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Evaluation and Management of Dyspnea: A Case series analysis

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Abstract:
Dyspnea is one of the most common symptomfor which patients seeks medical attention. Various etiologies and multiple associations makes it a diagnostic challenge to clinicians. Acute causes maybe life threatening if not diagnosed and treated on time. Acute pulmonary embolism, acute myocardial infarction, Acute respiratory distress syndrome, spontaneous pneumothorax, pulmonary hemorrhage, chest wall injury and foreign body aspiration are some causes of acute dyspnea. Dyspnea can be considered as an alarm sign of disease severity and related to disease prognosis and outcome.Initial assessment include history taking, clinical examination, routine blood tests, chest radiographs, electrocardiogram, spirometry and arterial blood gas analysis.
In this article we presents four different cases of dyspnea and their evaluation with management.
Key Words*:Dyspnea,Pneumothorax, Antituberculartreatment, Pulmonarytuberculosis, Intercostal tube drainage, MTB, AFB, CBNAAT, HRCT, ABG, UIP, IPF, 6MWT, ANA, Pleural effusion, Thoracentesis, Carcinomalung.*

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I. INTRODUCTION

Dyspnea is defined as the subjective awareness of breathing discomfort. Causes can be multifactorial including cardiac, pulmonary, malignancy, metabolic and psychological.[1] It may be a diagnostic challenge for clinicians and proper evaluation is required to find out the cause. Although pathogenesis is unclear and underlying mechanism comprises afferent signals from respiratory system to central nervous system and efferent signals from central nervous system to respiratory muscles. Peripheral and central chemoreceptors activated by hypoxemia, hypercapnia and acidemia. Mechanoreceptors in lungs and chest wall include stretch receptors, irritant receptors, j-receptors, muscle spindles and golgi tendon organs. These receptors activates in increased work of breathing produces airway resistance high. Efferent signals transmitted by corollary discharge to sensory cortex causing sensation of air hunger[2]. Clinicians should know various causes of dyspnea in different age groups to provide better treatment and prevent life threatening complications[3]. Dyspnea plays significant role in prognosis of chronic respiratory diseases.[4] Initial evaluation include proper history taking,general physical examination, chest radiograph, spirometry and electrocardiogram along with routine blood tests.[5]In this article we present four different cases of dyspnea and their management.

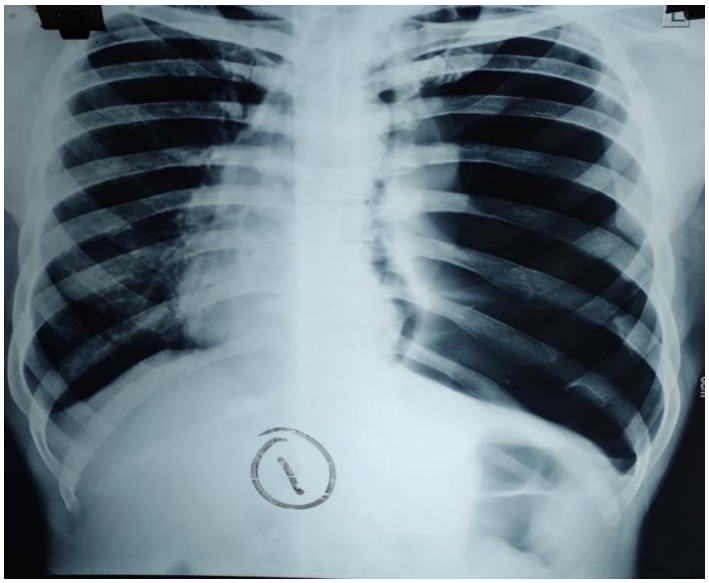
II. CASE REPORTS

Case 1

A 57 years old male presented with cough with minimal expectoration since 20 days, low grade fever since 5 days,dyspneaon exertion since 4 days and left sided chest pain since 4 days. He giveshistory of hemoptysis 20 days ago. He took antitubercular treatment(ATT) for pulmonary tuberculosis(PTB) 2 years ago for 6 months. He was not a known case of diabetes or hypertension. He was a former smoker (for20 years, 2 bundle /day),Occasional alcoholic since 20 years.

