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# THE CHEST & HEART ASSOCIATION OF BANGLADESH

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Vol. 38, No. 1, January 2014

## ORIGINAL ARTICLE

# Association Between Metabolic Syndrome and Obstructive Sleep Apnoea (OSA)

**Pulak Kumar Dey[1], Md. Touhidul Islam Khan[1], Md. S.M.Lutfor Rahman[2], Md. Zakir Hossain Sarker[2], Md. Abdus Shakur Khan[2], Md. Abdur Rouf[3], Mohammed Shahedur Rahman Khan[3], Bashir Ahmed[3], Md. Khairul Anam[2], Md. Abu Raihan[3], Md.Ali Hossain[4]**

**Abstract:**

***Background & objective:*** *Obstructive sleep apnea (OSA) is characterized by recurrent episodes of partial or complete upper airway obstruction during sleep and is manifested as a reduction (hypopnea) or cessation (apnea) of airflow with ongoing respiratory effort and are associated with frequent disruptions of sleep. It has immense importance in terms of its complications like obesity, hypertension, insulin resistance, altered lipid profile leading to metabolic syndrome (MS). The present study was undertaken to find the association between metabolic syndrome and OSA.*

***Patients & Methods:*** *This case-control analytical study was conducted on 84 consecutive subjects. Of them 54 were cases (excessive day-time somnolence not explained by any other factors with at least any two of the symptoms like choking / gasping during sleep, recurrent awakening from sleep, unrefreshing sleep, daytime fatigue, or impaired concentration) and 30 were controls. Metabolic syndrome was diagnosed based on WHO criteria. In polysomnographic study if AHI was found < 5 no OSA was considered, AHI 5-15 was mild,15-30 was moderate and AHI >30 was regarded as severe type of OSA.*

***Result:*** *More than three-quarters (77.6%) of metabolic syndromes (MS) were observed to be associated with OSA compared to 11.5% of the subjects who had not metabolic syndrome. The severity of MS increased sharply with types of OSA, 85% with moderate type while 96% cases of severe OSA had metabolic syndrome. Among 4 variables (Age, sex, obesity and OSA which were found significantly associated with MS in univariate analysis), age of the subjects and OSA were observed to be the independent predictors of MS in multivariate analyses. The older subjects (50 years and more) were 2.6 (95% CI = 1.3 – 4.1) times more prone to have MS than their younger counterparts (p = 0.021). Likewise the subjects having OSA was 7.4(95% CI = 2.3 – 20.9) times more likely to have MS than the subjects without OSA (p < 0.001).*

***Conclusion:*** *Obstructive sleep apnoea is associated with metabolic syndrome and should be treated early to prevent its complications.*

***Key words:*** *OSA, Metabolic syndrome, Polysomnography, CPAP, BMI.*

***[Chest & Heart Journal 2014; 38(1) : 1-6]***

## Introduction:

OSA a neglected issue in developing country like Bangladesh but having immense importance because it is associated with various co-morbidities like IHD, cardiac arrhythmias, HTN, DM, dyslipidemia, obesity, cognitive impairment, pulmonary hypertension etc. Many patients often presents with uncontrolled DM and hypertension

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in spite of getting optimum medical treatment due to unrecognized OSA. Lacking knowledge regarding OSA among physicians giving rise to various miserable conditions which could be treated easily. OSA is a common and most importantly treatable disease. The current definition of obstructive sleep apnea (OSA) syndrome is based on the consensus conference statement of an American Academy of Sleep Medicine task force convened in 1999. OSA is characterized by recurrent episodes of partial or complete upper airway obstruction during sleep. These episodes are manifested as a reduction (hypopnea) or cessation (apnea) of airflow with ongoing respiratory effort and are associated with repeated disruptions of sleep. Overnight monitoring should demonstrate five or more events of obstructed breathing per hour of sleep. These events may include any combination of obstructive hypopneas/apneas or arousals related to respiratory effort. Mechanistically, recurrent obstructive events with cyclic intermittent hypoxia and sleep fragmentation are believed to be the key triggers of various pathogenetic mechanisms in OSA, including sympathetic activation, cellular oxidative stress, and systemic inflammation [1-3] leading to characteristic features of the metabolic syndrome. Intermittent hypoxia followed by reoxygenation may result in increased oxidative stress, which is a fundamental cellular pathogenic process in metabolic and cardiovascular function.[4,5] A number of observational studies have demonstrated that OSA is independently associated with increased markers of oxidative stress.[4-6] Intermittent hypoxia may selectively upregulate inflammatory pathways over adaptive pathways[7] and generate cytokines and other mediators that modulate metabolic and vascular functions.[8] OSA is associated with heightened sympathetic activation both at night and in the day[9] and is believed to be important mechanism for hypertension.

In addition to the polysomnographic criteria for OSA, the patient must exhibit excessive daytime somnolence that is not explained by other factors or at least two of the following: choking/gasping during sleep, recurrent awakenings from sleep, unrefreshing sleep and daytime fatigue or impaired concentration. The severity of OSA is often characterized according to the symptoms, frequency of breathing events recorded during overnight polysomnography, and degree of oxygen desaturation.

**The metabolic syndrome**

Metabolic syndrome was first described as a cluster of metabolic abnormalities, with insulin resistance as the central pathophysiological feature and it was labeled as "Syndrome X". There are different accepted criteria for its definitions and the most widely used criteria have been proposed by the World Health Organization (WHO), the European Group for the Study of Insulin Resistance (EGIR), the National Cholesterol Education Program – Third Adult Treatment Panel (NCEP ATP III) and International Diabetes Federation . All these organizations have suggested that the core features of metabolic syndrome are central/visceral obesity, hypertension, insulin resistance and dyslipidaemia but they have applied the criteria differently in identifying the cluster of syndrome components.

**Historical perspective of OSA:**

The sleep apnoea/hypopnoea syndrome is the most common medical disorder to be described in second half of the twentieth century. It is not a new condition, merely a recently recognized one, as cases can be identified from manuscripts thousands of years old.[25] One of the earliest reports in the British medical literature was in 1829. Joe, the fat boy in Dickens' *Posthumous Papers of the Pickwick Club* made his appearance in 1837. The medical discovery of sleep apnoea occurred simultaneously in Germany and France in 1965.

**Methods and Materials:**

This was an observational analytical case-control study and was conducted in the Seep Laboratory of National Institute of Diseases of the Chest and Hospital, Mohakhali, Dhaka and in homes of patients and controls over a period of one year from July 2013 to June 2014.Men or women of any age attending at out-patient department (OPD) and chambers of respected teachers with complaints of symptoms of obstructive sleep apnoea were the cases, while the apparently healthy population was selected as controls. Subjects with the complaints of increased day time sleepiness not explained by any other factors with at least any two of the following symptoms were included in the study:choking/gasping during sleep,

recurrent awakening from sleep, unrefreshing sleep or impaired concentration. Subjects having the symptoms of obstructive sleep hypopnea/apnea but were unwilling to participate in the study were excluded from the study. Based on above enrolment criteria a total 54 cases and 30 controls were purposively included in the study. Blood samples were drawn for fasting blood sugar (FBS) and fasting lipid profile. Before polysonographic study, patients name, identification number, age, sex, height (in centimeter) and weight (in kilogram) and neck circumference (in centimeter) were recorded on a structured questionnaire. Data were collected using a structured questionnaire (research instrument) containing all the variables of interest. Using computer software SPSS (Statistical Package for Social Sciences), version 17, data were processed and analyzed.

**Result:**

The present study intended to find the association between metabolic syndrome and obstructive sleep apnea (OSA) included a total of 84 subjects. Of them 54 were cases (subjects who exhibit excessive daytime somnolence, had a snoring history from the spouse and repeated awakening from the sleep, choking/gasping during sleep) and 30 controls (who were free from such symptoms). The findings of the study derived from data analyses are documented below:

**Obesity status:**

Nearly three-quarters (74.1%) of the OSA patients were obese (BMI e" 30 kg/$m^2$) as opposed to only 13.3% in non-OSA subjects.

**Waist circumference:**

The mean waist circumference was significantly higher in OSA subjects than that in the non-OSA subjects ($p = 0.014$). However, there was no significant difference between the groups in terms of neck circumference ($p = 0.488$).

**Biochemical investigations:**

The OSA subjects had a significantly higher level of fasting blood sugar (144.5 ± 52.0 mg/dl) than the non-OSA subjects (84.4 ± 4.1 mg/dl) ($p < 0.001$). The level of serum total cholesterol was also much higher in the former group (349.7 ± 94.0 mg/dl) than that in the latter group (183.5 ± 60.6 mg/dl). The incidences of low HDL cholesterol and high LDL and triglyceride were staggeringly higher in the former group compared to those in the later group ($p < 0.001$).

**Metabolic syndrome and demographic characteristics:**

The mean age of the subjects with MS (48.9 ± 10.9 years) was significantly higher than that of the subjects without MS (39.2 ± 13.6 years) ($p < 0.001$). Females were more prone to have MS than their male counterparts ($p = 0.003$). Obesity was also observed to be a significant determinant of MS ($p < 0.001$).

Association between metabolic syndrome and demographic characteristics

| Variables | MS | | p-value |
|---|---|---|---|
| | Yes (n = 58) | No (n = 26) | |
| Age (yrs)# | 48.9 ± 10.9 | 39.2 ± 13.6 | 0.001 |
| Sex* | | | |
| Male | 32(55.2) | 23(88.5) | 0.003 |
| Female | 26(44.8) | 3(11.5) | |
| Obesity* | | | |
| Obese | 44(75.9) | 0(0.0) | < 0.001 |
| Non-obese | 14(24.1) | 26(100.0) | |

Figures in the parentheses indicate corresponding %;
# Data were analyzed using Unpaired t-Test and were presented as mean ± SD.
* Chi-squared Test ($c^2$) was done to analyzed the data.

**Types of OSA:**

More than two-thirds (68.5%) of the case group had severe OSA (AHI > 30), 13%had moderate OSA (AHI 15 – 30) and 18.5% normal AHI (< 5). A few subjects (13.3%) in the control group had severe OSA.

**Types of OSA and MS:**

Metabolic syndrome increases sharply from normal AHI (36%) to moderate OSA (85%) and severe OSA (95%) indicating that greater the severity of OSA, the higher is the frequency of MS.

Risk of developing OSA in patients with metabolic syndrome:

More than three-quarters (77.6%) of metabolic syndromes were observed to be associated with OSA compared to 11.5% of the subjects who had not metabolic syndrome. The OSA patients carry 26.5(95% CI = 6.9 – 102.6) times higher risk of developing metabolic syndrome than those without having OSA.

Risk of developing OSA in patients with metabolic syndrome (n=84)

| OSA | MS | | Odds Ratio (95% CI of OR) | p-value |
|---|---|---|---|---|
| | Yes (n = 58) | No (n = 26) | | |
| Yes | 45(77.6) | 3(11.5) | 26.5(6.9 – 102.6) | <0.001 |
| No | 13(22.4) | 23(88.5) | | |

Regression analysis showing predictors of underweight

| Variables of interest | Univariate analysis (p-value) | Multivariate analysis Odds Ratio (95% CI of OR) | p-value |
|---|---|---|---|
| Age (years) | 0.001 | 2.6(1.3 – 4.1) | 0.031 |
| Sex | 0.00 | 1.0(0.5 – 1.2) | 0.584 |
| Obesity | < 0.001 | 0.9(0.3 – 2.4) | 0.157 |
| Presence of OSA | < 0.001 | 7.4(2.3 – 20.9) | < 0.001 |

**Multivariate analysis:**

Table demonstrates the binary logistic regression analysis of Odds Ratios for characteristics of the subjects likely to develop metabolic syndrome. The variables revealed to be significantly associated with MS in univariate analyses were all entered into the model directly. Of the 4 variables (found significantly associated with MS in univariate analysis), age of the subjects and OSA were observed to be the independent predictors of MS in multivariate analyses. The older subjects (50 years and more) were 2.6 (95% CI = 1.3 – 4.1) times more prone to have MS than their younger counterparts (p = 0.021). Likewise the subjects having OSA was 7.4(95% CI = 2.3 – 20.9) times more likely to have MS than the subjects without OSA (p < 0.001).

**Discussion:**

Obstructive sleep apnoea (OSA) is a highly prevalent disease characterized by recurrent episodes of upper airway obstruction that result in recurrent arousals and episodic oxyhemoglobin desaturations during sleep. Significant clinical consequences of the disorder cover a wide spectrum including daytime somnolence, neurocognitive dysfunction, cardiovascular disease, metabolic dysfunction, and cor pulmonale. The major risk factors for the disorder include obesity, male gender, and age.

The present observational analytical case-control study was aimed at finding the association between metabolic syndrome and obstructive sleep apnea (OSA). In the case group over 80% of the subjects had obstructive sleep apnoea (OSA) and in the control group a few subjects (13.3%) had OSA. Over three-quarters (77.6%) of metabolic syndromes were observed to be associated with OSA compared to 11.5% of the subjects without metabolic syndrome. The OSA patients carry 26.5(95% CI = 6.9 – 102.6) times higher risk of developing metabolic syndrome compared to their non-OSA counterparts. Over two-thirds (68.5%) of the cases had severe OSA (AHI > 30), 13% had moderate OSA (AHI 15 – 30) and 18.5% had normal AHI (< 5). The severity MS of OSA patients increased sharply with types,85% with moderate type while 95% of severe OSA patients had MS. While male-female distribution was almost identical in both types. The binary logistic regression analysis of Odds Ratios for characteristics of the subjects likely to develop metabolic syndrome. The variables revealed to be significantly associated with MS in univariate analyses were all entered into the model directly. Of the 4 variables (found significantly associated with MS in univariate analysis), age of the subjects and OSA were observed to be the independent predictors of MS in multivariate analyses. The older subjects (50 years and more) were 2.6 (95% CI = 1.3 – 4.1) times more prone to have MS than their younger counterparts (p = 0.021). Likewise the subjects having OSA was 7.4(95% CI = 2.3 – 20.9) times more likely to have MS than the subjects without OSA (p < 0.001).

Over the years, many studies have investigated the relationship between OSA and metabolic syndrome[10,11,18,20,24,26] and the findings are more or less consistent with the findings of the present study. Compared with non-OSA control individuals, OSA was found to be independently associated with individual metabolic parameters in the metabolic syndrome as well as with an increased prevalence of metabolic syndrome, with an odds ratio of 9.1.[11] A study of similar design found OSA to be independently associated with metabolic syndrome but not with insulin resistance.[26] A community-based study conducted in Chinese adults in Hong Kong identified a five-fold increased risk for having metabolic syndrome in individuals with OSA and the severity of OSA correlated with number of components of the metabolic syndrome.[11]

Majority (94.4%) of the OSA patients in the present study was hypertensive. Epidemiologic and large cross-sectional studies have consistently demonstrated an association between OSA and hypertension independent of obesity.[14] The association was found even in mild OSA [13] whereas those with moderate OSA had almost three times greater risk for developing hypertension than did control individuals with no documented SDB events.[13] A number of observational studies have demonstrated that OSA is independently associated with increased markers of oxidative stress.[2-6]

In the present study the OSA patients had a significantly higher level of fasting blood sugar (144.5 mg/dl) than the non-OSA subjects (84.4 mg/dl) ($p < 0.001$). A number of studies have looked at the association between OSA and insulin resistance/glucose intolerance. [7] Epidemiologic data from the Sleep Heart Health Study suggested that patients with mild or moderate to severe OSA have increased risks for fasting glucose intolerance after adjustment for confounding factors.[17] Previous clinical studies including smaller samples of patients have yielded conflicting results, whereas recent studies have more consistently demonstrated an independent association between OSA and insulin resistance in adults, [15,16,17,19] although this is by no means a universal finding. [21]The present study demonstrated a significantly altered lipid profile in OSA patients compare to their non-OSA counterparts. While the level of serum total cholesterol, LDL cholesterol and triglycerides were much higher in the former group than that in the latter group, the HDL-cholesterol was lower in the former group than that in the latter group. Obesity (based on BMI) was also a common occurrence in OSA patients. Abnormal lipid profiles have frequently been reported in patients with OSA in the Sleep Heart Health Study of over 6000 men and women.[12] There were an inverse relationship between AHI and HDL-cholesterol levels and a positive associate in between AHI and triglycerides especially in younger individuals on adjustment for confounding factors. Case control studies also demonstrated that OSA patients had more adverse lipid profiles than did BMI-matched individuals without OSA. [1, 21,24] Although the affected lipid parameters were different, low-density lipoprotein (LDL)-cholesterol is much more atherogenic in an oxidized form and individuals with OSA have been reported to exhibit lipid peroxidation with higher levels of oxidized LDL-cholesterol compared with non-OSA individuals.[22] Furthermore, a lower capacity for HDL-cholesterol to protect LDL-cholesterol from oxidation was found in OSA, and the degree of HDL dysfunction correlated with the severity of OSA and oxidative stress. [23]

**Conclusion:**

From the findings of the study it can be concluded that OSA patients carry a much higher risk of developing metabolic syndrome compared to their non-OSA counterparts. Majority of the OSA patients is hypertensive and diabetic and possess a significantly higher level of fasting blood sugar than their non-OSA counterparts. The OSA patients also exhibit a significantly altered lipid profile with high total cholesterol, LDL cholesterol and triglycerides and low HDL-cholesterol.

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## ORIGINAL ARTICLE

# Comparative Yielding of Acid Fast Bacilli from 3 Specimen Examinations – Experience from a High Burden Laboratory in Bangladesh

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**Abstract:**

***Setting:*** *National Tuberculosis Reference Laboratory (NTRL), National Institute of Diseases of the Chest and Hospital (NIDCH), Bangladesh.*

***Background:*** *Smear microscopy is still the gold standard for diagnosis of pulmonary tuberculosis in high burden countries like Bangladesh. Though WHO recommended 2 smear (spot and morning) examinations, but still there is no study regarding this experience in Bangladesh.*

***Objective:*** *This retrospective study was carried to see effectiveness of morning and spot smear examination for diagnosis of pulmonary tuberculosis in Bangladesh.*

***Patients and methods:*** *All patients attending in and out patient departments of NIDCH with respiratory symptoms and or abnormal chest X-rays provided three sputum samples each for acid-fast bacilli smear microscopy were considered. Systematic external quality assurance was done by designated EQA center as per national tuberculosis control program guideline. A total of 16,144 TB suspects submitted three samples on two consecutive days (spot, early morning, spot) had been considered for this study between January 2013 and December 2013. Smears were prepared and stained by Auramine O staining method under LED Fluorescence Microscopy as per standard operating procedure.*

***Results:*** *Out of total 48432 smears examined a total of 4476 smears including 1806 cases were positive. The smear and case positivity rate were 9.24% and 11.1% respectively. Total 3 samples were positive in 1069 cases. Only 1st and 2nd samples detected 486 cases, 1st and 3rd 27 cases and 2nd & 3rd 19 cases only. 1st sample alone detected 160 cases, 2nd sample alone 31 cases and 3rd sample 14 cases. Therefore it is evident that spot and early morning sample will be enough for the diagnosis of pulmonary tuberculosis.*

***Conclusion:*** *This retrospective analysis from a high burden laboratory indicated that spot and early morning smears examination will be sufficient for routine sputum microscopy.This observation therefore correlative with the WHO recommendation.*

***[Chest & Heart Journal 2014; 38(1) : 7-10]***

**Introduction:**

Tuberculosis (TB) is an infectious disease caused by acid-fast bacillus, which belongs to the Mycobacterium tuberculosis complex. TB transmission begins with a human source, most often a person with cavitary, pulmonary TB. When

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an infectious patient coughs, sneezes or talks, aerosols are formed in the lungs and expelled. These aerosols contain the micro-particles that carry the bacilli, and can be inhaled by others.[1] The disease affects the lungs in approximately two thirds of cases, but almost all other organs can be the site of TB infection.[9] It is estimated that about one third of the world's population is infected with TB. However, the infection is contained by the immune system in about 90% off those infected. The TB bacilli can lie dormant for years, being protected by a thick waxy coat. If the immune system is weakened, for example by an HIV infection or treatment with immunosuppressive agents, the chances of developing active TB become much higher.[1] Tuberculosis is both preventive and curable. One third of the world's population (Two billion people) carries the TB bacteria. More than nine million of these become sick each year with active TB that can be spread to others, but the latent disease cannot be spread. It disproportionately affects people in resource-poor settings, particularly in Africa and Asia. It also poses significant challenges to developing economies as it primarily affects people during their most productive years.[1]

Early diagnosis and treatment is the cornerstone of TB control. It is a global public health problem. Bacteriological diagnosis of new smear positive pulmonary tuberculosis in low resource, high prevalent country like Bangladesh where HIV infection is low mostly dependent upon direct smear microscopy.[2] National Tuberculosis Control Program (NTP) Bangladesh adopted DOTS strategy since November 1993, introducing Ziehl-Neelsen microscopy as a primary tool for case finding. Considering advantages of light emitting diode (LED) microscopy especially higher sensitivity (60% vs 80%) and high turnover (20 slides vs 100 slides), World Health Organization (WHO) endorsed this technique for NTP of the country to increase case detection. WHO also endorsed two specimen examinations (spot - morning or spot – spot) to reduce work load, cost, early case detection and drop out of the patient in a setting with well organized external quality assessment (EQA) system.[3]

Bangladesh is a high TB burden country ranking 6$^{th}$ among 22 high TB burden countries.[2] The current estimated prevalence, incidence and TB mortality is 411, 225 & 45 per 100000 populations respectively.[2] In 2012, 1072 microscopy centers tested 1422910 suspects with 8% case positivity by conventional Z-N microscopy. NTP Bangladesh is phasing LED microscopy since April 2012 starting from National Tuberculosis Reference Laboratory (NTRL) to up to 150 microscopy centres under USAID funding with well trained microscopist. Initially starting with 3 specimen examination and phasing to two specimen examination (spot & morning) from April 2014. All the microscopy centers are under the supervision of 40 EQA centers situated at chest disease clinic (CDC). After starting LED microscopy as well as two specimen examination, there was no verified report, how much cases are missing by testing two specimens. WHO metanalysis report indicated that the incremental effect of 3$^{rd}$ specimen varies between 2-5%. NTRL is the central lab of NTP Bangladesh and also the pioneer of introducing LED microscopy of the country. Every year examining >60000 smear with well experienced microscopist under NTP EQA system. NTRL also assisted to develop a well accepted training module and EQA SOP for NTP for Z-N and LED microscopy. NTRL performed 3 specimen examinations with LED microscopy from April 2012 and is continuing. In this study we retrospectively analysed first 3 quarter of 2013 of LED fluorescence microscopy to see the comparative yielding of AFB from 3 specimens examination using LED fluorescence microscopy as well as the incremental effect of 3$^{rd}$ specimen examination in routine AFB microscopy at NTRL.

