Original Research Article

Anaesthetic Management of Anaphylactic Shock Caused by ruptured Hydatid Cyst of the Liver: A case report

ABSTRACT

Hepatic hydatid disease is very common in developing nations. Although anaphylactic shock is a rare complication during anaesthesia, it can present with severe cardiovascular shock and hypersensitive reactions during surgical removal of hydatid cyst, requiring prompt emergency management. We report a case of 43 year old female posted for elective removal of hydatid cyst in the right lobe of liver under general anaesthesia. An anaesthesist should be aware of the "tell-tale" signs of anaphylaxis and be prepared for early and prompt treatment of anaphylactic shock. Anaphylactic prophylaxis and close monitoring for early diagnosis and prompt treatment of anaphylaxis are essential to avert any complications associated with anaphylactic shock.

KEYWORDS: Hydatid cyst, anaphylactic shock, anaesthesia.

INTRODUCTION

Hepatic hydatid cyst caused by "Echinococcus granulosus" is endemic in several developing countries and is a common health problem [1,2]. Anaphylaxis during anaesthesia is a rare phenomenon. It usually presents as mild hypersensitive reaction like urticaria or life-threatening circulatory shock [3].The number of incidences of this complication vary between 1 in 5000 and 1 in 20,000 [4] surgical procedures with a mortality rate of 3% to 6% [5]. All anaesthetic drugs especially muscle relaxants, inhalational agents, hypnotics, opioids, colloids, antibiotics and latex are most often involved in anaphylaxis [6]. We report a case of anaphylactic shock during hydatid cyst removal surgery under general anaesthesia.

PATIENT AND OBSERVATION

PATIENT INFORMATION:

A 43 year old female was posted for elective surgery for three well defined multilocular cystic lesions of sizes 8.8 ×7.9 cm, 7×5.8cm and 5.5×5.4 cm located in segment V, VII and VIII in the right lobe of liver diagnosed by computed tomography. Peripheral calcifications are noted within the lesions. Patient has minimal ascites with bilateral pleural effusion. She gives a history of pulmonary tuberculosis 8 years back for which she completed an anti-tubercular therapy (ATT) course. There was no other significant medical or surgical history.

CLINICAL FINDINGS: On general examination, patient has a height of 156 cm and weighing 52 kg with a body mass index of 21.3 kg/m^2. Her blood pressure was 108/78 mmHg and heart rate was 100 beats/min. Palor, icterus and bilateral pedal edema were present. No clubbing or cyanosis were present. On airway assessment, patient had an adequate mouth opening of three fingers with Mallampatti class 2 and neck and temporomandibular joint movement are adequate. All teeth were present. Cardiovascular, respiratory and central nervous systems were normal on systemic examination. The abdomen was distended, tender

on palpation and bowel sounds were heard. Chest X-ray showed bilateral effusion. Both conjugated and unconjugated bilirubin values were raised. All the rests of laboratory tests including complete blood count, alanine transaminase, aspartate transaminase, prothrombin time, international normalized ratio, urea, creatinine, sodium, potassium and random blood sugar were normal. Electrocardiography was unremarkable

TIMELINE OF CURRENT EPISODE-

Patient was apparently well 3 months back when he developed spikes of fever associated with vomiting and gradual onset of jaundice which progressed over the past 3 months and was eventually admitted. She was diagnosed with multiple hydatid cyst and was planned for elective removal of hydatid cyst on 03/12/2021.

On the day of surgery, an 18 G iv cannula was secured on the right arm and was patient was adequately preloaded. Premedication of glycopyrrolate (0.02 mg/kg) was given after which patient was shifted to operative room and standard monitors including ECG, non-invasive blood pressure cuff and oxygen saturation probe were attached. An intravenous 1 g of ceftriaxone was administered without any reactions or hemodynamic changes. The baseline parameters of the patient were a heart rate 88 beats /minute, NIBP 134/86 mmHg and SpO_2 99%. Patient was preoxygenated with 100 % oxygen for 4 minutes and induced with intravenous propofol (2 mg/kg), fentanyl (2µg/ kg) and vecuronium (0.1mg/kg). Tracheal intubation was done with a normal endotracheal tube following laryngoscopy. Anaesthesia was maintained with vecuronium, sevoflurane and a mixture of nitrous oxide and oxygen (50%:50%). A tidal volume of 420 mL and respiratory rate of 14 breaths /minute was maintained. SpO_2 was 99%, capnography ($ETCO_2$) was 34-38 mmHg and peak airway pressure (PAwP) was 20 cmH_2O. A bolus of fentanyl (50 µg) was given at time of surgical incision. Injection hydrocortisone 100 mg was given intravenously before commencement of surgery. 10 minutes after the opening of the cyst, patient went into hypotension (NIBP: 58/36 mmHg) and tachycardia (HR 118 beats/min).

