Ephraim Teffera. *Ethiop Med J, 2016, Vol 54, No.3*

CASE REPORT

RE-EXPANSION PULMONARY EDEMA FOLLOWING REPAIR OF MISSED POST TRAUMATIC DIAPHRAGMATIC HERNIA A CASE REPORT AND REVIEW LITERATURES

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ABSTRACT

Reexpansion pulmonary edema following repair of missed diaphragmatic hernia is very rare .Here reported is a case of bilateral reexpansion pulmonary edema occurring after reconstruction of left sided post traumatic diaphragmatic hernia. The patient was re intubated and ventilated later put on supplemental oxygen via a catheter through the endotracheal tube followed by extubation and non re breather face mask to compensate for hypoxemia. He was given intravenous diuretics and inotropic support. Forty-eight hours after the acute event, the patient recovered with minimal residual hypoxemia. Reexpansion pulmonary edema after repair of missed diaphragmatic hernia is a rare complication with a high mortality rate of up to 20%. It should be considered in cases of hypoxemia following any chest reexpansion procedure. The exact pathophysiology leading to this complication is not clear but it is believed to be permeability pulmonary edema as a result of pulmonary micro vascular damage. Risk factors for reexpansion pulmonary edema should be evaluated and considered prior to insertion of chest tubes. Treatment is supportive and emphasis should be given for preventive measures.

Key Words: *Reexpansion Pulmonary Edema, Diaphragmatic Hernia*

INTRODUCTION

Re-expansion pulmonary edema (REPE) has been a known entity since the first reports during the late 1950s. REPE is a rare phenomenon which might result from any chronically collapsed lung that re-expands in a faster rate. However there is paucity of reports REPE following repair of missed Diaphragmatic Hernia. The associated high mortality rate the evolving understanding of it pathophysiology and the relative frequency of a physician dealing with re-expansion of the lungs in one way or another make it an important clinical state.

CASE REPORT

Here reported is a case of a 27-year old male patient who was referred to our hospital from a rural hospital with an initial diagnosis of post traumatic left sided Empyema Thoracic following a stab injury three weeks prior to his presentation to our hospital. An initial chest tube placed at the district hospital initially drained blood and later serosanguinous minimal fluid. On the second week the patient experienced vomiting of ingested matter of only one episode otherwise he had no abdominal symptoms. The patient showed some improvement but persisted to have dyspnea on exertion with left sided chest pain. After failure of radiographic resolution patient was referred to our hospital the chest drain removed after two weeks.

At admission, the vital signs of the patient were stable. He had decreases air entry on the left hemithorax posteriorly, otherwise he had no remarkable finding. He had undergone laparotomy with a single lumen intubation and naso-gastric tube in place. We found approximately a six centimeter defect at the dome of the left hemi diaphragm with part of the transverse colon and the omentum herniated through and adhered to the defect and the chest wall. The adhesions were released and the abdominal contents reduced. The left lung appeared compressed but showed some signs of expansion. A chest tube was left in place connected to a underwater seal system bottle and the diaphragmatic defect was closed with No. 2 Silk. The patient was stable throughout the operation and the procedure was uneventful.

He was extubated in the operation theater following standard procedures. After one hour of the procedure

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and while in the recovery room, the patient started to have dyspnea with frothy excessive secretions and tachypneia of 40-57 per minute. He had labored breathing with bilateral fine creptations on auscultation. His oxygen saturation dropped to 70%-75%.He was re-intubated and frequent suctioning done via the endotracheal tube. Intravenous Frusemide was given at 20 mg doses incrementally up to 80 mg.Since there was no mechanical ventilator, the patient was initially put on supplemental oxygen with catheter via the endotracheal tube and later ventilated using an anesthesia machine.

The right side creptations in the chest cleared soon following re-intubation and diuretic treatment, while the left side creptations persisted. His blood pressure (BP) dropped, ranging between 60/45 and 70/50 mmHg, and his pulse rate was between 112/min and 136/min. He was started on epinephrine drips at 0.1mic gram/kg /min. On the next day, the patient was extubated and put on non-re breather face mask. He maintained his oxygen saturation with respiratory rate coming down to 28/min, which normalized over the subsequent days. He only had minimal hypoxemia and tachypneia after 48 hrs. He was discharged improved with no residual hypoxemia after 7 days of treatment.