**Patients & Methods:**

- Study type: Retrospective analysis of routine AFB microscopy data.
- Study Period: January to December 2013.
- Patient types: All patients attending in and out patient department of NIDCH submitting three sputum specimens (Spot-S1, Morning M & Spot-S2 )
- Smears were prepared and stained in batches by Auramin O & examine under LED fluorescence microscope (Primo Star, Carl-Zeiss) following standard operating procedure.[6-8]

- Initial IUATLD/WHO scale was followed for grading. IQC and EQA were performed routinely.[6]

**Results:**

Smear microscopy still remains as the primary tool for the diagnosis of new smear positive pulmonary tuberculosis (PTB). LED microscopy is the superior to ZN microscopy with respect to sensitivity, cost and turn over. Third specimen examination implicates minimal incremental effect on AFB microscopy.

**Case detection and smear positivity by age and sex:**

A total of 1806 smear positive suspects submitted 4476 smears. The majority of the case and smear positivity were found between the age group 15-34 is 47.3% and 46.6% respectively. Among the total positive cases 73.6% were male and 26.4% were female and male-female ratio was approximately 3:1. But bellow 15 years female were predominate (table 1 &2).

**Cumulative case positivity and smear positivity:**

A total of 48432 smears from 16144 suspects were examined. Among these 4476(9.2%) smears and 1806(11.19%) suspects were positive (table 3).

**Case detection by smear:**

Out of 1806 smear positive cases 3 smear examination detected 1069(59.19%) cases. Spot(S1) and morning(M), spot(S1) and spot(S2), morning(M) and spot(S2) along detected 486(26.91%), 27(1.50%) and 19(1.05%) respectively.(table 4) In total spot(S1) and morning(M) detected 1792(99.22%) cases. Only 14(0.78%) cases were detected by examining 3rd sample (S2) alone (table 4 &5).

**Table-I**

*Case detection by age and sex*

| Age group | Male | Female | Total(%) |
|---|---|---|---|
| <15 | 19(41.3) | 27(58.7) | 46(2.5) |
| 15 – 24 | 231(62.4) | 139(37.6) | 370(20.5) |
| 25 – 34 | 353(73) | 131(27) | 484(26.8) |
| 35 – 44 | 260(75.8) | 83(24.2) | 343(19) |
| 45 – 54 | 196(79.7) | 50(20.3) | 246(13.6) |
| 55 – 64 | 156(83.9) | 30(16.1) | 186(10.3) |
| >64 | 115(87.8) | 16(12.2) | 131(7.3) |
| Total | 1330(73.6) | 476(26.4) | 1806(100) |

**Table-II**

*Smear positivity rate by age & sex*

| Age group | Male | Female | Total(%) |
|---|---|---|---|
| <15 | 57(42.9) | 76(57.1) | 133(3) |
| 15 - 24 | 570(62.7) | 339(37.3) | 909(20.3) |
| 25 - 34 | 862(73.1) | 317(26.9) | 1179(26.3) |
| 35 - 44 | 616(74.8) | 207(25.2) | 823(18.4) |
| 45 - 54 | 465(78.5) | 127(21.5) | 592(13.2) |
| 55 - 64 | 394(83.1) | 80(16.9) | 474(10.6) |
| >64 | 324(88.5) | 42(11.5) | 366(8.2) |
| Total | 3288(73.5) | 1188(26.5) | 4476(100) |

**Table-III**

*Cumulative Case Finding and Smear Positivity*

| Year | TB Suspects | AFB Positive Cases | | Total Smear Tested | Smear Positivity | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Scanty | | 1+2+3 | | Total | |
| | | No. | % | | No. | % | No. | % | No. | % |
| 2013 | 16144 | 1806 | 11.19 | 48432 | 750 | 16.76 | 3726 | 83.24 | 4476 | 100 |

**Table-IV**

*Case detection by smear single or in combination*

| Specimen Types | Case Positive | |
|---|---|---|
| | No. | % |
| S1 + M + S2 | 1069 | 59.19 |
| S1 + M | 486 | 26.91 |
| S1 + S2 | 27 | 1.50 |
| M + S2 | 19 | 1.05 |
| S1 | 160 | 8.86 |
| M | 31 | 1.72 |
| S2 | 14 | 0.78 |
| Total | 1806 | 100% |

Here Z value at the level of 1% significance is 2.58 and here calculated value for 3 sample examination is 26.509. So P is <0.001; Ho is rejected i.e. spot and morning specimens examination are enough for the diagnosis of pulmonary tuberculosis.

**Table-V**

*Smear/s positivity among cases*

| Sample types | Total Positive Cases | |
|---|---|---|
| | No. | % |
| S1 | 1742 | 96.46 |
| M | 1605 | 88.88 |
| S2 | 1129 | 62.51 |
| S1 + M | 1792 | 99.22 |
| Total | 1806 | 100% |

**Discussion:**

NTRL is the central laboratory of NTP laboratory network starting LED microscopy since April 2012 and starting 2 specimen examinations since April 2014. It is one of the high burden microscopy laboratories of the world. Reducing 3rd sample examination would not affect case finding rather will reduce one third of the work load.

Age, sex distribution of the detected cases revealed that out of 1806 the majority of the case and smear positivity were found between the age group 15-34(47.3% and 46.6% respectively). The overall male female ratio was 3:1 except the age group <15 where the female were predominant. This finding massage with the annual report of the 2013[2].

The overall case positivity was 11.19% and smear positivity was 9.24 %( Table 3). This finding is consistent at NTRL for last 10 years. But there is no comparative data of the country with respect to LED microscopy to compare NTRL data. But over all country case positivity rate was 8% in 2012 by ZN microscopy

Table 4 & 5 reveals that out of 1806 cases diagnosed and 14 (0.785%) cases were diagnosed by 3rd (S2)specimen examination only. This incremental effect is much lower than WHO metaanalysis.[4,5] This finding may be due to single centre study as well as high internal quality control under well experience microscopist working last 15 years. On the other hand WHO analysis was among multicenter study with different level of expertise of microscopist at different centre.

**Conclusion:**

Spot and morning specimen will be sufficient for routine smear microscopy by LED technique. The implemental effect of 3rd sample was minimal. This observation therefore comply with WHO recommendation.

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NB: The poster format of the study was presented in the 45th union conference at Barcelona ( PD-828-31) in 2014

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## ORIGINAL ARTICLE

# Prevalence of Chronic Bronchitis, Asthma and Allergic Rhinitis in a Rural Community of Bangladesh

**Niru Sultana[1], Md. Humayun Kabir[2], M Abu Sayeed[3]**

**Abstract:**

***Backgrounds:*** *Chronic respiratory diseases (CRD) are the major causes of morbidity and mortality worldwide. The CRD are increasing in both developed and developing countries with increasing rate of air pollution, which in turn due to deforestation and industrialization. The exhaust of different types of industries and transportations are worsening inhaling air and CRD. There are few published reports on CRD in Bangladesh.*

***Aims:*** *This study addressed the prevalence and risks of CRD (chronic bronchitis, asthma and rhinitis) in Bangladesh.*

***Subjects and Methods:*** *Six villages in a rural community were selected purposively. All people of age 10 years or more were considered eligible. A structured questionnaire was used to collect socio-demographic information including family history of CRD and exposure to smoking, dust, fumes and exhausts. Height (ht) and weight (wt) were taken and body mass index was calculated (BMI=wt in kg/ht in $m^2$). Finally, peak expiratory flow (PEF, L/min)) was estimated.*

***Results:*** *Overall, 1203 (m/f = 661/542) subjects volunteered. Their mean ±SD of age was 30.8±15.6 years. Compared with the female participants the males were older ($p<0.001$) and had significantly higher height ($p<0.001$) and weight ($p<0.001$) though BMI did not differ. The males had significantly higher PEF [mean (SD): 369 (101) vs. 246 (64) L/min; $p<0.001$] than the females. The prevalence rates of chronic bronchitis, asthma and allergic rhinitis were 6.7, 7.5 and 15.6%, respectively. Chronic bronchitis, asthma and rhinitis were significantly associated with increasing age, family history of respiratory illness, current smoking and dust exposure at work place (for all, $p<0.01$). Lower PEF had significant association with all three CRDs.*

***Conclusion:*** *The study revealed that the prevalence rates of chronic bronchitis, asthma and rhinitis in the rural population were remarkably higher and not negligible. Increasing age, family history of CRD, smoking and dust exposure were found to be related to CRD. Lower the PEF more is the chance of developing CRD. More study is needed encompassing the population in the urban and industrial areas for assessment of the magnitude and risk of CRD in Bangladesh.*

***Key wards:*** *Chronic respiratory disease (CRD), Asthma, Allergic rhinitis, chronic bronchitis, Rural Bangladesh*

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**Introduction:**

Chronic respiratory diseases (CRD) are chronic diseases of the airways and other structures of the lung.[1] Common chronic respiratory diseases include chronic bronchitis, chronic obstructive pulmonary disease, asthma, bronchiectasis, emphysema, lung fibrosis, allergic rhinitis.[1,5] However, the prevalence of CRD is probably underestimated, since it is not usually diagnosed until it is clinically apparent and moderately advanced.[2-4] Chronic bronchitis and asthma impose an enormous burden both in developed and developing countries.[5]

Asthma morbidity and mortality account for around 1% of all disability adjusted life years (DALYs), equivalent to 16 million DALYs lost per year worldwide[5]. A large proportion of asthma is developed in early childhood. For about 10% of the people who develop asthma in adulthood, the disease can be attributed to exposure to specific substances in the workplace.[6] The prevalence of an IgE sensitization to aeroallergens measured by allergen specific IgE in serum or skin tests is over 40% of the population in Australia, Europe, New Zealand and the United States of America.[7]

Bangladesh has also significant prevalence of chronic respiratory diseases like chronic bronchitis, asthma, allergic rhinitis and other respiratory illnesses.[8-10] A study was conducted (1998) among the children of 1-5 years of age in a coastal region of Bangladesh and found that the prevalence of asthma was 11.8%.[8] Another study "National Asthma Prevalence Study in Bangladesh 1999", jointly organized by Asthma Association and The Chest & Heart Association of Bangladesh found that the prevalence of asthma was 5.2%.[9] Another cross sectional study was conducted to measure the magnitude of the problem of COPD and estimated that the prevalence of COPD in total population of Bangladesh was 4.3%.[10]

Very few studies with regard to the risk factors associated with chronic respiratory diseases in rural areas have been conducted. The present study was conducted to study the prevalence of chronic bronchitis, asthma and allergic rhinitis and their associated factors in rural area of Bangladesh.

**Materials and Methods:**

The study was conducted in the purposively selected six villages of Sreepur Upazilla in the district of Gazipur in month of November 2012. The people aged 10 years or more residing in the study areas were considered eligible. The objectives and procedural details were explained to the villagers, heads of the families and village leaders and religious leaders. These leaders cooperated inviting the villagers to volunteer the study. Each interested participated was enlisted and interviewed by using a structured questionnaire, which included occupation (exposure to organic or inorganic dust), personal habit (smoking: biri, cigarettes), chewing tobacco leaf, cooking fuel [solid – wood, leaf, straw; liquid – kerosene and compressed natural gas (CNG)]. The participant was also interviewed about family history of chronic respiratory diseases (CRD) and taking of bronchodilators. The anthropometric measurements included height and weight. Body mass index (BMI: weight in kg / height in met sq) was calculated. Each participant was advised and trial was given to assess the peak (maximum) expiratory flow (PEF) using peak expiratory flow meter (L/min), commonly known as Wright's Peak Flow Meter. For measurement, it was first ensured that the pointer of Wright's Peak Flow meter was set to zero. The subject was preferably standing or sitting in a comfortable, upright position. Then the subject was told to hold the peak flow meter level (horizontally) and keep his or her fingers away from the pointer. The subject took a deep breath and close lips firmly around the mouthpiece. Then the subject blew as hard as he or she could. The pointer was looked at and the reading was checked and the pointer was reset back to zero for the next measure. This procedure was repeated three times and the average value of the three readings was taken as the peak expiratory flow (PEF, L/min) of the subject.

The standard criteria for chronic bronchitis used in the study was presence of cough and expectoration on most of the days for at least three months a year for two or more consecutive years.[11] Episodic wheezing, not due to other lung or heart disease, present in the year before the survey and labeled as asthma by the respondent and also by the physician. Allergic rhinitis - an inflammation or irritation of the mucous membranes of the nose caused by an allergic reaction and the common characteristics of the disease include nasal congestion, frequent sneezing, itchy and runny nose, itchy and watery eyes, and sore throat. [12] A smoker was defined by the presence of regular smoking of any type *i.e.* cigarettes, *bidi*s or *hookah*.[11] Information on number of cigarettes (or *bidi*s) and the duration of smoking was also obtained. Nonsmoker is a person who does not smoke and past smoker refers to who stopped smoking more than 1 month prior to interview. [13]

*Statistical analyses*: The biophysical characteristics were given in means with standard deviations (SD). The student's t-tests (unpaired) were used to show the differences between male and female participants. The prevalence rates were expressed in percentages. Chi square tests were applied to determine the associations of the CRD events (bronchitis, asthma and rhinitis with risk factors. The level of significance was taken at <0.05. The SPSS 16.0 was used for data analyses.

**Results:**

A total of 1203 (m / f = 661 / 542) subjects were investigated. The male to female ratio was 1.73:1. The comparisons of mean (SD) of age, height, weight, BMI and PEF between male and female subjects were shown in table1. The means (SD) of age height and weight were significantly higher in males than in females (for all, p<0.001). Overall, the prevalence rates of chronic bronchitis, asthma and allergic rhinitis among the total (male + female) participants were 6.7, 7.5 and 15.6%, respectively [table 2]. The prevalence rates of chronic bronchitis, asthma and allergic rhinitis according to sex, age-groups and other factors were shown in table2. Compared with the females the male subjects had significantly higher prevalence of chronic bronchitis (p<0.001), whereas, asthma and rhinitis showed no such difference. These three CRDs significantly increased with advancing age (p<0.001). The nutritional status of the subjects showed no association. Family history of respiratory illness, current smoking and dust exposure at work place (for all, p<0.01) were found to have significant association with these CRDs. Fuels for cooking whether solid (wood, leaf, straw), liquid (kerosene oil) or gas (methane) had no significant effect on chronic bronchitis or asthma. Whereas, compared with solid the liquid (or gas) showed significant association with rhinitis (14.9 vs. 33.3%, p= 0.004). The habit of chewing tobacco leaf was found to be related to bronchitis and asthma (for both p<0.001) but not to rhinitis. Bronchodilator was used in 90% of the asthmatic patients but only 19% of them with bronchitis. Lower PEF had significant association with all three CRDs.

**Table-I**

*Comparison of characteristics between male and female correspondents*

| Variables | Male (n=661) | | Female (n=542) | | p |
|---|---|---|---|---|---|
| | Mean | SD[1] | Mean | SD | |
| Age (y) | 32.4 | 15.0 | 28.1 | 14.2 | <0.001 |
| Height (cm) | 159.6 | 8.6 | 148.7 | 6.9 | <0.001 |
| Weight (kg) | 53.2 | 10.6 | 45.9 | 10.7 | <0.001 |
| BMI[2] | 20.7 | 3.5 | 20.6 | 4.2 | ns |
| PEF[3] | 369 | 101 | 246 | 64 | <0.001 |

1 – SD, standard deviation
2 – BMI, Body mass index (wt in kg / ht in met sq.)
3 – PEF, peak expiratory flow

**Table-II**

*Distribution of risk factors related to chronic bronchitis, asthma and allergic rhinitis*

| Variables | n | ChronicB ronchitis % | *p* | Asthma % | *p* | Allergic Rhinitis % | *p* |
|---|---|---|---|---|---|---|---|
| Total (male +female) | 1203 | 6.7 | | 7.5 | | 15.6 | |
| Male | 661 | 8.3 | | 7.1 | | 14.7 | |
| Female | 542 | 4.8 | <0.05 | 7.9 | ns | 16.8 | ns |
| Age | | | | | | | |
| <25 | 547 | 0 | | 3.8 | | 19.2 | |
| 25-44 | 408 | 9.3 | | 9.3 | | 13.7 | |
| >44 | 248 | 17.3 | <0.001 | 12.5 | <0.001 | 10.9 | <0.001 |
| BMI | | | | | | | |
| <18.5 | 366 | 6.0 | | 4.9 | | 20.3 | |
| 18.5-24.9 | 670 | 6.7 | | 7.5 | | 13.1 | |
| >24.9 | 167 | 8.4 | ns | 13.2 | <0.01 | 18.0 | <0.001 |
| Family history of Respiratory illness | | | | | | | |
| Yes | 52 | 19.6 | | 37.2 | | | |
| No | 1151 | 6.2 | 0.014 | 3.9 | <0.001 | | |
| Dust exposure at work place | | | | | | | |
| Yes | 817 | 8.3 | | 8.6 | | 20.2 | |
| No | 386 | 3.4 | <0.01 | 5.2 | <0.05 | 6.0 | <0.001 |
| Current smoking | | | | | | | |
| Yes | 362 | 11.6 | | 9.7 | | 13.6 | |
| No | 841 | 4.6 | <0.001 | 6.5 | <0.05 | 16.5 | 0.002 |
| Chewing tobacco leaf | | | | | | | |
| Yes | 340 | 12.1 | | 12.2 | | 13.6 | |
| No | 863 | 4.6 | <0.001 | 5.7 | <0.001 | 16.5 | ns |
| Cooking fuel | | | | | | | |
| Solid (wood, leaf, straw) | 1158 | 6.5 | | 7.4 | | 14.9 | |
| Liquid / gases | 45 | 13.3 | ns | 8.9 | ns | 33.3 | 0.004 |
| Bronchodilator used in past | | | | | | | |
| Yes | 52 | 19.6 | | 90.2 | | | |
| No | 1151 | 6.2 | <0.001 | 3.8 | <0.001 | | |
| Peak expiratory flow (l/min) | | | | | | | |
| <250 | 463 | 8.4 | | 11.7 | | 22.2 | |
| 250-350 | 344 | 7.0 | | 6.4 | | 13.1 | |
| >350 | 396 | 4.5 | <0.05 | 3.6 | <0.001 | 10.1 | <0.001 |

n = number; ns – not significant; *p* – following chi sq

**Discussion:**

Considering the increasing trend of CRD globally it is assumed that the prevalence of chronic respiratory diseases is increasing in Bangladesh. However, there have been a few population-based studies conducted in Bangladesh on the prevalence of chronic respiratory diseases (CRD). Thus, it is difficult to make any comparison with other reports – whether our study findings of chronic bronchitis (6.7%), asthma (7.5%) and allergic

rhinitis (15.6%) are consistent. Likewise, the risk factors investigated in this study neither could be confirmed nor could be challenged. The difficulties in comparing the other study findings were more profound due to lack of reliable diagnostic criteria. We avoided the term chronic obstructive pulmonary disease (COPD) because it included emphysema, which could not be clinically diagnosed. We could only include those CRDs that were possible for clinical diagnosis.

Had we included spirometric assessment it would have been better to conclude the study findings. However, the risk factors we included as genetic association with the diseases – chronic bronchitis, asthma and allergic rhinitis were found significantly associated with family history of CRD. This finding is consistent with other studies. [1-5,16,17] Other risk factors like inhalation exposure, occupational dust, indoor and outdoor air pollution were also found to have significant association with chronic bronchitis, asthma and allergic rhinitis. These findings are consistent with most of the studies. [13 – 14] .

Data showed that chronic bronchitis was predominantly occurring in males (75%) and the prevalence increases with increasing age. Smoking, dust, a lower than average PEF (adult: m/f <250L / <200L) were found to be the major factors associated with it. Similar findings were noted by Jindal et al. (2005).[11]COPD disease was diagnosed in 4.1% of 35295 subjects. As mentioned, significant risk association was seen with male sex, advancing age, low socio-economic status and tobacco smoking.