Respiratory examination reveals patientconcious,oriented, SpO_2: 90% on room air, BP: 126/80 mmHg, Pulse: 123/min, Trails sign positive, trachea appears to be shifted to right side, Chest movements decreased on left side of chest, Hyper resonant notes on left side,Decreased breath sounds on left side, Crepitations on right mammary and infra scapular area.

Chest radiograph advised

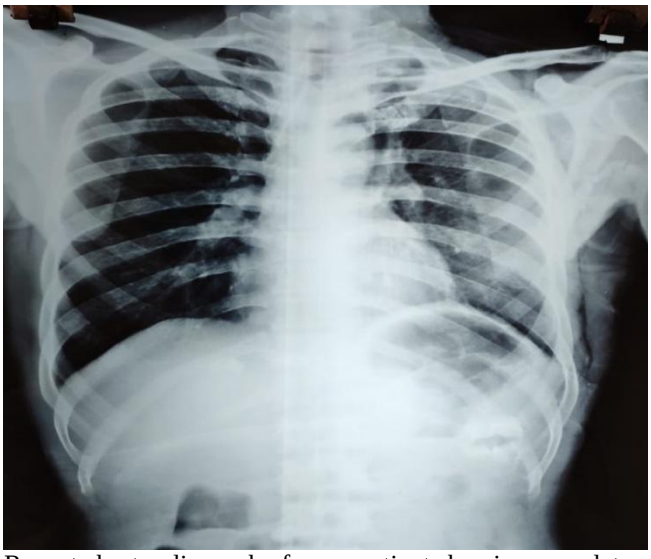


(Figure1: Chest radiograph showing left sided pneumothorax)

Left sided Intercostal tube drainage (ICTD) done on 4thIntercostal space (ICS) mid axillary line after taking consent, Tube was working (air column movement present, no BPF noted), No immediate complications, tube care explained to attender and repeat Xray chest advised, Postoperative vitals were stable, and shifted to ward, Blood Routine- Hb :13 g/dl, leukocytes: 4600/mm3, platelet: 4 lakh/µL, Liver and renal function: Normal, HIV & HBsAg: Non-reactive, Sputum for Acid fast Bacilli (AFB): Negative, Sputum for CBNAAT(catridge based nucleic acid amplification test): Mycobacterium tuberculosis (MTB) not detected. After 5 days, tube was not working as no air column moving in under water seal. Patient was not dyspneic, On auscultation bilateral air entry equal, Repeat Xray advised

Repeat chest Xray shows lung expanded, tube clamped for 24 hours, there was no collapse of lung after clamping the ICDT, Tube extubated and planned for discharge on Dry powder inhaler (DPI) and symptomatic medications.Diagnosis of Secondary spontaneous pneumothorax was made.

Xray at time of discharge given below.



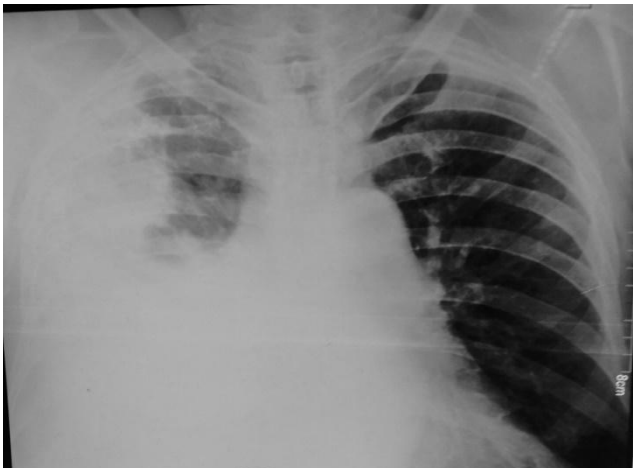
(Figure 2: Repeat chest radiograph of same patient showing complete expansion)

Case 2

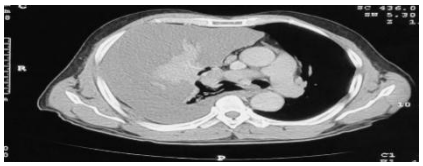
A 60years old male presented withDyspnea on exertion since 2 months, Right sided chest pain since 2months, Cough with expectoration since 2 moths, Low grade fever since 3 days,no hemoptysis or feet swelling. Past History revealsATT history: 25 years agofor PTB;taken for 6 months; Not a known case of Diabetes or Hypertension; No previous surgeries/hospitalization. Personal History: chronic smoker since 30 years(1 bundle /day), Occasional alcoholic

Respiratory examination showsPatient was conscious and oriented, SpO2 :96% on room air, BP :130/90 mmHg, pulse :98/min, Pallor and clubbing(grade 3) were present, Trail's sign positive, trachea appears to be shifted to left side, Chest movements and chest expansion decreased on right lung field, Vocal fremitus and vocal resonance were reduced on right side, Stonydull note on percussion– rightinframammary & infra scapular area.

