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Case Report:

Explosive Pleuritis

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Abstract: Pleural effusions associated with pneumonia (parapneumonic effusions) are one of the most common causes of exudative pleural effusions in the world. Approximately 20 to 40% of patients hospitalized with pneumonia will have an accompanying pleural effusion. The term 'Explosive pleuritis' was originally described by Braman and Donat in 1986 as pleural effusions developing within hours of admission. We report a 38 years old male patient with minimal pleural effusion which progressed rapidly within one day to involve almost whole of the hemithorax. There were multiple loculations on ultrasonography of thorax. Pleural fluid was sero-sanguinous and revealed gram positive diplococcic. The patient improved with antibiotics and pigtail catheter drainage.

Key Words: Explosive pleuritis; Hemithorax; Loculated; Pigtail catheter.

Introduction:

Pleural effusion, the result of accumulation of fluid in the pleural space, is a common medical problem. The term 'Explosive pleuritis' was originally described by Braman and Donat[1] in 1986 as pleural effusions developing within hours of admission. It is the rapid development of pleural effusion involving more than 90% of the hemithorax. We report a 38 years old male patient with minimal pleural effusion which progressed rapidly within one day to involve almost whole of the hemithorax with multiple septations and required catheter drainage in addition to medical therapy. This condition is rare and an early diagnosis and immediate treatment is essential to reduce morbidity and mortality.

Case Report:

A 38 years male patient from shimla presented in medicine OPD with high grade fever of 2 days duration and pain right side of chest and dyspnea for one day. He was smoker and labourer by profession. Past history was unremarkable. On examination, he was febrile and had dullness and decreased breath sounds on right infra scapular area. Chest roentgenogram revealed haziness over right lower zone and blunt right costophrenic angle (Fig.1). The patient was started on oral amoxicillin-clavulanic acid and was advised pleural fluid analysis. The patient did not come for pleural tap and landed up in casualty the next day with marked dyspnea and high grade fever. On clinical examination patient was febrile and had respiratory distress. Chest examination revealed massive pleural effusion on right side. Chest roentgenogram revealed homogenous opacity involving more than 90% of right hemithorax (Fig.2).

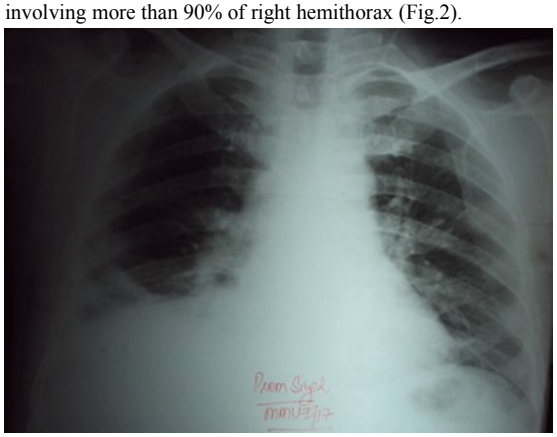

Figure 1: Chest X ray PA view on day 1 showing haziness right lower zone with blunt right CP angle

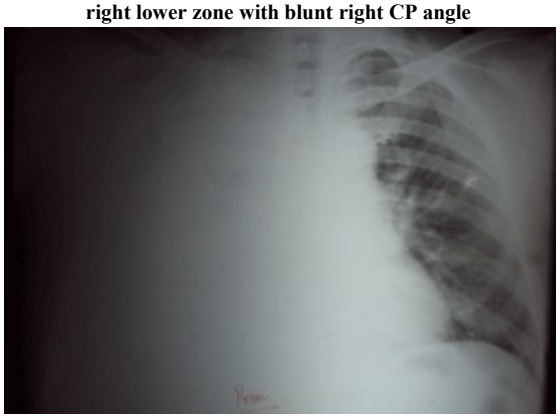

Figure 2: Chest X ray PA view on day 3 showing homogenous opacity involving more than 90% of right hemithorax.