A population-based epidemiologic survey of chronic obstructive pulmonary disease was carried out by Hossain and Islam (2009)[14] in Dhaka city using spirometry and found the prevalence was 11.4% among subjects at 35 years or older. The prevalence of this study[14] was higher because of urban population exposed heavily to the exhaust of automobiles and factories. Moreover, it was conducted in higher age group (>35y). Increasing age, sex, smoking duration and low socioeconomic condition were revealed as independent risk factors. The lower prevalence of COPD in our study may be due to less exposure to automobile gases. People living in cities are likely to face overcrowding and air pollution than rural areas.

Goel et al. also observed higher prevalence of chronic bronchitis (9.1%) and was significantly higher in men (11.1%)[15] are not inconsistent with our findings. It may also be noted that First National Asthma Prevalence Study (FNAP) in Bangladesh (1999) [9] showed that about 7 million people (5.2% of the population) are suffering from current asthma in at least three episodes of asthma attack in one year but in our study the prevalence of asthma (7.5%) was high and was exposed to dust and other associated risk factors (age, tobacco chewing, dust, family history, smoking and PEF) significantly more than the general population.

Allergic rhinitis (15.6%) reported by Kabir et al. [16] almost conforms our finding.

It is an exploratory study, and it provides a valuable summary about the burden of respiratory diseases in the rural population of Bangladesh. However, the limitation of this study is that the data was collected from only 6 villages out of 50 from Sreepur which may not truly represent the whole population.

**Conclusion:**

This exploratory study revealed that the prevalence rates of chronic bronchitis, asthma and rhinitis in the rural population were remarkably higher and not negligible. Advancing age, genetic predisposition (family history of CRD), smoking and environmental dust exposure were found to be related to chronic bronchitis, asthma and rhinitis. Lower the PEF more is the chance of developing CRD. Further study may be undertaken encompassing the population in the urban and industrial areas for assessment of the magnitude and risk of CRD in Bangladesh.

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## ORIGINAL ARTICLE

# Association between Dietary Habits and Asthma Severity

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**Abstract:**

***Objective:*** *To investigate association between dietary habits and asthma severity in children.*

***Design:*** *Cross-sectional study.*

***Setting:*** *Outpatient department of National Institute of Disesaes of Chest & Hospital (NIDCH) & Outpatient department of Bangabandhu Sheikh Mujib Medical University (BSMMU) Hospital.*

***Methods:*** *Children aged 6 to12 years with diagnosis of asthma were eligible for the study. The clinical criteria for diagnosis of asthma were based on the recommendations of the British Thoracic Society Guidelines, 2009 The cases were children with persistent asthma (mild, moderate or severe) while the controls were those with intermittent asthma. Due to the limited number of children with intermittent asthma attending the clinics, we recruited one control for two cases matched by age groups. Data collection was carried out through interview with the parents or guardians of the patient using a standard pre-coded questionnaire. All investigators were blinded to the classification of asthma severity. Dietary habits were determined based on the consumption of specific foods or food groups in the past 12 months. The frequency of food intake was classified into two categories adapted from criteria used in the literature: frequent when intake was three or more times per week, and infrequent when never consumed or intake twice per week or less. . The weight was measured by a mechanical platform scale with capacity up to 150 Kg and height was measured by a stadiometer. Measurements were made using standardized methodology. Nutritional status was classified into two categories according to WHO Reference Growth Standards: Obese, children with a Z-score of BMI-for age>2 and non-obese, children with a Z-score of BMI for-age <2.*

***Results:*** *After adjusting for confounding factors, maternal smoking during pregnancy, preterm birth and obesity were significantly associated with persistent asthma, with adjusted ORs (95% CI) of 2.11 (1.08- 4.13), 2.61(1.07-6.35) and 2.89 (1.49-5.61), respectively. No significant association was observed between frequency of consumption of specific foods, food groups, or dietary pattern and the severity of asthma.*

***Conclusions:*** *This study did not find a significant association between dietary habits and asthma severity in children. Maternal smoking during pregnancy, preterm birth and obesity were independent factors associated with persistent asthma.*

***Keywords:*** Asthma, Diet, Epidemiology, Risk factors.

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**Introduction:**

Asthma is the most common chronic disease in childhood. In spite of advances in the knowledge of pathophysiology and treatment of the disease, the prevalence and severity of asthma in children has increased over the last few decades[1,2] It has been proposed that changes in dietary habits may be one of the factors responsible for this increase.[3-5] Numerous epidemiological studies have been conducted to investigate the association between dietary habits and the risk of asthma in children.[3-5] These studies have identified the intake of fruits, vegetables and fish as protective factors against childhood asthma while fast food consumption as a risk factor for the disease.[6-11] Studies on dietary habits and severity of asthma are few and show inconsistent results.[11-13] The present study aimed to investigate the association between dietary habits and the severity of childhood asthma.

**Methods:**

This cross-sectional study was conducted at the Pulmonology Outpatient clinics of two teaching hospitals of Dhaka between March 2012 and February 2013.

Children aged 6 to12 years with diagnosis of asthma were eligible for the study. The clinical criteria for diagnosis of asthma were based on the recommendations of the British Thoracic Society Guidelines, 2009.[14] The diagnosis of asthma was made if all the following criteria were met: (*i*) recurrent episodes (>3) of one or more of the following symptoms – wheeze, cough, breathing difficulties and chest tightness, particularly at night or in the early hours of the morning; (*ii*) respiratory symptoms improve spontaneously or after treatment (bronchodilators with or without corticosteroids); (*iii*) presence of triggers or aggravating factors such as exposure to allergens or irritants, physical exercise, weather changes or emotional stress; and (*iv*) personal history of atopy (allergic rhinitis or eczema) and/or family history of atopy (asthma, allergic rhinitis or eczema) in first-degree relatives. The severity of asthma was assessed based on the clinical criteria recommended by the National Heart, Lung, and Blood Institute (NHLBI), and was classified as intermittent asthma or persistent asthma (mild, moderate or severe).[15] The cases were children with persistent asthma (mild, moderate or severe) while the controls were those with intermittent asthma. Due to the limited number of children with intermittent asthma attending the clinics, we recruited one control for two cases matched by age groups.

Data collection was carried out through interview with the parents or guardians of the patient using a standard pre-coded questionnaire. All investigators were blinded to the classification of asthma severity. The dependent variable was asthma severity, classified as persistent or intermittent, while independent variables were: dietary habits, demographic and socioeconomic data (gender, age and skin color, family income and educational level of parents), smoking during pregnancy, presence of allergens in the home (curtains, carpets, fluffy toys or pets), gestational age (preterm <37 weeks), birth weight (low birth weight <2500g), family history of asthma (first-degree relatives) and personal history of allergic rhinitis. Dietary habits were determined based on the consumption of specific foods or food groups in the past 12 months. The frequency of food intake was classified into two categories adapted from criteria used in the literature: frequent when intake was three or more times per week, and infrequent when never consumed or intake twice per week or less.[12,13] Specific food or food groups included: milk or yoghurt, meats (bovine, and poultry), vegetables (leafy and non-leafy), fish, eggs, fruit, legumes (beans, peas, lentils, and chick peas), roots and tubers (potato, sweet potato, manioc), grains (rice, pasta, bread), butter, soft drinks and processed foods including fast food. Nutritional status was measured using the Body Mass Index (BMI) calculated by dividing body mass (Kg) by height2 (m2). The weight was measured by a mechanical platform scale with capacity up to 150 Kg and height was measured by a stadiometer . Measurements were made using standardized methodology. Nutritional status was classified into two categories according to WHO Reference Growth Standards: Obese, children with a Z-score of BMI-for age>2 and non-obese, children with a Z-score of BMIfor-age <2.[17]

*Statistical analysis*: Double data entry was performed using the software EPI-data 3.2. Analyses were carried out using the statistics package Stata 11 (Stata Corp., College Station, USA). A descriptive analysis was conducted for each group with calculation of absolute and relative frequencies for independent variables. Crude and adjusted odds ratios (OR) and 95% confidence intervals (95% CI) were calculated using conditional logistic regression given the matching of cases and controls by age groups. Multivariate analysis was applied to control for potential confounding factors, with inclusion of variables according to the preestablished hierarchical levels as follows: level

1: gender, skin color, maternal schooling, income, paternal schooling, and smoking during pregnancy; level 2: allergens in the home, gestational age, birth weight, family history of allergic rhinitis, exposure to passive smoking; level 3: dietary variables; and level 4: obesity. Only the variables with $P$ <0.20 remained in the model. $P$<0.05 was considered statistically significant.

**Results:**

A total of 202 patients were screened for eligibility, of which 5 were excluded due to diagnosis of chronic neurological disease ($n$=1), pulmonary tuberculosis ($n$=3), congenital heart diseases ($n$=1). Thus, 197 patients were included in the study, of whom 134 were classified as the cases (persistent asthma) and 63 as the controls (intermittent asthma). *Table* I shows the characteristics of 197 patients. Bivariate analysis showed that male gender, maternal smoking during pregnancy and obesity were significantly associated with persistent asthma.

The frequency of intake of specific food or food groups was not significantly associated with asthma severity (*Table* II). After adjusting for confounding factors, maternal smoking during pregnancy, preterm birth and obesity were significantly associated with persistent asthma, with OR (95% CI) of 2.11 (1.08-4.13), 2.61(1.07-6.35) and 2.89 (1.49-5.61), respectively (*Table* III). No significant association was observed between dietary habits and asthma severity.

**Table-I**

*Characteristics of the Study Population (N=197)*

| Variables | Persistent asthma n=134 (%) | Intermittent asthma n=63(%) | OR (95% CI) | P value |
|---|---|---|---|---|
| Male gender | 77 (59.3) | 30 (48.4) | 1.58(1.03-2.42) | 0.03 |
| Maternal smoking | 12(4.0) | 3 (1.1) | 1.90(1.07-3.37 | 0.02 |
| Presence allergens in home | 128(95.1) | 60(96) | 0.81(0.28-2.31) | 0.68 |
| Gestational age <37 wks | 24(18.5) | 8(12.3) | 1.49 (0.79-2.79) | 0.21 |
| Birth weight <2.5kg | 18(14.5) | 9(15.4) | 0.93(0.51-1.72) | 0.83 |
| Maternal education <class V | 63(47.7) | 35(55.2) | 0.76(0.49-1.17) | 0.21 |
| Paternal education <class V | 81(64.9) | 39(69.0) | 0.82(0.51-1.32) | 0.41 |
| Family history of asthma | 80(59.5) | 35(56.0) | 1.13(0.74-1.75) | 0.55 |
| Family history of allergic rhinitis | 100(72.8) | 41(67.2) | 1.37(0.86-2.18 | 0.19 |
| Passive smoking | 62(46.1) | 23(36.5) | 1.47(0.95-2.27) | 0.08 |
| Obesity | 40(32.9) | 10(18.2) | 2.20(1.30-3.74) | <0.001 |

**Table-II**

*Bivariate Analysis of Association between Dietary Habits and Asthma Severity (N=197)*

| Food consumption* >3 times/wk | Persistent asthma n=134 (%) | Intermittent asthma n=63(%) | OR (95% CI) | P value |
|---|---|---|---|---|
| Milk | 125(94.3) | 58(98.4) | 0.95(0.37-2.40) | 0.91 |
| Vegetables | 91 (69.3) | 40(65.3) | 1.18(0.75-1.86) | 0.47 |
| Meat | 114(85.7) | 107(90.5) | 10.63(0.32-1.25) | 0.19 |
| Fish | 26(19.7) | 11(17.3) | 1.14(0.66-2.00) | 0.65 |
| Eggs | 57(42.9) | 27(42.9) | 0.98(0.64-1.51) | 0.93 |
| Processed food | 94(70.7) | 42(68.5) | 1.10(0.70-1.75) | 0.67 |
| Soft drink | 52(38.9) | 25(42.4) | 0.88(0.57-1.36) | 0.57 |
| Pulses | 118(88.4) | 57(90.5) | 0.80(0.40-1.61) | 0,53 |
| Potatoes | 68(50.9 | 27(45.6) | 1.22(0.80-1.87) | 0.36 |
| Cereals | 130(98.1) | 62(97.6) | 1.25(0.29-5.29) | 0.77 |
| Butter | 107(80.4) | 55(86.5) | 0.64(0.35-1.16) | 0.15 |
| Fruits | 106(80.5) | 51(82.3) | 0.85(0.48-1.50) | 0.58 |

**Table-III**

*Adjusted Analysis Of Association Between Patient Characteristics Variables And Asthma Severity*

| Variables | OR (95% CI) | P value |
|---|---|---|
| Male gender | 1.58(0.97-2.55 | 0.06 |
| Paternal education level <class VIII | 0.68(0.40-1.17) | 0.17 |
| Smoking in pregnancy | 2.11 (1.08—4.13) | 0.03 |
| Gestational age <37 wks | 2.61(1.07-6.35) | 0.04 |
| Butter e"3 times/wk | 0.60(0.30-1.21) | 0.15 |
| Obesity | 2.89(1.49-5.61) | <0.01 |

**Discussion:**

This cross-sectional study did not show significant association between frequency of consumption of specific foods, food groups, or dietary pattern and the severity of asthma in children aged 6 to 12 years.

Several limitations should be taken into account when interpreting the results of this study. The statistical power of the study may be insufficient for investigating the association between diet and asthma severity due to a relatively small sample size. The broad age range of the participants (6 to 12 year) may act as a confounding factor given that food consumption may vary substantially among children of different age groups. In order to control for the confounding effect of age, the cases and controls were matched by age-groups. We did not recruit non-asthmatic children as controls because this study aimed to investigate association between dietary habits and asthma severity rather than risk of asthma. This study was hospital-based, and therefore the results may not necessarily be extrapolated to general population of asthmatic children. Mild persistent, moderate persistent and severe persistent asthma were combined into a single "persistent asthma" category given that inter-observer agreement increases with reduced number of categories.[26] oreover, this simplified classification for asthma severity (intermittent *vs.* persistent) had a practical implication because only children with persistent asthma need long-term controller medications.

To date, there is limited and inconsistent evidence about association between diet and asthma severity in children. Recently, Ellwood, *et al.*[4,13] reported the global results of the ISAAC study (Phase III) on the association between food consumption in the last 12 months and atopic diseases such as asthma, rhinoconjuctivitis and eczema. Fruit intake >3 times per week was found to be a protective factor against severe asthma in both adolescents and children, with OR (95% CI) of 0.89 (0.82-0.97 and 0.86 (0.76-0.97), respectively. Fast food consumption >3 times per week was a risk factor for severe asthma in two populations, with OR (95% CI) of 1.39 (1.30-1.49) and 1.27 (1.13-1.42), respectively. However, some inconsistent findings were observed between two populations, and there was also heterogeneity of findings across different study centers and countries.[13] In the present study, fruit consumption e"3 times a week appeared to be a protective factor against persistent asthma, with an OR of 0.85 (95% CI 0.48-1.50), although the result was not statistically significant. The conflicting findings regarding the association between Mediterranean diet and asthma severity in children were also found in two studies with similar research methodology and population. The inconsistency of the results on diet and asthma severity across different studies, even though among different populations within the same study, may be attributable to sampling error and/or other associated factors such as memory bias, accuracy of the diagnosis and classification of asthma, variation in food types among different geographical regions as well as biological variation among study populations. These factors should be taken into account in the future researches on diet and asthma severity in children.

The present study showed that maternal smoking during pregnancy was associated with more severe asthma in children. Smoking represents a modifiable risk factor for respiratory infections and

asthma in childhood. *In utero* exposure to maternal smoking has a direct effect on the development of respiratory system of the fetus, with compromised development and function of the lungs in infants.[19,20]

This study identified preterm birth as an independent factor associated with persistent asthma. The relationship between gestational age and asthma severity has been investigated in previous studies with conflicting results.[21,22] The present study showed that obesity was significantly associated with more severe childhood asthma. This finding is consistent with that reported in previous studies.[23-25]

In conclusion, this study shows that obesity rather than dietary habits is significantly associated with asthma severity in children. Other independent factors associated with persistent asthma included maternal smoking during pregnancy and preterm birth.

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## ORIGINAL ARTICLE

# Study on Prognostic Evaluation of COPD Patients Using BODE Index

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**Abstract:**

***Background:*** *Chronic obstructive pulmonary disease (COPD) is a common disease characterized by airflow limitation which is irreversible or partially reversible. The risk of death in patients with COPD is traditionally graded with the use of the forced expiratory volume in one second (FEV1), but it correlates better with the BODE index.*

***Objective****: The aims of this study were to assess that component of BODE index have more impact on mortality of COPD patients than post bronchodilator FEV1 & to see the relative contributions of respiratory& non-respiratory causes of death in COPD patients.*

***Methods & Materials:*** *It was a prospective study during the period of July 2010 to June 2014 was conducted in outpatient at Sher-E-Bangla Medical College Hospital, Barisal, Bangladesh.. Total 252 patients were included who were COPD.*

***Results:*** *Among the study population of 252,188(74.60%) were male & 64(25.39%) were female and 188(74.60%) patients were smokers and 64(25.39%) nonsmokers. Out of 188 smokers 176(69.84%) were male & 12(04.76%) were female. Out of 64 nonsmokers, 8(12.50%) were male and 56(87.50%) were female who were exposed to biomass fuel while cooking.We assessed the severity of COPD in each patient at the beginning according to ATS guideline. Most of the study population-188(74.60%) were in stage III COPD. During the study period of 48 months (4yrs), 52(20.65%) patients died out of 252. All patients who died, had BODE score point 10 i.e. in 4$^{th}$ Quartile.Out of 52 deaths 28(53.84% of death) died from respiratory causes (corpulmonale and respiratory failure) &24(46.15% of the death) died from non-respiratory causes (like malignancy, IHD and complications of HTN).*

***Conclusion:*** *A recognizable number of patients suffer from COPD due to exposure of Biomass fuel particularly females of Bangladesh. The higher the BODE score points, higher the risk of death from respiratory causes. Co-morbidities are accountable for the death of COPD patients in addition to variables in BODE index.*

***Clinical implication:*** *COPD is a heterogeneous disease without a simple prognostic trajectory. Co-morbidities & exacerbations contribute to the overall severity in individual patients. The BODE index measures functional limitation, nutritional status and symptoms, in addition to airflow obstruction, and is therefore well placed to assess clinical risk and the integrated response to treatment.*

*Key words: COPD, Prognosis, BODE index.*

***[Chest & Heart Journal 2014; 38(1) : 23-27]***

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**Introduction:**

Chronic obstructive pulmonary disease (COPD) is a common disease characterized by airflow limitation which is irreversible or partially reversible.[1-3] The risk of death in patients with COPD is traditionally graded with the use of the forced expiratory volume in one second ($FEV_1$), [5-9,11] but it correlates better with the BODE index.[17] The BODE is a multi-dimensional index which combines four important variables into a single score- (B) Body mass index; (O) airflow obstruction measured by the forced expiratory volume in one second ($FEV_1$); (D) dyspnea measured by the modified Medical Research Council (MRC) scale; and (E) exercise capacitymeasured by the 6 minute walk distance (6MWD). Each variable is graded and a score out of 10 is obtained, with higher scores indicating greater risk. The risk of death from respiratory causes increases by more than 60% for each one point increase in BODE index (Celli et al 2003). We use BODE index in our study.

**Methods & Materials:**

It was a prospective study during the period of July 2010 to June 2014 and was conducted in outpatient at Sher-E Bangla medical college hospital, Barisal, Bangladesh. Total 252 patients were included who were diagnosed as COPD. Diagnosis were established by respiratory physicians based on medical history, current symptoms & spirometry following GOLD guidelines ( GOLD Expert Panel 2003) ie COPD patients were defined by $FEV_{1>}$80% andFEV1/ FVC ratio <70% measured 20 minutes after the administration of salbutamol.[1,2,12] Other dyspnic condition like Asthma, CCF, IHD, TB, Bronchiectasis were excluded from this study. A regular follow-up was given as per study protocol.

**Result:**

The characteristics of the study population are shown in Table 1.

Among total 252, mean age was 60.63 ± 10.05, 188(74.60%) were male and 64(25.39%) were female shown in fig-1.

Among 252 patients, 188(74.60%) are smokers and out of them176(69.84%) were male & 12(04.76%) were female & 64(25.39%) non smokers all of them were exposed to biomass fuel & 8(12.50%) were male and 56(87.50%) were female.

