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Case Report

Hypernatremia post hydatid cyst resection: case report

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ABSTRACT

Hypernatremia or increased sodium level is defined as the plasma sodium level over 145 m mol/l. The electrolyte abnormality is associated with severe mortality (40-60%). Patients presenting acutely with this entity have varied clinical spectrum ranging from unresponsive state, dysphagia, shortness of breath, vomiting to seizures, coma or to even death. Iatrogenic hypernatremia usually a rare entity ensues after usage of hypertonic saline as scolicidal agent during hydatid cyst resection surgeries. There is a need of high level of suspicion, to be maintained in all patients being managed for clinical conditions requiring use of hypertonic saline (3-20% NS). Here we present a case of iatrogenic hypernatremia, post-surgical resection of hydatid cyst, with clinical manifestation and subsequent management in our tertiary care hospital.

Keywords: Hypernatremia, Iatrogenic, Seizures, Hypertonic Saline, Scolicidal, Hydatid cyst

INTRODUCTION

Acute increase of serum sodium concentration with values more than 145 m mol/ L within 48 hours is caused due to a net water fluid loss and/or hypertonic sodium gain.[1,2] Intravenous infusion of hypertonic saline, excessive salt ingestion, gastric lavage, topical application of salt on burned areas, and bicarbonate administration during cardiac arrest are all possible causes. Intra operative development of hypernatremia is a rare entity, and patients exposed to hypernatremia are susceptible to major neurological complication even to extent of coma or death.[2] The spectrum of studied mortality rate with elevated serum sodium (Na) levels with 160 m mol/L is as high as 70%, 150 m mol/l -38-45%. The chances of survival are somehow better in Pediatric age group as compared to adults with tolerability and survivability profile of children even for extreme hypernatremia 200 m mol/L.[3,4]

We report a case of an adult patient who survived an iatrogenic acute hypernatremia (160 m mol/L) resulting from hypertonic saline irrigation of intra-abdominal hydatid cyst while undergoing laparoscopic de roofing. This is a unique report of an intraoperative developed serum sodium increase and resolved without any subsequent residual neurological damage.

CASE REPORT

41-year-old male, 80 kg was admitted to hospital duly worked up, after initial presentation of abdominal pain on multiple episodes. The patient was evaluated, diagnosed as case of hydatid cyst segment III liver and planned for elective laparoscopic de roofing and excision of cyst by GI surgery dept. Pre anesthesia evaluation was done up a week prior and salient finding included adequate airway assessment, nil medication and allergy history, no co morbidity, no seizure episode history. Radiological investigations: X-ray chest-WNL lung fields with

elevation of rt hemi diaphragm, CT scan Abd-hydatid cyst segment III liver (Figure 1). Blood investigations: Electrolytes Na^+ -135 m mol/L, K^+ -4.2 m mol/L, CBC-WNL, liver function test-SGOT-60, SGPT- 70, total bilurubin-1.5 m mol/L, renal function test-WNL. Patient accepted in ASA II (overweight, smoker, hyper bilirubinemia) and standard anti aspiration and NPO protocol followed. As per institutional protocol 02 units of packed red blood cells arranged and kept ready in blood transfusion dept with standby ICU bed and ventilator.

On surgery day, patient underwent laparoscopic de roofing of cyst, irrigation of cyst cavity with 20% hypertonic saline for contact period of 6-8 minutes. The duration of surgery was 120 mins and uneventful. Patient was extubated on table in view of uneventful surgery under adequate multimodal analgesia cover. Patient reviewed in immediate post op period in PACU. Altered sensorium of patient was observed and immediately oxygen inhalation at 4 LPM by face mask started and head end propped up. Arterial Blood gas sample analysis done and findings pointed towards hypernatremia (Na^{+}-160 m mol/L) and respiratory alkalosis. Patient electively intubated, in order to have airway protection and controlled correction of acute hypernatremia along with concomitant work up for other causes of altered sensorium.

Immediate plan of action for correction of hypernatremia was formulated with care of under mentioned points: (a) Rate of correction: <0.5 m mol/L (to prevent hyponatremic de myelosis), (b) Choice of fluid: D5 (dextrose 5%) (c) Free fluid requirement on basis of formula: Normal total body water (0.6 X body weight)-Current body water (0.6 X body weight X140/Na^+); volume of fluid: H_2O deficit X (140/Na^+ in I V fluid) * (d) Serum osmolarity: 2x(Na^+) + Glucose/18+ urea/2.8

Osmoles in relative excess: 0.6 x lean body weight (kg) x (current osmolarity-280). *Na^+ in D5%: 0 m mol/L.[10]

On basis of above Free water requirement was 6 liters and total D5% requirement was 6 L and correction required from 160-140 m mol (20 m mol), 20 m mol to be corrected over 40 hours by 6000 ml D5% i.e., 150 ml/hr. With correction 4 hourly electrolyte charting was mandated with ongoing mechanical ventilation. Patient's fluid status was monitored on cumulative fluid balance concept and over next 36 hours dyselectrolytemia improved along with improved respiratory mechanics (Table 2). Concomitantly neurological evaluation carried out and was WNL. Patient was extubated on POD_3 after due weaning and being satisfied with weaning indices. Patient was extended BiPap support for next 4 hours and post that provided O_2 by nasal cannula at 2 L min. In view of adequate recovery and stable hemodynamics he was discharged to home on POD_7.

In our patient the active and prompt response by attending anesthesiologist prevented any adverse neurological or cardiovascular compromise. Since the neurological and other systemic examination was essentially with in normal limits, we attribute the episode of altered sensorium to hypernatremia.

