eliana S. Mendes. 2004. Nongenomic actions of glucocorticosteroids on the airway vasculature in asthma. Proc Am Thorac Soc Vol. 1: pp 235-238.

2. Benjamin Volovits, Lea Bentur, Yaron Finkeistein, Yasser Mansour et al. 1998. Effectiveness and safety of inhaled corticosteroids in controlling acute asthma attacks in children who were treated in the emergency department : A controlled comparative study with oral prednisolone. J Allergy Clin Immunol Vol 102: pp605-609.

3. Collin JV, Clark TJH, Brown D & Toownsend J. 1970. Intravenous corticosteroids in the treatment of acute bronchial asthma. Lancet. Vol 2: pp 1047-9.

4. Corbridge TC & Hall JB. 1995. State of the art: The assessment and management of adults with status asthmatics. Am J Respir Crit Care Med. Vol. 151: pp 1296-1316.

5. Devidayal, Singhi S, Kumar L, Jayshee M. 1999. Efficacy of nebulized budesonide compared to oral prednisolone in acute bronchial asthma. Acta Pediatric Vol 88: pp 835-40.

6. E.R. Mc Fadden Jr. 1998. Inhaled glucocorticoids and acute asthma. Am J Respir Crit Care Med. Vol 157: pp 677-678.

7. Global initiative for asthma, 2004. Global burden of asthma. Pp120.

8. Gustavo J. Rodriggo. 2005. Comparison of inhaled fluticasone with intravenous hydrocortisone in the treatment of adult acute asthma. Am J Respir Crit Care Med. Vol 171: pp 1231-1236.

9. Gustavo J. Rodrigo,Carlos Rodrigo. 1998. Inhaled flunisolide for acute severe asthma. Am J Respir Crit Care Med. Vol 157:pp698-703.

10. Gustavo J. Rodrigo. 2006. Rapid effects of inhaled corticosteroids in acute asthma. Chest. Vol 30:pp 1301-1311.

11. Gustavo J. Rodrigo. 2006. Inhaled corticosteroids in the treatment of asthma exacerbations: Esseential concepts. Arch Bronconeumol. Vol 42:pp 533-540.

12. Kavuru MS & Widemann HP. 1998. In: Diagnosis and management of asthma. 2nd edition. Professional Communication Inc. Cleveland, Ohio, p23.

13. Matthew Masoli, Denise Fabian, Shaun Holt, Richard Beasley. 2004. Global Burden of Asthma – Develop for GINA.

14. Matthew Masoli, Richard Beasley. 2004. Diagnosis and management of adult asthma. Medicine International, Bangladesh edition. Vol 4: pp 63-68.

15. Masoli International, Bangladesh edition. M, Beasley R. 2004. Diagnosis and management of adult asthma. Medicine. Vol 4:pp63-68.

16. McFadden Jr ER, Elsanadi N, Strauss L, Galan G, Dixon L, McFadden CB et al. The influence of parasympatholytics on the resolution of the acute attacks of asthma. Am J Med. 1997. Vol 102:pp7-13.

17. National Guidelines 2005. Asthma Bronchiolitis COPD. Asthma Association, Bangladesh. Vol 3:pp46-51.

18. Rodrigo GJ, 2003. Inhaled therapy for acute adult asthma. Curr Opin Allergy Clin Immunol. Vol 3(3):pp 169-75.

19. Sunil D. Kumar, Jorge L. Brieva, Ignacio Danta, adam Wanner. 2000. Transient effect of inhaled fluticasone on airway mucosal blood flow in subjects with and without asthma. Am J Respir Crit Care Med. Vol 161: pp918-921.

Chest & Heart Journal
Vol. 39, No. 2, July 2015

ORIGINAL ARTICLE

Infection Pattern of Bronchiectasis and non-Bronchiectasis Patients in NIDCH in Relation to C/S of Sputum

Md. Hasanur Rashid[1], Mir Mahbubul Alam[2], Pulak Kumar Dey[3], Olia Sharmeen[4], Habib Uddin Ahmad[5], Md.Naimul Hoque[6], Md. Abdur Rouf[6], Md. Rashidul Hassan[7]

Abstract:

Background: *the prevalence of bronchiectasis reflects the socioeconomic conditions of the population under study. Bronchiectasis remains a major cause of morbidity in less-developed countries like Bangladesh. On the other hand, non-bronchiectatic diseases like Chronic Obstructive Pulmonary Disease (COPD) are major causes of chronic morbidity and mortality throughout the world. Many people suffer from this disease for years and die pre-maturely of it or its complications Methods: An observational analytical study with group comparison design was conducted in National Institute of Diseases of the Chest & Hospital (NIDCH), Mohakhali, Dhaka from October 2012 to September 2013 to know the infection pattern of bronchiectasis and non-bronchiectasis patients in relation to culture and sensitivity test of sputum. Results: One hundred and forty two patients were enrolled for the study of which 43 were the patients of bronchiectasis and the rest 99 were non-bronchiectasis patients. The mean age of the bronchiectasis patients was 49.79 (±15.43) years and that of the non-bronchiectasis patients was 57.91 (±12.79) years. Most of the families (85%) were from below average social class and most of the respondents were smoker (88, 62%). Severe dyspnoea was more common in non-bronchiectasis patients (65.3%) than bronchiectasis patients (2.1%). Pseudomans was the mostly observed organism in sputum culture of bronchiectasis patients while Klebsiella was the leading organism in sputum culture of non-bronchiectasis patients. Against Klebsiella, Tozabactum was 100% sensitive and Tobramycin was 90% sensitive while most of the common antibiotics like Amoxycilliin, Amoxiclav, Cefipime Cephradine, Cephalexen, Piperacillin, and Cefuroxime were 100% resistant. Against Pesudomonas Ampicillin was 100% sensitive. Other sensitive antibiotics were Tobramycin (88.9%), Piperacillin (86.7%) and Tozabactum (80%). Conclusion: Resistance of the organism to widely prescribed antibiotics appeared to be a major concern. Patients may be prescribed with less commonly used sensitive antibiotics on the basis of Culture and Sensitivity (C/S) reports. Further in-depth research with large sample size and more powerful study design is required to extract further information in this regard.*

[Chest & Heart Journal 2015; 39(2) : 103-114]

Introduction:

Background

Currently no systematic data are available on the incidence or prevalence of bronchiectasis. A general theory is that the emergence of vaccines and antibiotics in the 20th century resulted in a decline in the rate of bronchiectasis in developed countries [1].

1. Assistant Professor, Respiratory Medicine, NIDCH, Dhaka
2. Junior Consultant, Chest Diseases Clinic, Madaripur
3. Medical Officer, NIDCH, Dhaka
4. Associate Professor, Dept. of Haematooncology, Sir Salimullah Medical College & Mitford Hospital
5. Assistant Professor, Respiratory Medicine, NIDCH, Dhaka
6. Associate Professor, Respiratory Medicine, NIDCH, Dhaka
7. Professor cum Director, NIDCH, Dhaka

Correspondence to: Md. Hasanur Rashid, Assistant Professor, Respiratory Medicine, NIDCH, Dhaka

The best data available suggest that the prevalence of bronchiectasis mirrors the socioeconomic conditions of the population under study, with significantly lower prevalence in areas where immunizations and antibiotics are readily available. Bronchiectasis remains a major cause of morbidity in less-developed countries, especially in countries with limited access to medical care and antibiotic therapy [2,3].

Bronchiectasis is an abnormal dilation of the proximal and medium-sized bronchi (>2 mm in diameter) caused by weakening or destruction of the muscular and elastic components of the bronchial walls. Affected areas may show a variety of changes, including transmural inflammation, edema, scarring, and ulceration, among other findings. Distal lung parenchyma may also be damaged secondary to persistent microbial infection and frequent post-obstructive pneumonia. Bronchiectasis can be congenital but is most often acquired [4]. Congenital bronchiectasis usually affects infants and children . Acquired forms occur in adults and older children and require an infectious insult, impairment of drainage, airway obstruction, and/or a defect in host defense. The tissue is also damaged in part by the host response of neutrophilic proteases, inflammatory cytokines, nitric oxide, and oxygen radicals. This results in damage to the muscular and elastic components of the bronchial wall. Additionally, peribronchial alveolar tissue may be damaged, resulting in diffuse peribronchial fibrosis[5].

Impaired clearance of secretions causes colonization and infection with pathogenic organisms, contributing to the purulent expectoration commonly observed in patients with bronchiectasis. The result is further bronchial damage and a vicious cycle of bronchial damage, bronchial dilation, impaired clearance of secretions, recurrent infection, and more bronchial damage [6].

Causes of bronchiectasis include the following:

- Primary infections
- Bronchial obstruction
- Aspiration
- Cystic fibrosis
- Primary ciliary dyskinesia
- Allergic bronchopulmonary aspergillosis
- Congenital anatomic defects
- Connective-tissue disorders
- Alpha1-antitrypsin (AAT) deficiency
- Autoimmune diseases
- Idiopathic inflammatory disorders
- Autosomal dominant polycystic kidney disease
- Traction from other processes
- Toxic gas exposure

Primary infections

Bronchiectasis may be the sequel of a variety of necrotizing infections that are either inadequately treated or not treated at all. Primary infection (i.e. in the absence of intrinsic defects or noninfectious extrinsic insults) was a particularly common cause of bronchiectasis in developed countries prior to the widespread use of antibiotics [7] and it remains important in developing countries, where antibiotics are used inconsistently [2,3] .

Typical offending organisms that have been known to cause bronchiectasis include the followin[1,7]:

- *Klebsiella species*
- *Staphylococcus aureus*
- *Mycobacterium tuberculosis*
- *Mycoplasma pneumonia*
- Pseudomonas
- Nontuberculous mycobacteria
- Measles virus
- Pertussis virus
- Influenza virus
- Herpes simplex virus
- Certain types of adenovirus

Infection with respiratory syncytial virus in childhood may also result in bronchiectasis.

Chronic obstructive pulmonary disease (COPD) is a major cause of chronic morbidity and mortality throughout the world. Many people suffer from this disease for years and die pre-maturely of it or its complications. Its pulmonary component is characterized by airûow limitation that is not fully reversible. The airûow limitation is usually

progressive and associated with an abnormal inûammatory response of the lung to noxious particles or gases. The chronic airûow limitation characteristic of COPD is caused by a mixture of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema), the relative contributions of which vary from person to person. Airûow limitation is best measured by spirometry, because this is the most widely available, reproducible test of lung function. Because COPD often develops in longtime smokers in middle age, patients often have a variety of other diseases related to either smoking or aging [8] . COPD itself also has signiûcant extrapulmonary (systemic) effects that lead to co-morbid conditions [9]. Thus, COPD should be managed with careful attention.

Although asthma can usually be distinguished from COPD, in some individuals with chronic respiratory symptoms and ûxed airûow limitation it remains difficult to differentiate the two diseases. In many developing countries, both pulmonary tuberculosis and COPD are common[10]. In countries where tuberculosis is very common, respiratory abnormalities may be too readily attributed to this disease [11]. Conversely, where the rate of tuberculosis is greatly diminished, the possible diagnosis of this disease is sometimes overlooked. Therefore, in all subjects with symptoms of COPD, a possible diagnosis of tuberculosis should be considered, especially in areas where this disease is known to be prevalent [12].

The organisms detected include most commonly nontypeable Haemophilus influenzae, but also Streptococcus pneumoniae, Moraxella catarrhalis, and Pseudomonas aeruginosa. A number of studies have now shown conclusively, using protected specimen brush catheters at bronchoscopy, that LABC does exist in COPD and may be detected right down to the peripheral airways and is not caused by contamination from nasopharyngeal commensals (Monso, 1999, Zalacain, 1999 and Soler 1999). Evaluation of risk factors for LABC in COPD shows that current cigarette smoking and the FEV_1 were related to colonization, suggesting that LABC is related to disease progression.