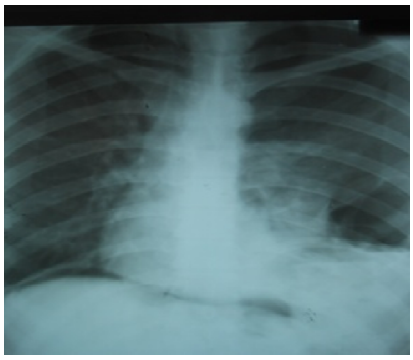

Figure1: The initial chest X-ray showing left sided diaphragmatic hernia

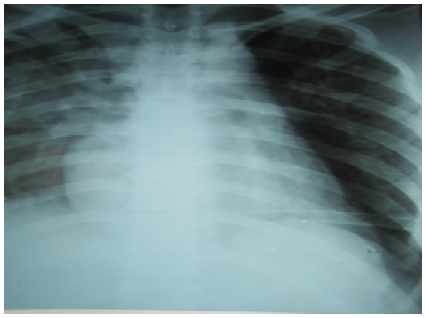

Figure 2: Post-operation chest-X-ray showing a transradiant left hemithorax& collapsed lower lobe

DISCUSSION

Penetrating diaphragmatic injuries usually result from stab or gunshot wounds to the lower chest inferior to the nipple line, upper abdomen, the flanks, or the back. There are no specific physical signs suggesting to diaphragmatic injury when there is no associated herniation. The acute tear of the diaphragm may become apparent only when problems related to the herniated abdominal viscera arise. More commonly, however, the defect is discovered incidentally when radiographic evaluations for unrelated conditions take place. The key to an early diagnosis is a high level suspicion. The presence of abdominal complaints in a patient who has sustained a chest wound is strongly suggestive of a diaphragmatic injury, and vice versa. Occasionally, a diaphragmatic rupture is entirely missed when there is no surgical exploration. This occurs most often with stab wounds. Those from penetrating trauma tend to contain only colon or a portion of the stomach (1).

Reexpansion pulmonary edema (REPE) following drainage of Pneumothorax was first described in 1958 by Carlson (2). Although a rare phenomenon, (only 60 cases described between 1958 and 1985) unilateral REPE is well defined complication of treatment for pneumothorax and pulmonary effusion (3,4). Even though post non-traumatic pneumothorax drainage REPE is the flagship disorder, there are reports of REPE occurring flowing decortications, drainage traumatic pneumothorax, drainage of giant hepatic cyst, excision of giant mediastinal masses and repair of missed diaphragmatic hernia (5-8). Based on these reports, it can reasonably be inferred that REPE can occur associated with re-expansion of a chronically collapsed lung. However, despite a wide range of reports on REPE associated with many predisposing clinical conditions, there is only one report on REPE following repair of missed diaphragmatic hernia (9) based on the author's review in the English literature.

REPE is a rapid process, developing within one hour of re-expansion in two-thirds of cases and within 24 hours in the remainder. It is unlikely to occur in lung collapses of less than 3 days duration (8).Although the process can be localized to the area around the collapsed lung, it can be generalized and life-threatening. The incidence is <1%, but some studies reported incidence of over 14% in those with larger pneumothoraces, collapse of longer duration, and in younger patients (< 30 years of age). It is more common in males with a ratio of 38:9.Most (94%) of the cases occur within the ipsilateral, collapsed lung and 64 % occur

within one hour of expansion of the lung. REPE has been seen bilaterally in up to 6.7% and contra-lateral lung in 0.3%. All cases seem to occur within 24 hours (8,9). Clinical severity ranges from asymptomatic radiologic abnormalities to severe cardiopulmonary insufficiency and death, with one clinical study reporting a death rate of 20%.Increased mortality is associated with bilaterality and sudden onset and age > 50 years (10).