DIAGNOSTIC ASSESSMENT

Surgeon was asked to halt the procedure. Inhalational agent (sevoflurane) was immediately stopped and 100% oxygen. $EtCO_2$ (32 mmHg) and airway pressure (20 cmH_2O) were normal. Chest was clear on auscultation with bilateral air entry and there was no wheeze. No changes were noted in ECG and there was no significant blood loss (approximately ≈200 mL). A provisional diagnosis of anaphylactic shock was made.

THERAPEUTIC INTERVENTION

A bolus of injection hydrocortisone 100 mg and injection pheniramine maleate 45.2 mg was administered. A central line catheter was secured in the right subclavian vein and fluid resuscitation was started with ringer's lactate. Intravenous mephentermine (30 mg) was put in the drip, but the NIBP showed minimal improvement of 62/45 mmHg. Intravenous norepinephrine infusion (12 mcg/ min) was started via the central venous catheter.

FOLLOW UP AND OUTCOME OF INTERVENTION

The hemodynamic parameters were eventually stabilized with NIBP of 113/54 mmHg and heart rate of 98 beats/min and surgery was commenced. Norepinephrine infusion was titrated to 4 mcg/ min. After completion of surgery, presence of rashes all over the body and a hot and flushed skin drew suspicion towards anaphylactic shock. The patient was then transferred to ICU with endotracheal tube in situ. Postoperative vitals were stable and extubation was done 2 hours later without any further complications.

DISCUSSION

The incidences of anaphylaxis during anaesthesia vary from 1 in 6000 to 1 in 20 000 [8] with an estimated mortality rate of 3%– 6%. [7]Muscle relaxants, local anaesthetics, hypnotics, opioids, inhalant agents, colloids, latex and antibiotics are the common causal factors of anaphylaxis.[8]Anaphylaxis occurs immediately effecting various organ systems and can present with several symptoms with hypotension, bronchospasm and urticaria being the major ones during general anaesthesia.[9,10]

During surgical cyst removal of the hydatid cyst, IgE-mediated anaphylactic reaction occurs when there is spillage of the highly antigenic hydatid fluid into the systemic circulation. [7,11]The allergic reactions vary from mild hypersensitivity reaction to a fatal anaphylactic shock, convulsions and coma.[12] During the cyst excision, the patient went into rapid and severe hypotension and bradycardia. The sudden appearance of these symptoms despite minimal blood loss and preloading the patient points the diagnosis towards anaphylaxis resulting from the spillage of the highly antigenic fluid into the systemic circulation by the hydatid cyst rupture.

The primary aim should be to immediately restore the haemodynamic stability.[13,14]Some reports have advised use of vasopressors.[7,13,14] We started norepinephrine infusion (12mcg/ minute) for the management of our case which was later tapered to 4mcg/ minute once haemodynamic stability was achieved. In addition, intravascular volume and cardiac output should be maintained with colloid or crystalloid fluids.[7] In our case, haemodynamic stability was achieved within 10 minutes of starting norepinephrine infusion and crystalloid fluids. Inhalational agents should be stopped and 100% oxygen should be administered to prevent bronchospasm and desaturation.

Prophylactic antihistamines may prevent further worsening of anaphylactic reactions. [7,13]Injection pheniramine maleate 45.2 mg bolus was administered in our case. Corticosteroids should be given to attenuate the airway oedema and inflammation. We have given injection hydrocortisone 100 mg and injection dexamethasone 8 mg in our case. Extubation should be done 5-6 hours later after the airway oedema has subsided. [9]

CONCLUSION

Hemodynamic instability in the absence of bleeding and hypovolaemia should raise suspicion towards anaphylaxis and specific management should be immediately started. The anaesthesist should be aware of the “tell-tale” signs of anaphylaxis and be prepared for any emergency. Vigilant monitoring for early diagnosis and prompt treatment of anaphylaxis are essential to avert any disaster or complications of anaphylactic shock.

LIST OF TABLES AND FIGURES

Figure 1. Ruptured hydatid cyst in a kidney tray.

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