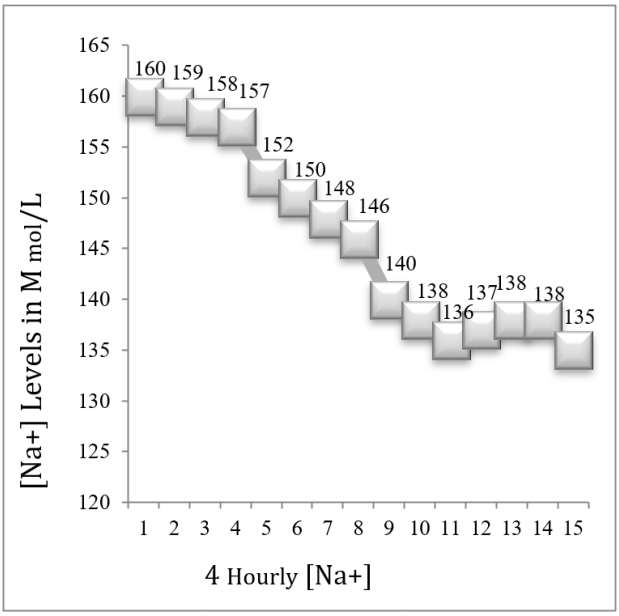

Figure 1: Values of monitored (Na+) electrolyte during the period of convalescence.

Table 1: Resolution of hypernatremia.

Post-op day	Clinical spectrum	Na^+ values	
		0900 H	1700 H
1	Intubated, mech ventilation, CT Head -WNL	160	158
2	Intubated, mech ventilated, Fundus Exmn-WNL	148	140
3	Weaning trial-stable resp mechanics, Extubated, Bi PaP; IPIP:12, EPIP:6	138	135
4	Bi PaP; IPIP:10, EPIP:4, intermittently with O_2 by NP	135	138
5	Bi PaP; IPIP:8, EPIP:4, intermittently with O_2 by NP	136	136

NP-nasal prongs

DISCUSSION

Hydatid disease is caused by *Echinococcus granulosus* that generates cysts, most often in the liver, the lung, and, occasionally, in other sites.[5] *Echinococcus* species of medical importance include *granulosus* and *multilocularis*. *Echinococcus granulosus* causes *cystic echinococcosis* (CE) and *Echinococcus* multilocularis causes alveolar echinococcosis (AE). The consensus for management of AE and CE has been arrived at by experts committee of WHO.[6] The management modalities are tabulated below (Table 2).

Table 2: Image based, stage specific approach for management of AE and CE.

Cystic echinococcosis (CE)	*Alveolar echinococcosis* (AE)
Percutaneous treatment	Early diagnosis and radical surgery followed by anti-infective prophylaxis with albendazole
Surgery	
Sterilizing drug treatment	
Wait and watch	

Surgical removal of the intact cyst is the preferred form of therapy. Conservative approaches include sterilization of the cyst contents with various scolicidal drugs, Formalin, hydrogen peroxide, silver nitrate, cetrimide, absolute alcohol, povidone iodine, and hypertonic saline. These agents are efficacious but most can have undesirable side effects limiting their use. A few cases have indicated that sterilizing hydatid cysts using hypertonic sodium chloride carries a risk of resorption with acute hypernatremia.[6] Regular and frequent monitoring of sodium levels during therapy is inescapable need to prevent any catastrophe.[6-8]

In our case, hypernatremia may have resulted from absorption of hypertonic saline through cysts walls and from exchange of both salt and water through the peritoneal membrane. Hypernatremia after a hypertonic saline injection or absorption via a semi permeable membrane is attributed to free water loss and solute movement across gradient. Hypernatremia is classified as per (Na^+) levels mild -146-149 m mol/l, moderate-150-169 m mol/l and severe >170 m mol/l.

Mortality and morbidity due to severe hypernatremia is studied in detail but still remains a topic of interest for clinicians managing them. Even after correction of hypernatremia, the possibility of residual neurological complication can't be ruled out completely and requires frequent monitoring. Various neurological complications occurring due to hypernatremia can be attributed to: acute hypernatremia-leading to reduction in brain water content and associated dehydration of cerebral cells with brain shrinkage may result in intracranial vascular damage and subdural hematomas.[9]

Brain demyelinating lesions (central pontine and/or extrapontine myelinolysis) are a well- known complication of rapid correction of pre-existing hyponatremia.[10]

In response to hypernatremia-Brain undergoes adaptive responses to minimize osmotic shrinkage. Intracellular accumulation of osmoles tends to counteract brain water loss. This protective change predisposes the patient to cerebral edema which may arise from failure of the osmoles to dissipate at the same rate as hypernatremia during treatment.[9-10]

Hypernatremia correction-based on experimental models and available guidelines and literature optimal rate of correction of hypernatremia is by decreasing (Na+) by 0.5 m mol /l/ h by use of suitable fluid and calculation of total body water deficit. The same was adhered to in our case. Peritoneal dialysis or hemodialysis may be used, although they have not been shown to improve survival benefits.[3] Such modalities can be of use while adopting natriuresis based approach and since exogenous sodium excess was thought to be the cause, active urinary sodium depletion combined with hypotonic fluid therapy were used to restore (Na) to normal in 36 hours. In our patient early identification and aggressive management together with young age was the positive outcome determinant.

CONCLUSION

Iatrogenic hypernatremia post irrigation with hypertonic saline in cases of hydatid cyst can cause severe hypernatremia and can be cause of significant mortality and morbidity with neurological sequalae. This case highlights the significance of prompt and effective management of hypernatremia and complete resolution without any residual neurological deficit. Any case being administered hypertonic saline must be monitored carefully and religiously for any electrolyte disturbance with high suspicion for hypernatremia in post op period.

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