Our current understanding of the pathophysiology of REPE is that it is likely a multifactorial process with the edema resulting from increased capillary permeability due to hypoxic and mechanical damage to the alveolar capillary membrane during lung compression. It has been shown that there is decreased blood flow to the compressed lung and this is thought to cause hypoxic damage to the endothelium of the capillary walls, leading to increased vascular permeability. Analysis of the edema fluid has shown a high ratio of protein to serum, as would be expected with increased permeability as opposed to hydrostatic edema.

In animal models, Jackson M, et al. (11) has shown free radical generation in lung tissue contributes to the pathogenesis of REPE, although re-initiation of lung perfusion and ventilation require a rapid change in intrathoracic pressure. Hypotension results from the shift of intravascular fluid to the alveolar space and expectoration of voluminous fluid is evidenced by the increment of the lungs' weight in post mortem examinations and estimated to be as much as three liters. Symptoms may vary from the completely asymptomatic patient with only radiographic findings to overt clinical presentation consisting of tachypneia, tachycardia, hypotension, cyanosis, pink frothy sputum production, cough, dyspnea and chest pain with decreased air entry and audible rales over the affected lung. Symptoms may last 24 – 48 hours and disappear in five to seven days or may evolve to be lethal (12).

Management of REPE is mainly supportive. The cornerstone in the management of REPE is positive-pressure mechanical ventilation and utilization of positive end-expiratory pressure (PEEP) to help re expand collapsed alveoli, increase functional residual capacity, and reduce shunting. While one is preparing for tracheal intubation patients may be tried using noninvasive ventilation, as good results are obtained even in serious cases, as a first line of action. Positioning the patient on lateral decubitus the affected side down is recommended which, in unilateral cases, contributes to reducing the pulmonary shunt and improving oxygenation. Asynchronous/ differential lung ventilation is said to be rarely necessary but in severe cases i.e. failure of mechanical ventilation might be a treatment alternative. The usefulness of manometry in measuring pleural pressures during Thoracentesis remains controversial.

Inotropic support should address the lower hemodynamic state of these patients. The use of diuretics is somewhat controversial while several authors s including review articles on the topic (3,4,8,14-19).has argued for diuretics as part of the management of REPE, some authors have suggested otherwise describing diuretics to be detrimental as they may compromise the already hypotensive state (19,20). In the author's opinion the fact that the pathophysiology of REPE is multi factorial, including sudden increase in hydrostatic pressure and that inotrops can offset the potential risk of diuretics judicial use of these medications may be imperative in the management of REPE.

Considering the significant degree of overall mortality of greater than 20 %, prevention should be the mainstay of management. One should be ready for endotracheal intubation and ventilation if a patient develops features of pulmonary edema following any decompression of the lungs. Cough, chest pain and shortness of breath with or without sputum production should raise suspicion of REPE and is an indicator to stop further drainage. There is no standard limit to the amount of drainage. Most authors recommend drainage of 1000- 5000 ml as the safe limit. Even though treatment of the histological changes of the pulmonary vasculature secondary to hypoxic and mechanical changes is the ideal treatment, supportive management remains the current mode of treatment. The proven capillary leak in the pathology beckons that steroids may a have a role in stabilizing the inflammatory response together with non-steroidal anti-inflammatory drugs (NSAIDs).

Our patient had a typical picture of missed post traumatic diaphragmatic hernia following a stab injury presenting with failure of resolution of radiologic findings. He had larger than average defect and contents (omentum and transverses colon), which is in line with the previous reports. In our case, the identifiable risk factors for REPE were young age, lung compression of greater than three days duration. Our patient had exhibited a classical pattern of post operative REPE, manifesting within one hour of the procedure, and a relatively rare state of bilateral involvement. He was managed with supportive measures with intubation, inotrops and diuretics.

Conclusion: REPE is a rare clinical entity with significant mortality. REPE may occur following nearly any type of re-expansion of a compressed lung. It is a type of permeability pulmonary edema. Physicians should be aware of the risk factors for REPE, Including long duration of compression, volume, and speed of de-compression. One should be familiar with the clinical pictures of REPE, i.e. most occur within one to two hours of re-expansion with dyspnea hypotension and cough. Almost all REPEs occurs within 24 hrs and it could be bilateral. Management is supportive with endotracheal intubation and diuretics and hemodynamic support with inotrops.