**Table-I**

*Characteristics of study population, according to whether they survived*

| Characteristics | Survived (N = 200) | Died (N = 52) | P value |
|---|---|---|---|
| | Mead±SD | | |
| Age (yr) | 59±10 | 59±12 | 0.03 |
| Smoking history (pack-yr) | 59±10 | 64±14 | 0.28 |
| Body mass index | 20±3 | 20±4 | 0.07 |
| FEV1 | | | |
| Liters | 0.75±0.28 | 0.56±0.13 | 0.002 |
| Percent of predicted | 0.32±0.10 | 0.25±0.03 | 0.04 |
| FVC | 1.40±0.09 | 0.53±0.12 | 0.53 |
| FEV1/FVC | 0.55±0.09 | 0.53±0.12 | 0.53 |
| MMRC dysp. | 3.55+0.64 | 3.80+0.42 | 0.02 |
| 6MWD | 215.26+76.20 | 126.90+90.21 | 0.002 |

**Table-II**

*Severity of COPD in study population*

| | No. of patients (%) |
|---|---|
| | (N=252) |
| Stage I ($FEV_1$>50% of predicted) | 16(6.34) |
| Stage 11 ($FEV_1$ - 36-50% of predicted) | 48(19.04) |
| Stage III (FE 1<$^3$5 of predicted) | 188(716) |

**Table-III**

*Variables and point values used for the computation of the body mass index, degree of airflow obstruction and dyspnea and exercise capacity (BODE) index.*

| Variables | Points on BODE Index | | | |
|---|---|---|---|---|
| 0 | 1 | 2 | 3 | |
| FEV1 (% of predicted) | >65 | 50-64 | 36-49 | X35 |
| Distance walked in 6 min (m) | >350 | 250-349 | 150-249 | 5149 |
| MMRC dyspnea scale | 0-1 | 2 | 3 | 4 |
| Body-mass index | >21 | <21 | | |

**Table-IV**

*Score points of study population*

| BODE index score | No. of patients(%) (N=252) |
|---|---|
| 0 | 0(0) |
| 1 | 0(0) |
| 2 | 4(1.58) |
| 3 | 4(1.58) |
| 4 | 4(1.58) |
| 5 | 8(3.15) |
| 6 | 32(12.64) |
| 7 | 40(15.80) |
| 8 | 36(14.28) |
| 9 | 56(22.22) |
| 10 | 68(26.98) |

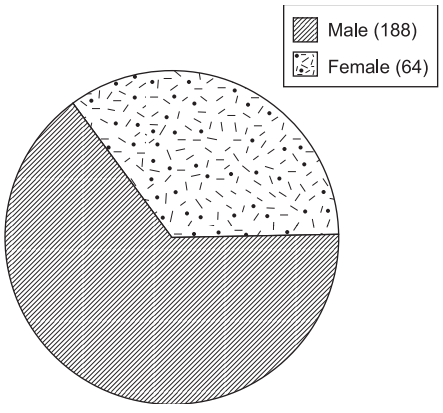



**Fig.-1:** *Sex distribution in study population*

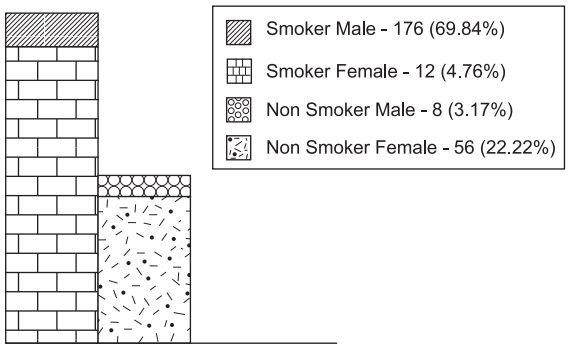
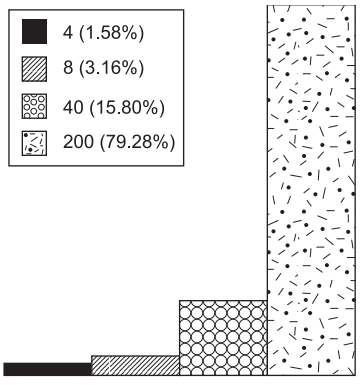



**Fig.-2:** *Smoking habit.*




| |
|---|
| Quartile 1 (score 0 to 2) |
| Quartile 2 (score 3 to 4) |
| Quartile 3 (score 5 to 6) |
| Quartile 4 (score 7 to 10) |

**Fig.-3:** *No. of study population according to quartile (Q)*

During the study period of 48 months (4yrs), 52(20.65%) patients died out of 252which was lower than original study. All patients who died, had BODE score point 10 i.e. in 4th Quartile.

We found that out of 52 death patients 28(53.84% of death) died from respiratory causes (corpulmonale and respiratory failure) & 24(46.15% ) died from non-respiratory causes(like malignancy, IHD and complications of HTN). All patients died from respiratory causes had BODE score points 10.

**Discussion:**

Chronic obstructive pulmonary disease (COPD) is a common disease characterised by airflow limitation which is irreversible or poorly reversible.[1,2,12,13] COPD is predicted to be the third common cause of death in the world by 2020 and causes more death than bronchial asthma.[6-11]

The forced expiratory volume in 1second (FEV1) has traditionally been used to assess COPD severity.[1,12,13] According to American Thoracic society (ATS), FEV1 more than 50% of predicted value represents mild disease (stage-I), 36-50% moderate(stage-II) and d" 35% severe disease(stage-III). 25% of patients with severe disease will die within 2 years and 55% by 4 years.[12,13,15] But a number of other factors such as age, body mass index, hypoxaemia, hypercapnia are independent predictors to survival. [18-21]

The BODE scale, consisting of body mass index BMI(B), the degree of airflow obstruction(O), functional dyspnoea(D) and exercise capacity(E) as assessed by the six minute walk test, has been shown to help predict survival over 1-3 years(Celli 2004).[17,23]

In our study over the period of between July 2010 and June 2014, we assessed a total of 252 COPD patients (as diagnosed by FEV1 and FEV1/FVC ratio 20 minutes after administration of nebulised salbutamol) by using BODE index and other variables like smoking, co-morbidities etc.

The characterstics of the study population are shown in table-1. Among the study population of 252, 188 patients were male and 64 female; 188 patients were smokers and 64 nonsmokers. Among nonsmokers, 8 were male and 56 were female who were exposed to biomass fuel while cooking. We assessed the severity of COPD in each patient at the beginning according to American thoracic society guideline[12] which is shown in table-3.

Most of the study population (188(74.6%)) were in stage III COPD. Variables and point values used for the computation of the body mass index, degree of airflow obstruction, dyspnoea and exercise capacity(BODE) index are shown in table-2.

The points for each variable were added, so that the BODE index ranged from 0 to 10 points, with higher score indicating a greater risk of death.[17]

BODE index score are shown in table-4. And depending on the scores patients are grouped into four Quartiles which have been shown in fig-2.

During the study period of 48 months (4yrs), 52(20.65%) patients died out of 252. 28(53.84% of death) died from respiratory causes(corpulmonale and respiratory failure) & 24(46.15% of the death) died from non-respiratory causes(like malignancy, IHD and complications of HTN). All patients who died, had BODE score point 10 i.e. in 4th Quartile.

**Conclusion:**

1).A recognizable number of patients suffer from COPD due to exposure of biomass fuel particularly females of Bangladesh.

2).The higher the BODE score points, higher the risk of death from respiratory causes.

3). Co-morbidities are accountable for the death of COPD patients in addition to variables in BODE index.

**Clinical Implication:**

1.COPD is a heterogeneous disease without a simple prognostic trajectory.

2.Co-morbidities & exacerbations contribute to the overall severity in individual patients.

3.The BODE index measures functional limitation, nutritional status and symptoms, in addition to airflow obstruction, and is therefore well placed to assess clinical risk and the integrated response to treatment.

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## *ORIGINAL ARTICLE*

# Stenting of Ductus Arteriosus : Single Centre Experience in a Tertiary Cardiac Centre of Bangladesh

**Nurun Nahar Fatema**

**Abstract:**

***Objective:*** *Stenting of Ductus Arteriosus (PDA) is indicated mainly in duct dependent cyanotic lesions where Blalock Taussig (BT) Shunt is indicated to improve pulmonary circulation. Application of this procedure of stenting of PDA is not so universal as ductus anatomy is complicated in cyanotic duct dependent cases.*

***Methods :*** *From October 2005 to October 2013, 30 patient had successful PDA stenting in Cath Lab of Combined Militery Hospital, Dhaka. All 30 cases had reduced blood flow to pulmonary circulation, 12 were getting injectable Prostaglandin also. Work up to confirm the diagnosis were done with thorough clinical examination and investigations. All the cases had initial Aortogram in lateral and four chamber projection to decide about possibility of stenting and then to select an appropriate stent. Thirteen cases were excluded from study for vertical or tortuous course of PDA and presence of major aortopulmonary collateral arteries (MAPCA) instead of PDA. All procedures were performed retrograde through femoral artery.*

***Results:*** *Forty percent of patient in this study were neonates and rest were infants.*

*Median age was three months. Majority of patients (56.66%) were male. Pulmonary atresia with ventricular septal defects (VSD) was seen in 56.66% cases, Pulmonary atresia with intact ventricular septum (IVS) was seen in 20% cases, Tricuspid and Pulmonary atresia was seen in 13.33% cases and critical pulmonary valve stenosis was seen in 10 % cases. Median weight of the patient was 3.2 kg. Oxygen saturation of the patients ranged from 30% to 60% on admission. All twelve newborn were treated with injection prostaglandin before stenting. PH of the patient ranged from 7-7.25. Stent diameter varied from 3.5 to 5 mm and stent length varied from 10 mm – 15 mm. Stent migration was experienced in one patient which was later repositioned, spasm of the duct was noticed in 4 cases. Mean procedure time was 35+/_ 5 minutes.*

***Conclusion:*** *Ductal stenting is a practicable, safe, effective method compared to palliative surgery in neonates and infants. Paediatric cardiologists, cardiac surgeons , relatives can buy some time to allow growth of the pulmonary artery with optimum flow before planning next corrective or palliative surgery.*

***[Chest & Heart Journal 2014; 38(1) : 28-34]***

**Introduction:**

In a heart with left sided aortic arch, the arterial duct connects the junction of pulmonary bifurcation with the left pulmonary artery to the descending aorta just distal to the origin of the left subclavian artery, and has a short, straight course.

This anatomy is seen in patient with pulmonary stenosis or atresia with intact ventricular septum

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late in fetal life. The arterial duct in neonate with severe right heart obstructive lesions early in fetal life has a very different anatomy : the ducts are long tortuous and mostly have [1,2] a vertical origin from the arch.

Duct dependent pulmonary circulation may be associated with a single ventricle or also with normally or borderline developed right or left ventricle.

Blalock Taussig were the first to create a surgical communication as an alternative to arterial duct.

Ductal stenting is indicated in duct dependent cyanotic lesions chiefly in the neonatal period.

Ductus arteriosus stenting, although not widely accepted, is also considered as less invasive alternative for first stage palliation.

**Materials and Methods:**

Most of the cases in this study were young infants or neonates who were getting PGE1 Infusion.

In most of the cases infusion were stopped two hours before procedure.

All the cases were clinically examined, SPO2 recorded , Prostaglanding infusion given in neonates only with SPO2 less than 60%.

All the cases had thorough Echocardiography to look for anatomy specially ductal anatomy.

After that they were planned for ductal stenting .

We prefer the procedure to be done under sedation and allow the baby to breath spontaneously if there was no additional respiratory problems. In those who were in ventilation already or had very low SPO2 (< 40%) were done under general anaesthesia. A central line was secured for giving durgs before starting the procedure.

**Procedure:**

Before going for ductal stenting as first stage palliation, detail diagnosis of the congenital heart disease has to be established by two- dimensional and Doppler echocardiography .

Interventional strategy should be planned depending on single or dual pulmonary supply.

Patients were drapped and sedated first, some of them were intubated . Prophylactic antibiotic was used routinely.

After cannulation of right or left femoral artery and vein with 4/5 Fr sheath, patients were heparinized with 50-100 u/kg heparin.

The duct with a common orientation, arising from descending aorta or from subclavian artery can be done through femoral artery approach.

Materials used:

1. 4/5 Fr short sheath.
2. 4 Fr cobra catheter.
3. Berman angiographic catheter.
4. Pigtail catheter.
5. Judkins Right 4 Fr catheter.
6. Long sheath 4 Fr cook, 5 Fr PDA delivery sheath for ADO II.
7. 0.014 guide wire with floppy tip.
8. 0.35 Terumo guide wire.
9. Stents- coronary stents from Medtronic, Boston Scientifice, Jhonson and Jhonson.

Diameter 4-5 mm, Length 9-15 mm.

The stent length was chosen slightly larger than the PDA size to prevent constriction of the uncovered end and after deployment.

After cannulation aortogram is performed in lateral and four chamber projection.

We use Judkins right 4 french for crossing PDA with coronary floppy tipped 0.014 wire. Later, Guiding cath 5 french or cook long sheath 4 french or ADO II delivery 5 french is placed near the aortic end of ductus.

Prostaglandins were manipulated in some cases for allowing constriction of ductus or opening of narrow PDA. In few cases prostaglandin was stopped after crossing PDA.

We did not require any pre dilatation with balloon in any of our cases.

Pre mounted coronary stents were used. Stent diameter selected on the basis of weight of the patient, diameter varied from 3.5 to 5 mm and length varied from 10-15 mm.

For patient weighting 3-4 kg , Stents selected were 3.5-4 mm, patient weighting 4-5 kg had stent of 4.5 mm diameter and those more than 5 kg had stent of > 4.5 mm to 5 mm diameter .

**Results:**

Fig 1 : Showed age distribution of cases. Most of the cases (40%) were neonates and only 6.6% were in more than one year age group.

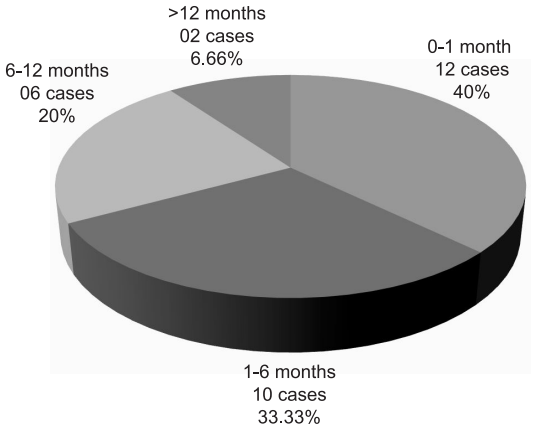


**Fig 1:** *Age distribution of patients. N=30*

Fig. 2: Showed sex of the patient. Most of the patients in this study (56.66%) were male.

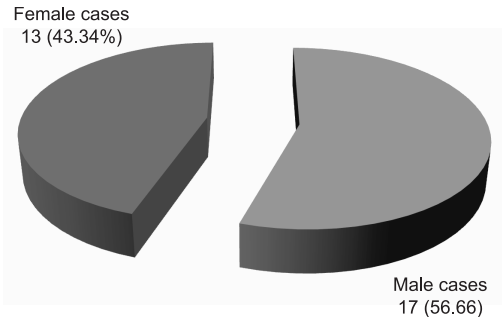


**Fig -2:** *Sex distribution of the patients n -30*

Fig. 3: showed weight distribution of patient. Majority of patient,s weight ranged between 2.8-3.5 kg 53.33%.

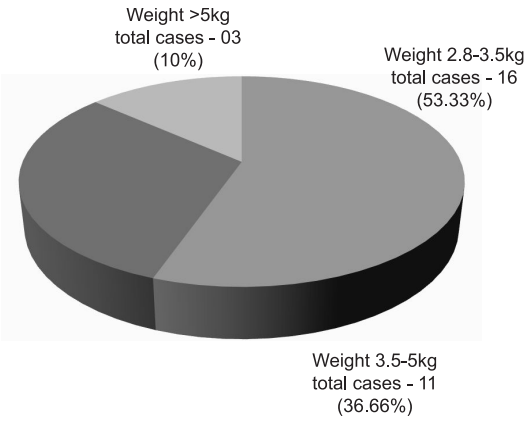


**Fig.-3:** *Weight distribution of the patients n -30*

Fig. 4: Showed pH range of the patient on admission. In majority (56.66%) of cases, pH ranged from 7.10-7.20.

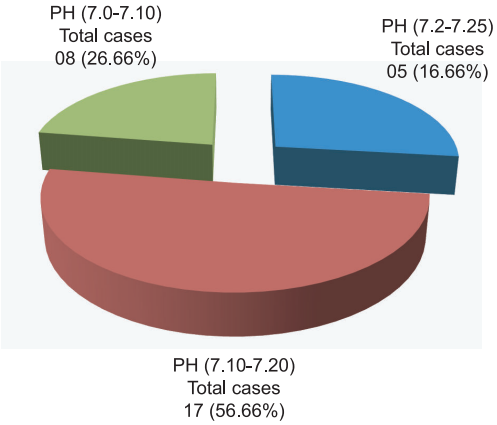


**Fig-4:** *PH range of the patients in study cases n -30*

Fig. 5: Showed stent diameter of the cases. In fourteen (46.66%) cases stent diameter was 4.5 mm, in eleven (36.66%) cases stent diameter was 4.0 mm, in three (10%) cases stent diameter was 5.0 mm and in two (6.66%) cases stent diameter was 3.5 mm.

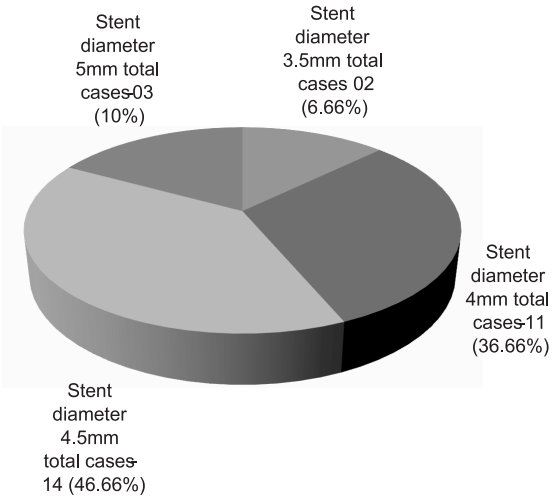


**Fig.-5:** *Diameter of the stent in mm used in study cases. n-30*

Fig. 6: Showed fluoroscopy time in the patients. Nineteen (63.34%) cases had fluoroscopy time ranged for 30-40 minutes. Eleven cases (36.66%) had fluoroscopy time of 40-50 minutes.

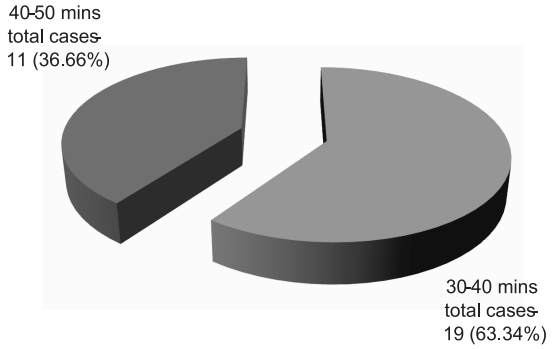


**Fig.-6:** *Total Fluoroscopy time, n -30*

Table 1: Showed type of the lesion. Majority of cases had pulmonary atresia with VSD (56.66%), 20% had pulmonary atresia with intact septum, 13.33% cases had both tricuspid and pulmonary atresia and 10% cases had critical pulmonary valve stenosis with hypo plastic right ventricle.

**Table-I**

*Types of lesion in study cases. n -30*

| Name of Lesion | No. of cases | Percentage |
|---|---|---|
| Pulmonary Atresia with VSD, PDA | 17 | 56.66% |
| Pulmonary Atresia with intact septum, PDA | 06 | 20% |
| Tricuspid and Pulmonary Atresia,PDA | 104 | 13.33% |
| Critical Pulmonary Stenosis, PDA | 03 | 10% |

Postponed after initi1al aortogram: Thirteen cases were postponed for vertical course, tortuousity and for wrong diagnosis of PDA for MAPCA.

**Table-II**

*Oxygen saturation of patients during admission. n-30*

| $O_2$ Saturation | No. of cases | Percentage |
|---|---|---|
| 30-40% | 11 | 36.66% |
| 40-50% | 15 | 50% |
| 50-60% | 04 | 13.33% |

Table 11: Showed oxygen saturation of the patient during admission. Most of the patient,s (50%) saturation ranged from 40%-50% on admission.

Table 111: Showed narrowest ductal diameters of patient in aortogram. Fourteen (46.66%) cases had

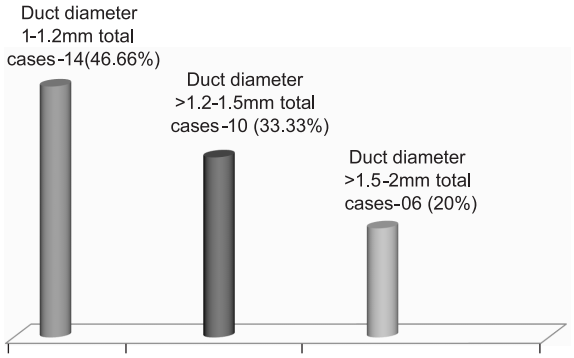


**Fig.-7:** *Narrowest ductal diameter measurement. n-30*

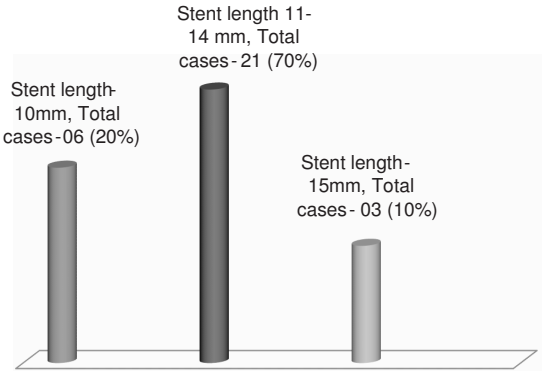


**Fig.-8:** *Length of the stent in mm used in study cases. n-30*

PDA diameter ranged from 1-1.2 mm. Ten (33.33%) case had diameter ranged from 1.2-1.5 mm. In six (20%) cases diameter ranged from 1.5-2.0 mm.

Table IV: Showed length of PDA stent in millimeter. Twenty one (70%) cases had PDA stent length ranged from 11-14 mm. In this study all atypical PDA with tortuous course were excluded.

