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Case Report of Intraoperative Anaphylactic Shock and Post Operative Methemoglobinemia –Cetrimide Induced During Hepatic Hydatid Cyst Surgery

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Abstract

Hydatid cyst is a parasitic infection most commonly localized in the liver. They may not be diagnosed early because they remain asymptomatic at small sizes and may reach large sizes at the time of diagnosis. In this situation, compression symptoms may occur and they may present with serious complications such as rupture and anaphylactic shock, which are rare. Treatment methods are determined by classification according to the imaging techniques used in the diagnosis.

Keywords:- Anaphylactic Shock,cetrimide induced hypotension,hydatic cyst surgery

CASE REPORT

Presenting concerns and clinical findings

A 60 years old female patient known to have dyslipidemia, coronary artery disease status post 2 cardiac stents 10 years ago presenting after the incidental finding of asymptomatic multiple hepatic hydatic cyst diagnosed on abdominal ultrasound and confirmed by abdominal CT scan ,for which she was started on albendazol 200 mg for 6 months. There was no history of fever, cough with expectoration, loss of appetite, or recent weight loss.

Diagnostic focus and assessment: Blood investigations (hemogram, renal and liver function tests) were within normal limits. Chest X-ray was normal, Computerized tomography (CT) scan of abdomen showed a 4.8*4.6*3.5 cm loculated cystic formationat the level of segment VII with diffuse well calcificationson relation with old hydatic cysts.a 6.9*6.7*5.5 cm complex cysts at the level of segment Vand 1.3 cm cystic formation at the level of segment VI of the liver, Thin-walled daughter cysts were seen within the cyst.

Surgical procedure: The patient was premedicated with 2 mg midazolam before induction, and she was monitored by electrocardiogram (ECG), noninvasive blood pressure and peripheral oxygen saturation.

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Induction of anesthesia was with injection propofol 2 mg/kg and paralysis with injection rocuronium 50 mg. Anesthesia was maintained with sevoflurane and nitrous oxide. The patient was intubated with 7.5 mm single lumen endotracheal tube fixed at 21 cm. The patient was continuously monitored by ECG, noninvasive blood pressure monitor, pulse oximetry and end-tidal capnography. Ventilation was maintained by intermittent positive pressure ventilation at the rate of 12 breaths/minute, and tidal volume of 550 mL. Anaesthesia was maintained with sevoflurane plus oxygen and nitrous oxide (50:50). Muscle relaxation was provided by using repeated doses of rocuronium. Cetrimide was prepared preop 1g in 1L Normal Saline.

Hydatid Cyst Operative Report: Patient in supine position under General endotracheal anesthesia .Insertion of foley catheter under sterile conditions and orogastric tube without complications .All bony prominences were padded. Preoperative antibiotics were given. The abdomen was prepped using Chlorhexidine and draped in the usual sterile fashion. Right subcostal incision was made with a scalpel from the xyphoid to the anterior axillary line. Fascia was opened with electrocautery. The peritoneal cavity was entered sharply. Adhesiolysis was carried out using a combination of sharp dissection and electrocautery. Identification of a large hydatid cyst occupying segments V, VI, and VII Adhesiolysis and exposure of the segment completely .The pericystic area and operating field are covered with pads soaked with scolicidal agent- Cetrimide to prevent the spillage of parasites into the surrounding tissue and peritoneal cavity. The cyst is punctured and 70cc of aspirated fluids that is clear and colorless was aspirated. Before instilling the scolicidal agent, as much fluid as possible is aspirated to prevent dilution of the scolicidal agent. Then, the scolicidal agent is planned to be instilled into the cyst cavity and left for approximately 5-15 minutes. 60cc were injected without complications .Upon injecting an additional 60cc without pressure nor force hypotension was recorded .Surgery was paused awaiting clearance from anesthesia .Surgery resumed after clearance from anesthesia. Cetrimide was aspirated after 8 minutes if being kept in the cyst and the cyst is unroofed. The cyst contents, such as the germinative membrane was evacuated. the cavity was explored carefully for any gross communication with the biliary tract and for the presence of exogenous cysts embedded in the wall of the cyst.Pad was placed in the cavity.A small 1x1cm cyst next to the first one slightly on the posterior side of segment VI was injected aspirated and injected with 15cc of cetrimide .Complete excision of cyst was performed .Hemostasis . Identification of a third cyst at the superoanterior side of segments VII and VIII was identified.Right triangular and the right anterior fold of the coronary ligaments were divided for proper exposure of the cyst.The pericystic area and operating field are covered with pads soaked Cetrimide .Aspiration of the cyst and injection of 20cc Cetrimide.Aspiration of the content. Unroofing of the cyst, evacuation of daughter cysts and complete cystectomy was done. Placement of gelfoam into the two cavities of which large cysts were excised .The peritoneum of the gallbladder was incised over the fundus and the gallbladder dissected off the liver bed. The cystic duct and cystic artery were identified and dissected circumferentially to

