Case Report

Delayed traumatic diaphragmatic hernia mimicking hydropneumothorax

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ABSTRACT

Traumatic diaphragmatic hernia (TDH) is generally a consequence of thoraco-abdominal trauma. Anaesthetic problems arise due to herniation of abdominal contents into the thoracic cavity causing diaphragmatic dysfunction, lung collapse, mediastinal shift and haemodynamic instability. Diagnosis depends on history, clinical signs and radiological investigations. Sometimes, it may be misdiagnosed as hydropneumothorax due to the presence of air and fluid in the viscera lying in the pleural cavity. We report a case of TDH mimicking hydropneumothorax on radiological investigations and subsequent surgical management, which led to serious complications.

Key words: Hydropneumothorax, thoraco-abdominal trauma, traumatic diaphragmatic hernia

INTRODUCTION

Thoraco-abdominal trauma may cause traumatic diaphragmatic hernia (TDH). Diagnosis of TDH is a challenge especially in cases of blunt trauma and requires a high index of suspicion. Diagnosis is often delayed because of co-existent prominent features of haemodynamic instability, respiratory insufficiency, gastrointestinal symptoms and cardiac arrest following trauma, which divert attention away from diaphragmatic injury. We report a case of delayed TDH following a fall from height mimicking as a case of hydropneumothorax.

CASE REPORT

A 45-year-old female presented with a history of fall from 18 feet height three weeks ago and difficulty in breathing for last 2 days. She had a past history of completely treated pulmonary tuberculosis 10 years back.

The patient was conscious, oriented with a respiratory rate 30/min, heart rate 120/min and blood pressure 112/68 mm Hg. Air entry was reduced on the left side of the chest. Abdominal examination revealed tenderness in the epigastrium without guarding or rigidity. Bowel sounds were present all over the abdomen.

X-ray chest posteroanterior view [Figure 1] revealed large air-fluid level in left hemithorax with collapsed underlying lung and obliteration of cardiophrenic angle with rightwards shift of the mediastinum. It also showed infiltrates and scarring in upper zones bilaterally. Plain computerised tomography (CT) scan chest [Figure 2] also revealed hydropneumothorax and lung collapse. Ultrasonography of abdomen, obtained 7 days before admission, did not reveal any evidence of solid organ injury or free fluid in the abdominal cavity.

A provisional diagnosis of traumatic hydropneumothorax was made on the basis of chest X-ray and CT scan. An intercostal drain tube (ICT) was inserted in left 5th intercostal space in the midaxillary line through which 50 ml seropurulent fluid drained out. After few hours, gastric contents appeared in the intercostal drainage system. Nasogastric tube (Ryle's tube) was inserted and repeat chest X-ray was done which revealed the presence of Ryle's tube in the area of left hydropneumothorax [Figure 3]. Diagnosis was revised as TDH with iatrogenic perforation of stomach

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due to ICT insertion. Surgical repair was planned under general anaesthesia.

On preoperative evaluation, patient was tachypnoeic with respiratory rate 36/min and SpO_2: 92% on oxygen by face mask (FiO_2 0.4). Heart rate was 118/min and blood pressure was 130/78 mm Hg. Blood gas analysis showed PaO_2 64 mm Hg and $PaCO_2$ 39 mm Hg with pH 7.38. Other investigations were within normal physiological limits.

In the operation theatre monitoring for her vital parameters with electrocardiogram, non-invasive blood pressure and SpO_2 was instituted using Collin 306 monitor. Rapid sequence induction was performed with injection thiopentone 250 mg and succinylcholine 100 mg intravenously (i.v.). The trachea was intubated with oral cuffed endotracheal tube size 7.5 mm ID and intermittent positive pressure ventilation (IPPV) was initiated. Anaesthesia was maintained with O_2, sevoflurane 2-4% and i.v. atracurium bolus and top ups. Thoracic epidural catheter was placed at T9-T10 level and injection bupivacaine 0.25% 8 ml was administered once the test dose response was negative. On surgical exploration of the abdomen, a large diaphragmatic rent of 6 cm × 5 cm was detected on the left side with stomach and spleen herniating into the thoracic cavity. Both viscera were reduced into the abdominal cavity and tear was repaired. A fresh ICT was inserted and a feeding gastrostomy was performed. The patient remained stable intraoperatively and after surgery she was shifted to intensive care unit (ICU) with endotracheal tube *in situ* for further management.

Mechanical ventilation of the patient was continued in ICU by using ventilator Vela (M/S Viasys - USA). Still, she had reduced air entry on the left side of the chest. In ICU, chest X-ray revealed extensive infiltrates in the left lung [Figure 4] and arterial blood gases (ABG) showed hypercarbia ($PaCO_2$ 58 mm Hg).

