Povidone-iodine toxicity in a child posted for laparoscopic removal of hepatic and renal hydatid cysts

Sir,

Povidone-iodine (PI) is an antiseptic solution consisting of polyvinylpyrrolidone with water and iodine (available iodine 1%). The 10% solution has been used effectively as a scolicidal agent for hydatid cysts. We hereby discuss the case of a child posted for laparoscopic removal of hepatic and renal hydatid cysts, who developed metabolic acidosis with acute renal failure.

A 10-year-old child (weight 26 kg) presented with history of pain in epigastrium. Pain was dull, mild to moderate, and there was no radiation. Systemic examination revealed hepatomegaly. Computed tomography scan abdomen revealed well-defined cystic lesion of 12.5 × 8.5 × 11.4 cm in the left lobe of liver extending to lesser sac and perihepatic area along with small cysts in right kidney. Routine blood investigations and liver function tests were normal. Anti-Echinococcus IgG antibody was positive by Enzyme Linked Immunosorbent Assay. Diagnosis of hydatid cyst of liver and kidney was made, and the child was posted for elective laparoscopic removal of hepatic and renal cysts. Baseline haemodynamic parameters were normal for age. Inhalational induction performed with sevoflurane (8%) in oxygen. After administering fentanyl (60 μg) and vecuronium (3 mg), trachea was intubated. A total of 2.5 L of 10% PI (Microwin® 10% w/v) was injected into hepatic hydatid cyst. An hour into the procedure, suddenly heart rate increased from 110 to 180/min along with hypercarbia (end-tidal CO_2 55mmHg) and hyperthermia (from 35.1 to 37.4°C). Injection paracetamol (500 mg) and a bolus (0.5 L) of normal saline were infused. An arterial blood gas analysis showed pH 7.14, pCO_2 36.5 mmHg, pO_2 135 mmHg, SO_2 97.7%, K^+ 7.4 mmol/L, Na^+ 127 mmol/L, Ca^+ 0.81 mmol/L, Cl − 249 mmol/L, lactate 4.3 mmol/L, base deficit - 15 mmol/L, HCO_3 12.7 mmol/L with Hb of 16 gm%. Blood pressure and airway pressures were normal. Urine output decreased and did not improve after first fluid bolus so a further fluid bolus of 0.5 L of balanced salt solution (sterofundin®) and injection frusemide 15 mg intravenous (IV) were administered. Urine output improved and presumptive diagnosis of anaphylaxis due to systemic entry of hydatid cyst fluid was suspected. Injection hydrocortisone and pheniramine maleate were administered. Patient was shifted to intensive care unit (ICU). Investigations revealed haemoglobin 17 g/dL, leukocyte count 49.6 × 10^3/μL, serum creatinine 0.9 mg/dL, serum albumin 1.9 g/dL, and potassium 5.2 mEq/L in the immediate post-operative period. Liver, renal, and thyroid function tests were in normal range. Severe metabolic acidosis persisted and thus plan for haemodialysis was made. Tachycardia was fluid responsive and Sterofundin® 100 ml/h, albumin (20%) 25 ml/h and hydrocortisone 6 mg/h were started. Bedside echocardiography revealed good cardiac contractility, under filled heart chambers and inferior vena cava diameter of 0.4 – 0.8 cm with collapsibility. After 3–4 h of initiation of haemodialysis, acidosis was corrected and heart rate settled. On post-operative day 1, the patient was fully awake and obeying commands. Trachea was extubated after completion of dialysis. Hydrocortisone infusion was tapered and stopped. Patient was kept for another 2 days in ICU for observation before shifting to ward.

In its complexed form (i.e. PI), iodine vapour pressure is reduced essentially to zero, and it becomes soluble in water. The available iodine is released at very slow rate from the complex, prolonging germicidal action of free iodine. Povidone-iodine is highly nephrotoxic and due to its renal elimination, iodine intoxication can result in lethal vicious cycle and anuria.[1] Clinically, patients with PI intoxication can present with hypotension, nausea, vomiting, acute renal failure, hypothyroidism, confusion, metabolic acidosis, seizures, blindness, fever, rash, uterine infarction, elevated hepatic enzymes and iodine-induced haemolysis with decreased serum haptoglobin and lactate dehydrogenase levels.[1] Serum iodine levels could not be done as this analysis was not available in our hospital. Labbé *et al*. reported a case where abnormalities of cardiac conduction, lactic acidosis, acute renal failure, hypocalcaemia and thyroid dysfunction were features of intoxication.[2] In

another case report of iodine toxicity in a child with mediastinitis, after median sternotomy, continuous PI irrigation was carried out and the child died due to its toxicity.[3] Iodinated contrast medium (IV) can cause contrast-induced acute kidney injury, but even with non-intravascular use of iodinated compounds, iodine absorption through mucosal, burned skin or interstitial tissues, can cause nephrotoxicity.[4]

Early implementation of renal replacement therapy can be lifesaving.

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