Subdural Haematoma in an Adult Due to Hypernatraemia

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Abstract
Neurological manifestations are quite common in hypernatraemia but subdural haematoma due to hypernatraemia is very rare in adult population. We report a case of 50 year old female patient who presented with acute acalculus cholecystitis who subsequently developed persistent hypernatraemia with multiple subdural haematomas and patient died. Patient died because of persistently raised intracranial tension, before she could be taken up for surgical evacuation of subdural haematoma.

Introduction
Hypernatraemia is defined as an increase in the plasma sodium concentration more than 145 mmol/l. Hypernatraemia can develop after loss of water via both renal and non-renal routes. Symptoms of hypernatraemia are predominantly neurological. Neurological manifestations of hypernatraemia are subarachnoid haemorrhage, intra-cerebral haemorrhage and subdural haematoma.

Case Report
A 50 year old female patient reported with complaints of pain in right hypochondrium radiating to back and was associated with high grade fever with chills of 2 days duration prior to admission. Patient also had 2 episodes of non-projectile vomiting containing food particles and giddiness on the day of admission.

No similar history in the past, clinically patient was febrile with temp 101°F, Pulse 110/min regular low volume; B.P: 80/50 mmHg; R.R: 40/min; SpO₂: 96% at room air; jugular venous pressure: normal. No pallor, icterus, cyanosis, lymphadenopathy seen and no signs of dehydration. Abdominal examination revealed soft abdomen with generalised tenderness more so in right hypochondrium, murphy’s sign was negative, no organomegaly, and bowel sounds normally heard. Chest examination revealed reduced movements on right infra-axillary and infra-scapular regions with stony dull note over the above areas along with diminished vesicular breathing; no pleural rub or any adventitious sound was heard. Cardiovascular system was normal. Neurologically she was conscious, oriented, no neurological deficit.

Investigations: ESR : 30 mm in 1 hr, Haemoglobin: 12 gm%, Total leucocyte count : 14,000/cumm, Platelet count : 1.8 lakhs/cumm, Blood sugar level-random : 98 mg%, Peripheral blood smear : normocytic and normochromic, C-reactive protein(qualitative) : positive, Liver fuction tests and Renal function tests were within normal limits, Blood culture : no growth. Urine routine was within normal limit, S. Sodium : 136 mmol/l and S. Potassium : 4 mmol/l.

Chest X-ray P/A view: Homogeneous opacity in right lower zone with obliteration of costophrenic angle, suggestive of right sided pleural effusion. USG abdomen and thorax: Showed acalculus cholecystitis with peri-gallbladder fluid collection with right sided pleural effusion.

Treatment: she was managed as a case of acute acalculus cholecystitis with Ryle’s tube aspiration, IV fluids, parenteral antibiotics and surgical consultation was sought and they advised conservative management as the diagnosis of acalculus cholecystitis
in this patient was established 72 hours after the onset of symptoms.

On day-3, patient was afebrile; Pulse: 88/min; BP: 120/80 mmHg; RR: 20/min; P/A: soft slight tenderness in right hypochondrium, bowel sounds present. CNS: conscious and oriented no neurological deficit. R/S: was normal. Total leucocyte count: 7,000 /cumm; Renal functions were normal; S. Sodium 142 mmol/l; S. Potassium 4 mmol/l. Patient responded to conservative management so surgical intervention at that time was deferred. Conservative management was continued; Ryle’s tube aspiration was continued as the patient did not tolerated oral fluids.

On day -6, patient developed double vision and generalised headache. On examination patient was afebrile; Pulse: 68/min; BP: 130/90 mmHg; RR: 20/min. CNS: Conscious, oriented. Partial ptosis present on left side with weakness of all extraocular muscles supplied by 3rd nerve, B/L pupils : normal size reacting to light, Fundoscopy showed no papilloedema, No signs of meningeal irritation. No motor-sensory deficit. Blood sugar level-random-101 mg%; S. Sodium 167 mmol/l; S. Potassium 4.2 mmol/l. patient was encouraged to drink plenty of salt free oral fluids. Ryle’s tube aspiration was stopped.

