Cetrimide Induced Metabolic Acidosis: A Rare Intraoperative Complication in a Case of Hydatid Disease of Liver

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Abstract: Liver is the most common organ involved in hydatid disease. Surgery remains the mainstay of treatment. During surgery of hydatid disease of liver, scolicidal agents (like cetrimide, hypertonic saline, savlon etc.) are used to kill the parasites and prevent intraperitoneal contamination. Use of cetrimide can rarely cause metabolic acidosis intraoperatively, which is a serious complication. Intraoperative arterial blood gas analysis (ABGA) is very helpful in early detection of metabolic acidosis. Patient who develops metabolic acidosis should be managed in a critical care unit. We hereby report a case of 31 year old female patient having hydatid cyst of liver operated laproscopically and developed metabolic acidosis due to use of cetrimide during surgery.

Keywords: Hydatid Disease, Scolicidal Agents, Metabolic Acidosis, Abga-Arterial Blood Gas Analysis

1. Introduction

Hydatid disease is caused by the larval stage of Echinococcus granulosus (also known as Taenia echinococcus). Humans are accidental intermediate hosts, whereas animals can be both intermediate hosts and definitive hosts. In humans, 50–75% of the cysts occur in the liver, 25% are located in the lungs, and 5–10% distribute along the arterial system. Cetrimide is commonly used scolicidal agent during surgery. Metabolic acidosis is a rare but serious complication of cetrimide which requires critical management. But many surgeons are unaware regarding this complication, so here we are sharing our experience of a case that developed metabolic acidosis during surgery due to cetrimide use and was managed in critical care unit.

2. Case Report

A 31 year old female patient presented with pain in abdomen since one year in right upper quadrant, intermittent, mild to moderate in intensity, non radiating without any associated complains like vomiting, fever or yellow discoloration of eye and urine. Patient was taking symptomatic medications for pain but she was not relieved by that. History of contact with domestic animals was present. There were no medical co morbidities. On examination patient’s abdomen was soft with mild tenderness in right hypochondrium and epigastric region. There were no any other significant findings on general and systemic examinations.

Blood Investigations: Hemogram, liver function test and renal function test were within normal limits. Ultrasonography: Approximately 5cm*4cm well defined heterogeneous echotexture lesion with hyperechoic rim in right lobe of liver, another approximately 39 mm*31mm sized similar characterized lesion in left lobe of liver, possibility of hydatid cysts. CECT Abdomen: 48mm*43mm lesion in segment III and 43mm*48mm lesion in segment VII, possibility of benign lesions most likely hydatid cyst.
Coronel section showing both Hydatid cysts

Preoperatively patient was given Tablet Albendazole 400 mg twice a day for 2 weeks before surgery. Patient was posted for laparoscopic excision of hydatid cyst. To prevent anaphylactic shock patient was given Injection Hydrocortisone 200mg IV stat. Injection Dexona 8 mg IV stat and Injection Avil 1amp IV stat just before induction.

Anesthesia: Injection glycopyrrolate 0.2 mg IV stat, injection emset 4 mg IV stat, injection fentanyl 80 mg IV stat were given as pre anesthetic medications. For induction of anesthesia injection propofol 200 mg iv and injection suxamethonium 100 mg iv were given and for maintenance of anesthesia N2O, sevoflurane as inhalation agents and vecuronium 4 mg iv stat and 1mg iv sos was given as neuromuscular blocker.

After creating pneumoperitoneum, following ports were inserted-10mm port just above umbilicus, 10 mm port in epigastrum just below xiphoid, 5mm port at right midclavicular line subcostally, 5mm port in right anterior axillary line at level of umbilicus. 5mm port in left midclavicular line subcostally and 5mm port in left axillary line at level of umbilicus were placed for left side hydatid cyst.

Intraoperatively 2 cysts were found-one was at posterosuperior aspect of right lobe of liver and second was at posterosuperior aspect of left lobe of liver. Operative field was filled with roller gauzes soaked in 0.5% cetrimide(20 ml of 1.5%cetrimide was diluted with 40 ml of normal saline to form 0.5% cetrimide-40 ml solution was used for filling the operative field with roller gauzes and rest was used to irrigate the cyst cavity). Thus for both cysts total diluted cetrimide solution was 120 ml. De-roofing of the cysts was done. Cysts were containing daughter cysts, membranes and fluid as its content which were removed and saline and cetrimide wash was given in the cyst cavity. A drain was kept. Surgery lasted for 2 hours and there was no fluctuation of pulse rate, blood pressure and there were no other signs of anaphylactic shock.