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confirm that these were the only structures entering the gallbladder. A clip was placed on the cystic duct close to the neck of the gallbladder. A nick was made in the cystic duct and a cholangiogram catheter inserted. A cholangiogram was obtained showing minimal bile leak at the site of excision of segments' V VI VII cyst. Cystic duct ligated and divided.Biliary communications with visible fistulas were sutured intraoperatively with nonabsorbable sutures. Hemostasis attained. Placement of two lamellated drains each in a cavity.

Intraoperative events: There was no problem with respect of anaesthesia during the first 30 minutes. After starting the cyst excision, sudden and persistent hypotension (60/30 mmHg), hypocarbia (EtCO2 14 mmHg) and bradycardia (heart rate 30/minute) were noticed. Therefore, the operation was stopped. At this time, sevoflurane and nitrous oxide were discontinued, and the lungs were ventilated with 100% oxygen for hypoxaemia. Then fluid resuscitation was started for hypotension.Hypotension did not respond to fluid replacement. The diagnosis was an anaphylactic reaction, and a 200 µg bolus of IV adrenaline was given, followed by an infusion of 5 µg/kg/minute. After, hydrocortisone (200 mg) were used. Invasive arterial catheter was inserted and arterial blood gas analysis was done. Severe acidosis were seen (pH:7.1)The patient responded to fluid and epinephrine infusion within 10 minutes. The blood pressure, SpO2 and heart rate returned slowly to normal. Following haemodynamic stabilization, the surgery was continued. The surgery team reported that there was an aberrant venous drainage detected in the HC. After the surgery, the patient was admitted to the intensive care unit and epinephrine infusion was continued for inotropic support for a day.

DISCUSSION

The incidence of anaphylaxis during anaesthesia and the perioperative period is rare and range from 1 in 6000 to 1 in 20 000 anaesthetics.[1] The estimated mortality rate is 3%– 6% . The common anaphylactoic agents are muscle relaxants, local anaesthetics, antibiotics, latex, chlorhexidine, hypnotics and inhalant agents, protamine, colloids, opioids and antibiotics.

Anaphylactic reaction occurs immediately, systemically and can influence various organ systems, and several symptoms have been observed such as respiratory (bronchospasm and upper airway obstruction), cardiovascular (hypotension and arrhythmias), skin (urticaria and angioedema) and gastrointestinal [nausea and vomiting].

A large portion of these symptoms may not be seen under general anaesthesia. During general anesthesia, hypotension, bronchospasm and urticaria are the main symptoms.

In a previous study, the incidence of intra-operative anaphylaxis with hydatid cyst was reported to be low at 0.2–3.3%.[2] During surgical cyst removal,[3] or percutaneous drainage of the liver, hydatid cyst IgE-mediated anaphylactic reaction occurs when there is spillage or release of the highly antigenic hydatid fluid into the systemic circulation.[4] The allergic reactions vary from mild hypersensitivity reaction to a fatal anaphylactic shock, convulsions and coma.

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Some reports have recommended that the usage of the prophylactic corticosteroid and antihistamines could avoid anaphylactic reactions.[5] In the present study, anti-histamines and corticosteroids were admitted for the risk of anaphylaxis after induction. However, during the cyst excision, sudden and persistent desaturation, severe hypotension, hypocarbia, bradycardia and diffuse erythema were observed. These symptoms appeared to be primarily related to anaphylaxis due to the spillage of the highly antigenic HC fluid into the bloodstream by the HC rupture. However, the surgical team reported that the cystic wall was intact but there was a vein in the cyst cavity.

Hydatid cyst has a high intracystic pressure and contains antigenic fluid. In a previous study, the authors claimed leakage of cystic fluid into the bloodstream due to high intracystic pressure and blunt dissection .In our case, we believe that the high intracystic pressure coupled with blunt dissection must have been the cause of leakage of cystic contents through this vein into the circulation with no apparent macroscopic rupture. So, the same etiology may also be effective in our case. In reviewing the literature, a few anaphylactic reactions have been reported with no apparent macroscopic rupture of the hydatid cysts.

