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METABOLIC ACIDOSIS AND ACUTE KIDNEY INJURY: ADVERSE EFFECTS OF INJUDICIOUS SCOLICIDAL (CHLORHEXIDINE 0.05%) USE IN HYDATID DISEASE.

Gastroenterology

Gopalakrishnan Gunasekaran Department of Surgical Gastroenterology, Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry, India.

Debasis Naik* Department of Surgery, Jawaharlal Institute of Postgraduate Medical Education and Research, Puducherry, India *Corresponding Author

ABSTRACT

Surgery remains the mainstay of treatment for hydatid disease, with the risk of intra-operative spillage of scolices, giving rise to secondary hydatidosis. To minimize this scolicidal agents are injected to the cyst and the operative field is walled off with sponges soaked in scolicidal agents.[1] However, this is associated with adverse effects like sclerosing cholangitis and fibrosing peritonitis.[12-14] Chlorhexidine (0.05%) is one the commonly used scolicidal agent in hydatid disease with the least toxicity profile. We here report a case of metabolic acidosis with acute kidney injury following the use of Chlorhexidine as scolicidal.

Summary: As there are no safe recommendation of upper limit of volume and concentration of Chlorhexidine as scolicidal, future research is needed.

KEYWORDS

Chlorhexidine, hydatid disease, metabolic acidosis, acute kidney injury.

INTRODUCTION

Surgical treatment remains the mainstay of treatment of hydatid disease, with the risk of intraoperative spillage of scolices. Measures like, injecting scolicidal solution into the hydatid cyst and preserving the operative field with sponges soaked in scolicidal agents have been used to avoid secondary dissemination of the parasite during surgery.[1] However, there is no ideal scolicidal agent that is both effective as well as without any adverse effects. Chlorhexidine (0.05%) is one of the scolicidal agents used in hydatid disease with the least toxicity profile. We here present a rare case of chlorhexidine induced metabolic acidosis and acute kidney injury following surgery for hydatid disease in a 45year lady.

CASE REPORT

A 45year lady presented with complaints of dull aching pain in the right upper abdomen for 1 year associated with early fullness of stomach and decreased appetite. There was no history of vomiting or fever or jaundice. She had history of Laparotomy with cystectomy for hydatid cyst in the greater omentum and partial cystectomy for liver hydatid cyst 2 year back. On examination, her vitals were stable, per abdomen – tenderness was present in the right hypochondrium, 6 x 8 cm, tender, firm lump was present in the right hypochondrium extending into the right lumbar region, with rounded margins and moving with respiration. Superiorly, the lump was going beneath the right subcoastal margin. Liver was not enlarged and spleen was not palpable. Her routine blood investigations were normal, except for eosinophilia. Hydatid serology was positive and Contrast Enhanced Computed tomography (CECT) Thorax and Abdomen revealed fluid attenuating lesions in liver and splenic parenchyma and in the peritoneal cavity. Two lesions, 8 x 7.5 cm (segment VII and segment VIII) and 7 x 6 cm (segment IV) in the liver, another 6 x 5.5 cm lesion in the superior pole of the spleen and 8.5 x 8.7 cm left sub diaphragmatic lesion posterior to spleen, exerting mass effect over the spleen and left kidney, all suggestive of multiple hydatidosis (Figure 1 & 2). A course of Albendazole 400 mg twice a day for 3 weeks was given, she was vaccinated against Pneumococci, Meningococci and H. influenza and then taken up for elective surgery. Intraoperatively, the liver cysts in segment VII and VIII were bulging out of inferior surface of liver displacing the duodenum, right kidney and pancreas anteriorly and inferiorly. The cyst at the superior pole of the spleen was adhered to under surface of left lobe of liver, and left sub-diaphragmatic lesion was adhered to the posterior border of the spleen, extending to the lesser sac behind the body and tail of the pancreas. Partial cystectomy of liver hydatid cysts with drainage of cyst contents with irrigation using around 200 ml of chlorhexidine 0.05% was done with excision of splenic and the sub-diaphragmatic lesions in toto with the spleen (Figure 3). Post-operatively, in the recovery room she developed tachycardia and tachypnea. ABG revealed metabolic acidosis with pH – 7.13, HCO3- 12 mEq, pCO2 – 26 mmHg, Lactate - 1.6 and her serum creatinine rose from 0.6 mg/dL to 1.35 mg/dL indicating Acute kidney injury. This was corrected with I.V. fluids and Sodium bicarbonate infusion. On second postoperative day, her acidosis got corrected. She was discharged on fifth postoperative day after removal of abdominal drains and tolerating normal diet with advice to take two more courses of Albendazole.

Figure1 & 2: CECT Abdomen showing liver hydatid cyst occupying segment VII and VIII, and the sub diaphragmatic cyst coursing along the posterior border of spleen into the lesser sac behind the body and tail of pancreas.