After Completion of Surgery (0hours):

Patient did not come out of anesthesia effect, so patient was kept intubated and immediate ABGA was done, which was suggestive of severe metabolic acidosis (pH 7.1,HCO₃⁻ 16, pCO₂ 48). Patient’s Urine output was 75ml (concentrated) though 2.5 liter fluid was given intraoperatively. These findings suggest that patient had developed metabolic acidosis due to some insult during surgery. Patient was given Injection Soda Bicarbonate 5amp (50cc) in 100 cc NS IV slowly over 1 hr, Injection Lasix 40mg IV stat.

After 1 Hour of Surgery

Patient was unconscious, not following verbal command and pH was 7.24, HCO₃⁻ 20, pCO₂ 42, so patient was electively shifted to ICU from OT and was put on SIMV+PSV Mode ventilation.

After 4 Hour of Surgery

Patient became conscious, following verbal command, but muscle tone and power was not adequate, so patient was kept intubated on mechanical ventilator. At this time...
patient’s pH was 7.41 and HCO₃⁻ 22.8, pCO₂ 34.7 and urine output was 800 ml (clear).

After 15 Hour of Surgery

Patient was conscious, following verbal commands, muscle power was adequate and vitals were within normal limits. So extubation was done. Patient was kept on ventilator with O₂ at 6-8 liter/min for next 24 hours.

Post Operative Investigations

<table>
<thead>
<tr>
<th>Post op days</th>
<th>0hr</th>
<th>1 hr</th>
<th>4hr</th>
<th>Day 1</th>
<th>Day3</th>
<th>Day 5</th>
<th>Day10</th>
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<tr>
<td>Creatinine</td>
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<td>1.79</td>
<td>2.48</td>
<td>3.28</td>
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<td>1.12</td>
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<tr>
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<td>28</td>
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<td>29</td>
<td>62</td>
<td>89</td>
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<tr>
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<td>3.6</td>
<td>3.4</td>
<td>3.6</td>
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<tr>
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<td>-</td>
<td>13.2</td>
<td>13.1</td>
<td>12.6</td>
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<td>10200</td>
<td>-</td>
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<td>16,100</td>
<td>11,200</td>
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<tr>
<td>pH</td>
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<td>7.24</td>
<td>7.41</td>
<td>7.4</td>
<td>-</td>
<td>-</td>
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</tr>
</tbody>
</table>

"-" Indicates Test Not Done

Patient’s renal function started to deteriorate after surgery and serum creatinine level raised up to the value of 3.28 mg/dl on 3rd post operative day and urea raised up to 89 mg/dl. Adequate hydration and input output was maintained, and nephrotoxic drugs were avoided. Patient did not require dialysis as urine output became more than 30-50ml/hr after 4 hours of surgery. Urea, creatinine levels which were high started to decrease and reached to normal level on 10th post operative day. Patient’s blood pressure was also increased to the value of 150/90 from 1st post operative day and remained static up to 10th post operative day and then it came to its normal level. These findings suggest that patient has suffered renal insult due to metabolic acidosis. Patient was discharged on 12th post operative day. On discharge patient was vitally stable, taking all orally, per abdomen-soft, non tender, passing stool, flatus normally.

3. Discussion

Hydatid disease is caused by dog tape worm- echinococcos granulosus. Liver hydatid cysts may be asymptomatic initially, but can exert pressure as they grow on adjacent tissues and can cause symptoms such as vomiting, abdominal pain and abdominal distension. Complications like cyst rupture into bile ducts, obstruction of the biliary ducts, bacterial infection, intraperitoneal rupture, portal hypertension, gastrointestinal bleeding can occur [1]. Liver hydatid cyst can be managed by medical therapy, percutaneous therapy and surgery. Medical therapy is (albendazole and flubendazole) curative in only 20% to 30% of all cases. Preoperative treatment with albendazole softens the cyst, reduces intracystic pressure and simplifies its removal and prevents recurrence. Preoperative chemotherapy with albendazole should begin at least 4 days preferably 1 month before surgery and continued for 2 to 6 months after surgery [2, 3]. Chemotherapy is preferred when the disease is inoperable, surgery is not available, or the cysts are numerous. Chemotherapy is also used to prevent secondary echinococcosis [4].