On day -7, patient was drowsy, Pulse: 50/min regular; BP: 140/90 mmHg; Neurologically patient was drowsy but arousable easily, dilated normally reacting pupil on left side, complete ptosis on left side, bilateral plantars were mute and other previous findings persisted. Serum sodium was 160 mmol/l, CT brain showed multiple subdural haematomas with midline shift to the right side (Figures 1, 2). Patient was given IV Mannitol and IV Phenytoin Sodium and neurosurgical consultation was sought. But patient’s relatives were not afforded for surgery and so were advised referral to Govt. Medical Institute. Patient relatives refused for referral despite explaining them all the consequences for not going for surgical intervention.

On day -7, patient was deeply comatose, Pulse: 40/min regular; BP: 80/50 mmHg; R.R: 6/min. Patient was not able to maintain saturation while breathing 100% oxygen. Patient was intubated provided ventilator support and was given vasopressor support also. ECG: bradycardia with sinus rhythm, GCS was poor, B/L pupils dilated not reacting to light, and Brain stem reflexes were absent. Serum Sodium: 154 mmol/l. Patient expired.

Discussion

Hypernatraemia is not an uncommon electrolyte abnormalities but development of subdural haematoma in hypernatraemia is quite rare in adults as compared to neonates. In this case cause of hypernatraemia is due to water loss via Ryle’s tube aspiration, vomiting and fever. Gastric juice and saliva contain 99.5% water and 0.5 % is inorganic and organic substances.

Patient had no present/ past history of head trauma and sodium levels were normal on admission and there was no neurological deficit on admission. Patient developed sudden neurological deficit after raised serum sodium levels suggesting raised sodium levels as its cause.

In infants incidence of subdural haematoma secondary to hypernatraemia is rare, few cases have been reported by Mocharla’s, Herzberger’s, Luttrell and Finberg, Finberg. Mocharla’s reported one case, Herzberger’s reported one case, Luttrell and Finberg reported one case, Finberg also reported development of subdural haematoma in hypernatraemia in animal model study.

Search of literature did not reveal any case report of subdural haematoma in adults. The symptoms of hypernatraemia in these studies were disturbance
of consciousness, headache, hyperreflexia, muscle twitches, skeletal muscle rigidity, seizures, coma and death.4-6 Our case presented with disturbance in consciousness, 3rd cranial nerve involvement, coma and death. The main cause of hypernatremia in these studies were vomiting, diarrhoea, fever, dehydration, meningitis, diabetes insipidus and high salt water intake.4-6 Our patient had vomiting, fever, sweating and ryles tube aspiration as its cause. The other neurological abnormalities associated with hypernatremia were thrombosis of the superior, longitudinal and other dural sinus and cortical venous thrombosis,5 subarachnoid haemorrhage,6 subependymal vessels and intraventricular haemorrhage.6

Sudden shrinkage of brain cells in acute hypernatremia may lead to parenchymal or subarachnoid haemorrhages and subdural haematomas, due to tear of cortical veins.1

Subdural haematoma is also caused by direct cranial trauma, anticoagulation medication, thrombocytopenia and leukaemia.

Main diagnostic investigation for subdural haematoma is non contrast CT brain. Our patient had multiple subdural haematomas with midline shift on non contrast CT brain scan.

Patient requires neurosurgical intervention, burr hole or an emergency craniotomy for evacuation of subdural haematoma.

Conclusion

Though hypernatremia is a common electrolyte abnormality but it can cause some serious and rare neurological manifestations and subdural haematoma is a very rare manifestation and can occur even in adult population apart from paediatric age group. This case highlights the rarest occurrence of subdural haematoma as a manifestation of hypernatremia in adults.

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References