Chest radiograph followed by Computed tomogram (CT) of chest advised.



(Figure 3: Chest radiograph showing right pleural effusion)



(Figure 4: Computed tomography of chest showing Right massive pleural effusion, hilar mass obliterating upper and lower lobe bronchus causing distal collapse)

Diagnostic and therapeutic thoracentesis was performed, pleural fluid analysis reveals lymphocytic exudative nature with very low ADA (adenosine deaminase) level and cytology doesn't shows malignant cells.

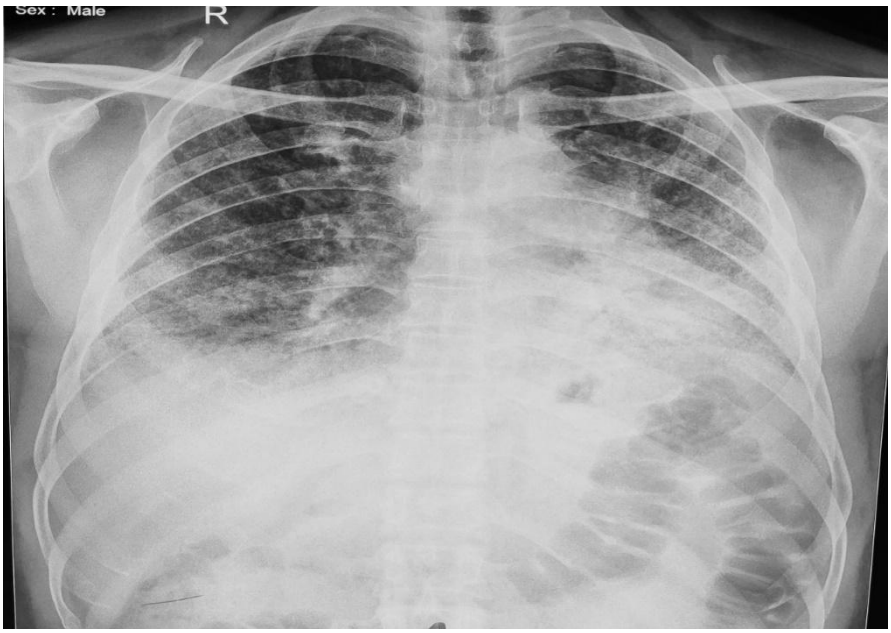
Flexible bronchoscopy (FOB) was done -Right intermediate bronchus was occluded by growth & bronchial biopsy was taken from growth. Biopsy specimen sent for histopathological examination- Revealed poorly differentiated Adeno carcinoma, EGFR was negative. A diagnosis of Poorly Differentiated Adenocarcinoma Of Right lung was made. Patient started with chemotherapy (paclitaxel +carboplatin) after prechemotherapy evaluation. Patient tolerated well with chemotherapy and pleurodesis was advocated with sclerosing agents.Discharged with an advice to come for next cycle of chemotherapy after 21 days.

Case3

A 60year old male presented with complaints of dry cough for past 6 months, dyspnea on exertion for past 1 month, loss of appetite since 2 weeks. There was no history of fever, chest pain, pedal edema or hemoptysis. He was not taking any regular medication, No symptoms suggestive of Rheumatic diseases(arthralgia, dry eyes, dry mouth, muscle weakness, numbness, tingling, Raynaud phenomenon). Nohistory of exposure to fumes, dust, birds, molds, desert cooler or therapeutic irradiation.No history suggestive of sarcoidosis; rashes, nodes, ankle swelling. He was a Policeman by occupation and chronic smoker for 30years but not alcoholic. Family history was non-contributory.