Table V: Showed complication in our cases. Only one case had stent migration to Descending Aorta which was later repositioned and reinflated with a slightly larger balloon. Spasm of ductus were observed in 4 (13.33%) cases.

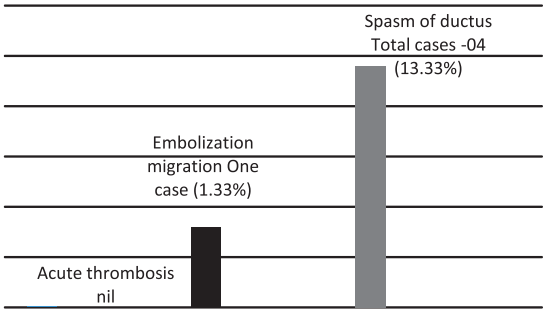


**Fig.-9:** *Complications observed in study cases: n-30*

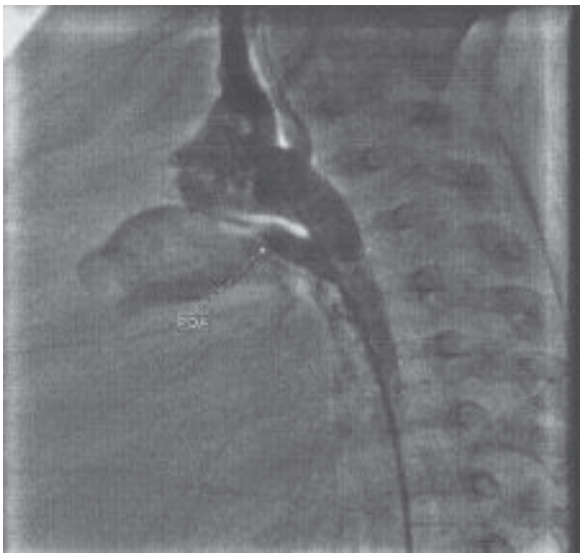

**Fig-10:** *Aortogram showing tiny PDA*

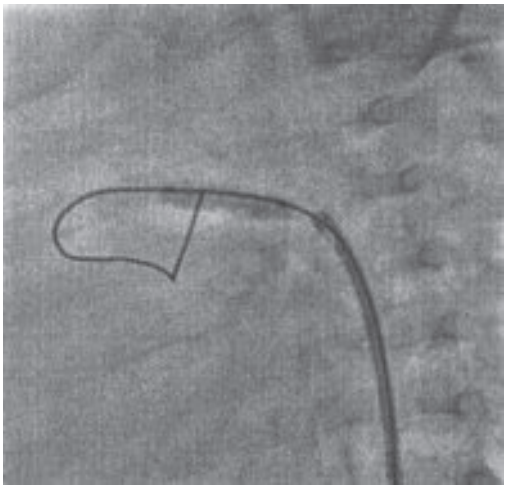

**Fig-11:** *Showing stent inside PDA after balloon inflation*

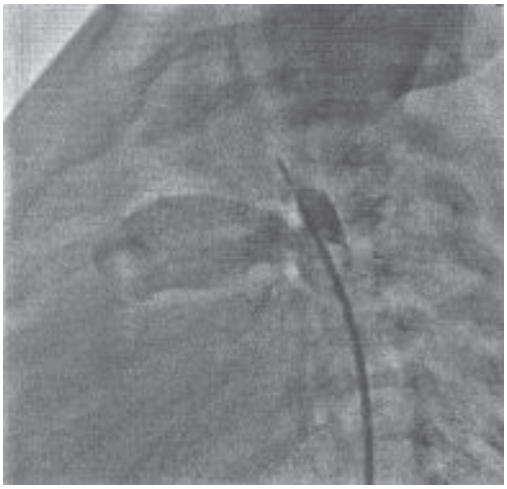

**Fig-12:** *Aortogram showing large PDA with inflated stent inside*

**Discussion:**

Ductal stenting has proved itself as an attractive alternative to conventional shunt surgery in duct dependent congenital heart diseases.

Ductal stenting may be done by the retrograde femoral arterial route or the antegrade transvenous route depending on the ductus morphology and type of lesion.

In our series, all the cases were performed through retrograde transarterial route. Unlike PDA of isolated lesion, these PDA´s are most of the time showed difficult anatomy and it become challenging to implant a stent successfully.

A simple ductus connects the junction of pulmonary bifurcation with the left pulmonary to the descending aorta just distal to the origin of the left subclavian artery and they always has short, straight course [1-5].

This kind of ducts are seen in critical pulmonary valve stenosis, Pulmonary Artesia with Intact Ventricular Septum cases.

The ductus in neonate with right heart obstruction from early fetal life has longer, tortuous and vertical ducts. The duct may be left or right sided or even bilateral.

In our study only 3 cases had right sided ductus.

Underlying lesions commonly found are:

1. Pulmonary Atresia with intact septum.
2. Pulmonary Atresia with ventricular septal defect.
3. Pulmonary Atresia with double outlet right ventricle.
4. Critical Pulmonary Stenosis.
5. Tricuspid Artesia with Pulmonary Stenosis or Atresia.
6. Transposition of Great Arteries, Ventricular Septal Defect and Pulmonary Atresia.
7. Double outlet right ventricle, Ventricular septal defect and Pulmonary Atresia.[6-9]

Majority of cases in our study had Pulmonary Atresia with VSD (56.66%) and 10% cases had critical PS. There anatomies were carefully analyzed by 2D and Doppler echocardiography before selecting the patient for stenting. Patient were established by giving Prostaglandin

10-20nanogm/kg/min if oxygen saturation was found less than 60%. Patient,s blood gas was analyzed and acidosis corrected. Ideally patient should arrive in the catheterization laboratory with the smallest acceptable size of the patent duct. If the duct is not constricted, then some constriction can be allowed by stopping the prostaglanding for several hours. Prostaglandin can be restarted at any time if duct constriction has occurred. In case of acute constriction during procedure local application of small dose of prostaglandin is also useful.

Ductus arteriosus stenting in cases with abnormal anatomy has likelihood to accelerate branch pulmonary artery stenosis and is like to be very challenging.

Initial aortogram is very critical to look for the ductus origin from aorta, its shape , course, tortuousity, site of insertion into pulmonary artery, prelace of branch pulmonary artery stenosis, specially at the site of insertion.

In oue series all vertical and tortuous PDA´s were excluded from the study.

In our study only 40% cases were neonates but in other study [4,5] all of this cases were neonates.

This indicates delay in referring the cases, delay in identification of symptoms by the parents and also inability of the parents to avail treatment from tertiary centre.

Type of the lesions in this study indicates that all of the cases had duct dependent pulmonary circulation. We do not find any cases with ductus dependent systemic circulation in this study.

Possibly patient with hypo plastic left heart syndrome, interrupted aortic arch ect, could not reach tertiary center for availing treatment and die even before diagnosis.

Common complications of ductal stenting are:

1. Acute stent thrombosis.

2. Spasm of the ductus

3. Migration of the stent.

We have embolization of stent into descending Aorta in one case which we repositioned again and re inflated with one size more balloon (3.5mm to 4mm). After completion of procedure, patients were kept in paediatric cardiac ICU for 48 hours. Those who were ventilated, extubated by 12 hours. Central lines were removed after stopping heparin infusion. Symptoms of heart failure from over shunting were not experienced in our series. We used combination of clopedogral and aspirin in all of our cases. Stent stenosis was not experienced in any of our cases so far. We refer the cases to surgeons for total correction or Bidirectional Glenn operation depending on single ventricle or biventricular anatomy in 6-18 months time. All the patients were closely followed up at 3 monthly interval before referral to surgeon for next surgery.

**Conclusion:**

Findings of this study showed that PDA stenting could be a good alternative to surgery for initial palliation. It should be considered in those who will need multiple surgeries in future. It is safe, effective and feasible but it is not a permanent solution. Patient should go for next procedure after 6 months as its efficiency reduces usually after 6 months. so long term palliation without stent restenosis might be a concern.

Stenting Of Ductus Arteriosus : Single Centre Experience In a Tertiary Cardiac Centre of Bangladesh

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Chest & Heart Journal
Vol. 38, No. 1, January 2014

## REVIEW ARTICLE

# Obesity Paradox in Heart Failure

**Dilruba Ahmed[1], Md. Roushon Ali[2]**

**Abstract:**

*Obesity is a worldwide health problem that results in a significant increased risk of morbidity and Mortality specially cerebrovascular and coronary heart disease. But it may not always be true. A higher body mass index (BMI) carries a survival benefit in patients with heart failure. Data from more than 28000 patients with heart failure support the view that the optimal BMI ranges between 30 and 35 kg/m$^2$ as opposed to the common belief that weight loss and being slim is generally good. In chronic diseases such as heart failure, chronic kidney disease, or chronic obstructive pulmonary disease, this assumption no longer holds true, because of a phenomenon called the "obesity paradox", and weight loss is no longer advisable. The origin of this clinical observation is not entirely clear, but some factors may have an influence: obese patients with heart failure are on average younger, have better nutritional status and appetite, present at an earlier stage of the disease, are less catabolic, have lower levels of natriuretic peptides and have higher muscle mass. An optimal BMI has not been defined, and it is not clear if fat mass is as beneficial as muscle mass when looking at absolute BMI values.*

***Keywords:*** *Heart failure, Muscle, Obesity, Obesity paradox.*

***[Chest & Heart Journal 2014; 38(1) : 35-38]***

## Introduction:

Obesity paradox was first describe in 1999. However, many physicians have still never heard of the obesity paradox. The reason may be that the time frame is simply not long enough to allow for paradigm shifts, and it seems that nothing less is necessary with regard to our perception of obesity in patients who are chronically ill. Therefore, it is not surprising that current guidelines for heart failure issued by different bodies do not mention the existence of an obesity paradox.[1,2] Clinicians involved in the care of patients may thus not be aware that the advice to lose weight commonly advocated for overweight or obese individuals with cardiovascular disease may not make sense for all of them. So, clinician should approach the obesity paradox in an orderly manner. The term describes a common phenomenon seen in many chronic illnesses, including coronary artery disease, arterial hypertension, heart failure, diabetes mellitus, chronic obstructive pulmonary disease, chronic kidney disease and several others. Among patients with these illnesses, overweight and mild obesity are counterintuitively but commonly associated with better survival than underweight or what is usually called normal weight, ie, a body mass index (BMI) up to 25 kg/m $^2$.In terms of survival, heart failure patients seem to fare best with a BMI between 30 and 35 kg/m$^2$.

## Survival Benefit with Higher BMI

Obesity paradox first described from data of more than 1300 patients with chronic kidney disease

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undergoing hemodialysis.[3] The authors' conclusion was that "nutrition aimed to achieve the high end of normal body mass index may help to reduce the high mortality and morbidity in hemodialysis patients". This notion is of particular interest, because the authors of that study did not even analyze nutritional intake and thus extrapolated far beyond their data using proxies such as serum albumin or prealbumin. However, the study paved the way for an avalanche of publications dealing with the subject. Indeed, only one year later, Davos et al.[4] described the existence of an obesity paradox in an abstract at the annual meeting of the American Heart Association, only to be published in 2003 as a full report. In the mean time, other groups had taken up the issue, leading to a larger publication using data from 1203 patients with advanced heart failure to show that cardiopulmonary exercise testing, pulmonary capillary wedge pressure and serum sodium were strong predictors of survival in this group of patients. Importantly, the authors concluded "higher body mass index was not a risk factor for increased mortality, but was associated with a trend toward improved survival".[5] Several independent groups have confirmed these results using large databases mainly from prospective trials now involving more than 28,000 patients. A meta-analysis of those patients[6] with a mean follow-up of 2.7 years has shown that individuals with a BMI between 25.0 and 29.9 kg/m$^2$ had lower all-cause mortality than individuals with a normal BMI. This finding could be confirmed for cardiovascular mortality, and it remained true after adjusting for several risk factors. However, it has to be taken into account that the available data do not permit an upper threshold to be given for the beneficial effects of obesity, simply because the number of individuals with a BMI greater than 40 kg/m2 remains small, both in real life and in clinical studies.

**Metabolic Differences in Obesity**

A matter of ongoing debate is whether the obesity paradox really does exist and if it does, what is the metabolic basis for its existence? Indeed, a number of factors need to be considered when looking at the data. On average, obese patients with heart failure are younger, have better nutritional status and appetite, present at an earlier stage of the disease, are less catabolic, have lower levels of natriuretic peptides, have higher muscle mass and potentially higher left ventricular ejection fraction.[7,8] It has recently been shown that patients with heart failure and appendicular skeletal muscle mass 2 SD below the mean of a healthy young population are significantly older and have significantly less body weight than patients with normal skeletal muscle mass.[9] Indeed, obesity leads to unavoidable exercise simply by carrying one's own weight, and such exercise as recommended by the guidelines may help to maintain skeletal muscle.[8]

Obesity may thus primarily be a marker of a different status.[8] On the other hand, the guidelines state that the presence of obesity is a risk factor for the development of heart failure, because obesity is in many cases associated with the clustering of cardiovascular risk factors, ie, the metabolic syndrome, and because obesity leads to an increase in circulating blood volume and consequently to higher cardiac output, cardiac work and systemic blood pressure.[1] Other changes in obese individuals include an enhanced turnover of free fatty acids, increased sympathetic tone, the activation of inflammatory mediators, and a hypercoagulable state.[10] In addition, obesity itself may be involved in the chief complaint of heart failure patients, shortness of breath, thus creating an overlap of symptoms derived from the obese status and from the failing heart.[5]

**The Metabolic Basis for the Obesity Paradox in Heart Failure**

Body composition is an important issues in heart failure. Obesity is a risk factor for developing heart failure, but obesity also carries a survival benefit when heart failure has become manifest. A low BMI or the development of cachexia are certainly detrimental in patients with heart failure;[6,10,11] mild or even moderate obesity, on the other hand, may well be acceptable. There is no need to ask heart failure patients to gain weight, but there is good reason to make them stop losing it. Discussion needs to be extended to matters of body composition, as it is not clear if there is such a thing as an optimal body composition,[12] ie, content of lean mass versus fat mass, in patients with heart failure. The BMI, originally described by Adolphe Quetelet in 1832 (Quetelet index) and renamed

"body mass index" in 1972, was originally used as an estimation tool of body fat content.[13] Critics of the BMI have argued that it fails to distinguish between fat mass and lean mass, and that for that reason muscular people are frequently misclassified as overweight or obese. Body composition changes over the lifespan need to be considered, ie. after the age of 30 years, lean mass decreases at the expense of increases in fat mass.[14] Despite the loss in muscle mass, this usually leads to a net increase in body weight. Usually 5 to 10 years before death, BMI usually starts to decline as a consequence of inactivity, anorexia and poor nutritional intake.[7] The term "sarcopenia" describes loss of muscle mass and strength with advancing age. On average, 5–13% of elderly people between 60 and 70 years are affected by sarcopenia, and the numbers rise to 11–50% for those aged 80 years and above.[15,16] It was therefore surprising to see that the criterion of sarcopenia or muscle wasting, ie, muscle mass 2 SD below the mean of a healthy young cohort, was present in almost 20% of stable heart failure outpatients with a mean age of 67 years.[9] The mean BMI of patients affected by muscle wasting was significantly lower than that of those not presenting with muscle wasting. It is clear now that a higher BMI is beneficial in heart failure, but it is tempting to speculate that higher muscle mass is even better than fat mass, even though fat mass as an energy depot may also help in decreasing mortality rates.

**Conclusions:**

Obesity paradox in heart failure is gradually becoming clear but many questions still remain unanswered. However, the obesity paradox is there as the data are more than convincing and it is there to stay. The optimal BMI in heart failure seems to be somewhere between 30 and 35 kg/m$^2$ and certainly not in the region commonly considered as normal BMI. It cannot be stressed too often that patients with chronic disease and healthy individuals are different. The influence of age, nutritional status, appetite, disease severity, catabolic status and muscle mass all need to be considered as they all contribute to the obesity paradox. In particular, muscle mass and strength require more research, as we do not yet know whether only a higher BMI is beneficial or whether higher muscle mass is also required. An upper threshold for the BMI needs to be defined. In the meantime, clinicians' advice to their patients should be to stop losing weight once heart failure is present.

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Vol. 38, No. 1, January 2014

## REVIEW ARTICLE

# Cough in Children: Diagnostic Approach and Management Update

**Md. Saifuddin Khaled[1], Mohammed Shahidullah[2], Firoza Akter[3]**

**Abstract:**

*Cough is a forced expulsive maneuver, usually against a closed glottis and is associated with a characteristic sound. It is a critical reflex action designed to promote clearing the airways of secretion, protects the airway from foreign body aspiration and can be the manifesting symptom of a disease. Cough is one of the most common complaints for which parents bring their children to a health care practitioner. Although we have some understanding about the cough reflex through animal studies, the exact mechanism has not yet been completely elucidated. Pediatric cough can be classified in different ways with overlapping categories and a number of etiologies are there. Careful history taking and physical examination together with appropriate investigations, the correct diagnosis can be made in most cases within a reasonable time frame. The current review provides essential informations to approach and manage this common childhood problem.*

***Key words:*** *bronchial asthma, Childhood, Diagnosis, Management.*

***[Chest & Heart Journal 2014; 38(1) : 39-44]***

**Introduction:**

Cough is the most common pediatric problem managed by family physicians and it is more common in pre-school children than in older children. Two out of 3 children aged between 0 and 4 years visit their physicians at least once a year with acute respiratory infections and up to three quarters of them will have cough.[1] While most children with cough don't have serious lung conditions, coughing can be troublesome and difficult to treat sometimes. Normal children wearing cough meters have been recorded as coughing 10-11 times per day but rarely at night. During respiratory infections or in children with recurrent cough, coughing may occur 60-100 times by day and less often at night.[2] Coughing in children may be distressing and has a major impact on child's sleep, school performances and ability to play. It may similarly disturb other family member's sleep and be disruptive for school teachers. Considerable parental anxiety is generated in families with a child with coughing.[3]

Community based surveys show that, parental reported cough as an isolated symptom has a higher prevalence.[4] Reported cough without colds has a prevalence of 28% in boys and 30% in girls.[5] The prevalence of chronic cough in Chinese children is about 6.4%. In USA cough accounts for 3% of medical consultation.

Physiology of cough:

Cough is a rapid and ballistic movement. A typical cough starts with a deep inspiration to at least 50% of the vital capacity. This is followed by the compression phase, where the glottis closes for about 0.2 sec and there is contraction of respiratory

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musculature. The glottis then opens suddenly. This unleashes the high intrapleural pressure that develops during the glottis closure, creating a high expiratory flow rate and narrowing the central airways, which can be as high as 12L/sec and reaches a peak flow of 30 to 50 msec following the start of the expiratory phase. Finally, the relaxation phase completes the act when the respiratory musculature relaxes with a reversal of the intrathoracic pressure.[6] Normal function of the mucociliary apparatus is critical in maintaining an effective cough, as it brings secretions from the periphery to the proximal airways where they can be cleared by coughing.

**Classification of pediatric cough:**

Pediatric cough can be classified in several ways, based on time frame, etiology, characteristics (moist/wet vs dry cough), and specific or non specific cough from a good history and thorough physical examination-

Acute cough: A recent onset of cough lasting <3 weeks.

Chronic cough: A cough lasting >8 weeks.

Prolonged acute cough: A cough lasting for 3-8 week period. It's a "grey" area between acute and chronic cough sometimes called 'subacute cough'.[7]

Recurrent cough: A recurrent cough without a cold is taken as repeated (e"2/year) cough episodes apart from those associated with head colds, that each last more than 7-14 days.[8]

Post viral cough: Post viral cough is a cough originally starting with an upper respiratory tract infection but lasting >3 weeks.

Specific cough: A specific cough is one in which there is a clearly identifiable cause.

Non specific isolated cough: The term "non-specific isolated cough" has been used to describe children who typically have a persistent dry cough, no other respiratory symptoms (isolated cough), are otherwise well with no signs of chronic lung disease and have a normal chest radiograph.

**Etiologies:**

Common causes of acute cough in children-

Simple head cold

Bronchiolitis

Pneumonia

Croup

Bacterial tracheitis

Seasonal allergic rhinitis

Inhaled foreign body

First presentation of some chronic diseases

**Diagnostic approach:**

Diagnostic evaluation of a child having cough should be initiated from a good history taking, proper physical examination and relevant laboratory investigations. A detailed history should be taken carefully to address the following specific points:

Time of onset of coughing (neonatal, infancy or child hood)

Nature (dry or productive) and quality (brassy, croupy, bizarre/honking, paroxysmal or staccato)of cough.

Timing (persistent, intermittent bouts, nocturnal or on awaking )

Triggering factors (cold air, exercise, feeding, seasonal variations, starts with a head cold)

Alleviating factors (bronchodilators, antihistamins, antibiotics)

**Table-I**

*Common causes of chronic cough in children (in descending order of likelihood)[9]*

| Infancy | Early childhood | Late childhood |
|---|---|---|
| Gastroesophageal reflux | Post-viral airway hyper-responsiveness | Asthma |
| Infection | Asthma | Post-nasal drip |
| Congenital malformation | Passive smoking | Smoking |
| Congenital heart diasease | Gastroesophageal reflux | Pulmonary tuberculosis |
| Passive smoking | Foreign body | Bronchiectasis |
| Environmental pollution | Bronchiectasis | Psychogenic cough |

Presence of associated symptoms (eg, wheezing, shortness of breath)

Any family history of respiratory symptoms, disorders or atopy

Risk of contact with tuberculosis or HIV

Smoking behavior of parents and

Vaccination status of child.

