CASE REPORT

Perioperative reexpansion pulmonary edema

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ABSTRACT

Reexpansion pulmonary edema is a rare form of acute lung injury following rapid re inflation of collapsed lung parenchyma. It can rarely be associated with anaesthesia and repair of traumatic diaphragmatic hernia.

We report a case of reexpansion pulmonary edema (REPE) occurring during repair of traumatic diaphragmatic hernia in an adult male. He was a victim of occupational trauma and presented to casualty with complaints of dyspnoea and left sided chest discomfort soon after the injury. Oxygen supplementation was administered via simple face mask to compensate for his hypoxemia. Chest radiograph revealed left diaphragmatic hernia and he was taken up for surgery. Under general anaesthesia with two lung ventilation and epidural analgesia via a thoraco-abdominal approach reduction of abdominal contents with diaphragmatic repair was undertaken. Intraoperatively within an hour of lung expansion he developed pulmonary edema. In spite of altered ventilation perfusion dynamics in the lateral decubitus position he was successfully treated on table. He recovered after a couple of days of invasive ventilatory support. Prompt diagnosis and treatment can reduce the lethality of this condition.

Key words: Reexpansion pulmonary edema; Traumatic diaphragmatic hernia; Chest.

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INTRODUCTION

Reexpansion pulmonary edema is a rare (1%) form of acute lung injury following rapid re inflation of collapsed lung parenchyma[1]. Though it is a well described complication following drainage of pleural effusion and pneumothorax it has very rarely been reported in association with anaesthesia and repair of traumatic diaphragmatic hernia[2]. Nevertheless reexpansion pulmonary edema is frequently fatal and the most effective clinical approach is prevention[3]. Herein we report a case of re expansion pulmonary edema occurring during repair of traumatic diaphragmatic hernia in an adult male.

CASE REPORT

A 30 year old male with history of fall from height while working, presented to the emergency department within an hour, complaining of shortness of breath and left sided chest pain. His medical history was unremarkable. Physical examination was normal except for a respiratory rate of 28/min and pulse oximetry (SpO_2) reading of 85% on ambient air. Chest examination showed decreased expansion, hyper resonance and decreased air entry to the left side of the chest. Abdomen was soft and non tender. His laboratory tests, ECG and echocardiography were normal. Chest X ray showed the stomach in the left hemithorax (figure 1). Computerised tomography of chest (figure 2) confirmed the diagnosis of diaphragmatic hernia. He was administered oxygen, 6 litres per minute, via simple face mask and maintained a SpO_2 of 95 %. He was kept nil by mouth, Ryle's tube was inserted and aspirated regularly. He consented for surgery only on the 4th day. The case was accepted under ASA physical status 2.

Reduction of abdominal contents with diaphragmatic repair was planned under general anaesthesia with two lung ventilation and epidural analgesia. Inj ranitidine and inj metaclopramide were given intravenously an hour before surgery. The patient was pre medicated with inj glycopyrrolate 0.01 mg/kg intravenously. 18 gauge epidural catheter was inserted through T9-T10 inter vertebral space with patient in sitting position. Anaesthesia was induced in a rapid sequence order with inj thiopentone 5 mg/kg and inj pentazocine 0.3 mg/kg intravenously followed by cricoid pressure. Endotracheal intubation was facilitated with inj

succinylcholine 1.5 mg/kg intravenously. Single lumen endotracheal tube of 8.5 mm internal diameter was used. Patient was then placed in right lateral decubitus position. Anaesthesia was maintained using 1 MAC halothane in N_2O and O_2. Epidural analgesia was provided using 0.25% bupivacaine and morphine. Muscle relaxation was maintained using inj. vecuronium. The following parameters were continuously monitored; electrocardiogram (ECG), end tidal CO_2, SpO_2, noninvasive blood pressure and urine output.

The abdominal contents were reduced. The diaphragmatic tear was about 10 cm long and was repaired using polypropylene mesh. Intercostal tube was then inserted and the ribs were approximated. The lung was then expanded applying Valsalva manoeuvre through Bain's circuit. While the thoraco-abdominal closure was underway, pink frothy secretion started gushing out from the endotracheal tube. Auscultation of the chest revealed bilateral crepitations, extensive on the left. The SpO_2 ebbed down to 60%, end tidal CO_2 was 25 mm of Hg. Blood pressure and pulse rate were 150/90 mmHg and 98/min respectively. A diagnosis of pulmonary edema was made and the speculated causes were REPE, myocardial infarction, fluid overload or left ventricular failure due to arrhythmias or cardiac tamponade . The ECG was normal and our intravenous fluid management was judicious, thus ruling out cardiac causes and fluid overload and pointing out re expansion pulmonary edema. Inj furosemide 80 mg intravenously and inj morphine 6 mg intravenously were given. Surgical closure was hastened and patient was then turned to supine position. The left subclavian vein was cannulated and the central venous pressure measured was 6cm of water. SpO_2 was improving by then and the patient was transferred to the intensive care unit.