Post operative period: In OR, after cetrimide injection, the patient developed hypotension and was started on adrenaline by SP as well. She received 6 Liters of IV fluids and steroids for possibility of anaphylactic shock.

Patient was transferred to the ICU. Upon arrival , SBP was 66 mmHg, HR was 130-140 beats/min,irregular, saturation was 75% on 100% FiO2,pulips were dilated non- reactive ,corneal reflexes were absent bilaterally, lips edematous, lungs clear, abdomen soft but drain was yielding bloody fluid. No lower limbs edema was seen. Laboratory tests were taken and showed a hemoglobin level of 8 mg/dL (preoperatively it was 14). 3 units of pRBCs were given flush by the central line. Aggressive fluid management was on board.IV cordarone by SP was given for rapid AFib. Urgent TTE was done by cardiology team and showed no pericardial effusion, a good EF of 65%, a non dilated IVC. No pulmonary hypertension nor right ventricule strain were seen. After patient was hemodynamically stable, urgent CT brain and CT angiography of the chest abdomen and pelvis were done. Radiology team reported no remarkable changes on CT brain, no pulmonary embolism on CT chest, but small bilateral pleural effusion was seen due to the aggressive hydration with bilateral atelactesis. Edema was seen arround the thyroid gland.

CT of the abdomen and pelvis showed post operative surgical changes at the level of the liver, and bowel walls were diffusely thickened, and the small bowel contains proximally a hyperdense content to rule oozing secondary to wall edema. After ruling out the major diagnosis (pulmonary edema, post operative bleed, seading of daughter vesicles tamponade, myocardial infarction, aortic dissection), we were left with 2 differential diagnosis: methemoglobunemia secondary to cetrimide and anaphylactic shock.Urgent methemoglobunemia level was ordered (not available in our hospital), and urgent TSH level as well. IV methylene blue of 100 mg IV once was given to which patient responded by increasing Saturation from 82% to 100% on 100% FiO2), solumerol was given for possible

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anaphylaxis, mechanical ventilation was resumed with PEEP 14 mmHg and FiO2 100%. Levophed was kept having for target a SBP bwtween 120 and 130 mmHg. Albendazole 400mg twice daily was continued per NG tube (instead of 200 mg pre operatively). Bronchodilators were started.

Day 1 post operatively patient developed seizure, keppra loading dose was given and the anti epileptic drug was initiated for therapy. CT brain rule out any changes. CT abdomen of control showed no new findings as well despite her elevated LFTs. Patient was stable on levophed, intubated, acidotic on ABGs (pH = 7,02). Sodium bicarbonate was given. Hypokalemia and hypomagnesemia were corrected by giving potassium chloride and magnesium sulfate. Patient was switched to metronidazole instead ofalbendazole.

Day 2 post operatively, levophed was stopped. Patient was awake oriented to space, person and time.

When there is unexpected or sudden, rapidly progressive haemodynamic and respiratory problems, the possibility of an anaphylactic reaction should always be considered in respect to an HC rupture, and the diagnosis should be made immediately. Initially, the aim of treatment should be to restore both adequate cardiac output and circulatory competency. Some reports have advised use of vasopressors with alpha as well as beta stimulating properties .Epinephrine is the most appropriate drug of choice in the management of bronchospasm and massive peripheral vasodilation, which occur in anaphylaxis and sometimes a high dose may be necessary and the infusion may have to be maintained for a long time.

In addition to epinephrine, intravascular volume, vascular tone and cardiac output should be supported with colloid or crystalloid fluids. In our case, we received a response within 10 minutes to epinephrine, colloid or crystalloid fluids. Besides, inhalation anaesthetics should be stopped and 100% oxygen administered for control of the airway and bronchospasm. Antihistamines are useful prophylactically and may also prevent further histamine binding after the development of anaphylactic reactions .Corticosteroids should be given prophylactically to diminish the airway swelling and prevent recurrence of symptoms. Extubation should not be immediate. Airway swelling and inflammation may continue for hours.

CONCLUSIONS

It should be kept in mind that anaphylaxis can occur despite all the precautionary measures and absence of rupture of the hydatic cyst or spillage of the cyst to the circulation or into the surrounding tissues.

The anaesthesist should be aware of anaphylactoid reaction and be prepared for treatment. Consequently, a close monitoring for early diagnosis and appropriate management of anaphylaxis are essential to stabilize the patient and produce the best outcome.

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