Thorough physical examination should include sings of ear, nose and throat infection, nasal polyps, chest deformities, abnormal chest auscultations, evidence of atopic diseases including eczema, failure to thrive as evident from low birth weight for height and sometimes a low height for age. Deviation of the growth curve is a particularly important finding that indicates severe disease. The child may be asked to perform his/her usual cough, this may help to differentiate between dry and productive cough and may reveal tracheobronchomalacia.

Laboratory investigations should be specific to underlying etiology defined from history and clinical examination. Tests should include chest radiography and CT scanning, sputum microscopy, cultures and serology, blood tests and screening for immunodeficiencies, lung function tests for appropriate age, allergy testing (skin prick or RAST specific testing) bronchoscopy, assessment of reflux, sweat test and ciliary function studies to exclude primary cilliary dyskinesia.

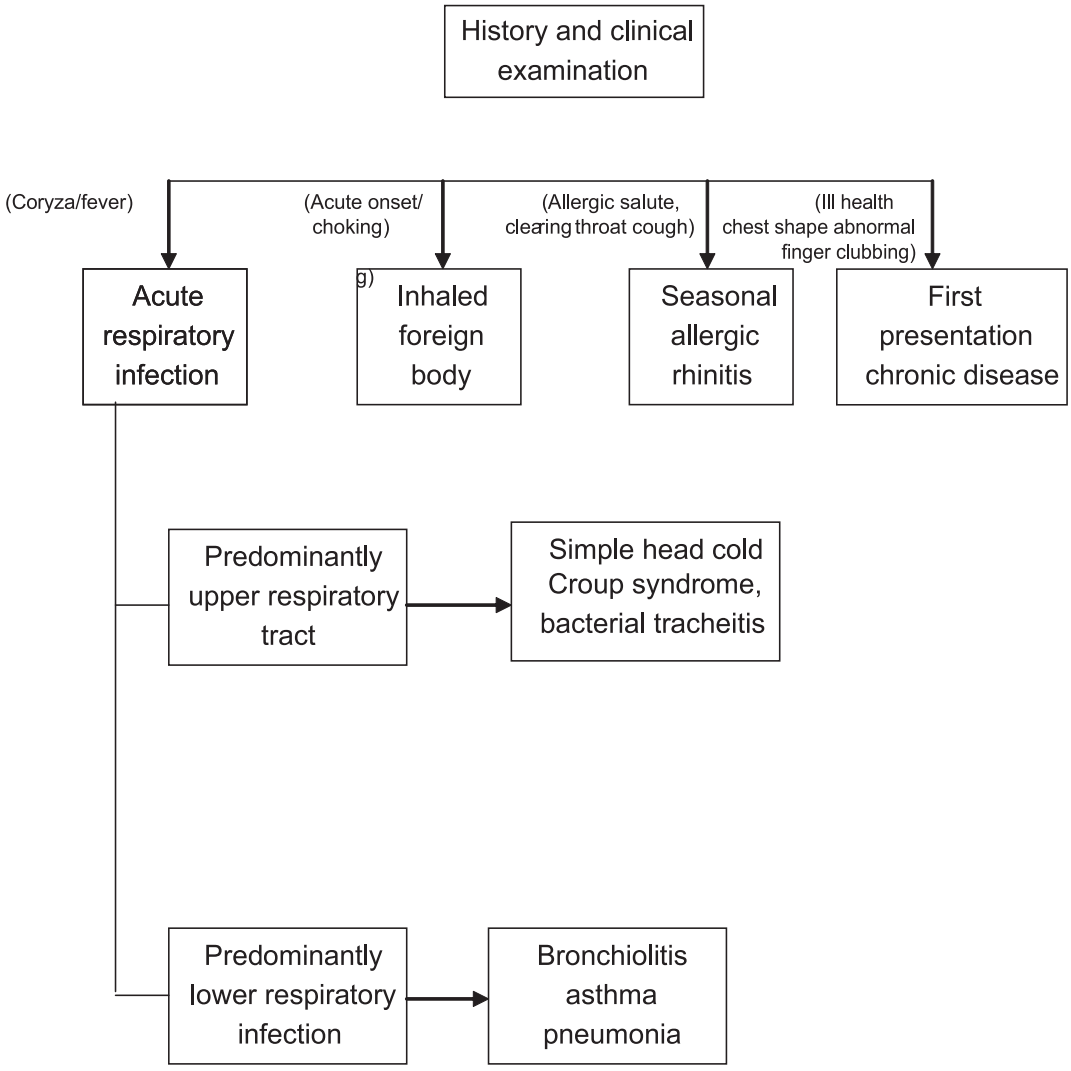


**Fig.-1:** *A simplified overview of the assessment of the common causes of acute cough (<3 weeks).*

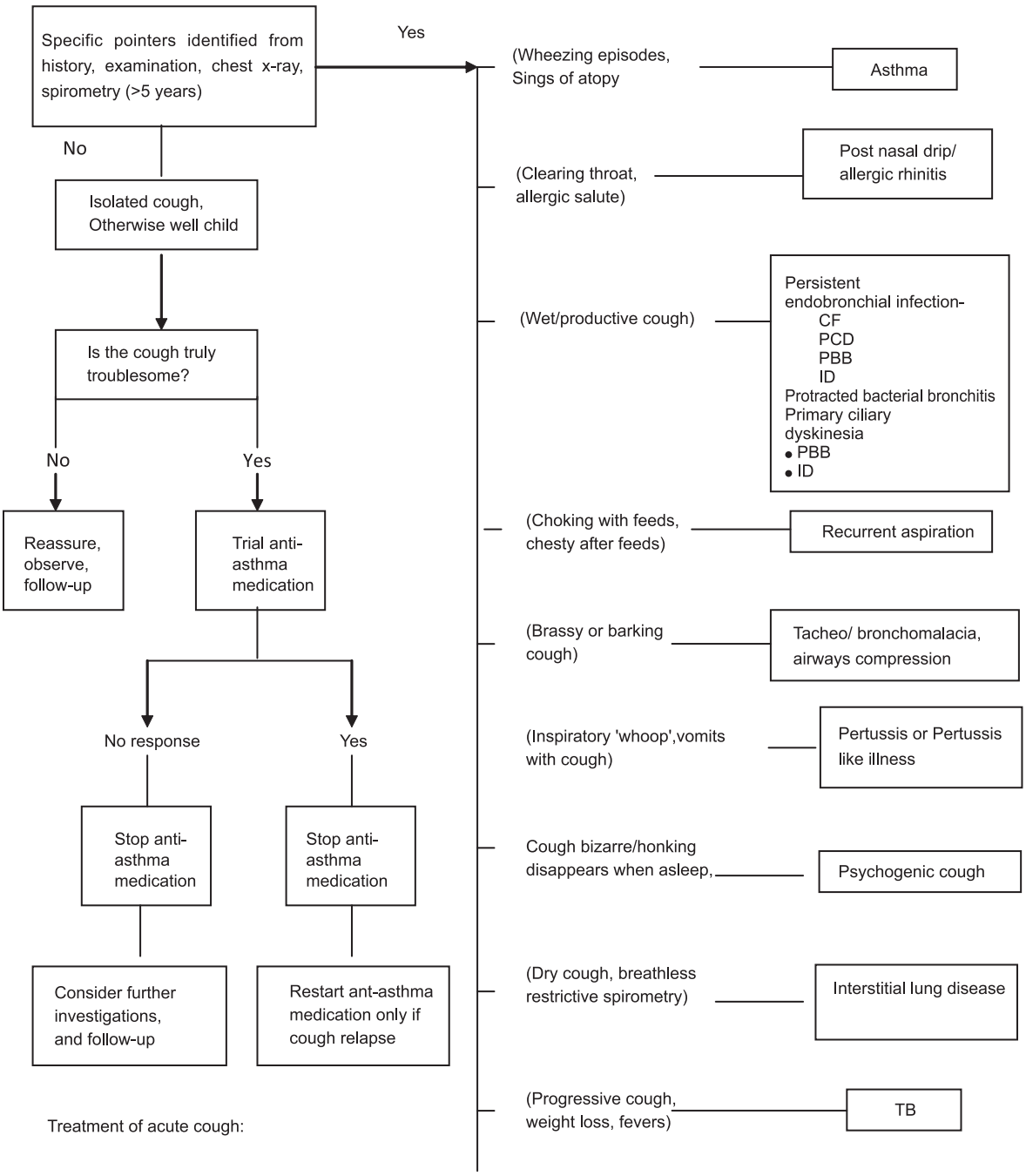


**Fig.-2:** *A simplified overview of the Assessment of the Common causes of chronic cough (>8 weeks)*

**Treatment of acute cough:**

The majority of children with acute cough have a viral respiratory tract infection and may not need any specific treatment but good supportive care and reassurance. During pediatric acute cough management, the following recommendations should take in consideration-

1. Over-the-counter medications are as effective as placebo for acute cough with simple head colds in children with the potentials for causing side effects. [10,11]
2. Bronchodilators are not effective for acute cough in non-asthmatic children. [12]

3. Antibiotics are generally not effective or recommended for treating acute coughs caused by simple head colds.[13]
4. Evidence-based guidelines should be followed for the treatment of following common specific causes of acute cough in children e,g community acquired pneumonia, croup, bronchiolitis, asthma. Macrolid antibiotics should be given early (in the first 1-2 weeks)to children with pertussis, when clinically suspected. [14]
5. Antihistamines and intranasal steroids are beneficial for children with anj allergic cough in the pollen season.[15]
6. There may be benefits from educating the community and medical professional about the natural history of cough and head cold. For majority of children the cough will have resolved by 14 days. However, for an important minority the cough will take 3-4 weeks to resolve. Giving this information to parents may reduce the need for subsequent doctor consultations, providing the cough is subsiding and provided that warnings are given about when to reconsult eg, ongoing fever, tachypnoea. [16]
7. Parents should be warned that informations obtained from internet on cough may be unreliable. [18]

**Treatment of chronic cough:**

The treatment of chronic cough in children should always be preceded by a systemic effort to first making an accurate underlying diagnosis and then applying specific treatment for that condition. The use of adult based cough algorithms, most often are unsuitable for application in children.[18] important recommendations should be followed as below-

1. An attempt should be made to remove the children with chronic cough from exposure to aeroirritants such as environmental tobacco smoking.[19]
2. Children with 'protracted bacterial bronchitis' should first have other underlying conditions excluded and sputum cultured before this diagnosis is made. A trial treatment of intensive physiotherapy and a prolonged course (eg, 4-6 weeks) of appropriate antibiotics may be tried.[20]
3. In children with non specific isolated cough (which includes post viral cough and recurrent viral bronchitis) no treatments seem particularly effective and parental reassurance is important. A period of observation with no diagnostic tests or treatments should be considered.[21]
4. In an otherwise well children with non specific isolated coughing with no specific disease pointers, empirical trials of anti-asthma, anti-allergic rhinitis or anti gastro-oesophageal reflux therapy are unlikely to be beneficial and are generally not recommended.[22]
5. If a trial of anti-asthma therapy is used to diagnose problem coughing as being caused by asthma, the treatment should be effectively delivered in adequate doses for a definite period of time (eg, 8-12 weeks). [23]
6. Empirical gastro-oesophageal reflux therapy is not indicated for non specific cough in children.[24]
7. In arriving at a diagnosis of psychogenic or habit cough, the physician should first be sure that organic causes are unlikely that the suggestive features of non-organic coughing present. Psychotherapy such as behavior modification regimes may be helpful in treating psychogenic coughing. [25]

**Conclusion:**

Cough is a common childhood problem and in the majority is reflective of expected childhood respiratory infections. However cough may also be representative of a significant serious disorder and all children with chronic cough should have a thorough clinical review to identify specific respiratory pointers. Cough in children should be treated based on etiology and there is no evidence for using medications for symptomatic relief of cough or for an empirical approach. Irrespective of diagnosis, environmental influences and parental expectations should be reviewed and managed accordingly.

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## CASE REPORT

# A Case with Pulmonary Fibrosis and Low Back Pain

**Bashir Ahmed[1], Shah Md. Saifur Rahman[2], S.M. Lutfor Rahman[2], Md. Rustom Ali[3], Pulak Kumar Dey[4], Shah Sayeed Md. Iqbal Hooda[4], Md. Tahsin Ahmed Chowdhury[4], Md. K. M Saifulla[4], Touhiduzzaman[5]**

*[Chest & Heart Journal 2014; 38(1) : 45-48]*

**Introduction:**

Pulmonary fibrosis is relatively a common issue for pulmonologists. Pulmonary fibrosis having various causes but in the perspective of Bangladesh pulmonary tuberculosis seems to be the common cause. But before treating PTB for proper management and to avoid unnecessary treatment with anti-TB drugs we should think of other causes. The other causes includes systemic connective tissue disease, pneumoconiosis etc. The history , clinical examinations , investigations, the diagnostic criteria(For systemic connective tissue diseases) and distribution of fibrosis help us reaching a diagnosis . Use of diseases modifying drugs and biologic agents may often prevent complications including pulmonary fibrosis.[1]

**Case report:**

Mr. Mizanur Rahman , a 55 years old businessman hailing from Comilla with the complaints of occasional coughing out of scanty amount of blood and increased breathlessness for 1 month. He has been suffering from cough and respiratory distress for last 15 years without seasonal and diurnal variation. He also complained of low back pain for 21 years. The pain was worse in the morning after waking from sleep and improved with physical activities. He gradually developed difficulty in movement of spine. He did not have palpitations, mouth ulcer, chest pain and nasal congestion, skin rashes, urinary and bowel abnormalities with no history of taking methotrxate or sulfasalazine. Contact history with active tubercular patients was also negative. He was treated as a case smear negative PTB 12 years back on the basis of chronic cough and radiological findings with no improvement after treatment. He was smoker and smokes for 30 pack year. On general examination patient was anxious but cooperative. There was no anaemia, jaundice, cyanosis,

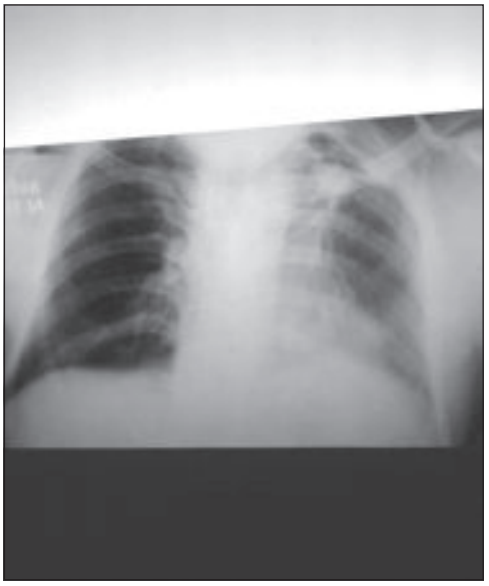

**Fig.-1:** *X-ray chest*

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**Fig.-2:** *CT-scan of chest*

clubbing, leukonychia, engorged neck veins or lymphadenopathy. Examination of respiratory system revealed features of pulmonary fibrosis and bronchiectasis of left lung. Examination of musculoskeletal system revealed lumbar lordosis, increased thoracic kyphosis and hyper- extension of neck. The patient was unable to look up and any side without movement of whole body. There was restricted movement of spine. Straight leg raising test about 70. There was tenderness over sacroiliac joint. Modified Schober's test was also positive. Other system examination revealed no abnormalities. Investigations showed: ESR- 70 mm in 1st hour. Liver function test (LFT) and renal function test (RFT), ECG, echocardiogram were normal. CRP was raised and HLA-B27 was positive. RF, anti-CCP antibody, ANA were negative. Sputum for AFB was negative; sputum for X-pert MTB/RIF did not detect MTB. Sputum for malignant cell was also negative. Chest X-Ray and CT-scan of chest revealed pulmonary fibrosis and bronchiectasis of left lung. X-Ray of dorso-lumbar spine showed bamboo spine appearance. Fusion of sacroiliac joint was also noted. Spirometry revealed restrictive lung disease. DLco corrected was 36% of predicted but DL/AV was 147% of predicted. 6MWT showed moderate desaturation with limitation of walking distance. FOB showed inflammatory changes.BAL was negative for malignant cell and also for AFB. So final diagnosis was pulmonary fibrosis with bronchiectasis due to ankylosing spondylitis.

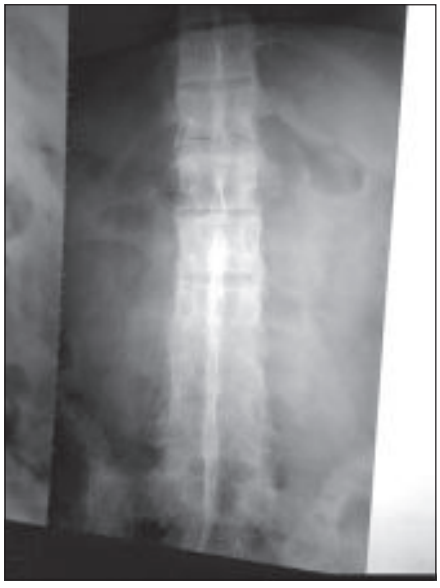
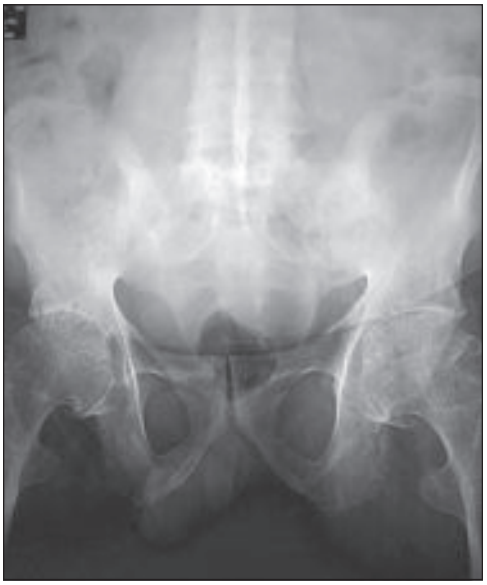


**Fig.-3:** *X-ray of lumbar spine*



**Discussion:**

Ankylosing spodylitis is characterized by a chronic inflammatory arthritis predominantly affecting the sacroiliac joints and spine which can progress to bony fusion of spine. The age of onset is usually between the age of 20 and 30 years. With a male preponderance of about 3:1[2]. The overall prevalence is less than 0.5% in most population.

Ankylosing spondylitis is thought to arise from the yet ill-defined interaction between pathogens and the host immune system in genetically susceptible

persons. The HLA-B27 itself is implicated through its antigen presenting function (It is a class-1 MHC molecule) or because of its propensity to form biodimers that activates leucocytes. The cardinal feature is low back pain and early morning stiffness with radiation to buttocks and posterior thighs. Symptoms exacerbated by inactivity and relieved by movement. As the disease progresses the spine become increasingly rigid as ankylosing occurs. Early physical signs include a reduced range movement of lumbar spine in all directions and pain on sacroiliac stressing. As the disease progresses the chest expansion becomes restricted. A few patients develop marked kyphosis of cervical and lumbar spine that may interfere with forward vision. Pleuritic chest pain is aggravated by breathing. Achilis tendinitis, planter fasciitis and tenderness over bony prominence are common. Abnout 40% patients develop peripheral arthritis. [2] This is usually assymetrical affecting large joints such as the hips ,knees, ankles and shoulder joint. Fatigue is a major complaints. [3] Disease activity in ankylosing spondylitis is detected by Bath Ankylosing Spondylitis Disease Activity Index (BASAI).[2,4] Extra-articular features include anterior uveitis, conjunctivitis, prostatis, cardio-vascular diseases like mitral incompetence, aortic incompetence, conduction defect, pericarditis, amyloidosis. Pulmonary manifestations are fibrosis of upper lobe, ILD, ventilatory impairment due to chest wall restriction, sleep apnoea, spontaneous pneumothorax, bronchiectasis etc. [2]

Apical fibrosis often appears > 5 years after the onset of the arthritic symptoms associated with the disease. The fibrosis may be unilateral or bilateral in nature and can be followed by cystic changes to the lung. [5] The causes of fibrosis is unknown but recurrent aspiration leading to pneumonitis from defective ventilatory alteration in apical mechanical stress from a rigid thoracic spine, recurrent impaired cough secondary to alteration in respiratory mechanisms.

The abnormal lung parenchyma is a fertile bed for super infection with a variety of organisms may contribute to development of bronchiectasis. [5]

Interstitial lung disease, beyond apical fibrosis, is now a recognized feature of pulmonary involvement in ankylosing spondylitis[6] and is detected by better visualization of the parenchyma with HRCT[7].

Mechanism of sleep apnoea is thought to arise from restriction of the oropharyngeal airway by compression of cervical spine. [8]

Pneumothorax is associated with fibrosis and smoking .