Percutaneous therapy in form of PAIR (puncture, aspiration, injection, and reaspiration) is another option, but the need for prolonged hospital stays or repeated visits and the development of complications such as spillage, biliary leakage, and deep abdominal abscesses have prevented its widespread use [5].

Surgery remains the mainstay of treatment for hydatid disease. Laparoscopic hydatid cystectomy has been found to be feasible and safe; advantages are less pain, good cosmesis, and rapid recovery with a shorter hospital stay [6]. During surgery prevention of spillage into the peritoneal cavity and wound edges is very important to prevent recurrence and anaphylaxis. Walling off the surgical field with sponges or packs soaked in sclocidal agents is an effective and logical means of using scloidal agents [7]. Sclocidal agents available are formalin, betadine, hypertonic saline, cetrimide, chlorhexidine, hydrogen peroxide and ethyl alcohol. Formalin, betadine and hydrogen peroxide are no longer used because of their toxicity. Ethyl alcohol is preferred for ultrasonic-guided PAIR, but it can cause caustic damage to the epithelium of communicating bile ducts may lead to sclerosing cholangitis. Hypertonic saline has been used in various concentrations but only 20% saline appears to be effective. It should not be used in patients who have cysts communicating with biliary tree because of the danger of causing caustic sclerosing cholangitis [8]. Cetrimide is a potent disinfectant and effective sclodical agent. It is used alone or in combination with chlorhexidine (savlon). Cetrimide (0.1-0.5%) is used in lower concentration (0.1-0.5%) [9]. Sclerosing peritonitis, metabolic acidosis and methemoglobinemia are reported complications of cetrimide use [10, 11, 12, 13].

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In our case patient developed metabolic acidosis during surgery due to which patient did not come out of anesthesia effect in immediate post operative period and patient had also developed deranged renal function.

Here anaphylactic shock can be ruled out as there was no fall in blood pressure, no skin lesions, no angioedema development, no spillage of hydatid material in peritoneal cavity during surgery and we had given injection dexona, hydrocortisone and avil before surgery to prevent it. Volatile anesthetic agents and neuromuscular blocker suxamethonium are associated with malignant hyperthermia and metabolic acidosis, but here the patient did not develop any rise in temperature or any signs symptoms suggesting malignant hyperthermia. Prolonged propofol administration (>48 hours) at high doses (>4 mg/kg/h) may cause a rare, but frequently fatal complication known as propofol infusion syndrome (PRIS) [14]. PRIS is characterized by metabolic acidosis, rhabdomyolysis of both skeletal and cardiac muscle, arrhythmias (bradycardia, atrial fibrillation, ventricular and supraventricular tachycardia, bundle branch block and asystole), myocardial failure, renal failure, hepatomegaly and death. However neither propofol was given in a very high dose nor any signs suggestive of PRIS were there. CO2 retention due to creation of pneumoperitoneum can cause delayed recovery from anesthesia but it is also ruled out as there was no significant rise of pCO2. As cetrimide has been documented in the literature as a cause of metabolic acidosis and other causes of metabolic acidosis have been ruled out, cetrimide appears to be the cause of metabolic acidosis in this patient.

ABGA measurement had helped us in early detection of metabolic acidosis but if done in all cases intraoperatively after use of cetrimide than it may help us in earliest detection of metabolic acidosis. After development of metabolic acidosis patient was managed successfully with judicious use of intravenous drugs and fluids in intensive care unit. As critical care management is very important in such patients, hydatid cyst surgery should be done in a setup having well equipped critical care unit. From our experience we feel that cetrimide should be used in a lower concentration (0.1-0.2%) with intraoperative ABGA measurements to avoid and detect acidosis at earliest.

4. Conclusion

Metabolic acidosis is a rare but serious complication of cetrimide use and one should keep it in mind while using cetrimide in hydatid surgery. Cetrimide should be used in a lower concentration and if used, intraoperative ABGA measurement is recommended.

5. Abbreviations

NS - Normal Saline
IV - Intravenous
HB - Hemoglobin
ABGA - Arterial Blood Gas Analysis
CECT - Contrast Enhanced Computed Tomography
OT - Operation Theatre
SIMV - Synchronized Intermittent Mandatory Ventilation
PSV - Pressure Support Ventilation

PRIS - Propofol Infusion Syndrome

References


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