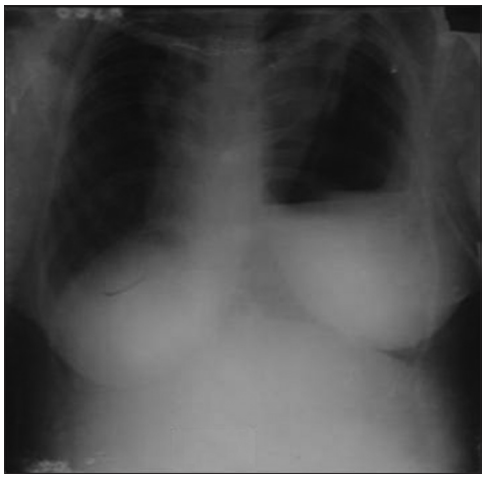

Figure 1: X-ray chest posteroanterior view resembling a picture of hydropneumothorax

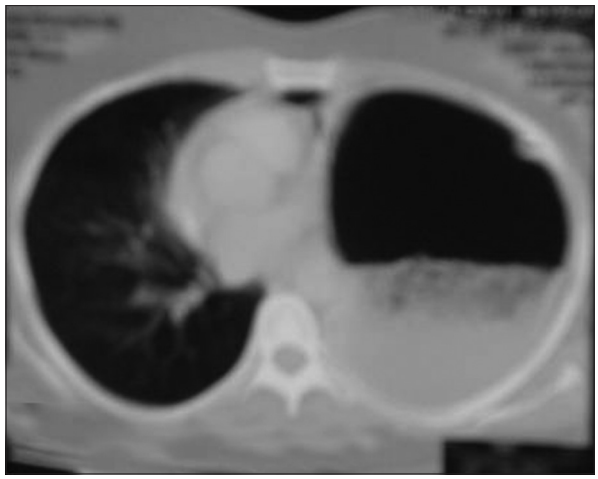

Figure 2: Enlarged view of single slice of computerised tomography scan of chest reported as large left hydropneumothorax with collapsed lung

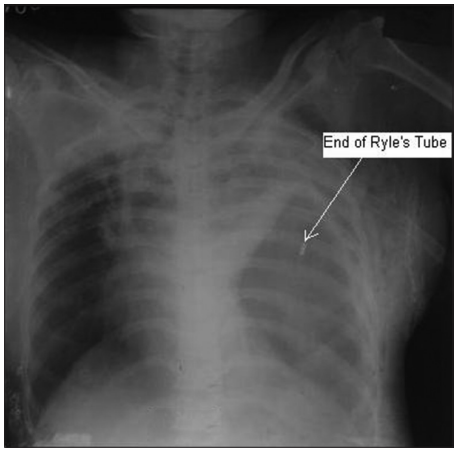

Figure 3: X-ray chest posteroanterior view showing end of Ryle's tube in left thorax

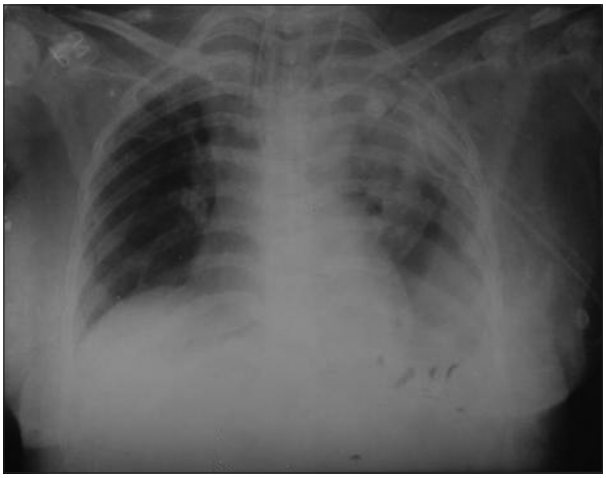

Figure 4: X-ray chest posteroanterior view with infiltration of left lung

Culture and sensitivity of pleural fluid showed growth of enterobacter species sensitive to meropenem and amikacin. Early weaning from the ventilator could not be done due to chest complications. She was managed on the ventilator in ICU and ABG revealed PaO_2 55 mm Hg and $PaCO_2$ 58 mm Hg with FiO_2 0.4, tidal volume 400 ml and respiratory rate 12. Subsequently tracheostomy was performed on 14th day in view of prolonged ventilation. Nutrition was maintained by enteral feeds through gastrostomy. Later on, patient was put on minimal ventilatory support (continuous positive airway pressure/adaptive servo-ventilation). In the course of events patient had high grade fever, leucocytosis and tachycardia indicating systemic inflammatory response syndrome. She responded to the therapy gradually, infection was under control, left lung expanded, gases exchange improved with haemodynamic stability. Her ICT was removed on 28th day. On the 30th postoperative day, she was transferred to the surgical department with tracheostomy and gastrostomy for further management.