Pleural tap was done but only 50ml of sero-sanguinous fluid could be tapped. Ultrasonography thorax was suggestive of multiple septations in pleural effusion. Pig tail catheterization was done under ultrasonographic guidance and fibrinolytic therapy with streptokinase was injected through the catheter and sero-sanguinous fluid was drained. CT thorax after 2 days revealed loculated right pleural effusion with mediastinal lymph node (8.4mm) with alveolitis right lung field (Fig.3). On detailed work up, investigations revealed: Pleural fluid: ADA-24.8U/L, Cytology- 180 WBC (N-70%, L-30%) second time repeated pleural fluid analysis revealed 480 cells mostly neutrophils, protein- 5.2mg%, culture - sterile, Gram stain- gram positive diplococci, AFB- negative, glucose- 39.4mg/dl(40-60), LDH- 1963u/l (more than 3 times serum LDH value of 448.7). Haemogram: TLC- 16050/cmm, Polymorphs 84%, lymphocytes 15%,(repeated after 5 days of treatment - 11810/cmm with P-80%,L-19%), Hemoglobin - 12.1g%, Platelets-209000/cmm, ESR- 50mm 1st hour. Biochemistry: Random blood sugar – 101mg%, Urea-36mg/dl, creatinine- 1.0mg/dl, sodium- 137meq, potassium- 4.5meq, chloride- 101meq. Liver function tests were normal. Blood culture was sterile. No organisms were detected on gram staining of sputum, sputum culture was sterile and it was negative for AFB. On these evidences it was diagnosed to be a case of explosive pleuritis probably caused by streptococcal infection. Patient was started with injectable amoxicillin-clavulanic acid and levofloxacin was added. After streptokinase fibrinolysis about 500-700 ml of sero-sanguinous pleural fluid was drained daily for 5 days, later the volume gradually decreased (fig.4). The pigtail catheter was removed and the patient was discharged on 14th day with advice to follow up.

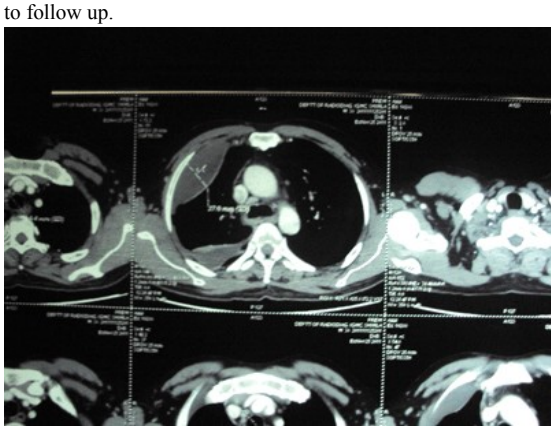

Figure 3: CT Thorax showing pleural effusion with loculations and septations on the right side (after pig tail catheter drainage).

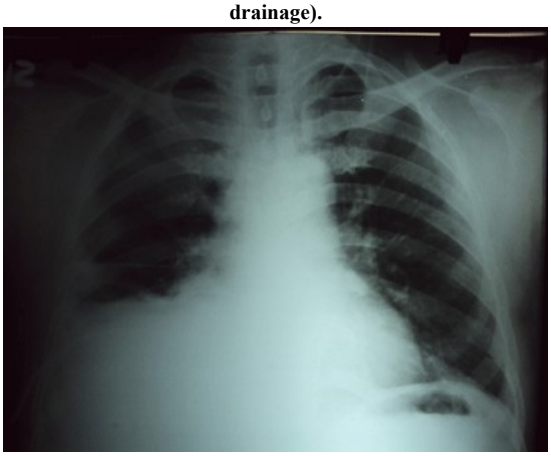

Figure 4: Chest X ray on day 7 after pig tail catheter drainage of right pleural effusion.

Discussion:

Pleural effusions associated with pneumonia (parapneumonic effusions) are one of the most common causes of exudative pleural effusions in the world. Approximately 20 to 40% of patients hospitalized with pneumonia will have an accompanying pleural effusion.[2] The term 'Explosive pleuritis' was originally described by Braman and Donat[1] in 1986 as pleural effusions developing within hours of admission. In their original article, clinical and roentgenographic evidence for two cases of explosive pleuritis caused by group A beta-hemolytic streptococci, in the absence of bronchopneumonia, were presented .They proposed that the pathogenesis of explosive pleuritis relates to the observation that streptococcal infections have a unique propensity to cause blockage of the peribronchial and subpleural lymphatics with cellular and necrotic debris.[1] Jasdeep K Sharma et al[3] in their article defined explosive pleuritis as the rapid development of pleural effusion involving more than 90% of the hemithorax within 24 hours, causing the compression of pulmonary tissue and a mediastinal shift to the contra lateral side.

The bacteriology of pleural infection differs somewhat from that of pneumonia. In one study of 434 patients with pleural infection, of whom nearly 60% achieved a microbiological diagnosis using standard conventional methods, the most prevalent organisms cultured in community-acquired pleural infections were streptococcal species (*Streptococcus milleri* [32%], *Streptococcus pneumoniae* [13%], other streptococci [7%]) followed by anaerobes (16%) and staphylococci (11%).[4] More than 40% of patients with pleural effusion do not ever have a positive bacterial culture. This may be partly dependent on the use of antibiotics prior to pleural fluid sampling and the care with which the sample is handled and cultured, On the other hand, blood cultures are positive in only a few cases of pleural infection (12%).[5] In our patient, bacteriological diagnosis could not be achieved as both blood culture and pleural fluid culture samples were taken after antibiotics were started. Though there were gram positive diplococci in the pleural fluid smears and serum LDH levels were markedly raised.