REFERENCES

1. Symbas PN, Vlasis SE, Hatcher CR Jr. Blunt and penetrating diaphragmatic injuries with or without herniation of organs into the chest. Ann. Thoracic Surg 1986;42:158.
2. Carlson RI, Classen KL, Gollan F, et al. Pulmonary edema fol-lowing the rapid expansion of a totally collapsed lung due to pneumothorax: A clinical and experimental study. Surg Forum1958;9:367-371.
3. Eduardo Henrique Genofre, Francisco s. Vargas, Lisete r. Teixeira, Marcelo Alexandre Costa Vaz, Evaldo Marchi. Re expansion pulmonary edema J Pneumol 2003;29(2):101-6)
4. Rasha Alqadi, MD, Valencia CF, Viscusi M, Latif S.Re-expansion pulmonary edema following thoracentesis. Rhode Island Medical Journal 2013;38- 40
5. Malota M, Kowarik M, Bechtold B, Kopp R. Re-expansion pulmonary edema following a posttraumatic pneumothorax: a case report and review of the literature. World Journal of Emergency Surgery 2011;6:321.
6. Matsumiya N, Dohi S, KimuraT, Naito H. Re-expansion pulmonary edema After Mediastinal Tumor Removal. Anesth Analg 1991;73:646-48.
7. SoharaY. Re-expansion pulmonary edema . Ann Thoracic CardivascSurg 2008;14(4):205-9.
8. Mahfood S, Hix WR, Aaron BL, Blaes P, Watson DC. Re-expansion pulmonary edema. *Ann Thorac Surg* 1988;45:340-5.
9. Inaba Kenji, Snider J, Holliday.Re-Expansion Pulmonary Edema After Repair Of A Missed Diaphragmatic Hernia Canadian Journal Of Surgery 2001; 44(4): 295-7
10. DuBose J, Perciballi J, Timmer S, Kujawaski EJ. Bilateral re-expansion pulmonary edema after treatment of spontaneous pneumothorax. Current Surg 2004;61:376-379.
11. Jackson R, Veal C, Alexander B, Brannen, FulmerJ. Re-expansion Pulmonary Edema: A Potential Role for Free Radicals in Its pathogenesis". Am Rev Resp Dis1988;137(5):1165-171
12. Mahajan VK, Simon M, Huber GL. Reexpansion pulmonary edema. Chest 1979;75:192-4.
13. Iqbal M, Multz A, Rossoff L, Lackner R. Re-expansion Pul-monary Edema After Vats Successfully Treated With Continuous Positive Airway Pressure. Ann Thorac Surg. 2000; 70:669-71.
14. Heller Bj, Grathwohl K. Contralateral Reexpansion Pulmonary Edema. South Med J 2000;93:828-31.
15. Sewel R, Fewel J, Grover F, Arom K. Experimental Evaluation of Re-expansion Pulmonary Edema. Ann Thorac Surg 1978;26:126-32.
16. Besalti1 O, Pekcan Z, Caliskan M, Aykut Z.A retrospective study on traumatic diaphragmatic hernias in cats. Ankara Üniv Vet Fak Derg 2011;58,175-79.
17. Malota M, Kowarik, Bechtold B. Kopp R. Re-expansion Pulmonary Edema Following A Posttraumatic Pneumothorax: A Case Report and Review of The Literature. World J EmergSurg 2011;6:32
18. Cho S, Lee J, Kim M. New Treatment Method for Re-expansion Pulmonary Edema: Differential Lung Ventilation. Ann Thorac Surg 2005,80:1933-1934.
19. Janmeja A,. Mohapatra P, Saini, KhuranaA; Re-expansion Pulmonary Edema- A Case Report. Lung India 2007;24:72-74
20. Light R. Thoracentesis and Pleural Biopsy. In Light R, Editor Pleural Disease. 4th Ed. Baltimore:Williams & Wilkins, 2003; 358-377.