

World Journal of Pharmaceutical research

Volume 3, Issue 2, 3201-3206.

Research Article

ISSN 2277 - 7105

ANAESTHETIC MANAGEMENT OF A PATIENT WITH DIAPHRAGMATIC RUPTURE FOR EMERGENCY SURGICAL REPAIR- A CASE REPORT

Dr Geeta Ahlawat, *Dr Mangal Ahlawat, Dr Nikita Jain

Department of anaesthesia, Pt B D Sharma PGIMS, Rohtak.

Article Received on 05 January 2014,

Revised on 27 January 2014, Accepted on 28 February 2014

*Correspondence for Author
Dr. Mangal Ahlawat
Department of anaesthesia, Pt
B D Sharma PGIMS, Rohtak,
India.

ABSTRACT

Traumatic diaphragmatic injury (DI) is a unique clinical entity that is usually occult and can easily be missed. An emergency laparotomy and thorough exploration followed by the repair of the defect is the gold standard for the management of these cases. Traumatic diaphragmatic injury can lead to herniation of abdominal contents into thorax and present a challange to anaesthesiologist. We report a case with diaphragmatic injury coming for emergency laparotomy.

Keywords: diaphragmatic injury, laparotomy, anaesthesiologist.

INTRODUCTION

Diaphragmatic rupture (also called diaphragmatic injury or tear) is a tear of the diaphragm, the muscle across the bottom of the ribcage that plays a crucial role in respiration. Diaphragmatic rupture is a life-threatening condition and can result from blunt or penetrating trauma and occurs in about 5% of cases of severe blunt trauma to the trunk. When a tear is discovered, surgery is needed to repair it. Since the pressure is higher in the abdominal cavity than the chest cavity, rupture of the diaphragm is almost always associated with herniation of abdominal organs into the chest cavity, called traumatic diaphragmatic hernia. and produce a challenge to anaesthesiologist This herniation can interfere with breathing, and blood supply can be cut off to organs that herniate through the diaphragm, damaging them. Accompanying electrolyte disturbances and shift of the mediastinum from distended viscera may compromise ventilation and circulation during induction of anesthesia. Regurgitation and aspiration are additional serious complications. We present a case with diaphragmatic injury coming for emergency laparotomy.

CASE REPORT

24 year male came to accident and emergency with history of road side accident with a truck and was taken up for emergency laparotomy in view of diaphragmatic perforation. On pre anaesthetic check-up, patient had history of loss of consciousness for few minutes with no history of head injury, vomiting, seizure or ent bleed. History of difficulty in breathing since the accident was present for which intercostal drain (ICT) was put on left side. No other significant past, personal or family history was present. He was adequately fasted as per ASA guidelines. On admission patient was in hypovolaemic shock and was resuscitated with intravenous fluids.

On examination patient was conscious, oriented to time place and person and cooperative. He was heavy built and moderately nourished. His pulse rate was 136 per minute regular, blood pressure -136/90 mm hg, respiratory rate 36 breaths per minute. On respiratory examination, tenderness was present on left side and air entry was markedly decreased and gurgling sounds was heard in inframammary area. Bilateral coarse crepitations were present. I.C Drainage was present with column movement. Around 100 ml of haemmorragic fluid was present in the drain. Abdomen was distended with tenderness present in whole of abdomen. Guarding was present. Abdominal organs could not be palpated. Cardiovascular and neurological examination was normal. On airway examination, patient was MPG grade III with bruises present over lips and left cheek. Inter incisor gap was around three fingers. Thyromental distance was 6.5 cm and neck and jaw movement was adequate. Ryles tube was present with 50 ml of fluid present in drain, billous in colour. Patient was catheterized with input-output of + 200 ml.

His haemoglobin was 10.8 g %, Bleeding time 2.38 mins, clotting time 5.45 mins, Total leucocyte count was 12000, Differential leucocyte count was 75/18/5/2, Platelet count- 2.2 Lac / cubic mm, Blood urea -42g, blood sugar- 98 mg%, serum sodium- 144 meq, serum potassium- 3.8 meq, ECG- sinus tachycardia, BGA- ph-7.24, po₂- 52, pco₂- 54, hco₃-28.2, with so₂ – 78%. Chest x ray showed elevated left lobe of diaphragm with bowel loops seen with air fluid level in left lobe of chest. trachea was pushed toward right and ict drain tubing was seen on left.

Patient was immediately shifted to operation theatre for emergency laparotomy and monitors for ECG, Spo₂, NIBP attached. Pre-op vitals recorded and two large bore i.v line started with 16 G, 18 G cannula with ringer lactate solution. Patient was pre-oxygenated for 3 minutes,

General anaesthesia was induced using I.V thiopentone 5mgkg-1, then adequacy of mask ventilation assessed and endotracheal intubation was performed after administering succinylcholine 1.5 mgkg-1 using cricoid pressure. Patient was maintained on controlled ventilation using 100% oxygen and a muscle relaxant. Anaesthesia was maintained with 0.5% halothane and oxygen 33% with nitrous oxide 67%, injection vecuronium bromide as required. Injection hydrocortisone 100 mg was given. Arterial line was secured in left radial artery for sampling. And central venous line triple lumen was inserted in right internal jugular vein and secured. Intraoperatively fluid was maitained according to cvp and blood loss. A midline laparotomy showed a large defect of the left hemidiaphragm. The left hemithorax was decompressed by returning the viscera to the abdominal cavity Intraoperatively after repairing diaphragm, his saturation improved to 100 % and chest was comparatively clear. Intraoperative BGA showed respiratory acidosis with so₂ 88% and po₂ 64. Intraoperative vitals were within range and urine output was adequate. In ondansetron 4 mg was given at the end of procedure. Smooth reversal was done with inj neostigmine and inj glycopyrolate after adequate respiratory efforts. Post operative course was uneventful. Post operative BGA showed compensated respiratory acidosis with mild hypoxia. Adequate post operative analgesia was given and nebulization was done with budecort and duolin alternatively. Patient was advised chest physiotherapy post operatively. Oxygen @ fio₂ 0.6 was given with propped up position. Post op BGA improved gradually and chest drain was removed on day 3 post op. Post op chest x ray showed normal lung fields with small lobulated pleural effusion which too disappeared. Respiratory distress gradually improved with improvement in exercise tolerance. Pt's post-operative period was uneventful. Patient was discharged on day 8 in stable condition.