Bacteria have a number of important effects on airway epithelium, including stimulation of mucous production, reduction of mucociliary clearance, and airway epithelial cell injury [13]. These mechanisms lead to increased airway inflammation and production of neutrophil elastase, interleukin 8 (IL-8), and other chemoattractants that contribute to the neutrophil influx. Once LABC is established the process persists as described in the "vicious cycle hypothesis"[14]. There is now evidence that H. influenzae, in addition to being present in the airway lumen, is also found in the submucosa and intracellularly; both these sites encouraging persistence of the organism and evasion from the effects of antibiotics [15]. Although mucous hypersecretion was originally considered not to be related to disease progression in COPD, there is now evidence that chronic mucous hypersecretion is related to decline of FEV1 in COPD, and one of the most important stimuli to hypersecretion may be LABC. Evidence has shown conclusively Evidence has shown conclusively that airway inflammation increases with higher airway bacterial loads determined from quantitative sputum cultures in patients with COPD[16]. In this study the bacterial species also influenced the severity of airway inflammation, with colonization by P. aeruginosa showing the greatest effect on inflammatory markers. More direct evidence that bacterial colonization contributes to airflow obstruction comes from a study that showed that H. influenzae colonization was associated with increased airway inflammatory markers in patients with chronic bronchitis and airflow obstruction, compared with patients with chronic bronchitis without airflow obstruction, where airway inflammation was reduced[17].

Although there has been some previous controversy regarding isolation of bacteria at COPD exacerbation, data from Stockley and colleagues show that an exacerbation with purulent sputum is associated with increased airway bacterial load . In another study Sethi and colleagues showed that exacerbations associated with detection of H. influenzae are associated with higher levels of airway inflammatory markers, compared with pathogen-negative exacerbations; thus providing further evidence that airway infection leads to increased inflammation and thus decline in lung function [15]. The majority of COPD exacerbations are associated with airway infection, either viral or bacterial. There is now evidence that not all COPD exacerbations recover to baseline with

respect to symptoms and lung function and as airway inflammatory markers are increased at exacerbation (Bhowmik, 2000), this suggests that airway inflammation may persist and lead to progressive decline in the FEV_1.

Some patients with COPD develop frequent exacerbations and this susceptible patient group has increased stable airway IL-6 and IL-8 levels compared with those patients with infrequent exacerbations (Bhowmik, 2000). Patients with frequent exacerbations are more likely to be colonized with H. influenzae when stable (Patel, 2001) and as COPD exacerbations increase with disease progression, then bacterial infection may play an important role in this process. Exacerbation frequency is an important determinant of health status (Seemungal, 1998) and eradication of bacterial colonization may have considerable impact on the consequences of COPD.

The bacteria in bronchiectatic airways have specific survival strategies. For instance *P. aeruginosa* organisms reside in a biofilm on the brochiolar surface. In this survival strategy, the organisms use the micromilieu of nutrients but avoid elimination by local phagocytic, antibody and other immune responses. Frustrated phagocytosis leads to the liberation of proteolytic chemicals, such as elastases and oxygen radicals, that cause further local tissue injury. It has been shown that patients with bronchiectasis who are colonized with *Pseudomonas* species have worse lung function and more progressive disease (Evans et al., 1996).

Rationale of the study

In clinical practice we very often encountered with the patients of bronchiectasis, COPD, lung abscess, pneumonia etc. at National Institute of of Diseases of the Chest & Hospital (NIDCH). Bacterial colonization of the respiratory tract is a major challenge for the treating physician. At first, empirical antibiotic is started to control infection, along with broncho-dilatation and reduction of broncho-inflammation. So it is utmost importance to know the common bacterial colonization in these patients. In the era of developing newer drug resistance – sensitivity profile of currently used antibiotic is also to be monitored. As a result, it will be possible to give appropriate antibiotic empirically at the first instance with faster relive of symptoms and early recovery of the patients. That is why the current study was undertaken.

Materials and Methods

Study design:

Observational analytical study with group comparison design.

Study setting:

Time: October 2012 to September 2013.

Place: National Institute of the Diseases of the Chest and Hospital (NIDCH), Dhaka.

Study population:

Patients suffering form bronchiectasis and non-bronchiectasis who attended NIDCH for treatment

Sample size: sample size was limited to 142.

Inclusion criteria:

Diagnosed patients of bronchiectasis and non-bronchiectasis i.e. COPD, lung abscess, bronchogenic carcinoma, CAP etc.

Exclusion criteria:

Not willing to participate.

Sampling method: Purposive sampling technique was used.

Data collection technique & instruments: After admission of the patients of bronchiectasis and non-bronchiectasis including COPD, bronchial carcinoma, lung abscess and CAP a complete history was taken and through clinical examinations was performed. A pretested structured questionnaire was used to extract information. The aims & objectives were informed to all patients in easy and understandable way. They were also informed that they could withdraw from the study any time they want. Written informed consents were obtained from each patient before start of the study. Sputum was collected immediately after admission before initiation of antibiotic under direct physician/investigator supervision following through mouth washing.

Sputum sample was collected in a sterile bottle and the characteristic of the sample was noted. Then the sample was sent to the Microbiology Laboratory for culture. High Resolution Computerized Tomography (HRCT) was performed within few days after admission. Those who could not perform HRCT were excluded from the study. Those patients who needed antibiotic on urgent basis were also excluded from the study. All the necessary routine investigations were also done. Finally patients were selected on the basis of history, clinical examinations and investigations reports.

Outcome variables:

- Age
- Sex
- Socio-economic status
- Prevalent pathogens
- Sensitivity profile of pathogens to antibiotic

Results:

Figure 1 shows the age distribution of the patents. The mean age of the bronchiectasis patients was 49.79 (±15.43) years and that of the non-bronchiectasis patients was 57.91 (±12.79) years. The age followed almost normal distribution. A significant difference was observed between these two groups (t=-3.260 (df=140); p<.001).

Out of 142 patients 103 were male (72.5%) and remaining 39 were female (27.5%). The male female ratio was 2.6:1. By category 24 bronchiectasis patients were female and 15 non-bronchiectasis patients were from the same sex. The sex difference between two groups was statistically significant ($\div^2$ =24.88 (df=1); p<.001) (Fig 2).

Figure 3 shows the socio-economic status of the patents. On the basis of monthly family income respondents were classified into two categories. Families earning less than 10000 taka per month were labeled as below average and taka 10000 or more earning families were marked as average group. Most of the families in both bronchiectasis and non-bronchiectasis groups were below average in socio-economic status (88.4% and 70.7% respectively).

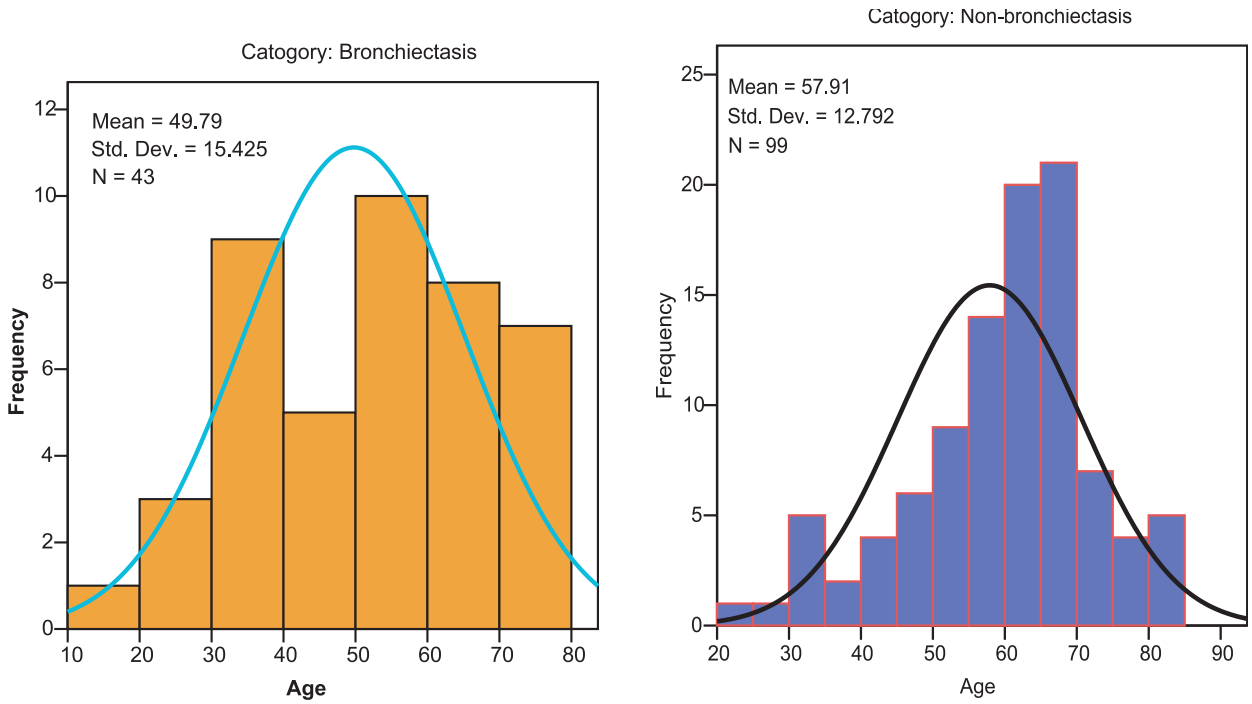


Fig.-1: *Age distribution of the patients*

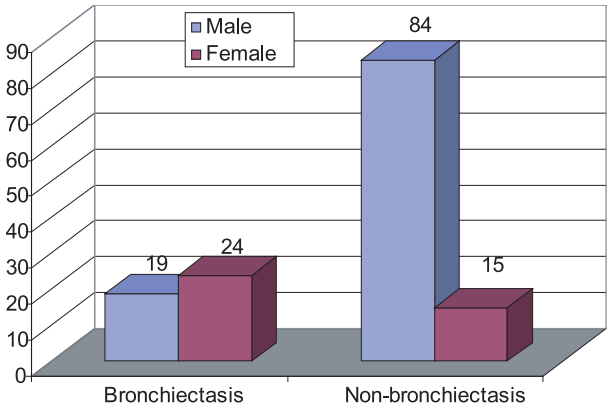


Fig.-2: *Distribution of the patients by sex*

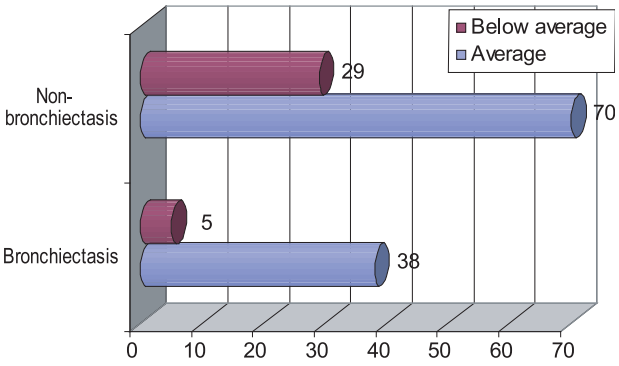


Fig.-3: *Distribution of the patients by socio-economic status*

Table-I

Distribution of the patients by occupation

Occupation	Category		Total
	Bronchi-ectasis (N=43)	Non-bronchi-ectasis (N=99)	
Service	3 (7.0)	6 (6.1)	9 (6.3)
Business	5 (11.6)	18 (18.2)	23 (16.2)
Day laborer	1 (2.3)	4 (4.0)	5 (3.5)
Farmer	6 (14.0)	29 (29.3)	35 (24.6)
Housewife	22 (51.2)	15 (15.2)	37 (26.1)
Aged/retired	2 (4.7)	24 (24.2)	26 (18.3)
Others	4 (9.3)	3 (3.0)	7 (4.9)
Total	43 (100.0)	99 (100.0)	142 (100.0)

• Percentage is given in parenthesis

Most of the female patients were housewives in both groups. Among male, in bronchiectasis group the leading profession was farming (14%), business (11.6%) and service (7%); in non-bronchiectasis group those percentages were 29.3%, 18.2% and 6.1% respectively. A considerable proportion of the patients was retired or aged in the both groups (Table I).