Diagnostic criteria: AS can be diagnosed when x-ray evidence of sacroilitis occurs with one of the features of history (Back pain >3 months improved by exercise and not relieved by rest) or clinical examinations ( Restriction of movement of spine or chest). [2]

Investigations which includes x-ray of sacroiliac joint, may shows widening in earlier stage and fusion in later stages. X-ray of dorso-lumbar spine may show syndesmophyte. Ossification of anterior longitudinal ligament and facet joint give rise to bamboo spine. MRI is more sensitive to detect early sacroilitis. [2] The ESR , CRP are usually elevated. Testing of HLA-B27 is helpful. Auto antibodies such as RF, ACPA and ANA are negative.[2]

Management includes education and physical activities. [2] Swimming is an ideal exercise. Poor posture should be avoided. A long acting NSAIDS is helpful. Patients of arthritis may be treated with Methotrixate or Sulfasalazine. Anti-TNF therapy(Adalimumab, Golimumab) may be considered in patients with BASADI e" 4.[2,9] Systemic steroid are used in uveitis. Surgery is helpful in restriction of hip or shoulder joint. Management for pulmonary fibrosis include medical, surgical treatment and physiotherapy. Azithromycine and other anti-inflammatory drugs may be prescribed along with bronchodilators. Surgical resection of affected lung/lobe may be considered when scaring, cavity develops and massive haemoptysis occurs. Chest physiotherapy is considered to improve breathing difficulty.

**Conclusion:**

Pulmonary fibrosis may have various causes but in the perspective of Bangladesh pulmonary tuberculosis is the most common cause. But smear negative pulmonary tuberculosis should be treated carefully as we can diagnose tuberculosis nowadays more confidently by virtue of X-pert MTB//RIF and sputum AFB culture. Other causes of pulmonary fibrosis including systemic connective tissue disease should not be forgotten. The early

diagnosis and proper treatment may save a patient from developing many complications.

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## CASE REPORT

# Cystic Hygroma with Thoracic Extension: A Case Report

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**Abstract:**

*Introduction: Cystic hygroma is a rare congenital malformation of the lymphatic system. It is most frequently detected in the head and neck region. Other rare sites include axilla, mediastinum, groin and retroperitoneum. It is usually evident at birth. Sometimes diagnosis is possible during prenatal period. The medical treatment of CH consists of the administration of sclerosing agents. Definitive treatment includes complete surgical excision.*

*Material and Methods:In this paper, a case is reported of cervical cystic hygroma with mediastinal extension in a female child which was evident at 5th day after her birth. She was taken to various hospitals but treatment delayed due to diagnostic dilemma. At last definitive treatment was done at age of 3 years and 8 months.*

*Results: Complete excision of both the neck mass and it's thoracic extension were done. She attended hospital after 1 month of surgery for follow up presenting herself free of symptoms.*

*Discussion: Any children presenting with a mass in the neck since birth, the first diagnosis should be considered as cystic hygroma.Though it is sometimes medically treated with sclerosing agents, surgery is the definitive treatment. Prognosis of cystic hygroma is excellent if complete surgical removal is possible.*

*Key Words: Cystic Hygroma (CH), Cystic Lymphangioma, Mediastinum.*

***[Chest & Heart Journal 2014; 38(1) : 49-53]***

## Introduction:

Cystic hygroma is a rarely encountered congenital malfor-mation of the lymphatic system, which is most often ob-served in the head and neck region[1]. Other infrequent sites include axilla, mediastinum, groin and retroperitoneum. It is usually evident at birth. 80-90% cases are evident within 2 years after birth. Diagnosis is usually done by clinical examination, chest x-ray, CT Scan or MRI. Ultrasonography is used as the first step in radiological diagnosis during intrauterine period.The medical treatment of cystic hygroma consists of the administration of sclerosing agents. Definitive treatment includes complete surgical

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excision. This particular patient visited many hospitals and there was diagnostic dilemma. The aim of this case report was to present diagnosis and treatment of a girl havingcystic hygroma of neck since birth with extension to the chest, together with the relevant literature.

**Case Report:**

A girl of 3 years and 8 months of age, presented with painless swelling over right side of the neck since birth;fever, pain and sudden increase in size of the mass for 20 days. At first it was evident as a small reddish nontender swelling which was noticed onfifth day after her birth which was measuring about2.5X1.5cm. Then her parents started homeopathy treatment and after that size increased to about 7.5X5.0cm within 1 week. The mass was very soft, non-tender, non-mobile. There was no difficulty in feeding or moving the neck. It was not associated with fever or jaundice. She was taken to a specialized hospital for children and FNAC was done which revealed benign vascular lesion suggestive of Haemangioma.

At age of 13 months, the neck mass increased in size and the overlying skin became red. This time it was associated with high fever (temperature raised up to 104°F). Her parents took her admission to a medical college hospital in paediatric surgery department. They described the mass as a 7X8cm swelling on right side of the neck, tender, increased local temperature, consistency firm, not fixed to overlying skin or underlying structures. It was diagnosed as Infectedcystic hygroma. Then 3 doses of Ethanolamine Oleate was injected into the mass once at every 1 month interval. But the size remained same.

At age of 2 years, the parents again started Homeopathy treatment for 3 months. Still there was no improvement. At that time she did not face any difficulty in eating, talking or moving the neck.

Twenty days back she had high fever with sudden increase in the size of the mass. Gradually the mass became painful and she was unable to move her neck. There was difficulty in eating and speech. Chest X-Ray and CT scan of neck &chest were done which revealed mediastinal extension of the mass. After attending 3 hospitals ultimately she was referred to NIDCH.

On examination, the baby was found cooperative, mildly anaemic. A large swelling on right side of the neck measuring about 7X8cm, non-pulsatile, tender, firm in consistency, not fixed to overlying skin or underlying structures, transillunimant, overlying skin of the swelling was smooth, reddish. Local temperature was raised. Regional lymph nodes were not palpable. There was no wasting of the muscles of neck but neck movement was restricted. Movement of the chest was reduced on right side. Other systemic examination reveal normal. X-ray of the chest reported a dense homogeneous opacity in right upper and mid zones, right paratracheal and paracardiac region (Figure-1). CT Scan of the neck and chest reported a large well demarcated anterior-superior mediastinal mass with extension superiorly right side of the neck at parapharyngeal space upto submandibular region; suggestive of mature teratoma (Figure- 2).

First operation was done on chest with right posterolateral thoracotomy approach. Excision of thoracic extension of cystic Hygroma was done. Cystic hygroma was found encroaching the Superior Venacava and Azygos Vein.

Second operation was done 3 weeks later by horizontal incision on right side of the neck. Large loculated mass was identified. Multiple feeding vessels were found. Severeadhesion to the great vessels of the neck was seen. Excision of cystic

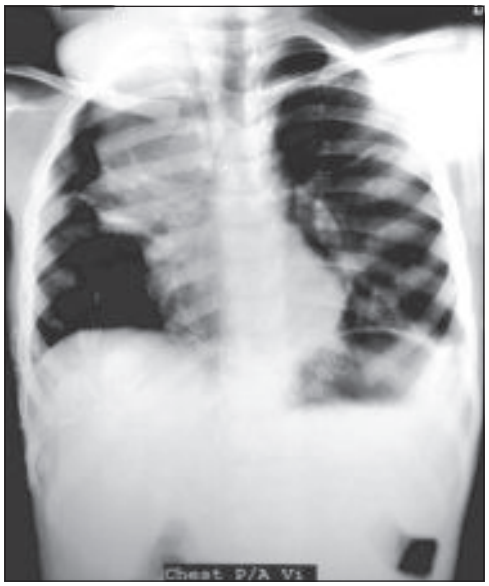


**Fig.- 1:** *Mass in right side of the neck and extension to right chest.*

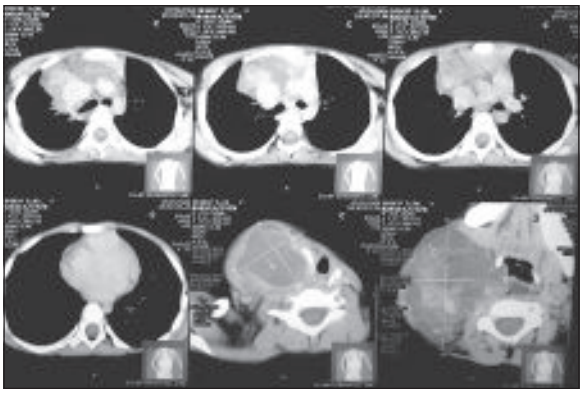

**Fig.-2:** *CT Scan of the Chest showing large anterior-superior mediastinal mass.*

hygroma of neck was done. Both the operations were planned for doing together. But patient's general condition did not allow stress of long time surgery, sooperations were done in two separate settings. Histopathology report of resected tissue from both the masses revealed cystic hygroma. After surgery the symptoms like difficulties in swallowing, speech or moving the neck were subsided. The baby was discharged from hospital with good physical condition and advice of follow up after 1 month. On follow up visit she was found playful and relieved of symptoms.

**Discussion:**

Cystic Hygroma (CH) is defined as a congenital, benign, multi-loculated lymphatic lesion classically found in posterior triangle of neck. This cystic lymphatic lesion that can affect any anatomic site in the human body. CH usually affects the head and neck (approximately 75%). Within the neck, the posterior triangle tends to be most frequently affected. More infrequent sites include the axilla, mediastinum, groin and retroperitoneum.[1] CH is synonymous with cystic lymphangioma. The lymphangiomas are thought to arise from a combination of the following: a failure of lymphatics to connect to the venous system, abnormal budding of lymphatic tissue, and sequestered lymphatic cell rests that retain their embryonic growth potential. These lymphatic rests can penetrate adjacent structures or dissect along fascial planes and eventually become canalized. These spaces retain their secretions and develop cystic components. The nature of the surrounding tissue determines whether the lymphangioma is capillary, cavernous, or cystic.[2]

The incidence of CH is estimated to be 1 case per 6,000-16,000 live births.[3] Mortality has been reported to be as high as 2-6% in some series, usually secondary to pneumonia, bronchiectasis, and airway compromise. Most cystic hygromas (50-65%) are evident at birth, with 80-90% of CHs presenting by age 2 years.t CH can be visualized using abdominal or transvaginal ultrasonography by 10 weeks' gestation,

CHs are typically soft, painless, compressible (doughy) masses. A CH typically transilluminates. In children who present with CH of the neck, closely evaluate for tracheal deviation, the tongue, oral cavity, hypopharynx, and larynx because any involvement may lead to airway obstruction. CH has been noted to be more common in persons with Turner syndrome, Down syndrome, Klinefelter syndrome and trisomy 18 and 13.u wIntrauterine alcohol exposure has been associated with the development of lymphangiomas.

CHs are composed of large irregular sinuses with a single layer of flattened epithelial lining and fibrous adventitial coats. The thickness of the vessel wall varies, with both striated and smooth muscle components. Cysts can range from 1 mm to several centimeters in size and are filled with clear-to-straw–colored fluid, which is eosinophilic and protein rich.The surrounding stroma is fibrous or fatty and may contain lymphoid aggregates, smooth muscle or other local tissues.

Diagnosis is usually done by CXR, CT Scan of Neck and Chest. CH can be visualized using abdominal or transvaginal ultrasonography by 10 weeks' gestation, Fast-spin MRI. Elevated alpha fetoprotein levels in amniocentesis fluid can also detectv .Fluorescent in situ hybridization (FISH) can be used to evaluate for cystic hygroma (CH) in prenatal chromosomal analysis. Chromosomes 13, 18, 21, X, and Y are specifically mentionedw .

CHs often present after a sudden increase in size secondary to infection or intralesional bleeding. Spontaneous decompression or shrinkage is uncommon. Potentially life-threatening airway compromise that manifests as noisy breathing (stridor) and cyanosis.

There may be feeding difficulties, as well as failure to thrive.

Proposal for staging of lymphatic malformations of the head and neck are stage I –

unilateralinfrahyoid, stage II - unilateral suprahyoid, stage III - unilateral infrahyoid and

suprahyoid, stage IV - bilateral suprahyoid and stage V - bilateral infrahyoid and

suprahyoid (adapted from de Serresx ) (Table- 1).

**Table-I**

*Proposal for staging of lymphatic malformations of the head and neck (adapted from de Serresx )*

| Stage | Location of lesion |
|---|---|
| I | Unilateral infrahyoid |
| II | Unilateral suprahyoid |
| III | Unilateral infrahyoid and suprahyoid |
| IV | Bilateral suprahyoid |
| V | Bilateral infrahyoid and suprahyoid |

Medical treatment consists of intralesional injection of sclerosing agents include OK-432 (an inactive strain of group A *Streptococcus pyogenes*), bleomycin, pure ethanol, bleomycin, sodium tetradecyl sulfate, and doxycycliney . The mechanism of action is an inflammatory response to the inactive bacteria, leading to fibrosis of the hygroma. An infected CH should be treated with intravenous antibiotics, and definitive surgery should be performed once the infection has resolved. If acute infection occurs prior to resection, surgery should be delayed at least 3 months.

The surgical team should attempt to completely remove the lymphangioma or to remove as much as possible, sparing all vital neurovascular structures. Complete excision has been estimated to be possible in roughly 40% of cases. CHs are ideally removed in one procedure because secondary excisions are complicated by fibrosis and distorted anatomical landmarks. Laser therapy is a recent advancement in the treatment of microcystic lesions.

The exceptions to excision at the time of diagnosis are few and include premature infants who are small in size and those with involvement of crucial neurovascular structures that are small and difficult to identify (eg, facial nerve). If no airway obstruction is present, surgery can be delayed until the child is aged 2 years or older, especially when operating around the facial nerve in the parotid area.[10]

Complications include airway obstruction, hemorrhage, infection, and deformation of surrounding bony structures or teeth if left untreated. Complications from the surgical excision of a cystic hygromainclude damage to a neurovascular structure (including cranial nerves), chylous fistula, chylothorax, hemorrhage, and recurrence. Most recurrences occur within the first year but have been reported to occur as long as 10 years after excision. If residual tissue is left behind, the expected recurrence rate is approximately 15%.[2]

In this case, it was thought at first as a case of haemangioma and was treated accordingly. Later diagnosis was confirmed as cystic hygroma with extension to the chest. As the lesion was of macrocystic structure and located in both the cervical and thoracic region, sclerotherapy with Ethanolamine Oleate was applied. But there was no improvement. Later complete surgical excision was planned as multiloculated cystic hygroma may not respond to sclerotherapy. Success of surgery has been found to correlate with histology, encapsulation, complete excision, anatomical location and stage of the lesion.[7,8,11] Imaging appeared to show smooth margins, indicating a lack of infiltration, which is a good prognostic feature, facilitating complete removal and low recurrence. However, this was an extensive stage III lesion with close relations to major structures and therefore a difficult procedure was anticipated. It proved to be impossible to remove the cystic hygroma completely without rupture, a recognised problem as these tumours usually have a fragile thin wall. Intra-operative rupture of the lesion complicates complete removal as it obscures the limitsof the structure.t However, Riechelmann[11] reported very low levels of recurrence (1/9 patients) following subtotal excision when small plaques of tumour wall were known to be left in situ. To the best of our knowledge we were able to remove the cystic hygroma completely.

**Conclusion:**

Any children presenting with a mass in the neck since birth, the first diagnosis should be considered as cystic hygroma.[12] CT or MRI evaluation of chest, retroperitoneum should be done to rule out any extension of primary lesion or presence of a second lesion. It is a benign lesion,[13] so, parents should be

assured first. Though it is sometimes medically treated with sclerosing agents, surgery is the definitive treatment. Prognosis of cystic hygroma is excellent if complete surgical removal is possible.

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## CASE REPORT

# Rupture of Non Coronary Sinus of Valsalva Aneurysm into Right Atrium: A Case Report

**Syed Al-Nahian[1], Rakibul Hasan[2], Omar Sadeque Khan[3]**
**Asit Baran Adhikary[4], Md. Aftabuddin[5]**

**Abstract:**

*Aneurysm of the sinus of Valsalva is a rare cardiac abnormality; however, the most common complication is rupture into heart chembers, usually the right heart chambers or rarely towards the left chambers. A ruptured aneurysm typically leads to an aortocardiac shunt and progressively worsening heart failure. Commonly on auscultation, it presents with a continuous murmur in left lower sternal edge. Here we report a case of a 27 years old male who suffered an aneurysm of the sinus of Valsalva rupture into the right atrium with only a pansystolic murmur who underwent successful surgical repair.*

*Key Words: Sinus of Valsalva, aneurysm rupture, pansystolic murmur.*

***[Chest & Heart Journal 2014; 38(1) : 54-56]***

**Introduction:**

Sinus of Valsalva is a dilatation of the aortic wall located between the aortic valve and the sinotubular junction. Its location is related to the presence of coronary arteries termed as the right coronary sinus (RCS), left coronary sinus (LCS) and non coronary sinus (NOCS).[1] Sinus of Valsalva aneurysm (SVA) is a dilatation caused by the lack of continuity between the middle layer of the aortic wall and the aortic valve.[2] It is a rare cardiac abnormality which most commonly is congenital but they may be acquired ( trauma, atherosclerosis, syphilis, cystic medial necrosis or infective endocarditis etc.). This sinus can commonly rupture and create a aortocardiac fistula into atrium or ventricle. RCS is the most frequently affected followed by the NOCS and rarely the LCS.[3] It is associated with a severe left-to- right shunt if communicating with the right sided heart chamber. Uncorrected rupture almost always cause deterioration of heart function and early surgical correction is the treatment of choice.[4]

**Case:**

Mr. Abdul Jalil a 27 years old male came to us with the complaints of palpitation, sharp chest pain and dyspnoea on exertion which was gradually worsening over the period of 2 years. On examination patient appeared to be anxious, pulse was 84 beats per minute, regular, high volume and collapsing in nature. Precordium examination revealed apex beat was in 6$^{th}$ intercostals space 7

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cm from midline with no parasternal heave or thrill but P2 was palpable. Both s1 and s2 were audible in all four areas but P2 was loud in the pulmonary area and a pansystolic murmur of grade 3/6 best heard in the lower left parasternal region. His ECG was normal, Chest X-ray PA view revealed cardiomegaly and trans-thoracic echocardiography showed a ruptured aneurysm of non-coronary sinus causing a left to right shunt into the right ventricle (RV) with a turbulent flow visible on color doppler from aorta to RV. The ventricular septum was intact with mild aortic regurgitation and moderate pulmonary hypertension ( PASP- 42 mm of Hg). Cardiac catheterization was performed and aortography revealed contrast material passes from non coronary sinus to right ventricle with significant step up of Oxygen saturation in RV; Qp:Qs ratio was 3:1 with PVR 2.11 unit $m^2$ and left to right shunt 65%. After proper evaluation patient underwent surgery for repair of the ruptured sinus. After standard median sternotomy right atrium and ventricle found to be dilated. Cardiopulmonary bypass (CPB) established with bicaval cannulation, aortic cross clamp applied, oblique aortotomy made and antegrade selective cardioplegia delivered through both the coronary ostia. Aorta was then inspected and a 0.5×1 cm tear was discovered right above the non-coronary cusp (fig-1) following which right atriotomy was made and it was found that the tear was interconnecting the aorta with the right atrium (just above the septal leaflet of tricuspid valve) (fig-2). The fistulatous opening just above the tricuspid valve was repaired with 5/0 prolene with pericardial pledget. A PTFE pledgeted repair was performed for the defect on the aortic side using 5/0 prolene. Aorta was closed in two layers with 5/0 prolene.

**Fig-1:** *Rupture sinus of Valsalva through aortotomy*

Right atriotomy was closed in layers and CPB was successfully terminated with minimum inotropic support without any difficulty (cross clamp time: 71 min; CPB time: 115 min). The patient was discharged on tenth post operative day. On the out-patient follow up after one month, he was in good health with no residual shunt or aortic regurgitation on trans-thoracic echocardiography.

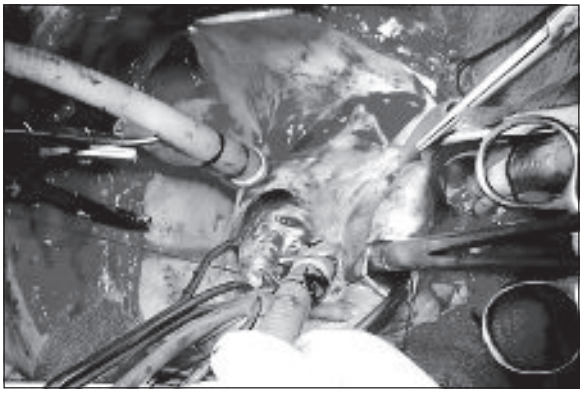

*Fig- 2: Communication of rupture sinus of Valsalva to right atrium above tricuspid valve*

**Discussion:**

Sinus of Valsalva aneurysm is a rare cardiac anomaly and arises mainly from a congenital defect of the aortic media or may follow bacterial endocarditis or degenerative disease. The aneurysms originate predominately from the right coronary sinus (70%) and are more prevalent in males and peoples from Asian desent.[4] Aneurysm of sinus of Valsalva can remain silent for several years and usually rupture during the 3rd or 4th decade of life (which is consistent with our patient's presentation) into any of the heart chambers - usually in the right chambers,[5] into the interventricular septum (causing complete atrioventricular block)[6] or in the pericardial cavity.[7] Until they rupture, sinus of Valsalva aneurysms are usually asymptomatic. Rupture of an aortic sinus aneurysm may commonly be associated with chest pain, difficulty in breathing and heart failure. Initially, a rupture in an endocardiac chamber can be silent and tolerated if it is small, gradual and progressive[8] but later manifests as progressive heart failure and the mean survival in a patient with an untreated ruptured sinus of Valsalva is about 4 years.[9] Generally on examination, the thrill and the

associated continuous murmur of ruptured sinus of Valsalva are said to be maximum at left lower sternal edge, however in this case unusually the thrill and the continuous murmur were absent and on the contrary there was only a pansystolic murmur present at lower left sternal edge. The murmur and other clinical findings were confirmed and crosschecked by five separate examiners of our department at different times but the findings were consistent. At present therapeutic treatment options for ruptured SVA include surgical methods such as direct suturing with or without pledget or suturing with the use of a patch, as well as the defect closure by intravenous placement of an occlude.[10] Surgical treatment presents very low per operative mortality risk (1%) in patient without infection.[7] Now-a-days, the ten years survival rate after surgical repair of a ruptured SVA is 90%[11] and the most used surgical technique is the 'dual exposure technique' where both the aorta and the chamber of termination are explored. Lukacs et al[12] report a zero perioperative mortality in a total of 30 patients with ruptured SVA and advised early intervention. In our center we also have performed surgical repair of around 10 cases with a zero perioperative mortality. In this case, surgical repair in the form of direct pledgeted suture was curative with absence of blood shunting from the aorta to right atrium on post operative clinical examination and color doppler echocardiography.