Volume controlled ventilation with positive end expiratory pressure of 10 mmHg was provided. Portable chest x-ray was sought but it was not available to us. Edema fluid collected from the side stream of capnometer chamber was analysed. It revealed a high protein to serum ratio (0.8) thus confirming REPE. Ventilation was continued till the lung fields were radiographically clear (Figure 3) and clinical signs of pulmonary edema subsided. Weaning and extubation were successfully done 36 hours after the surgery. Chest tube was removed after a week and he was discharged home on the 10th day.

DISCUSSION

The history of REPE is quite intriguing. The first reference to REPE was made by Pinault in 1853 following the removal of three litres of pleural liquid during pleurocentesis. The first well-documented report of REPE was presented by Foucart in 1875.[3] A case of REPE after TDH repair was reported as early as 1995 by Okuda et al[4] wherein acute ipsilateral pulmonary edema arose from the inflation of the collapsed lung with the application of positive pressure ventilation. In this case, immediately after the abdomen was opened and the stomach was withdrawn into the abdominal cavity, the SpO_2 fell and a large amount of yellowish fluid came out of the endotracheal tube. Postoperatively the patient improved after twenty four hours with the administration of oxygen, steroids, albumin and the application of positive end expiratory pressure under mechanical ventilation.

REPE may occur more commonly unilaterally (93%) and sometimes bilaterally (6.7%) or contra-laterally (1%) after rapid re expansion of collapsed lung.[5] The pathophysiology of REPE is complex and the phenomenon is multifactorial. Mechanical distress on the alveoli and oxygen radicals produced during re expansion of lung are primary factors leading to increased capillary permeability.[3] Reduction of surfactant in collapsed lung and alteration of blood flow following re expansion[6] are contributing factors. Moreover, the activity of different cytokines such as IL8 and monocyte chemoattractant protein 1, nitric oxide and xanthine oxidase have also been implicated in the pathogenesis of REPE.[1]

In literature, several risk factors have been associated with REPE - Younger age (<40 years), longer duration of lung collapse(>3 days), large space occupying lesions (>30% of single lung) and timing of lung re expansion.[5,7,8] Speed and volume of re expansion were important modifiable factors for REPE to occur.[9] Our patient had all these risk factors. A normal ECG and CVP helped us in ruling out cardiogenic pulmonary edema thereby avoiding disastrous consequences. REPE may also be related to excessive suction of tracheobronchial tree,[10] pulmonary hypertension[3] and decreased pulmonary lymphatic flow[1] but all these were not present in our case.

REPE is a noncardiogenic pulmonary edema with the edema fluid demonstrating a high protein index[11] as seen in our case. REPE commonly (64%) occurs within an hour of expansion[5] which was also another finding in our case.

Several strategies proposed in the literature, trying to reduce REPE morbidity and mortality, including occlusion of affected side pulmonary arteries with a balloon catheter[6,7] and high frequency jet ventilation,[12] have been advocated in resistant cases but were not needed in our patient because the pulmonary edema responded to routine management.

Use of double lumen tube and one lung ventilation mentions controversy. It is suggested that one lung ventilation during anaesthesia may alter the distribution of blood flow between the non dependent and dependent lung potentially predisposing to REPE.[13] On the contrary the use of double lumen tube from the outset of the procedure and continuation postoperatively upto 24 hours

after re expansion is supposed to protect the contralateral lung from spillage and reduces hypoxia.[2] However, double lumen tube can be used to provide differential lung ventilation for treating REPE.[14]

Thoracic epidural analgesia was used in our case to improve postoperative recovery. Although thoracic epidural blockade has got favourable effects in other forms of noncardiogenic pulmonary edema,[15] its role in REPE is yet to be elucidated. Nonetheless, in our case REPE developed despite using thoracic epidural blockade. This emphasises the role of oxygen derived free radicals rather than neurogenic factors as the major pathology in REPE.

CONCLUSION

REPE is a rare but severe complication and can occur after the rarer disease, traumatic diaphragmatic hernia, with the rarest encounter during anaesthesia. The clinical importance is ascribed to the high lethality in this condition. Avoiding modifiable risk factors greatly helps in reducing morbidity. Treatment is symptomatic but outcome is a matter of time.

Conflicts of interest: Nil

Informed consent from patient: Obtained.

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