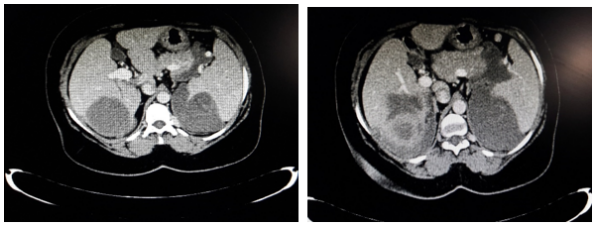

Figure 3: Resected specimen showing the splenic cyst, sub-diaphragmatic cyst and the spleen excised in toto.Figure1 & 2: CECT Abdomen showing liver hydatid cyst occupying segment VII and VIII, and the sub diaphragmatic cyst coursing along the posterior border of spleen into the lesser sac behind the body and tail of pancreas.

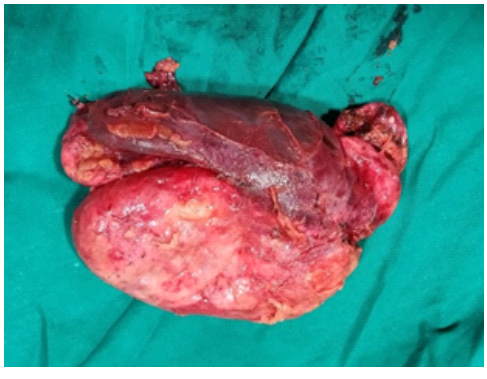

Figure 3: Resected specimen showing the splenic cyst, sub-diaphragmatic cyst and the spleen excised in toto.

DISCUSSION

Hydatid disease is a parasitic infestation of humans caused by *Echinococcus granulosus.* It is endemic in Australia and the Middle east, including Iran. The life cycle of parasite alternates between herbivores and carnivores. Man is an accidental intermediate host and an end point in parasite life cycle. Dogs and some wild carnivores are

the definitive host, harbouring the sexually reproductive forms of worms in their intestine. Eggs passed in the faeces when eaten up by intermediate host, hatch in the small intestine and the larval tape worms are released. These worms burrow through intestinal wall and encysts in the liver, lung, brain and other organs.[2]

Most patients are asymptomatic, the reason being slow growth rate of the cyst (1-5 mm per year). Symptoms usually develop in adulthood, the most common being discomfort in the right upper abdomen and loss of appetite. Other symptoms may include pain, anaphylactic reaction induced by rupture of cyst, hepatitis and cholangitis due to biliary obstruction caused by the daughter cysts, secondary infection of cyst, embolism, and subphrenic and intracystic abscess. Physical examination may reveal jaundice, abdominal distension, hepatomegaly and/ or palpable mass in the right upper abdomen.[3-7] Early diagnosis is usually incidental. The definitive diagnosis of most of the hydatid disease is by imaging like ultrasound, computed tomography scan and magnetic resonance imaging of abdomen. Immunodiagnosis also plays a complimentary role.[8]

Until three decades ago, surgery was the only treatment option available for liver hydatid disease.[9] Despite refinement in surgical techniques, there is considerable controversy as to what is the most effective technique, the role of cyst aspiration and external drainage, hepatic resection, management of residual cavity, cyst recurrence after surgery and high rates of complications and mortality related to reoperation in recurrent disease.[10] An immediate cure is claimed for surgical treatment but even with radical procedures this is far from being achieved with morbidity, mortality and relapse rates of 32%, 8% and 20%, respectively.[11]

In surgical management of the disease, neutralisation of the parasite, evacuation of cyst and management of residual cavity are the principal steps along with prevention of spillage into the peritoneal cavity and wound edge. Injecting a scolicidal agent into the unopened cyst and walling off the operative field with sponges soaked in scolicidal agent are the two most commonly employed measures.[1]
Formalin, hypertonic saline, cetrimide, chlorhexidine, hydrogen peroxide and ethyl alcohol are some the scolicidal agents used, however none has been found to be the ideal.[1] The use of scolicidal agent in hepatic hydatid cyst disease (formaldehyde, hypertonic saline) are reported to cause sclerosing cholangitis (in cysts with biliary duct communication) and fibrosing peritonitis (when used for peritoneal washout).[12-14] There are also a few case reports of metabolic acidosis and acute kidney injury following use of cetrimide-chlorhexidine solution.[15-17]

In our patient by ruling out sepsis (normal counts, sterile blood culture, normal chest radiographs) and malignant hyperthermia, we attribute the metabolic acidosis and acute kidney injury to be due to Chlorhexidine.

CONCLUSION

In spite of being the safest of the scolicidal agents, a regulated volume and concentration of chlorhexidine should be recommended. As there has been no recommended upper limit of volume and concentration of chlorhexidine to be used as scolicidal agent in human beings, research on the safe limits need to be undertaken in future.

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