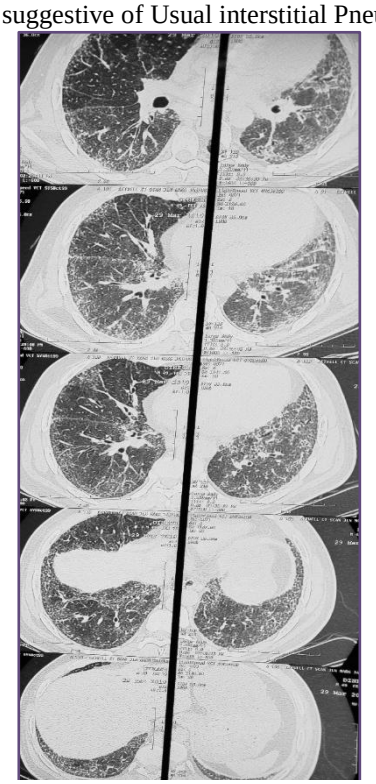
Respiratory examination revealspatient conscious and oriented, grade 2 clubbingpresent, PR-86/min, BP- 110/80 mmHg, afebrile, RR-24/min, SpO2- 90% room air, no lymphadenopathy, no pedal edema.

Trachea central, Bilateral chest movements equal,Bilaterallyresonanton percussion, air entry bilaterally equal, Bilateral Basal end inspiratory crackles and vocal resonance equal.



(Figure 5: Chest radiograph showing reticulonodular opacities)

Spirometry shows restrictive pattern,Sixminute walk test (6MWT) shows5% desaturation (distance covered <250metres). Arterial blood gas (ABG) on room air shows (pH: 7.45, pCo2: 32, pO2: 55, Hco3: 22, So2: 88), High resolution CT of chest was taken suggestive of Usual interstitial Pneumonia(UIP) pattern.



(Figure 6: HRCT chest showing honeycombing, interstitial septal thickening with subpleural and basal predominance)

Blood Investigations showsHb-14.1 g/dl,TLC -11,000/mm3, Random blood sugar- 158 mg/dl,Platelet-1.58 lakh/mm3, Renal and liver functions were normal, Antinuclear antibody (ANA) negative. Echocardiography reveals grade 1 left ventricular dysfunction, mild pulmonary hypertension and Mild tricuspid regurgitation. Based on history, clinical and radiological findings our patient got diagnosed as Idiopathic pulmonary fibrosis (IPF-UIP pattern).

Patient was treated with oxygen inhalation, injectionCefoperazon with sulbactam 1.5 gm 12^{th} hourly,

Tab Nintedanib 150mg 12th hourly, Cap Pantoprazole 40 mg 12th hourly and symptomatic medications.

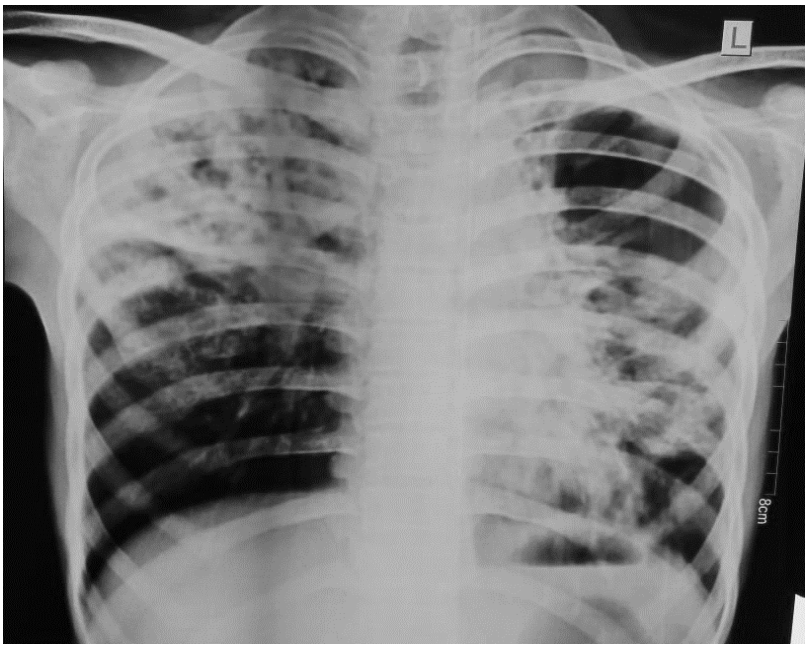
Case4

A 16years old male came with complaints ofcough with minimal expectoration since 3 months, shortness of breath since 3 months,Fever since 2 months, swelling of both legs since 1 month. He was taking ATT since 2 months for sputum positive PTB, Not a known diabetes or hypertension, no addictions. Family history suggests cardiac event in father at young age still on treatment.