The organisms implicated in explosive pleuritis include the broad spectrum of organisms responsible for major causes of pulmonary infection. These include gram-positive cocci such as *Streptococcus pneumoniae*, *Streptococcus pyrogenes* and other streptococci and staphylococci. Gram- negative cocci such as *Neisseria meningitides* and *Moraxella catarrhalis* are also included.[3] Laboratory results may reveal elevated polymorphonuclear leucocytes. There is a four fold rise in ASO (antistreptolysin-O) titres over several weeks or a single titre of more than 250 Todd units. Our patient had leucocytosis but ASO titres could not be obtained as it was not being done currently in the hospital. The clinical presentation and physical findings in our patient were consistent with variable degrees of respiratory distress and respiratory system findings of pleural effusion as described by Jasdeep k Sharma et al[3] in their patient having explosive pleuritis.

In streptococcal pneumonia there is a high frequency of pleurisy and pleural effusion that rapidly progresses to loculated empyema called explosive pleuritis.[6] This process can occur over a period of hours. Similar picture was there in our patient and the pleural effusion rapidly progressed in about 24 hours. Pathologically severe sero-sanguinous pleural effusion, hemorrhagic edema of the lung and dilated lymphatics in the interlobular septa are present.[6] The fluid drained in our patient was also sero-sanguinous and exudative.

The diagnosis and treatment of this condition make thoracotomy essential. Thoracentesis alone is ineffective. Although rapidly developing pleural effusions are best treated by early chest tube drainage because of a tendency toward early loculation, it is not unusual to have only minimal fluid drained from the pleural space.[7] In our patient also only a small amount of

fluid could be aspirated initially. It is only after pigtail catheter drainage the fluid could be drained. In addition the antibiotics (amoxicillin-clavulanic acid and levofloxacin) were started and the patient improved with the combined treatment.

In a trial by Nicholas A. Maskellet al[8], intrapleural streptokinase had a modest adverse-event profile in patients with pleural infection but was ineffective in reducing mortality, the need for surgical drainage, or the length of the hospital stay. Studies conducted earlier had established that fibrinolytic agents do lead to macroscopically effective in vivo lysis of intrapleural fibrin adhesions[9]and reduce the volume of infected pleural-fluid collections. Thus, there may still be a role for fibrinolytic agents in treating the small subgroup of patients who have an exceptionally large, loculated collection of pleural fluid that causes substantial dyspnea, hypoxemia, or hypercapnia by the mechanical impairment of lung function.[8] In our patient also the pleural fluid drainage increased substantially after streptokinase was injected in the pleural cavity and patient had marked symptom relief.

Our patient had clinical deterioration over a short period of time (about 24 hours) and rapid progression of radiological findings characteristic of explosive pleuritis though we could not arrive at a bacteriological diagnosis. The condition should be treated as a medical emergency and the patients usually require surgical intervention in addition to medical therapy.

References:

1. Braman SS, Donat WE. Explosive pleuritis. *Am J Med*.1986;81:723-726.
2. Light RW. Pleural Diseases. Fifth Edition. Lippincott, Williams and Wilkins, Baltimore. 2007.
3. Sharma JK, Marrie TJ. Explosive pleuritis. *Can J Infect Dis*. 2001 Mar-Apr;12(2):104–107.
4. Maskell NA, Batt S, Hedley EL, Davies CW, Gillespie SH, Davies RJ. The bacteriology of pleural infection by genetic and standard methods and its mortality significance. *Am. J Respir Crit Care Med.* 2006;174:817-823
5. Foster S, Maskell N. Bacteriology of complicated parapneumonic effusions. *Curr Opin Pulm Med*.2007;13:319-323
6. Johnson JL. Pleurisy, Fever and rapidly progressive pleural effusion in a healthy 29 year old physician. *Chest* 2001;119;1266-1269.
7. Thomas DF, Glass JL, Baisch BF. Management of streptococcal empyema. *Ann Thorac Surg* 1966;2;658-664.
8. Maskell NA. U.K. Controlled Trial of Intrapleural Streptokinase for Pleural Infection. *N Engl J Med* 2005;352:865-874.
9. Maskell NA, Gleeson FV. Effect of intrapleural streptokinase on a loculated malignant pleural effusion. *N Engl J Med* 2003; 348:e4-e4.