Table-II

Distribution of the patients by educational status

Educational status	Category		Total
	Bronchi-ectasis (N=43)	Non-bronchi-ectasis (N=99)	
Illiterate	11 (25.6)	28 (28.3)	39 (27.5)
Primary	26 (60.5)	36 (36.4)	62 (43.7)
Secondary	2 (4.7)	19 (19.2)	21 (14.8)
Higher secondary	2 (4.7)	8 (8.1)	10 (7.0)
Graduate and above	2 (4.7)	8 (8.1)	10 (7.0)
Total	43 (100.0)	99 (100.0)	142 (100.0)

• Percentage is given in parenthesis

Table II shows the educational statuses of the groups. In bronchiectasis group one in every four respondents had no education and more than 60% of the respondents had primary level education. In non-bronchiectasis group the percentage was almost identical for illiterate patients (27.5%). The numbers of highly educated patients in this group were higher than bronchiectasis group.

Table-III

Distribution of the patients by smoking habit

Smoking habit	Category		*p*-value
	Bronchi-ectasis (N=43)	Non-bronchi-ectasis (N=99)	
Yes	10 (23.3)	78 (78.8)	
No	33 (76.7)	21 (21.2)	<.001 (HS)
Total	43 (100.0)	99 (100.0)	

• Percentage is given in parenthesis
• HS= Highly significant

Table III depicts the smoking habit of the respondents. Most of the non-bronchiectasis patients were smoker (78, 78.8%) while 10 patients (23.3%) in bronchiectasis group were smokers. This difference was statistically highly significant (χ^2=39.23, df=1; p<.001).

Table-IV

Distribution of the patients by diagnosis

Diagnosis	Frequency	Percent
COPD	77	54.2
Bronchiectasis	43	30.3
Lung cancer	15	10.6
Lung abscess	4	2.8
CAP	3	2.1
Total	142	100.0

COPD = Chronic Obstructive Pulmonary Disease
CAP = Community Acquired Pneumonia

More than half of the respondents (54.2%) were the patients of COPD while about one third of the respondents (30.3%) were the patents of bronchiectasis. One in every ten respondents was the patient of bronchogenic carcinoma (Table 4).

Table-V

Distribution of the patients by characteristics of cough

Characteristics of sputum	Category		*p*-value
	Bronchi-ectasis (N=43)	Non-bronchi-ectasis (N=99)	
Type			
Mucopurulent	43 (100.0)	93 (93.9)	<.001 (HS)
Mucoid	0 (.0)	6 (6.1)	
Odour			
Fetid	16 (37.2)	4 (4.0)	<.001 (HS)
Non-fetid	27 (62.8)	95 (96.0)	
Increase sputum on posture change			
Yes	41 (95.3)	0 (.0)	<.001 (HS)
No	2 (4.7)	99 (100.0)	

In bronchiectasis group the sputum of all the patients was mucopurulent in nature and in non-bronchiectasis group it was 93.9%. In non-bronchiectasis group 6 patients had mucoid type sputum. Significantly more patients had fetid sputum in bronchiectasis group than non-bronchiectasis group. In 41 patients sputum volume increased with posture change in case bronchiectasis patients but in case of non-bronchiectasis patients no such thing happened. All these differences were statistically highly significant.

Table-VI

Clinical features by patient category

Clinical features	Disease Category		*p*-value
	Bronchi-ectasis (N=43)	Non-bronchi-ectasis (N=99)	
Wheeze			236 (NS).
Present	27 (62.8)	72 (72.7)	
Absent	16 (37.2)	27 (27.3)	
Haemoptysis			
Present	24 (55.8)	19 (19.2)	<.001(HS)
Absent	19 (44.2)	80 (80.8)	
Clubbing			
Present	43 (100.0)	42 (42.4)	<.001(HS)
Absent	0 (.0)	57 (57.6)	
Course crepitation altered by coughing			
Present	43 (100.0)	98 (99.0)	1.00 (NS)
Absent	0 (.0)	1 (1.0)	

Percentage is given in parenthesis
NS= Not significant
HS= Highly significant

Various clinical features of the patients are shown above. It is evident from the table 7 that presences of haemoptysis, clubbing and course crepitation were significantly higher in bronchiectasis patients.

Table-VII

Distribution of the patients by bacteria in sputum culture

Bacteria in culture of sputum	Disease Category		*p*-value
	Bronchi-ectasis (N=43)	Non-bronchi-ectasis (N=99)	
Pseudomoans	34 (79.1)	30 (30.3)	
Klebsiella	4 (9.3)	54 (54.5)	
Escherichia.coli growth	0 (.0)	12 (12.1)	
Acinetobacter spp growth	5 (11.6)	3 (3.0)	

The Gram-negative gammaproteobacteria *Pseudomans* was the mostly reported organism in sputum culture. In bronchiectasis patient pseudomonas growth was found in 34 patients (79%). In non-bronchiectasis patients this number was 30 (30.3%). Gram-negative bacteria *Klebsiella* was the second most reported organism in sputum culture. It was mainly isolated in non-broncheiectasis patients (54.5%). The other important bacteria were Escherichia coli and Acinetobacter spp (Table 9).

Sensitivity profile of some important antibacterial agents is shown in the above table. It is evident from the table that most commonly used antibiotics were not sensitive to tackle organism normally found in bronchiectasis and non-bronchiectasis patients. The most sensitive antibiotics were Polymixin B (100.0%), Tozabactum (85.7%), Tobramycin (84%), Colistin (92%), Levofloxacin (67.6%), Amikacin (67.3%), Piperacillin (57.1%), Pefloxacin (51.3%), Ampicillin & Tetracycline (50%) Azithromycin (39.8%), etc. Some widely used antibiotics like Amoxycilliin, Amoxiclav, Cephradine, Cephalexen, Carbenicilin, Cefipime, Nitrofurantoin, Nalidixic acid, Oxacillin, Cefuroximeand Vancomycin were 100% resistant. Other resistant antibiotics were Cefixime (92.7%), Co-trimoxazole (78.6%), Imipenem (75%) etc. (Table 10).

Sensitivity profile of some important antibacterial agents against two major organisms commonly found in bronchiectasis and non-bronchiectasis

Table-VIII

Sensitivity profile of some important antibacterial agents

Antibiotics	Sensitive		Resistant	
	Frequency	Percent	Frequency	Percent
Amikacin (n=98)	66	67.3	32	32.7
Amoxiclav (n=2)	0	.0	2	100.0
Amoxycillin (n=17)	0	.0	17	100.0
Ampicillin (n=6)	3	50.0	3	50.0
Azithromycin (n=118)	47	39.8	71	60.2
Aztreonam (n=68)	25	36.8	43	63.2
Carbenicilin (n=1)	0	.0	1	100.0
Cefipime (n=12)	0	.0	12	100.0
Cefixim (n=82)	6	7.3	76	92.7
Ceftazidime (n=14)	0	.0	14	100.0
Ceftrioxone (n=92)	22	23.9	70	76.1
Cefuroxime (n=18)	0	.0	18	100.0
Cephalexen (n=8)	0	.0	8	100.0
Cephradine (n=13)	0	.0	13	100.0
Ciproflxacin (n=31)	13	41.9	18	58.1
Colistin (n=102)	92	82.1	10	17.9
Co-trimoxazole (n=14)	3	21.4	11	78.6
Gentamicin (n=5)	2	40.0	3	60.0
Imipenem (n=4)	1	25.0	3	75.0
Levofloxacin (n=74)	50	67.6	24	32.4
Meropenem (n=78)	22	28.2	56	71.8
Nalidixic acid (n=2)	0	.0	2	100.0
Nitrofurantoin (n=2)	0	.0	2	100.0
Oxacillin (n=3)	0	.0	3	100.0
Pefloxacin (n=39)	20	51.3	19	48.7
Piperacillin (n=21)	12	57.1	9	42.9
Polymyxin B (n=3)	3	100.0	0	.0
Tetracyclin (n=18)	9	50.0	9	50.0
Tobramycin (n=25)	21	84.0	4	16.0
Tozabactum (n=7)	6	85.7	1	14.3
Vancomycin (n=2)	0	.0	2	100.0

Table-IX

Sensitivity profile of some important antibacterial agents against two major organisms commonly found in bronchiectasis and non- bronchiectasis patients.

Antibiotics	Klebsiella		*p*-value	Psedomonas		*p*-value
	S	R		S	R	
Amikacin (n=98)	36 (70.6)	15 (29.4)	0.003*	24 (68.6)	11 (31.4)	0.028*
Amoxiclav (n=2)	-	2 (100.0)	-	-	-	-
Amoxycillin (n=17)	-	6 (100.0)	-	-	7 (100.0)	-
Ampicillin (n=3)	-	-	-	3 (100.0)	-	-
Azithromycin (n=118)	24 (48.0)	-	-	19 (38.0)	-	-
Aztreonam (n=68)	2 (10)	18 (90)	<.001*	25 (64.1)	14 (35.9)	0.071
Carbenicilin (n=1)	-	-	-	-	-	-
Cefipime (n=12)	-	2 (100.0)	-	-	8 (100.0)	-
Cefixim (n=82)	3 (6.5)	43 (93.5)	<.001*	3 (10.3)	26 (89.7)	<.001*
Ceftazidime (n=14)	-	2 (100.0)		-	9 (100.0)	-
Ceftrioxone (n=92)	9 (19.6)	37 (80.4)	<.001*	13(34.2)	25 (65.4)	0.052
Cefuroxime (n=18)	-	3 (100.0)	-	-	12 (100.0)	-
Cephalexen (n=8)	-	3 (100.0)	-	-	4 (100.0)	-
Cephradine (n=13)	-	3 (100.0)	-	-	8 (100.0)	-
Ciproflxacin (n=31)	-	5 (100.0)	-	14 (63.6)	8 (36.4)	<.001*
Colistin (n=102)	32 (68.1)	15 (31.9)	0.013*	51 (92.7)	4 (7.3)	<.001*
Co-trimoxazole (n=14)	-	3 (100.0)	-	3 (42.9)	4 (57.1)	0.705
Gentamicin (n=5)	-	1 (100.0)	-	-	-	-
Imipenem (n=4)	-	-	-	2 (66.7)	1 (33.3)	0.56
Levofloxacin (n=74)	30 (68.2)	14 (31.8)	0.016*	18 (72.0)	7 (28.0)	0.028*
Meropenem (n=78)	8 (17.4)	38 (82.6)	<.001*	12 (48.0)	13 (50.0)	0.841
Nalidixic acid (n=2)	-	-	-	-	1 (100.0)	-
Nitrofurantoin (n=2)	2 (66.7)	1 (33.3)	0.564	-	-	-
Oxacillin (n=3)	-	-		-	-	-
Pefloxacin (n=39)	12 (70.6)	5 (29.4)	0.090	9 (75.0)	3 (25.0)	0.08
Piperacillin (n=21)	-	3 (100.0)	-	13 (86.7)	2 (13.3)	0.005*
Polymyxin B (n=3)	-	-	-	-	-	-
Tetracyclin (n=18)	1 (14.3)	6 (85.7)	0.059	5 (50.0)	5 (50.0)	1.00
Tobramycin (n=25)	9 (90.0)	1 (10.0)	0.011*	8 (88.9)	1 (11.1)	0.02*
Tozabactum (n=7)	2 (100.0)	-	-	4 (80.0)	1 (20.0)	0.18
Vancomycin (n=2)	-	-	-	-	1 (100.0)	-

* Significant S=Sensitive R=Resistant

patients are presented in the above table. Against Klebsiella, Tozabactum was 100% sensitive and Tobramycin was 90% sensitive while most of the common antibiotics like Amoxycilliin, Amoxiclav, Cefipime Cephradine, Cephalexen, Piperacillin, and Cefuroxime were 100% resistant. Against Pesudomonas Ampicillin was 100% sensitive. Other sensitive antibiotics were Tobramycin (88.9%), Piperacillin (86.7%) and Tozabactum (80%) (Table 11).