Respiratory examination reveals

Pulse-114/min, BP-100/60mmHg, SpO2- 90% room air, RR- 30 breath/ min, afebrile, swelling of both legs(with tenderness) noted with Homan's sign positive bilaterally, Trachea central, chest wall movement & expansion decrease on right side,bilateralcrepitations & wheeze present.

Chest Xray was taken.



(Figure 7: Chest radiograph shows bilateral lung involvement with fibrocavitatory disease)

ABGshows hypoxia,Sputum for AFB shows 3+, Sputum for CBNAAT-MTB detected, Rifampicin sensitive, routine Blood investigations were normal.He was treated with moist oxygen inhalation, intravenous fluids were given with strict monitoring, continued ATT along with IV antibiotics and symptomatic medication. Venous doppler showed Bilateral Lower limb DVT with IVC thrombosis. Started injection Enoxaparin 1 mg/kg 12thhourly subcutaneous.Sent blood and urine investigations as per haematology reference to find out cause for DVT.Urine homocysteine was positive, low serum vitB_{12} (1.96 ng/ml),Protein C and S found reduced (11% and 34% respectively).Thrombolysis was started with injectionHeparin along with aspirin and warfarin (Target INR :2.5 to 3). Echocardiography shows (Right ventricular dilation, pressure overload and dysfunction). Thrombolysis done with Injection Streptokinase 250000 IU in loading dose in 30 minutes followed by 100000 IU/hour maintenance dose over 12 to 24 hours.Success of treatment reviewed by repeat echocardiogram. After successful reperfusion treatment patient was discharged with advice to continue ATT and Tab Apixaban 5mg 12th hourly for next 3 months.

III. DISCUSSION

Evaluation and managementdiffers in various cases of dyspnea according to etiology. Four different cases described in article are Secondary spontaneous pneumothorax, Malignant pleural effusion, Interstitial Lung Disease-IPF and Pulmonary thromboembolism. Chest radiograph and clinical examination confirms diagnosis of Pneumothorax. Intercostal chest tube drainage is indicated in cases of tension pneumothorax, bilateral involvement and those with dyspnea. Clamping of chest tube before removal once lung expands found effective. [6]

Presence of Massive pleural effusion in malignancy indicates poor prognosis. Majority of cases present with dyspnea on exertion, but incidental radiographic appearances are not uncommon. Thoracentesis (Diagnostic& therapeutic) and pleural fluid analysis after chest radiography constitutes initial step in management followed by CT chest and tissue diagnosis based on location of lesion. Bronchoscopicguidedor CT-

guided biopsy can be adopted for central and peripheral lesions respectively. Treatment of malignancy after immunohistochemistry should be started and pleurodesis prevents recurrence of pleural effusion. [7]

IPF is one of the most common causeof pulmonary fibrosis. Dyspnea is an initial presentation in majority of cases and underlying mechanism is still uncertain. Proposed hypothesis includes reduced compliance and lung volume, high respiratory drive and dead space ventilation and pulmonary hypertension. A multidisciplinary approach is required to arrive at final diagnosis. Blood tests, spirometry, exercise testing, HRCT chest, bronchoscopy and lung biopsy include the diagnostic tools in diagnosis of IPF. Only two antifibrotic drugs found beneficial in IPF are Pirfenidone and Nintedanib. Other modalities of management are oxygen supplementation, Noninvasive ventilation and symptomatic medications. Lung transplantation recommended in refractory cases. [8-11]

Virchow's triad of thrombosis include hypercoagulability, endothelial injury and altered blood flow. Thromboembolic complications are life threatening if not diagnosed and adequately treated. DVT of lower limbs are most common causes of pulmonary thromboembolism. Risk stratification helps in early diagnosis and prevents serious adverse events. Systemic thrombolysis with streptokinase, urokinase or reteplase are recommended in those with hemodynamic instability. Anticoagulation is preferred after stabilization.[12,13]

IV. CONCLUSION

Dyspnea is a common symptom for hospital admission which requires proper evaluation for prevention of life-threatening complications. Multiple etiologies and association with various co-morbiditiesmakes it a clinical challenge.

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