DISCUSSION

The incidence of TDH caused by blunt thoraco-abdominal trauma is less frequent (5%) as compared to penetrating injury (10-19%).[1-4] The forces transmitted to the abdomen by blunt trauma can create the pressure gradient across the diaphragm up to 1000 cm H_2O, which may be sufficient to cause diaphragmatic injury and hence hernia. Sometimes diaphragmatic traumatic injury remains unnoticed and after repeated increased intra-abdominal pressure due to coughing, straining or constipation the weakened fibres get stretched out resulting in a diaphragmatic rent leading to delayed TDH.

The left hemidiaphragm is more prone to be ruptured as it is unprotected when compared to the right which is protected by the liver. The most common organ to be herniated is the colon.[5] Other common organs involved are the stomach, omentum, small bowel, spleen and liver.[5,6]

Diaphragmatic hernia induces respiratory and circulatory dysfunction due to herniation of abdominal contents in the pleural cavity compressing the lung and mediastinum. It causes collapse of the lung, intrapulmonary shunting and mediastinal shift. This cardiopulmonary embarrassment is further augmented by pulmonary contusion or acute blood loss.[6] TDH may be associated with pericardial tamponade due to herniation of bowel into the pericardial sac and/or accumulation of fluid into the pericardium.[7] Rarely, perforation of herniated organs may lead to pneumothorax and pneumomediastinum.[8] Mediastinal shift and deviation of the trachea to the opposite side may cause a difficult intubation.[5]

Positive pressure ventilation may be advantageous in patients of TDH to expand the atelectatic lung, increase functional residual capacity and deliver higher concentration of oxygen if needed after reducing hernia. Large tidal volume and high airway pressures are not recommended because of the risk of barotrauma to the non-affected lung, decreased venous return and cardiac output and hence blood pressure. Nitrous oxide is to be avoided because of risk of increasing the mediastinal shift due to expansion of air filled bowel.[6] Loehning *et al*. have recommended either spontaneous ventilation or IPPV with low airway pressure after induction until reduction of hernia is imminent.[9]

Our patient was initially misdiagnosed as a case of traumatic hydropneumothorax on the basis of chest X-ray and plain CT scan. ICT insertion perforated the stomach, which soiled the pleural cavity, resulted in empyema, septicemia and other complications and hence increased morbidity and ICU stay with gastrostomy and tracheostomy. Radiological investigations have their own limitations. If the chest roentgenogram displays multiple air fluid levels in hemithorax indicating presence of bowel loops in the chest, the diagnosis of diaphragmatic hernia is definite. However in the case of gastric herniation in left hemithorax, a single air fluid level is observed, thus making differentiation between hydropneumothorax and diaphragmatic hernia difficult after thoraco-abdominal trauma. However diaphragmatic injury may be missed even on exploratory laparotomy, still intra-operative identification is the gold standard for diagnosis of traumatic diaphragmatic injury.[10,11] Initial chest roentgenogram will detect only 23-73% of diaphragmatic ruptures, leaving the rest of the cases inconclusive.[10] In our case, differentiation between TDH and hydropneumothorax would have been possible if nasogastric tube was inserted prior to chest roentgenogram. In fact, CT scan is also found to have a sensitivity of 0-50% similar to that of chest X-ray. Multi-detector CT (MDCT) would have been a better choice to make a correct diagnosis, a rapid and highly accurate modality for detecting TDH.[12] Unfortunately, it is not available in our institution.

Poor condition of patient necessitated emergency laparotomy. In our patient, Ryle's tube was inserted after the induced perforation of the stomach in the pleural cavity with ICT. Subsequent X-ray chest revealed the presence of tip of Ryle's tube in thorax confirming the diagnosis of TDH. Ryle's tube should have been inserted before radiological investigations.

CONCLUSION

TDH caused by thoraco-abdominal blunt trauma is a diagnostic and therapeutic challenge. It may be observed as delayed phenomenon following trauma. Radiological investigations must be meticulously interpreted, correlated with history of presentation and clinical findings. Simple manoeuvres like insertion of a nasogastric tube before radiological investigations would be a good tool for aiding correct diagnosis. MDCT would be a better tool than plain CT and chest X-ray in diagnosing TDH.

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