Discussion:

To know the infection pattern of bronchiectasis and non-bronchiectasis patients in relation to

culture and sensitivity test of sputum a cross-sectional observational study was conducted from October 2012 to September 2013 at National Institute of the Diseases of the Chest and Hospital (NIDCH), Dhaka. We studied 142 patients. Of them 43 were diagnosed as the patients of bronchiectasis by HRCT of chest and the others were non-bronchiectasis cases. The mean age was 55.45 (±14.09) years and the percentage of elderly people was higher. While most of the previous international studies found a predominance of female subjects, our findings was somewhat reverse [18].

Regarding socio-economic status most of the families (85%) were from below average class. Most of the respondents were smoker (88, 62%). In male the number of smokers was significantly higher than female (p<.001). This finding is consistent with some other international studies 1.

Severe dyspnoea was less common in bronchiectasis patients (2.1%) than non-bronchiectasis patients (65.3%). This difference is statistically highly significant (p <.001). Underlying pathology in the development of COPD could be the cause for this difference.

Pseudomans was the mostly reported organism in sputum culture of bronchiectasis patients (79%) while *Klebsiella* was the predominant organism in sputum culture of non-bronchiectasis patients (54.5%). Pasterur (2000) reported *P. aeruginosaand H. influenzae* (55%) as predominating in bronchiectasis patients while Soler (1998) found community-acquired pathogens in sputum culture of non- bronchiactasis patients. %). Pasterur (2000) reported *P. aeruginosaand H. influenzae* (55%) as predominating in bronchiectasis patients[7] while Soler (1998) found community-acquired pathogens (*Streptococcus pneumoniae*, *Haemophilus influenzae* and *Moraxella catarrhalis*) in 19 of 34 (56%) and to gram-negative enteric bacilli (GNEB), Pseudomonas, and Stenotrophomonas spp. in 15 of 34 (44%) of isolates. Particular geographical and environmental factors may be the cause for such difference[23].

We found that most commonly used antibiotics were not sensitive to tackle organism normally found in bronchiectasis and non-bronchiectasis patients. Against Klebsiella, Tozabactum was 100% sensitive and Tobramycin was 90% sensitive while most of the common antibiotics like Amoxycilliin, Amoxiclav, Cefipime Cephradine, Cephalexen, Piperacillin, and Cefuroxime were 100% resistant. Against Pesudomonas Ampicillin was 100% sensitive. Other sensitive antibiotics were Tobramycin (88.9%), Piperacillin (86.7%) and Tozabactum (80%). Ever growing resistance to antibiotics is a global problem. Taking of inappropriate and inadequate antibiotics is the major culprit in this regard.

Bronchiectasis usually presents with recurrent lower respiratory tract infection and chronic mucopurulent sputum production. The characteristic features are abnormally dilated thick-walled bronchi that are inflamed and colonized by bacteria. Symptoms include chronic cough, mucopurulent sputum production, hemoptysis, breathlessness, and tiredness. The incidence is perceived to have declined over recent decades, but significant numbers of patients continue to present to respiratory physicians [19]. Sometimes regarded as a condition in which extensive investigation is unnecessary or unlikely to lead to treatable causes, one of the aims of this study was to characterize the underlying causative factors of patients with bronchiectasis referred to NIDCH.

Identifying the underlying cause of bronchiectasis can have major implications for management. Diagnosing bronchiectasis has become significantly easier with the advent of high resolution computed tomography (HRCT), which has proved to be a highly sensitive noninvasive technique for demonstrating bronchiectatic change in the airways of patients with bronchiectasis. On the other hand the natural history of chronic obstructive pulmonary disease (COPD) is characterized by frequent exacerbations with an increase of cough, purulent sputum production, and dyspnea. In some patients, mostly those with severe airflow obstruction, severe respiratory failure occurs and may require mechanical ventilation. Bacterial infections have generally been considered as the leading cause of exacerbations in COPD[20]. Notwithstanding, the role of bacterial infections in acute exacerbations is a matter of debate. Because lower respiratory tract bacterial colonization is frequently present in COPD

patients [21], the principal problem is to differentiate between bronchial colonization and infection. Most of the earlier studies have relied on sputum as a diagnostic technique [22].

Conclusion:

To know the infection pattern of bronchiectasis and non-bronchiectasis patients in relation to culture and sensitivity test of sputum an observational analytical study with group comparison design was conducted in National Institute of Diseases of the Chest & Hospital (NIDCH), Mohakhali, Dhaka from October 2012 to September 2013. One hundred and forty two patients were enrolled for the study of which 43 were the patients of bronchiectasis and the rest 99 were non-bronchiectasis patients. *Pseudomans* was the mostly observed organism in sputum culture of bronchiectasis patients while *Klebsiella* was the leading organism in sputum culture of non-bronchiectasis patients. Against Klebsiella, Tozabactum was 100% sensitive and Tobramycin was 90% sensitive while most of the common antibiotics like Amoxycilliin, Amoxiclav, Cefipime Cephradine, Cephalexen, Piperacillin, and Cefuroxime were 100% resistant. Against Pesudomonas Ampicillin was 100% sensitive. Other sensitive antibiotics were Tobramycin (88.9%), Piperacillin (86.7%) and Tozabactum (80%).

References:

1. Barker, A 2002,' Bronchiectasis', *N Engl J Med* 2002; 346. no.18:1383-93.
2. Singleton, R, Morris, A, Redding, G , 'Bronchiectasis in Alaska Native children: causes and clinical courses; *Pediatr Pulmonol*'2000;29. no.3:182-7.
3. Chang, A, Grimwood, K, Mulholland, E , 'Bronchiectasis in indigenous children in remote Australian communities', *Med J Aust*2002;117:200-204.
4. Luce, JM, 'Bronchiectasis', In: Murray, JF, Nadel, JA (eds), *Textbook of Respiratory Medicine*. 2nd edn1994;WB Saunders and Co, Philadelphia:1398-1417.
5. Morrissey, D, 'Pathogenesis of Bronchiectasis', *Clin Chest Med* 2007,28:289-296.
6. Cole, PJ, 'A new look at the pathogenesis, management of persistent bronchial sepsis: A 'viscious circle' hypothesis and its logical therapeutic connotations, In: Davies, RJ, *Strategies for the Management of Chronic Bacterial Sepsis*. Oxford: Medicine Publishing Foundation1984:1-20.
7. Pasteur, M, Helliwell, S, Houghton ,S , 'An investigation into causitive factors in patients with bronchiectasis', *Am J Respir Crit Care Med* 2000;162:1277-1284.
8. Soriano, JB, Visick, GT, Muellerova, H, Payvandi, N, Hansell, AL, 'Patterns of comorbidities in newly diagnosed COPD and asthma in primary care', *Chest*2005;128, :2099–2107.
9. Agusti, AG, 'Systemic effects of chronic obstructive pulmonary disease', *Proc Am Thorac Soc*2005;.2:367–370.
10. Fairall, LR, Zwarenstein, M, Bateman, ED, Bachmann, M, Lombard C,Van, Rensburg, D et al,, , 'Effect of educational outreach to nurses on tuberculosis case detection and primary care of respiratory illness pragmatic cluster randomised controlled trial', *BMJ*2005; 331:750–754.
11. de Valliere, S, Barker, RD ,' Residual lung damage after completion of treatment for multidrug-resistant tuberculosis', *Int J Tuberc Lung Dis*2004;8 : 767–771.
12. Bateman, ED, Feldman, C, O'Brien, J, Plit, M, Joubert, JR, 'Guideline for the management of chronic obstructive pulmonary disease (COPD)', *S Afr Med J*2004 ; 94 :559–575.
13. Wilson, R, Sykes, D, Rutman, A, 'The effects of Haemophilus influenzae lipopolysaccharide on human respiratory epithelium', *Thorax* 1986 ;41 :728–729.
14. Wilson, R, 'The pathogenesis and management of bronchial infections: the vicious cycle of respiratory decline', *Rev Contemp Pharmacother*1992 ; 3 :103–112.
15. Sethi, S, Murphy, TF, 'Bacterial infection in chronic obstructive pulmonary disease in 2000: a State of the Art Review', *Clin Microbiol Rev*2001*;* 14 : 336–363.
16. Hill, AT, Campbell, EJ, Hill, SL, Bayley, DL, Stockley, RA, 'Association between airway

bacterial load and markers of airway inflammation in patients with stable chronic bronchitis', *Am J Med* 2000 ; 109 : 288–295.

17. Bresser, P, Out, TA, van Alpen, L, Jansen, HM, Lutter, R, ' Airway inflammation in nonobstructive and obstructive chronic bronchitis with chronic Burrows, B, and Earle, RH 1969, 'Course and prognosis of chronic obstructive lung disease: a prospective study of 200 patients', *N. Engl. J. Med* 2000 *;* 280 : 397–404.
18. Nicotra, MB, Rivera, M, Dale, AM, Shepherd, R, and Carter, R, 'Clinical, pathophysiologic, and microbiologic characterization of bronchiectasis in an aging cohort', *Chest*1995 ; 108 : 955–961.
19. Keistinen, T, Saynajakangas, Tuuponen, OT, and Kivela, SL, 'Bronchiectasis: an orphan disease with a poorly-understood prognosis', *Eur. Eur. Respir. J* 1997 *;* 10 : 2784–2787.
20. Tager, I, and Speizer FE, 'Role of infection in chronic bronchitis', *N. Engl. J. Med*. 1975 ;1292 : 563–571.
21. Haas, H, Morris, HJF, Samson, S, Kilbourn, JP, and Kim, PJ, 'Bacterial flora of the respiratory tract in chronic bronchitis: comparison of transtracheal, fiberbronchoscopic and oropharyngeal sampling methods'*, Am. Rev. Respir. Dis*1977 *;*116 : 41–47.
22. Gump, DW, Phillips, CA, Forsyth, BR, McIntosh,K, Lamborn, KR and Stouch, WH, 'Role of infection in chronic bronchitis', *Am. Rev. Respir. Dis* 1976 ; 113 : 465–474.
23. Soler, N, Torres, A, Ewig, S, Gonzalez, J, Celis, R, Rodriguez-Roisin, R et al., 'Bronchial microbial patterns in severe exacerbations of chronic obstructive pulmonary disease (COPD) requiring mechanical ventilation', *AM J RESPIR CRIT CARE MED*1998 ; 157 : 41498–1505.

Chest & Heart Journal
Vol. 39, No. 2, July 2014

CASE REPORTS

Management of Complications in Malignant Pleural Effusion: A Case Report

Shahriar Moinuddin[1], Shantonu Kumar Ghosh[1], Golam Mohiuddin Akbar Chowdhury[2], Md. Kamrul Islam[3], Md. Mobarak Hossain[4], Md. Aftabuddin[5], Asit Baran Adhikary[5]

Abstract:

Introduction: Malignant pleural effusion is a common complication of end stage malignancy. To reduce symptoms aspiration and tube thoracostomy were commonly performed and empyema may occur as a complication.

Material and Methods: In this paper, a case is reported of management of empyema in malignant pleural effusion of a patient who suffered from adenocarcinoma of lung. After developing empyema we have given pleural wash with povidone iodine, streptomycin, colistimethate sodium, metronidazole, normal saline and amikacin

Results: Complete recovery from empyema occurs and patient is now free from symptoms for last 2 months.