DISCUSSION

Blunt injury to the diaphragm occur in approximately 1% of all trauma adminssions. The diaphragm is most commonly injured by a direct blow to the abdomen, causing a sudden increase in intra-abdominal pressure, or by direct laceration from rib fractures. Rupture of the diaphragm rarely occurs in isolation, and associated injuries to the thoracic aorta, liver & spleen and pelvis are often present.

Diagnosis is often difficult or missed, as the above case shows. A high index of suspicion is vital. Chest X-ray may reveal the injury if abdominal contents have herniated into the chest, may reveal a thickening or fuzziness of the diaphragmatic outline, an elevated hemidiaphragm or be completely normal - especially if the patient is intubated & ventilated. Haemopneumothoraces are a common associated finding. Overall the plain Chest X-ray has a 50% accurracy.

Diaphragmatic injury may be detected on ultrasound - but rarely. CT scanning has a sensitivity of only around 70-80%. MRI may be the best non-invasive examination for blunt diaphragmatic injury. Laparoscopy or thoracoscopy have become the primary means of

diagnosing diaphragmatic injury - though they are more frequently employed for penetrating trauma. As with CT and MRI scanning, the patient must be haemodynamically stable for these procedures. Delay in detecting and repairing diaphragmatic injury increased both morbidity and mortality. Operative repair is also technically more difficult, the longer the delay to surgery.

Large-bore i.v. access should be gained and fluid resuscitation commenced. Invasive arterial and central venous pressure monitoring should be considered, but if the patient is in extremis, these should not delay commencement of surgery. The patient is at high risk of aspiration because of gastrointestinal obstruction, and therefore antacid premedication should be given, and a nasogastric tube should be inserted and aspirated if possible before rapid sequence induction with cricoid pressure. If difficult intubation is anticipated, awake fibreoptic intubation or tracheostomy under local anaesthesia should be considered. If the patient is haemodynamically unstable, induction should be in theatre with the surgeon ready to operate immediately. Agents less likely to decrease MAP (etomidate and fentanyl) should be used. Expansion of the viscera is likely to worsen the mass effect and impair circulation and respiration. Face-mask ventilation, with potential gastric insufflation; and nitrous oxide anaesthesia should therefore be avoided. In theory, positive-pressure ventilation might preferentially ventilate the normal lung rather than the collapsed lung. However, any re-expansion of the collapsed lung may exacerbate the mass effect, with rapid and disastrous worsening of the circulation. The collapsed lung should therefore be isolated and ventilation of the normal lung started with small tidal volumes and pressures, using a double-lumen tube, until the affected hemithorax has been decompressed. If this is not possible, a single-lumen tracheal tube with a bronchial blocker should be considered.

In most cases, isolated diaphragmatic rupture is associated with good outcome if it is surgically repaired. The death rate (mortality) for diaphragmatic rupture after blunt and penetrating trauma is estimated to be 15–40% and 10–30% respectively, but other injuries play a large role in determining outcome.

CONCLUSION

Traumatic injuries of the diaphragm are often clinically occult and can be masked and disguised by other violent injuries associated with polytrauma. The best approach is the high index of suspicion in such cases. Chest X-ray is the initial screening option followed by spiral

CT (preferably with multidetector rows) to evaluate the diaphragm. Optimal treatment of DI consists of early repair on laparotomy with careful evaluation of other associated violent injuries; however, with an increase in experience and expertise, laparoscopy and thoracoscopy, especially VATS, are finding their places in both diagnosis and definitive management of thoracic trauma with occult diaphragmatic injuries.

REFERENCES

- 1. Dwivedi S, Banode P, Gharde P, Bhatt M, Johrapurkar S R. Treating traumatic injuries of diaphragm. J Emerg Trauma Shock. 2010 Apr-Jun; 3(2): 173–176.
- 2. Petrone P, Leppaniemi A, Inaba K, Soreid e K, Asensio JA. Diaphragmatic injuries: challenges in the diagnosis and management. Trauma. 2007;9:227–36.
- 3. Athanassiadia K, Kalavrouziotis G, Athanassiou M, Vernikos P, Skrekas G, Poultsidi A, et al. Blunt diaphragmatic rupture. Eur J Cardiothorac Surg.1999;15:469–74.
- 4. Bergin D, Ennis R, Keogh C, Fenlon HM, Murray JG. The "dependent viscera" sign in CT diagnosis of blunt traumatic diaphragmatic rupture. AJR Am J Roentgenol. 2001;177:1137–40.
- 5. Meyers BF, McCabe CJ. Traumatic diaphragmatic hernia. Occult marker of serious injury. Ann Surg. 1993;218:783–90.
- 6. Yilmaz M, Isik B, Ara C, Yilmaz S, Kutlu R, Kocak O, et al. Gastric perforation during chest tube placement for acute diaphragmatic rupture and review of the literature. Injury Extra.2006;37:71–5.
- 7. Calhoon JH, Grover FL, Trinkle JK. Chest trauma; approach and management. Clin Chest Med. 1992;13:55–67.