**Conclusion:**

Though rupture sinus of Valsalva is a uncommon condition, surgery is the treatment of choice for most occasion. Generally it presents with heart failure, chest pain or dyspnoea with a continuous murmur in left lower sternal area but a pansystolic murmur in the same area could be a presentation for this condition. Early detection and intervention in the form of surgery is curative in almost all the cases.

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## CASE REPORT

# Successful Removal of a Intrathoracic Diaphragmatic Lipoma : A Case Report

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**Abstract:**

*Introduction: Lipomas are benign soft tissue tumor and can occur anywhere in the body. However intrathoracic lipoma are extremely rare benign lesions. Most of the patients remain asymptomatic and incidentally diagnosed by imaging study.*

*Presentation of Case: A 55-year-old female housewife presented to our hospital suffering from mild chest pain and mild shortness of breath for two months. A subsequent chest X-ray and CT scans revealed a large homogeneous, fat density mass occupying the lower half of the right lung field. Following surgical resection the dull mass in pale yeallow color removed from diaphragm near the cardiophrenic angle and histological analysis conûrmed it as a lipoma.*

*Discussion: Intrathoracic lipomas are very slow-growing benign tumors usually without any symptom, which originate from the adipose tissue in submesothelial layers of the pleura parietalis, diaphragm, mediastinaland extrapericardial. They are common in obese people and fully encapsulate in most cases. Chest X-ray and CT scans are the most helpful tests in the diagnosis of intrathoracic lipomas. Though radiological follow up in asymptomatic cases are advocated by some, most of the authors suggest complete surgical resection to prevent recurrence and conversion to liposarcoma .*

*Conclusion: Complete enbloc removal of lipoma whenever possible, is the only deûnitive treatment option. Local recurrence is fairly uncommon after adequate surgery.*

*Key words: Lipoma, Diaphragmatic, Thoracic lipoma.*

***[Chest & Heart Journal 2014; 38(1) : 57-60]***

## Introduction:

Lipoma is the most common benign soft tissue tumor that can occur almost anywhere in the body. Although they account for approximately half of all soft tissue tumors and 80% of all benign fat-containing neoplasms, intrathoracic location is

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rare.[1] Among the intrathoracic locations lipoma can arise anywhere from the mediastinum, diaphragm, bronchus, lung, or thoracic wall, with diaphragmatic localization to be considered extremely rare.[1,2] The first report of a diaphragmatic lipoma case detected by autopsy was made in 1886 by Clark.[3] Most of the patients with intrathoracic lipoma remain asymptomatic and get diagnosed incidentally on a chest radiograph or a computed tomography (CT). Here we are reporting a successful removal of intrathoracic diaphragmatic lipoma through right lateral thoracotomy.

**Case presentation:**

A 55 years old female housewife admitted to our hospital with the complaints of vague chest pain and mild respiratory distress for 2 months. She was normotensive, nondiabetic, non-smoker and maintained a weight of 55 kg for her 140 cm height. She had no history of weight loss with no significant past medical history. Clinical examination revealed she was non-anaemic, chest movement slightly restricted in right side, percussion note dull and breath sound diminished in right side from $4^{th}$ space to downwards along anterior and from $6^{th}$ space to downwards along mid axillary line and from $8^{th}$ space to downwards along posterior scapular line. All routine blood tests were within normal limit. The results of lung function tests, which showed FEV1/FVC was 107%, furthermore FVC was 59% and $FEV_1$ 64% of its predicted value, were consistent with the impression of restrictive ventilatory disorder result from large thoracic tumor. Subsequent chest x-ray showed a large homogenous mass occupying almost the lower half of the right lung field (Fig-I). A Computed Tomography (CT) scan of chest revealed a large fatty density mass (-64 Houndsfield units) that compressed the right lower lobe (Fig-II). A provisional diagnosis of intra thoracic lipoma was made.In order to remove the whole tumor, surgical resection performed by incision made through right $6^{th}$ intercostals space . In the operative ûeld, a soft pale yellow mass was noted in the right chest cavity. In gross appearance, it was a thinly encapsulated, dull mass in a pale yellow color which has smooth surface and soft to the touch (Fig-III). The tumor was attached to the diaphragm in the cardiophrenic angle with its pedicle of 2.0 cm in diameter, compressing the right lower lobe of the lung. There was no adhesion between the tumor and the surrounding organs, and the entire mass was successfully excised en-bloc and the diaphragm was repaired, primarily with silk number 0. The histopathological examination of the tumor revealed that there was well-differentiated mature adipocytes, with absence of pleomorphism or mitotic activity, suggestive of lipoma. The patient had a uneventful post operative course and was discharged on $12^{th}$ post operative day.

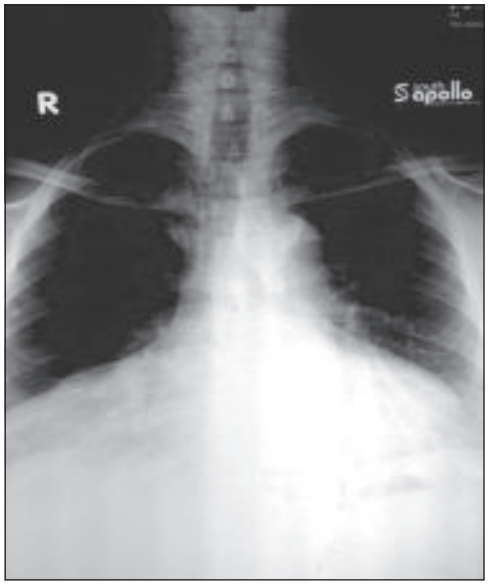


**Fig.-1 :** *Chest X-ray postero-anterior view showing the lesion occupying the right lower zone.*

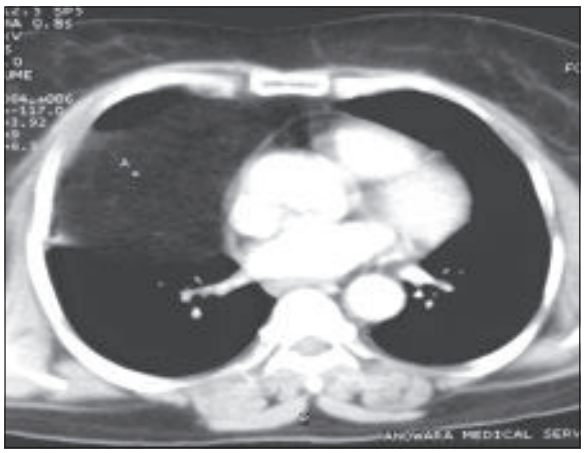

**Fig.-2:** *CT scan of the chest showing the mass occupying the right hemithorax near cardiophrenic angle.*

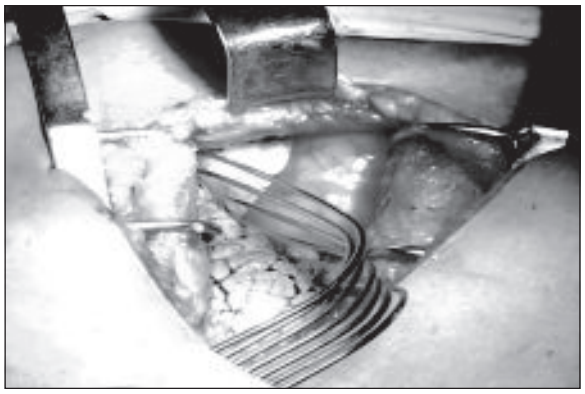

**Fig.-3:** *The encapsulated lipoma free from lung tissue*

**Disscussion:**

Lipomas are mostly found within the subcutaneous areas of the body. But intrathoracic lipomas are generally rare and slow-growing benign tumors. These tumors originate from the adipose tissue in submesothe-lial layers of the pleura parietalis, diaphragm, mediastinal and even extrapericardial. Williams and Parsons classified diaphragmatic lipomas according to anatomical localization as "intrathoracic lipomas" (total localization in the thoracic cage) and "sandglass thoracic lipomas" (localized both in the intrathoracic and extrathoracic parts, which are classified as cervicomediastinal and transmural lipomas),[4] in our report the case was purely intrathoracic. The incidence rate of diaphragmatic hernia is equal between male and female while the lesions usually emerge during the 4th or 5th decade of life.[5] Diaphragmatic lipomas are encapsulated, soft fatty tumors which are typically seen in obese people.[6] Similarly here, our patient was also overweight with BMI about 28.06 Kg/m$^2$. Generally diaphragmatic tumors do not have any characteristic symptoms but symptoms like chest pain, shoulder pain, back pain, dyspnea, cough, hemoptysis, and even diaphragmatic rupture have been reported in the literature.[7,8] Similarly in our case patients main symptoms were nonspecific chest pain and mild respiratory distress. CT scan allows a definitive diagnosis when it demonstrates a homogeneous fat attenuation mass ("50 to "150 Hounsfield units, or HU) which usually forms obtuse angles with the chest wall and displaced adjacent pulmonary parenchyma and vessels [9] (in our case it was -63 HU), though the density may not be entirely uniform because lipomas may often contain fibrous stroma or dystrophic calcification. Nevertheless, histological examination of the lesion is the most reliable method for a definite diagnosis.They are frequently found at the posterolateral part of the diaphragm at the area of Bochdalek hernia and are 2 times more likely to be seen on the left side[7] while, in our case we rather found the lipoma on the right side and near the cardiophrenic angle close to the area of Morgagni hernia. Aydin et al have reported several cases of diaphragmatic lipoma in which the lesion was excised either by thoracotomy or by vedio assisted thoracoscopic surgery and the diaphragm was repaired in all cases, primarily with silk number 0.[10] Recently, successful extirpation of a intrathoracic lipoma even with a single-port VATS has been reported.[11] In National Institute of Disease of Chest and Hospital (NIDCH) we have also performed several surgery for intrathoracic lipoma among which in case of diaphraghmatic lipoma we also repaired the diaphragm with 0 silk. The management strategy for diaphragmatic lipoma is still controversial as there is no consensus for the treatment of asymptomatic lipoma. However some authors suggested a periodic radiological follow-up of asymptomatic noninfiltrative diaphragmatic lipoma cases,[6] other authors insisted on surgical resection due to the risk of the development of diaphragmatic liposarcoma.[7,12,13] The outcome of resection of lipomas is generally very good as the recurrence rates after surgical excision have been re-ported to be less than 5%.[14] Most cases of recurrence are probably attributed to incomplete resection of the lesion.

**Conclusion:**

Although diaphragmatic lipoma very rarely may evolve towards liposarcoma in about 1% cases, surgical resection is still necessary in order to make proper diagnosis, as it is difficult to distinguish lipoma from well-differentiated liposarcoma preoperatively; as the compression symptoms could be fatal and lastly, complications such as intratumoral haemorrhage with pain and fever may exist during development of the tumor preoperatively. Local recurrence is generally uncommon after adequate surgery.

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## CASE REPORT

# Systemic Lupus Erythematosus Presented with Empyematous Pleural Effusion in an Adult Male Patient: A Diagnostic Challenge

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**Abstract**

*Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease specially of the females (9:1).Thus poses great difficulty in making an early diagnosis in males, often presenting with atypical manifestations. Pleural effusion occurs only 2.5-3% of cases.Rarely pleural effusion can be first manifestation of SLE occurs only in 2-3% of cases. Acute onset of empyematous pleural effusion has rarely been seen.Herein, we report a 30-year-old man with SLE presented with rapid progression of bilateral pleural effusion. Diagnostic thoracocentesis disclosed neutrophil-predominant sterile exudates and chest computed tomography revealed bilateral encysted pleural effusion. The diagnosis of SLE was readily established after serum antibody positivity specific for SLE.Nevertheless, optimal antibiotic therapy did not improve his condition. Intercostal chest tube drain was given associated with high dose corticosteroid therapy.*

***[Chest & Heart Journal 2014; 38(1) : 61-63]***

**Introduction:**

Systemic lupus erythematosus (SLE) is a diverse autoimmune inflammatory disease of unknown etiology.Principally affects women during child bearing age. Its pathogenesis is a complex contribution of hormonal, genetic and trigger factors(infection, ultraviolet light,radiation). Virtually all patients have skin and joint diseases. Between 30 and 50% will also develop renal, lung, cardiovascular and neurological involvement. SLE is diagnosed by American College of Rheumatology (ACR) criteria. However, a wide spectrum of clinical manifestations are common and early diagnosis of atypical clinical presentations is crucial to prevent the potentially fatal complications.

We report an adult male SLE patient who initially presented with right sided empyematous pleural effusion.

**Case Report:**

A 30-year-old non smoker cultivator was admitted through emergency department with fever and acute dyspnoea .The patient had been well until 2 years prior to this presentation, when he was noted recurrent episode of high grade fever,malar rash, discoid rash, polyarthritis, Raynaud's phenomenon,oral ulcer and alopecia. 4 months later he developed swelling of whole body.For this he was treated with some medications without any improvement. One week ago, he had been hospitalized for right sided pruritic chest pain and fever. After treatment with empirical antibiotics for 3 days, he felt better and was discharged with advice. At present physical examination revealed patient was dyspnoeic, blood pressure was 130/80 mmHg, body temperature 37.8°C, pulse rate was 110 beats/ minute and respiratory rate was 24 breaths/minute. Multiple small cervical lymphadenopathy, bilateral pedal edema, malar rash over nose sparing

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nasolabialfold, discoid lesion over forehead and back. Decreased breath sound over the right lower chest.The chest film revealed right sided moderate

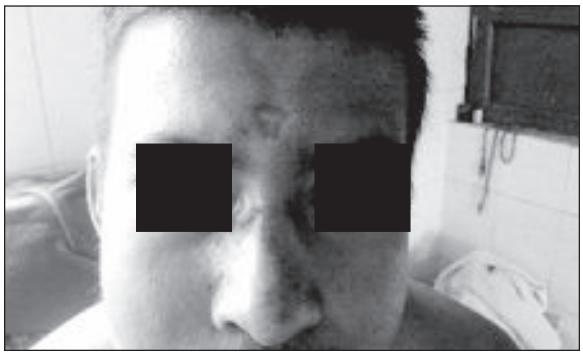

**Fig.-1:** *Butterfly rash over nose sparing nasolabial fold.*

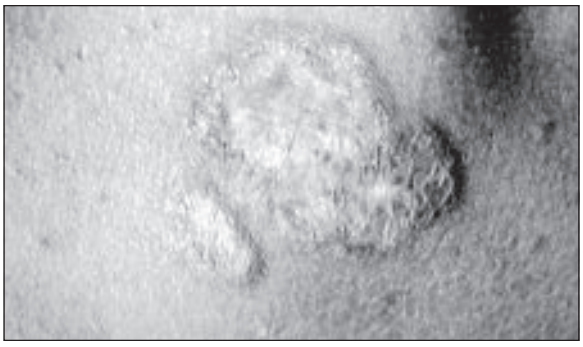

**Fig-2:** *Discoid rash on the back.*

pleural effusion. Laboratory analysis showed the white blood cell count was 10.47 $X10^9$/L (normal range 4.0-11.0$X10^9$ /L), hemoglobin concentration was 7.2g/dL (normal range 13.5-17 g/dL), ESR-15 mm/hr, serum albumin level was 2.5 g/dL (normal range3.5-.5.5 g/dL),serum glucose was 90 mg/dL (normal range 75-105 mg/dL). serum creatinine 0.6mg% and CRP was 7.26mg/L (normal<0.5mg/L).Serum fasting lipid profile, liver function tests were within normal limit.Urine analysis showed proteinuria (>300mg/dL), RBC 5-9/HPF. Diagnostic thoracocentesis resulted in an sterile exudate with predominant neutrophils (appearance-hazy, neutrophil-90%, lymphocyte-10%, protein-30.39gm/L, glucose-0.60 mmol/L).

Pleural fluid ADA 91.5 IU/L.Gram stain,AFB stain and culture was negative. Serology includes positive ANA&Anti dsDNA antibody and negative RF. Serum complement reports revealed hypocomplementemia $C_3$ 15.00mg/dL (normal 90.0-180.0) and $C_4$ 8.00mg/dL (normal 10.0-40.0) which indicates disease activity. Sputum for AFB and MT were negative. Chest computed tomography revealed bilateral encysted pleural effusion.

There was no improvement with the treatment of 3rdgeneration cephalosporin.A chest tube drain was placed and systemic corticosteroid was given.

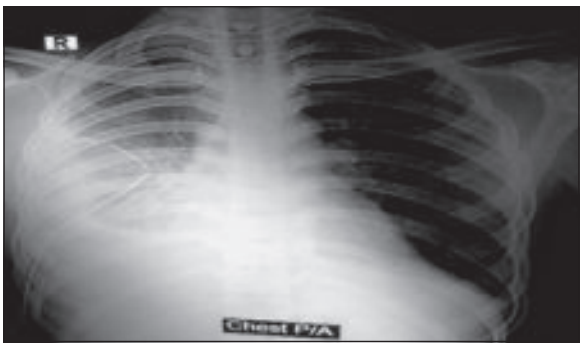


**Fig.-3:** *Chest X-ray P/A view shows right sided moderate pleural effusion*

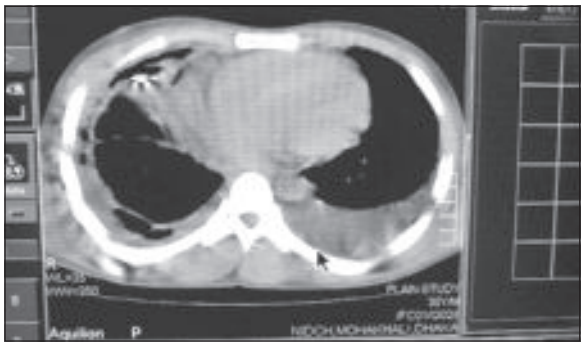

**Fig.-4:** *CT Scan of Chest, mediastinal window, shows bilateral encysted pleural effusion*

**Discussion:**

SLE is an imitator with various presentations. Overall, 90% of patients with lupus are female and most are between 15 and 50 years of age.[1]Therefore, making an early diagnosis is tough in elderly males without a classical expression.About 50% of patient of SLE experiences pulmonary involvement during the course of the disease. Although severe parenchymal lung disease is uncommon, pulmonary complications of SLE includes acute lupus pneumonitis, diaphragmatic dysfunction and shrinking lung syndrome, cavitating pulmonary nodules, pulmonary hypertension, pulmonary vasculitis, pulmonary embolism(often due to circulating anticardiolipin antibodies), alveolar haemorrhage (reflecting diffuse endothelial injury), chronic interstitial pneumonitis,[3-7]bronchiolitis obliterans (with or without organizing pneumonia),[8] and opportunistic pulmonary infections or drug toxicity from immunosuppressive therapy.[9]Lupus-related pleural effusion is currently described to immune complex deposition and the binding of anti-dsDNA antibodies to the mesothelium; it is usually

exudative with a low pH value, high protein, lactic dehydrogenase (LDH)and negative culture results.[10]The glucose level is often low in rheumatoid related pleural effusion but indeterminate in lupus.[11] Non-infectious pleural effusion, shown in this case also called sterile empyema or empyematous pleural effusion is usually seen in rheumatoid arthritis, pancreatitis and malignancy.[12]To our knowledge, it is rare in patients with SLE.[13]To distinguish it from infectious etiologies is crucial, since super-infection in lupus pleural effusion is possible. As for our case, the initial thoracocentesis was performed before antibiotic treatment started and only 3-days therapy was prescribed prior to the 2nd pleural effusion study. The results both implicated empyema (including a pH<7.2 and elevated LDH & total protein). The patient was initially treated with antibiotics under the impression of infectious empyema but in vain.

The presence of LE cells in the effusion, even though less sensitive, is highly specific for the diagnosis of lupus pleuritis and usually indicates an active disease status.[14]

They are composed of polymorp nuclear neutrophil phagocytosis of apoptotic bodies induced by antinuclear antibodies. However, LE cells are not frequently observed and cannot be depended on to make diagnosis, no longer belonging to the updating American College of Rheumatology revised criteria.[15]

**Limitations:**

In this case we could not perform pleural biopsy due to presence of moderate amount of empyematous effusion and lack of facility of detection of LE cells in pleural fluid.

**Conclusion:**

SLE does occur in adult males with an initial presentation as rapidly-progressive pleural effusion only. If laboratory analysis and thoracocentesis give an indeterminate result, a high clinical suspicion of autoimmune disturbance is reasonable and may facilitate early diagnosis.

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