Discussion: Malignant pleural effusion is a common complication of end stage malignancy. To reduce symptoms aspiration and tube thoracostomy were commonly performed and emapyema may occur as a complication. Pleural wash can be good option to reduce complications like empyema.

Key Words: Malignant Pleural Effusion, Empyema, Adenocarcinoma of Lung.

[Chest & Heart Journal 2015; 39(2) : 115-120]

Introduction:

Pleural effusions are commonly occur in malignancies.[1] The most common cause of this effusion is lung, breast, and ovarian cancers and lymphoma, with breast and lung malignancies alone account for approximately 75% of these effusions.[2] Among malignancies metastatic adenocarcinoma is the most common cause.[3] Malignant pleural effusion in patients with metastatic cancers or malignant pleural mesothelioma, may often appear at the late stage of disease and significantly reduce the patient's life quality and survival.[4,5] The annual incidence of malignant pleural effusion (MPE) in the United States is estimated to be around 150 000–175 000 cases per year.[6] The pathogenesis of MPE is by

1. MS Residency (Cardiovascular and Thoracic Surgery), Bangabandhu Sheikh Mujib Medical University, Shahbagh, Dhaka.
2. Professor, Department of Thoracic Surgery, National Institute of Diseases of the Chest and Hospital, Mohakhali, Dhaka.
3. Assistant Professor, Department of Thoracic Surgery, National Institute of Diseases of the Chest and Hospital, Mohakhali, Dhaka.
4. Registrar, Department of Thoracic Surgery, National Institute of Diseases of the Chest and Hospital, Mohakhali, Dhaka.
5. Professor, Department of Cardiac Surgery, Bangabandhu Sheikh Mujib Medical University, Shahbagh, Dhaka.

Correspondence to: Prof. Dr. Md. Aftabuddin, Chairman, Department of Cardiac Surgery, BSMMU, Tele: 9661438, 01711677713
e-mail- aftab12uddin@yahoo.com

hematogenous or lymphatic implantation of tumor cells or by direct extension of tumor cells from adjacent organs such as lung, breast, chest wall or pleura. Apart from the management of the primary disease process with chemotherapy, surgery or radiation, the main concern of MPE is palliation of dyspnea. The palliation of dyspnea in MPE is managed by the removal of fluid from the pleural space by a least invasive procedure with minimal morbidity within the limited survival period of these patients. The methods of removing fluid from the pleural space can be simple single time aspiration, tube thoracostomy, or video-assisted thoracoscopic surgery (VATS) and followed by pleurodesis. Pleural aspiration and intercostal tube cause transient and rapid relief of dyspnoea drainage are associated with a high recurrence rate (10%- 40%); risk of iatrogenic empyema and pneumothorax.[7,8] Mortality does not increase during pleurodesis process but the adverse side effects depends on the chemical agent used such as fever, gastrointestinal pain and discomfort, pleuritic chest pain, respiratory failure, cardiovascular disturbance, systemic inflammatory response, empyema and reduction of lung capacity that may occur.[9]

In the present report, we report a case of malignant pleural effusion complicated by empyema completely relieved after treatment with the several pharmacologic agents.

Case Report:

A 50 year old male smoker was admitted in a private hospital with shortness of breath, right sided chest pain and cough. His Chest X-ray shows right sided pleural effusion [Fig – 1], USG of whole abdomen reveals moderate pleural effusion and mild hepatomegaly with no ascites and lymphadenopathy. CT scan shows right sided massive pleural effusion with compression collapse of right lung. Right sided tube thoracostomy was done and fluid was drained. Pleural fluid cytology showed poorly differentiated malignant cells forming solid clumps or vague glandular spaces suggestive of adenocarcinoma. CT guided FNAC was done and diagnosed as a case of adenocarcinoma of right lung. pleurodesis was done with Bleomycin and chest tube was removed after 35 days [Fig – 2]. Four cycle of chemotherapy was given there with Paclitaxel and Carboplatin, each cycle at 3 weeks interval.

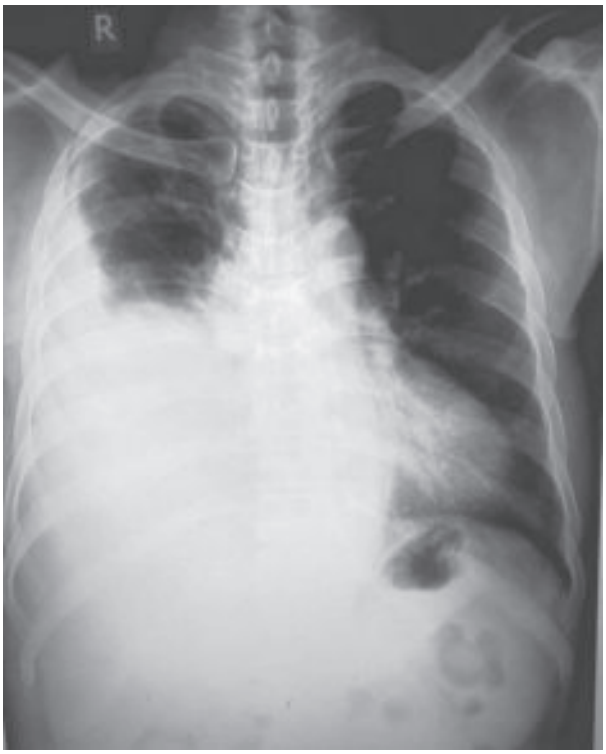


Fig.- 1: *Chest Xray showing malignant pleural effusion*

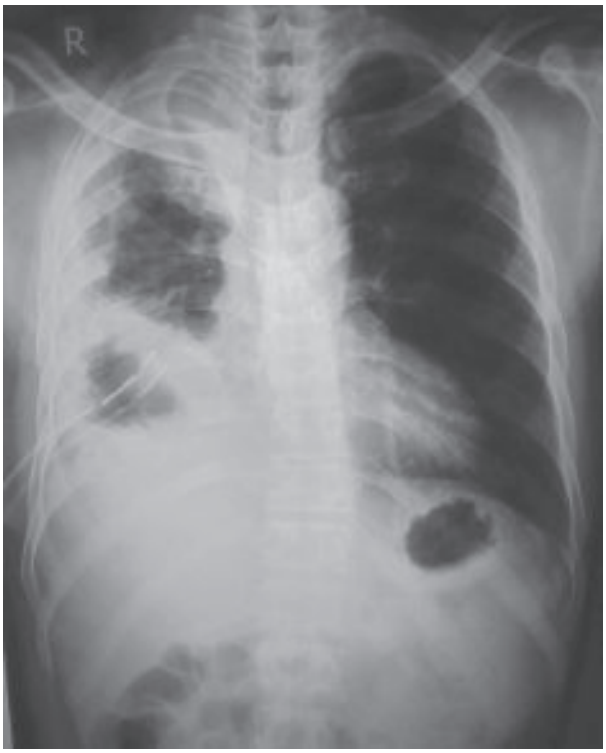


Fig.- 2: *Chest Xray following pleurodesis with bleomycin.*

One month after last chemotherapy patient developed respiratory distress, cough, chest pain, fever and swelling of previous tube thoracostomy site on coughing. After few days a cutaneous fistula developed at previous tube thoracostomy site, with discharge of frank pus.

With these complaints patient was admitted to NIDCH. On physical examination, patient was tachypnoeic and pallor. Physical examination revealed dull sound on percussion and decreased breath sounds in the middle and lower lung areas by auscultation. Chest X-ray was done and reveals right sided massive pleural effusion [Fig – 3] and CT scan shows encysted marked pleural effusion with pleural thickening and compression/collapse of adjacent lung (right) with Inflammatory changes in compressed right lung [Fig – 4] .

In view of massive pleural effusion and to relieve respiratory distress, tube thoracostomy was done under waterseal system and about 800 ml of pus was drained immediately and sent for biochemistry, cytology and for culture sensitivity. His dyspnoea was partly relieved. IT tube wash started with povidone iodine for 12 days with no response. Then Streptomycin wash was given for 6 days. Klebsiella species was found in C/S which was sensitive to Colistin and Polymyxin B. According to culture and sensitivity report IT tube wash started with Colistimethate Sodium (1 MIU)

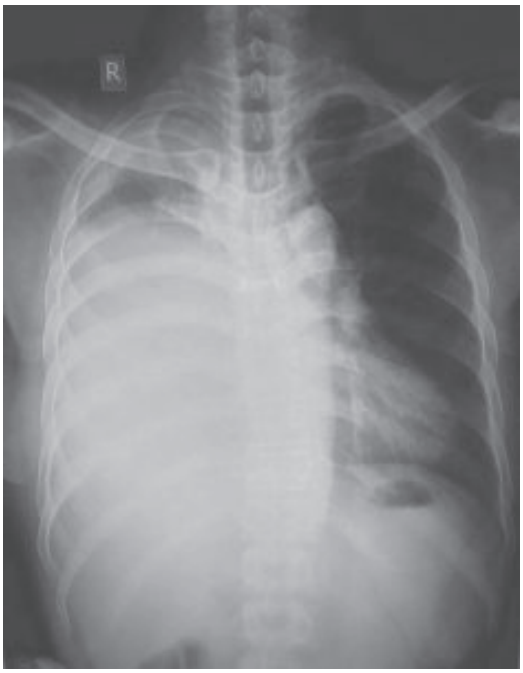


Fig.-3: *Chest Xray of recurrent pleural effusion 3 months following pleurodesis with bleomycin.*

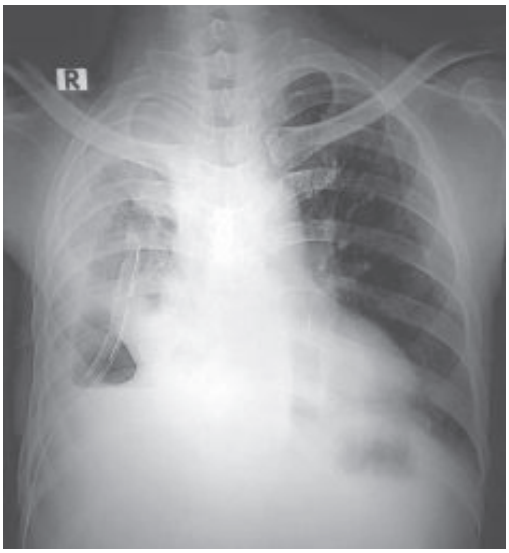


Fig.-4: *Chest Xray following Amaikacin wash before discharge.*

for 8 days. Initially response was good but gradually amount of pus increases with thickening of the pus. Metronidazole wash started and continued for 15 days. Then again povidone iodine wash given for 2 days and then with normal saline for 3 days. Again pus was send for C/S and Escherichia coli, proteus species and coagulase negative staphylococcus species were identified which are sensitive to Amikacin and Linezolid. Amikacin wash twice daily started continued for 14 days. Gradually daily IT tube collection decreases upto less 50 ml /day for consecutive 5 days and tube was removed and paitent was discharged with open drainage. [Fig – 5], 2 months after discharge patient is still free from pleural effusion.

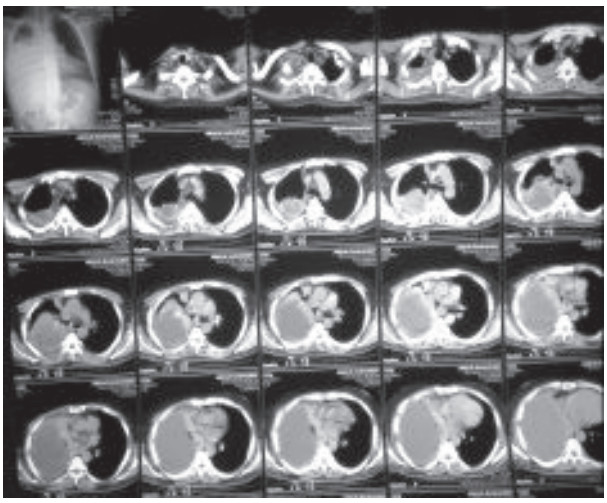


Fig.-5: *CTscan of chest showing recurrent malignant pleural effusion.*

Discussion:

We have presented a case a case of patient who had malignant pleural effusion from adenocarcinoma of lung and after tube thoracostomy followed by pleurodesis patient developed empyema and got a remarkable clinical remission by several pharmacologic agents.

Malignant pleural effusion is a common complication of end stage malignancy significantly impairing quality of life. The deteriorating effect on quality of life includes repeated hospitalization, altered pulmonary function, fatigue, and the requirement for numerous interventional procedures. Most effusions do not respond to systemic chemotherapy and the treatment is generally palliative[10].

Dyspnea was the most common presenting symptom in patients with malignant pleural effusion in our study. Although cough and pain can also be debilitating. All these greatly diminish the quality of the final phase of life for patients living with cancer. This is also true in studies done elsewhere. [10-12]

Chest radiographs confirm the size and location of the pleural collection. Thoracentesis is usually diagnostic and also therapeutic. Exudative and hemorrhagic collections should be considered metastatic until proved otherwise. Cytologically malignant cells are detected in approximately 50% of proven MPEs.[13-15] In our case pleural fluid cytology showed poorly differentiated malignant cells forming solid clumps or vague glandular spaces suggestive of adenocarcinoma. A CT scan may give information about loculations of MPE, the primary disease process, and anatomy of other organs in the thorax.

Symptomatic relief is frequently attained by removing a large amount of fluid. Tube thoracostomy involves making a hole in the intercostal space into the pleural cavity (blindly or with image guidance) under aseptic precautions with placement of a secured tube for continuous drainage into a water-sealed container. Tube thoracostomy can be preceded by VATS with operative visualization of the pleural cavity and pleural biopsy for diagnostic purposes. But, VATS can only be performed in those patients who can tolerate anesthesia and single lung ventilation.[16]

Either large-bore (28–36 F) or small-bore (7–16 F) chest tube can be used for reliable drainage and both have equivalent results. Initially tube thoracostomy was done in a private setting and after pleurodesis tube was removed. Then due to empyema we have done tube thoracostomy with 32 F tube to evacuate the collection which was pus.

Common complications of pleural fluid aspiration and intercostal tube drainage are associated with a high recurrence rate and risk of iatrogenic pneumothorax and empyema.[7,8] Thoracostomy tube should not be kept for a prolonged period for fear of infection, empyema, pneumothorax, etc. The recurrence of MPE is seen in around 80% of patients within 30 days after the removal of the tube.[17] In our case empyema developed 1 months after chemotherapy and about 2 months after removal of the tube.

Bleomycin is the most widely used antineoplastic agent for the management of malignant pleural effusion. Its mechanism of action is predominantly as a chemical sclerosant. Success rate after single administration varies from 58% to 85%.[12] There are other common agents used for pleurodesis. Talc has been proven to be one of the most effective sclerosing agents for treating malignant pleural effusion with success rate varies between 88%-100%.[18, 19] Tetracycline pleurodesis found to be effective with a success rate of 60%. Doxycycline has been proposed as an alternative to tetracycline with a similar success rate ranging from 25% to 100%.[18] Other agents used rarely include minocycline, mitoxantrone, corynebacterium parvum extract, interferons, interleukins and several chemotherapeutic agents. In our case initially pleurodesis was done by single administration of bleomycin, but empyema thoracis was developed.

Intrapleural therapy, many strategies have been piloted over the years to reduce the bacterial burden within an infected pleural space. Regular pleural lavage with physiological saline via chest tube has been a standard practice in some European centres. Irrigation with antiseptic or antimicrobial washouts to sterilize the pleural cavity has been reported in observation series/cases, predominantly after surgical intervention. Povidone iodine, normal saline and several

antimicrobial agents have shown good results in previous studies.[20-22] After development of empyema IT tube wash started with povidone iodine for 12 days then Streptomycin wash was given for 6 days. After getting the C/S IT tube wash started with Colistimethate Sodium (1 MIU) for 8 days. Initially response was good but gradually amount of pus increases with thickening of the pus. Metronidazole wash started and continued for 15 days. Then again povidone iodine wash given for 2 days and then with normal saline for 3 days. Due to no resonse again pus was send for C/S Amikacin wash, twice daily started continued for 14 days. Gradually daily IT tube collection decreases upto less 50 ml /day for consecutive 5 days and tube was removed and patient was discharged with open drainage and 2 months after discharge patient is still free from pleural effusion.

Conclusion:

Malignant pleural effusion is a common complication of end stage malignancy. To reduce symptoms aspiration and tube thoracostomy were commonly performed and empyema may occur as a complication. We reported a case of successful management of empyema thoracis followed by tube thoracostmy and pleurodesis due to adenocarcinoma of lung with several pharmacological agents like povidone iodine, streptomycin, colistimethate sodium, metronidazole, normal saline and amikacin.

References:

1. Light RW. Pleural Disease. 4th ed. Philadelphia, PA: Lippincott, Williams and Wilkins 2001; pp: 120-123.
2. Lynch TJ. Management of malignant pleural effusions. Chest 1993; 103: 385-389.
3. Awasthi A, Gupta N, Srinivasan R, et al. Cytopathological spectrum of unusual malignant pleural effusions at a tertiary care centre in north India. Cytopathology. 2007;18:28–32.
4. Basso SM, Mazza F, Marzano B, Santeufemia DA, et al. Improved quality of life in patients with malignant pleural effusion following videoassisted thoracoscopic talc pleurodesis. Preliminary results. Anticancer Res 2012; 32: 5131-5134.
5. Haas AR, Sterman DH. Novel intrapleural therapies for malignant diseases. Respiration 2012; 83: 277-292.
6. MarelM, Zrustova M, Stasny B, et al. The incidence of pleural effusion in a well deûned region. Epidemiologic study in central Bohemia. Chest 1993; 104:1486–1489.
7. Lambert C, Shah HH, Urschel HC Jr, et al. The treatment of malignant pleural effusions by closed trocar tube drainage. Ann Thorac Surg 1967;3:1–5.
8. Anderson CB, Philpott GW, Ferguson TB. The treatment of malignant effusions. Cancer 1974;33:916–922.
9. Snell RS. Clinical Anatomy for medical students. 6th ed. Lippincott Williams & Wilkins, 2000, 79-81.
10. Thomas A. D'Amico. Malignant Pleural effusion. www.mascc.org/ktml2/images/uploads/16th_presentation_summaries/Damicocompleteoverviewplen2.doc
11. American Thoracic Society. Management of Malignant pleural effusion. Am J Respir Crit Care Med 2000; 162: 1987-2001
12. Antunes G, Neville E, Duffy J, et al. BTS guidelines for the management of malignant pleural effusions. Thorax 2003; 58 (Suppl.11): 1129-1138
13. Irani DR, Underwood RD, Johnson EH, et al. Malignant pleural effusions: A clinical cytopathologic study. Arch Intern Med. 1987;147:1133–1136.
14. Prakash UB, Reiman HM. Comparison of needle biopsy with cytologic analysis for the evaluation of pleural effusion: Analysis of 414 cases. Mayo Clin Proc. 1985;60:158–164.
15. Salyer WR, Eggleston JC, Erozan YS. Efficacy of pleural needle biopsy and pleural fluid cytopathology in the diagnosis of malignant neoplasm involving the pleura. Chest. 1975;67:536–539.
16. De Campos JR, Vargas FS, DeCampos Werebe E, et al. Thoracoscopy talc poudrage:

A 15-year experience. Chest. 2001;119: 801–806.

17. Tassi GF, Cardillo G, Marchetti GP, et al. Diagnostic and therapeutical management of malignant pleural effusion. Ann Oncol. 2006;17:1–2.

18. Barbetakis N, Vassiliadis M, Kaplanis K, et al. Mitoxantrone pleurodesis to palliate malignant pleural effusion secondary to ovarian cancer. BMC Palliative care 2004; 3:4.

19. Steven A. Sahn. Malignant Pleural Effusion. Semin Respir Crit Care Med 2001; 22 : 607-615.

20. Ng T, Ryder BA, Maziak DE, et al. Treatment of postpneumonectomy empyema with debridement followed by continuous antibiotic irrigation. J Am Coll Surg 2008; 206:1178–1183.

21. Mennander A, Laurrika J, Kuukasjarvi P, *et al*. Continous pleural lavage may decrease postoperative morbidity in patients undergoing thoracotomy for stage 2 thoracic empyema. Eur J Cardiothorac Surg 2005; 27:32–34.

22. Mullins MM, Walker I, Standridge RD. Using povidone-iodine to treat empyema. J of Wound Care 2001; 10:155–156.

Chest & Heart Journal
Vol. 39, No. 2, July 2014

CASE REPORTS

Esophageal Diverticulum - A Case Report

Imran Ahmed[1], Md. Delwar Hossain[2], Md. Shamsul Alam[3], Omar Sadeque Khan[4], Md. Aftabuddin[5]

Abstract:

Patients with esophageal diverticulm may present with a variety of symptoms. Making diagnosis of esophageal diverticulum may be difficult in some cases. Delayed or missed diagnosis of this disorder may lead to more serious problems including gastrointestinal bleeding, aspiration pneumonia and cancer. We have reported a case of esophageal diverticulum with uncommon presentation. This 45 years age male patient had history of retrosternal chest pain and breathlessness for 1 year. Diagnosis was confirmed by echocardiography, CT scan of chest, barium swallow and rigid oesophagoscopy. We have done diverticulectomy with left posterolateral thoracotomy incision. There was no intraoperative and postoperative complications. After surgical removal of the diverticulum the patient remained fully asymptomatic. So appropriate diagnosis and management of esophageal diverticulum results in resolution of symptoms and decreases risk of morbidity.

Keywords: *Esophagus, diverticulum, chest pain, surgery, treatment outcome.*

[Chest & Heart Journal 2015; 39(2) : 121-125]

Introduction:

Esophageal diverticulum is an out-pouching or sac of the epithelial-lined tissue of the esophagus. It may be a true diverticulum involving all layers of the esophagus; or a false diverticulum involving only the mucosa and submucosal layers that protrude into the circular and longitudinal muscle of the esophagus[1]. It's prevalence ranges between 0.015% and 2% but only seldom they become sympto-matic[2]. Esophageal diverticulum is generally categorized by esophageal location: upper- zenker's diverticulum, middle and lower- epiphrenic diverticulum. It can be further categorized according to the mechanism of formation into: traction and pulsion diverticulum. Traction diver-ticulum occurs secondary to pulling forces on the outer aspect of the oesophagus such as midesophageal diverticulum. Pulsion diverticulum occurs secondary to increased intraluminal pressure such as Zenker's diverticulum[1]. Zenker diverticulum is located just proximal to the upper oesophageal sphincter in the posterior midline at the cleavage plane (known as Killian dehiscence) between the circular and oblique fibers of the crico-pharyngeus muscle[3]. Both Zenker's and epiphrenic diverticula are false diverticula with the mucosal layer protruding

1. Resident, Phase-B , MS (Cardiovascular and thoracic surgery), Bangabandhu Sheikh Mujib Medical University
2. House Surgeon, Department of Thoracic Surgery, *National Institute of Chest Diseases and Hospital* (NIDCH), Mohakhali, Dhaka
3. Associate Professor of Thoracic Surgery, *National Institute of Chest Diseases and Hospital* (NIDCH), Mohakhali, Dhaka
4. Medical officer, Department of Cardiac Surgery, Bangabandhu Sheikh Mujib Medical University
5. Professor and Ex- Chairman, Department of Cardiac Surgery, Bangabandhu Sheikh Mujib Medical University

Correspondence to: Dr. Md. Delwar Hossain, House Surgeon, Department of Thoracic Surgery, *National Institute of Chest Diseases and Hospital* (NIDCH), Mohakhali, Dhaka, Mobile no-01711851454, Esophageal diverticulum - A Case Report

through gaps in the outer muscular layer. The underlying mechanism in both cases is an abnormal motility of the esophagus with an impaired relaxation of the sphincter while propulsive contractions continue. Evidently, this leads to an increased intraluminal pressure just above the sphincters which then causes the mucosal layer to develop a pouch pushing through weakened sites of the muscular wall[4,5,6]. A large pro-portion of patients with esophageal diverticula may be asymptomatic. However, serious complications such as tracheal fistula, hemorrhage, vocal cord paralysis and retained foreign body may occur[7]. In addition, the risk of malignancy has also been described in case of esophageal diverticulum . The carcinogeneses may be caused by chronic irritation by food, inflammation and repeated injury[8,9]. So esophageal diver-ticulum needs prompt diagnosis and surgical intervention. We have described a successful diverticulectomy of a esophageal diverticulum with uncommon presentation.

Case Report:

Md. Abul kashem 45 years of age normotensive, non diabetic, non smoker, construction labour hailing from Tangail presented with retro-sternal chest pain and breathlessness for 1 year. Chest pain was moderate in nature, sharp and stabbing in character, having no radiation, provoked by lying condition and not relieved by taking rest. Patient also had history of breathlessness which was more marked during moderate to severe exertion and relieved by taking rest. For evaluation of chest pain and breathlessness patient having history of admisson in 'Badr Al Samaa' hospital of Oman. He had no his-tory of dyspagia, cough, fever, haemoptysis, swelling of ankle and weight loss. His bowel and bladder habits were normal. General examination findings were normal. Respiratory, cardiovascular, ali-mentary system and other systems examinations revealed normal findings. Chest X-ray, ECG, echocardiography and CT scan of chest were done in 'Badr Al Samaa' hospital. Chest X-ray and ECG showed normal findings. Echocardiography 2D & M Mode revealed 77% ejection fraction, normal valves and chambers, no regional wall motion abnormality and all other normal findings. CT Scan of chest (Plain and contrast) revealed a 82x68x67 mm thin walled, non-enhancing lesion in posterior mediastinum on right side, extending from the level of just below the tracheal bifurcation till right crus of diaphragm. Final impression of CT scan of chest (Plain and contrast) was esophageal diverticulum (Figure-1). Then patient came in Bangla-desh and was admitted in *National Institute of Chest Diseases and Hospital* (NIDCH) for definitive management. Barium swallow showed esophageal diverticulum at lower end of esophagus (Figure-2). Rigid oesophagoscopy was done in *National Institute of Chest Diseases and Hospital* (NIDCH) under general anesthesia .A diverticulum was found at 27 cm from upper incisor teeth which communicate with esophagus. Routine laboratory investigations including complete blood count, renal and liver function tests were within the normal range. With all aseptic precaution under general anesthesia with one lung ventilation left posterolateral thoracotomy at 8th intercostal space was done. After opening the left hemithorax, mobilization of oesophagus was done. Then diverticulum was identified. Neck of diverticulum was found at mid oesophagus level. After stappling, resection of the diverticulum was done from oesophagus (Figure-3).

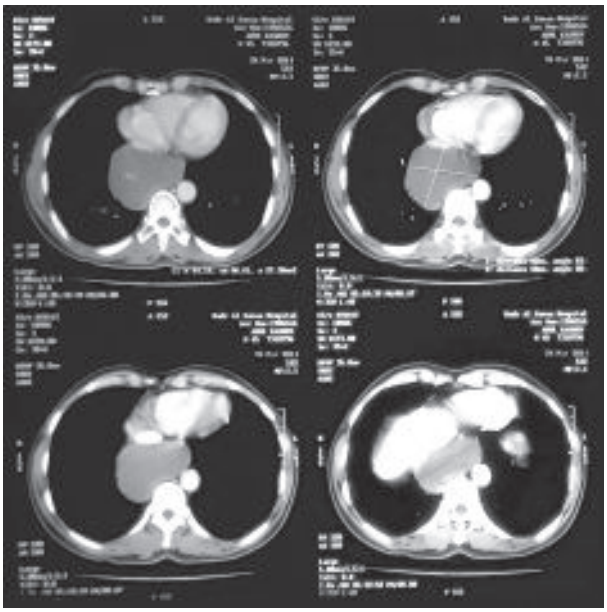


Fig.-1: *CT Scan of chest showed esophageal diverticulum.*

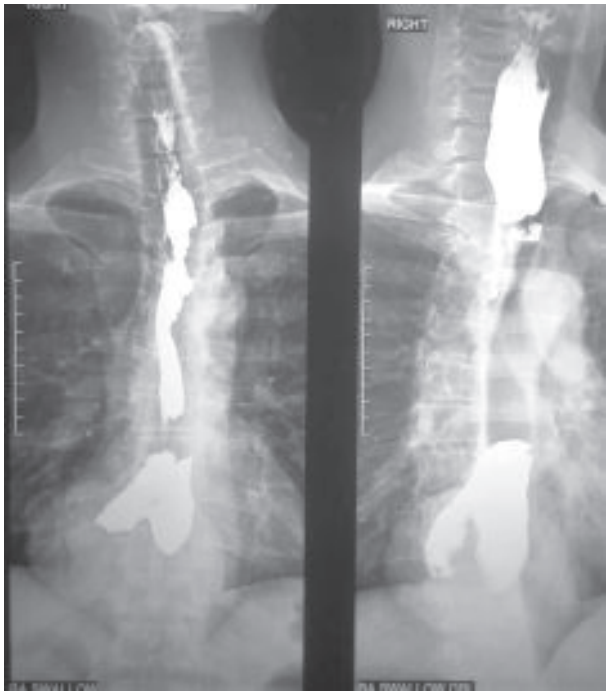


Fig.-2: *Barium swallow showed esophageal diverticulum at lower end of esophagus .*

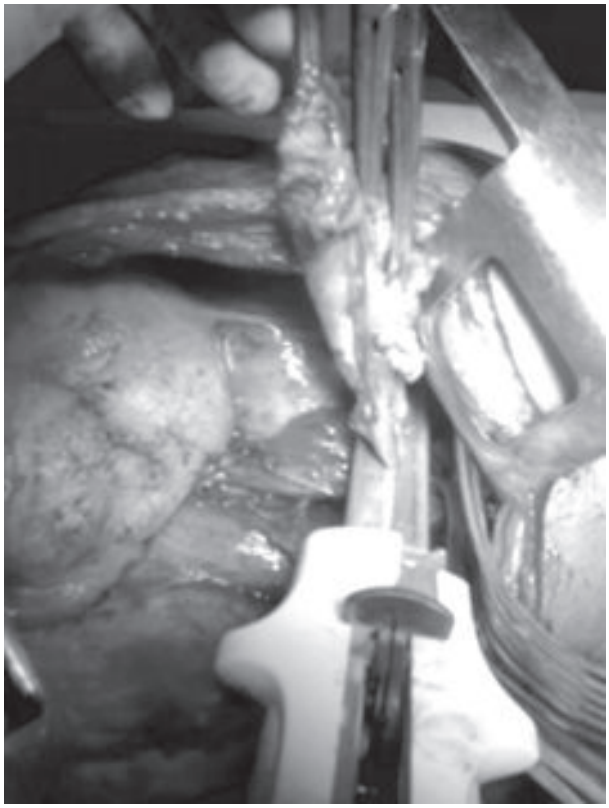


Fig.-3: *Diverticulectomy was done with linear stapler.*

After proper haemostasis keeping a chest drain tube in left hemi-thorax. Then chest was closed in layers. Histopathologic examination of the excised tissue revealed all coats of the esophagus with focal areas of keratinization of the squamous epithelium. There was absence of any cystic part in excised tissue and no malignancy was seen. On the 7th postoperative day the patient was asked to swallow 500 ml normal saline mixed with 2 ml gentian violet. There was no colour change of chest drainage tube. So oral feeding using first liquid food began from 8th postoperative day. There was no intra-operative and post-operative complications. Intercostal tube off was done on 8th post-operative day. The patient was discharged from *National Institute of Chest Diseases and Hospital* (NIDCH) on 14th post operative day. Currently he remained asymptomatic.

Discussion:

We have described a esophageal diverticulum with uncommon presentation. Neck of this diverticula was found at mid oesophagus level. This patient had history of retrosternal chest pain and breath-lessnes for 1 year. Schima et al.[10] stated that symptoms of mid-oesophageal diverticula were dysphagia, cough, choking, regurgitation, retrosternal pain and gurgling in neck. But in this study patient having only retrosternal chest pain and breathlessness. Ferreira et al.[11] showed that 80-90% predominant symptom of esophageal diverticula was dysphagia. But in this study there was no history of dysphagia. Pistorius et al.[12] stated that chest pain occurs between 38 to 60% of patients with midoesophageal diverticula. In this study predominant symptom was retrosternal chest pain. Hoffmann et al.[13] presented a case of midoesophageal diverticulum where retrosternal exertional chest pain radiating to the left arm. The clinical picture was suggestive of cardiac ischaemia until decompression of a food impacted midoesophageal diverticulum led to complete disappearance of the pain. Richter et al.[14] proposed that the underlying mechanism of retrosternal chest pain because of an impacted midoesophageal diverticulum was unclear. Cassivi et al.[15] stated midesophageal

diverticula were traditionally considered traction diverticula secondary to mediastinal inflammatory reaction. Thomas et al.[16] suggested that traction diverticula were rare and mostly result from scarring following an inflammation predominantly occurring in the mediastinal lymph nodes. Fekete and Vonns[17] proposed that many midthoracic diverticula were also associated with motility abnormalities such as spasm, achalasia or hypertension of the lower esophageal sphincter. But in this case report there was no such association. Laubert et al.[18] had done esophageal manometry in their esophageal diverticulum case. Laubert et al.[18] stated that esophageal manometry revealed functional disorders. But esophageal manometry was not performed in our case. Ferreira et al.[11] suggested that a sudden increase in the severity of dysphagia and regurgitation and/ or development of alarm symptoms such as local pain and hemoptysis or hematemesis may signal the presence of squamous cell carcinoma within the zenker's diverticulum. Midoesophageal diverticula were generally thought to be mostly asymptomatic not requiring diagnostic tests other than barium oesophagogram[19,20] . We had also done barium oesophagogram for diagnosis of esophageal diverticulum. Schima et al.[10] stated that complications have been reported such as major bleeding from a diverticulum and rupture with the development of oesophago-pleural or oesophago-bronchial fistulae. There was absence of any such complication in our case report. Yuan et al.[3] suggested that diverticulum might be either resected (diverticulectomy) or more conservatively suspended and fixed on the hypopharyngeal wall (diverticulopexy) or invaginated into esophagus itself (diverticular inversion). Laubert et al.[18] proposed that in case of esophageal diverticulum, surgical approach was open diverticulectomy with or without performance of cricopharyngeal myotomy. In our case report we had done diverticulectomy with left posterolateral thoracotomy incision. Yuan et al.[3] stated that complications for diverticulectomy were perforation, erosion with bleeding, nerve injury, mediastinitis, abscess formation, hematoma, fistula formation, stenosis and overall morbidity, mortality. But there was no intraoperative and postoperative complications in our case report.

Reference:

1. Matthews BD, Nelms CD, Lohr CE, Harold KL, Kercher KW, Heniford BT. Minimally invasive management of epiphrenic esophageal diverticula. Am Surg 2003; 69: 465-70.
2. Schima W, Schober E, Stacher G, et al. Association of midoesopha-geal diverticula with oesophageal motor disorders. Videofluoroscopy and manometry. Acta Radiol 1997; 38:108- 14.
3. Yuan Y, Zhao YF, Hu Y, Chen LQ. Surgical treatment of zenker's diverticulum. Dig Surg 2013;30:207–18.
4. Costantini M, Zaniotto G, Rizetto C, Narne S, Ancona E. Oesophageal diverticula. Best Pract Res clin gastroenterol 2004; 18:3-7.
5. Thomas Ml, Anthony AA, Fosh BG, Finch JG, Maddern GJ. Oesophageal diverticula. Br J Surg 2001; 88:629-42.
6. D'Journo XB, Ferraro P, Martin J, Chen LQ, Duranceau A. Lower oesophageal sphincter dysfunction is part of the functional abnormality in epiphrenic diverticulum. Br J Surg 2009; 96:892-00.
7. Sen P, Kumar G, Bhattacharyya AK. Pharyngeal pouch: associations and complications. Eur Arch Otorhinolaryngol 2006; 263: 463–8.
8. Honda H, Kume K, Tashiro M. Early stage esophageal carcinoma in an epiphrenic diverticulum. Gastrointest Endosc 2003; 57: 980–2.
9. Kimura H, Konishi K, Tsukioka Y. Superficial esophageal carcinoma arising from the diverticulum of the esophagus. Gastrointest Endosc 1997; 29: 53–4.
10. Schima W, Schober E, Stachepr G, et al. Association of midoe-Sophageal diverticula with motor disorders. Acta Radiologica 1997; 38: 108-14.
11. Ferreira LEVVC, Simmons DT, Baron TH. Zenker's diverticula: pathophysiology,

clinical presentation and flexible endoscopic management. Dis Esophagus 2008; 21: 1-8.

12. Pistorius GA, Hegenauer K, Pahl S, Feifel G. Intrathoracic diver- ticulum caused by a leiomyoma: a rare case of spontaneous oesophageal rupture. Eur J Surg 2000; 166: 426–8.
13. Hoffmann JC, Pistorius G, Muller P and Zeitz M. Unusual case of a large midoesophageal diverticulum mimicking unstable angina pectoris. Journal of Internal Medicine 2002;251:355–60.
14. Richter JE, Bradley LA, Castell DO. Esophageal chest pain: current controversies in pathogenesis, diagnosis and therapy. Ann Intern Med 1989; 110: 66–8.
15. Cassivi SD, Deschamps C, Nichols FC , Allen MS, Pairolero PC. Diverticula of the esophagus. Surg Clin North Am 2005; 85: 495–503.
16. Thomas ML, Anthony AA, Fosh BG, Finch JG, Maddern GJ. Oesophageal diverticula. Br J Surg 2001; 88:629-42.
17. Fekete R, Vonns C. Surgical management of esophageal thoracic diverticula. Hepatogastroenterology 1992; 39:97- 9.
18. Laubert T, Hildebrand P, Roblick UJ, et al. Mis approach for diverticula of the esophagus. Eur J Med Res 2010; 15: 390-6.
19. Baker ME, Zuccaro G, Achkar E, Rice TW. Esophageal diver-ticula: patient assessment. Semin Thorac Cardiovasc Surg 1999;11: 326–6.
20. Rice TW, Baker ME. Midthoracic esophageal diverticula. Semin Thorac Cardiovasc Surg 1999; 11: 352–7.

Chest & Heart Journal
Vol. 39, No. 2, July 2014

CASE REPORTS

An Adult Lady of Sarcoidosis Presenting with Cervical Lymphadenopathy

Md. Hasanur Rashid[1], Md. Abdur Rouf[2], Md. Shahedur Rahman Khan[2], Bashir Ahmed[2], Olia Sharmeen[3], Nigar Sultana Ahmed[4]

[Chest & Heart Journal 2015; 39(2) : 126-127]

Introduction:

Sarcoidosis is a multisystem granulomatous disorder of unknown aetiology characterized by the presence of non caseating granulomas. Over 90% of cases affect the lungs, but the condition can involve almost any organ[1]. The lymph nodes most frequently affected in sarcoidosis are those of the hilar and paratracheal groups. Of the superficial nodes, those of the right scalene group are most commonly affected but enlargement of any of the superficial nodes may be found[2]. Only 5% cases of sarcoidosis may present with superficial lymphadenopathy[1]. On the other hand, lymph nodes are the most common extrapulmonary site of tuberculosis where cervical and mediastinal glands are affected more frequently[3]. In tuberculosis the nodes are usually painless and initially mobile but become matted together with time. When caseation and liquefaction occur, the swelling becomes fluctuant and may discharge through the skin[3].

Case presentation

A 40 year old lady presented with fever, dysphagia, loss of appetite, weight loss, multiple cervical lympadenopathy and joint pain in June 2011. She was started anti tubercular therapy CAT 1 from 10 July'11 initially for six months on the basis of the clinical scenario and right supraclavicular lymph node biopsy which revealed multiple discrete epithelioid granuloma mostly non caseating type with focal central necrosis (Granulomatous Lymphadenitis, histologically consistent with Tuberculosis). But anti tubercular therapy was extended for further three months as a case of 'delayed responder'.

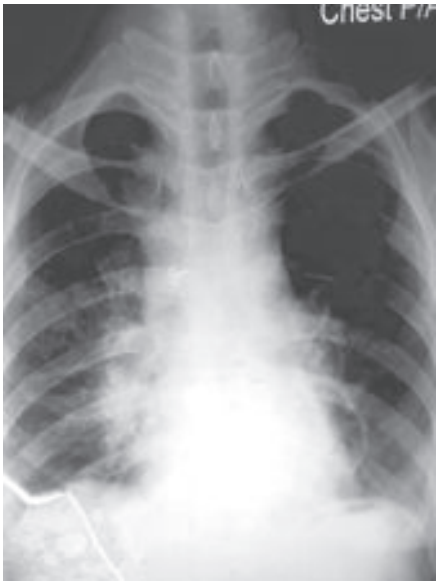


Fig.-1: *CXR (before treatment)*

Unfortunately the symptoms of the patient did not subside. So biopsy from cervical lymph node was again taken and histopathology revealed granulomatous lymphadenitis histologically tuberculosis. Ultimately she was started anti tubercular therapy with CAT2 regimen (modified) from 20 June'12. At the end of the therapy she was not improved rather developed bilateral parotid

1. Assistant Professor of Respiratory Medicine, National Institute of Diseases of the Chest & Hospital (NIDCH), Mohakhali, Dhaka
2. Associate Professor of Respiratory Medicine, National Institute of Diseases of the Chest & Hospital (NIDCH), Mohakhali, Dhaka
3. Associate Professor of Paediatrics (Haematology & Oncology), Sir Salimullah Medical College & Mitford Hospital, Dhaka
4. Emergency Medical Officer, National Institute of Diseases of the Chest & Hospital (NIDCH), Mohakhali, Dhaka

Correspondence to: Dr. Md. Hasanur Rashid, Assistant Professor of Respiratory Medicine, National Institute of Diseases of the Chest & Hospital (NIDCH), Mohakhali, Dhaka, Cell: 01711351587, Email: drhasanur.rashid@gmail.com

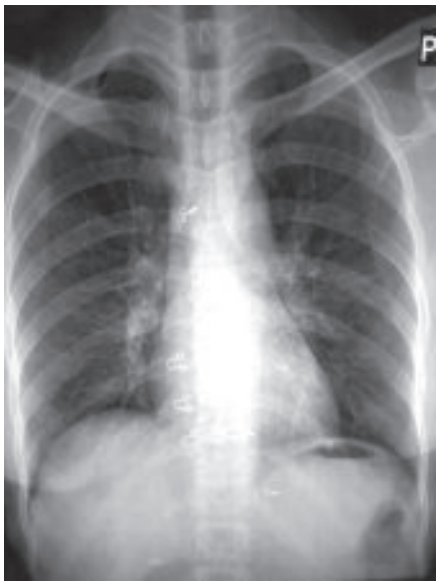


Fig.-2: *CXR (after treatment)*

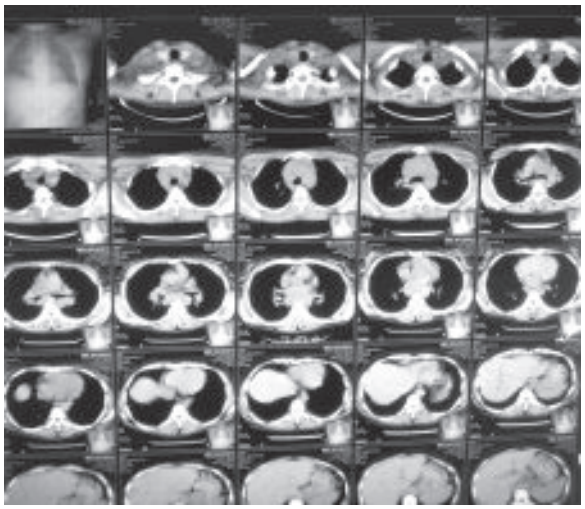


Fig.-3: *CT Scan of Chest showing BHL*

swelling, dryness of mouth, shortness of breath and multiple joint pain. Extensive survey was done that revealed Lymphopenia on Complete Blood Count; mediastinal lymphadenopathy and inflammatory change in posterior basal segment of right lower lobe on CT scan Chest with contrast; restrictive pattern of breathing on spirometry; cobblestone appearance of whole endobronchial mucosa on Fibre Optic Bronchoscopy; high level of Angiotensin Converting Enzyme in serum(203 U/L); splenomegaly (12.3 cm) on ultrasonography; non caseating discrete granuloma with fibrosis and foreign body type giant cell (consistent with sarcoidosis) on cervical lymph node biopsy. The results of serum calcium level, serum

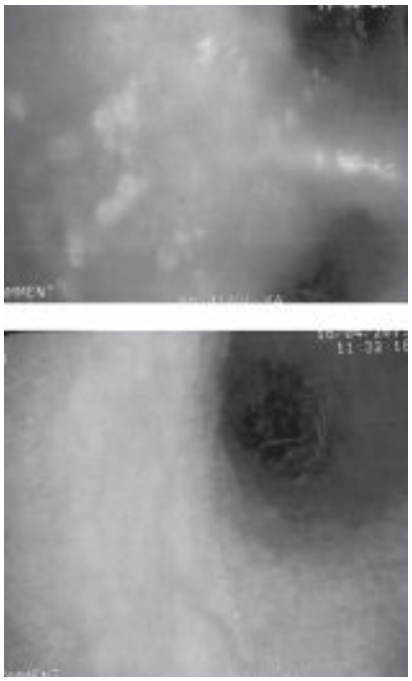


Fig.-4: *FOB showing cobblestone appearance of endobronchial mucosa*

aminotransferase level, Mantoux test and Xray of the bones of both her hands were normal.

So the diagnosis of "systemic sarcoidosis" was made. Accordingly the patient was treated with prednisolone from 19 May'13 initially 40 mg for 2 months then the dose was tapered to 5 mg by four months and continued 5mg per day for further two months. Now the patient is symptomatically improved, and disappearance of superficial lymphnodes also with mediastinal lymphnodes as evident on CXR.

Conclusion:

Though hilar and mediastinal lymphadenopathy is common presentation in sarcoidosis, cervical lymphadenopathy sometimes need meticulous evaluation to find out the exact etiology.

Tubercular lymphadenopathy is very common extrapulmonary tuberculosis in Bangladesh, the diagnosis of sarcoidosis presenting with cervical lympadenopathy yet to be considered.

References

1 Davidson's Principles & Practice of Medicine, 22nd edition, Page no-709, 710

2 Crofton and Douglas's respiratory Diseases2, 5th edition, Page No-1049

3 Davidson's Principles & Practice of Medicine, 22nd edition, Page no-690.