CASE REPORT

A woman with focal neurological deficit following treatment for cholera

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Abstract

A 41-year old woman was treated for cholera at one of the health centers in Blantyre. Two days after discharge from the treatment unit, she developed weakness of all 4 limbs and difficulties with speech. She was referred to the Queen Elizabeth Central Hospital. A CT scan of the brain showed hypodense lesions in the pons. A diagnosis of central pontine myelinolysis was made. She recovered slowly and was discharged from hospital 17 days after admission.

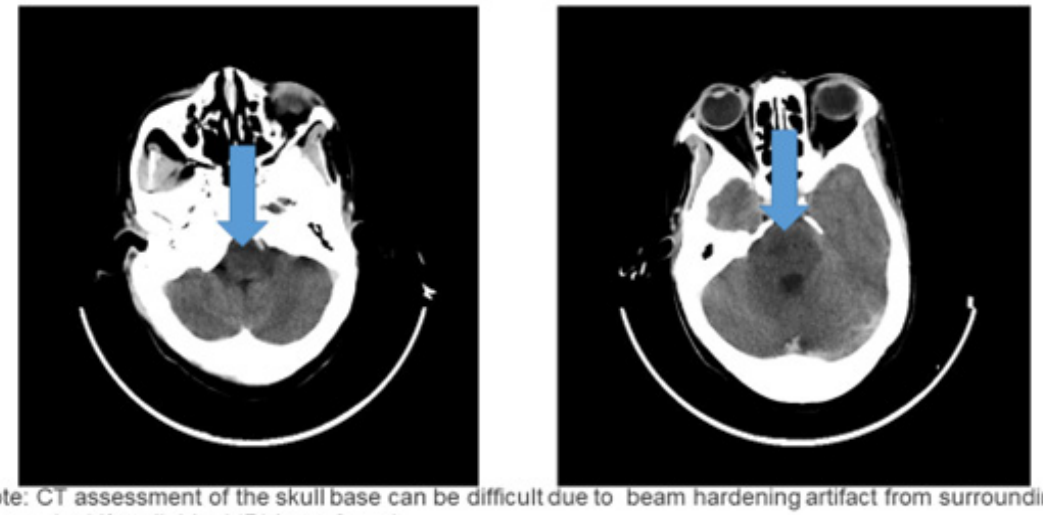
Case description

A 41-year old woman was admitted to the Queen Elizabeth Central Hospital with a 5-day history of weakness of both upper and lower limbs. She had earlier presented to one of the health centers within the City of Blantyre with a 3-day history of diarrhoea and vomiting. A diagnosis of cholera was made. She was admitted to this cholera camp for treatment. She was discharged a few days later seemingly well. Two days after leaving the cholera treatment camp, the patient's mother noted that the patient's speech was not as clear as it used to be. Within a day, the patient was unable to walk unaided and later was unable to walk at all and became mute. At this point her relatives brought the patient to the Queen Elizabeth Central Hospital.

There was no significant past medical history. She did not suffer from diabetes mellitus, or hypertension, she had not lost weight and she did not abuse alcohol. She did not take illicit drugs. The HIV test was negative at the time of presentation to the hospital. She was on no regular medication. On examination, she was not talking, she had features of upper motor neuron lesion with a power of 2/5 in all four limbs. She had horizontal nystagmus. Other cranial nerves were normal. The admitting officer thought the patient had developed a stroke secondary to severe dehydration.

Initial investigations showed a normal full blood count (Table 1), a normal urea and creatinine, a normal sodium but a low potassium (Table 2). Urinalysis was unremarkable (Table 3).

Two images from the patient showing hypodense pons suggestive of myelinolysis: Blue arrows



Note: CT assessment of the skull base can be difficult due to beam hardening artifact from surrounding bones. And if available, MRI is preferred.

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The patient was started on oral replacement of potassium and the physiotherapists were asked to come and review the patient. A CT scan was requested and is shown above.

Radiologist Report (Arthur Daire)

Images not of great quality in the given format.

However, there is some low density involving the pons. Given the history, my concern would be central pontine myelinolysis

Progress in the ward: Over the next 14 days, the patient gradually improved, first speech, then power. She was discharged home 17 days later walking albeit slowly with a set of exercises to continue at home.

Table 1: Full blood count report

	value	Reference range
Hemoglobin	13.5 g/dL	11.5-15 g/dL
White cell count	4.1 x10^3 /mL	3.6-8.7 x 10^3 /mL
MCV	84 fl	80-94 fl
Red blood cells	3.9 x 10^6/mL	3.5-5.5 x 10^6/mL

Table 2: chemistry results

	value	Reference range
Sodium	138 mmol/L	135-145/mmol/L
Potassium	3.2 mmol/L	3.5-4.5 mmol/L
Bicarbonate	24 mmol/L	22-28 mmol/L
Chloride	102 mmol/L	100-110 mmol/L
BUN	20 mg/dL	Up to 50 mg/dL
Creatinine	0.8 mg/dL	0.1-1.4 mg/dL

Table 3: Urine dipstix result

Parameter	Result
Color	Clear
Leukocyte Esterase	Negative
Bilirubin	Negative
Protein	Negative
Glucose	Negative
Blood	Negative
White blood cells	Negative
• Red Blood Cells: ≤2 RBCs/hpf	

Final Diagnosis and discussion

The CT scan findings and the clinical picture were suggestive of a diagnosis of central pontine myelinolysis. Central pontine myelinolysis is defined as the acute demyelinating disease, caused by fast serum osmolality fluctuation, resulting in symmetric demyelination of the central part of the basal pons[1]. Occasionally the disorder may affect the cerebral and cerebellar hemispheres. In this case it is called extra-pontine myelinolysis. The disorder most frequently occurs after a rapid correction of hyponatremia[1]. During hyponatremia, the decrease in serum osmolality results in an osmotic gradient between the intracellular and extracellular spaces. In response, extracellular water by osmosis shifts into cells. This happens in an attempt to normalize the osmotic gradient, thereby causing cerebral edema. Chronic hyponatremia is defined as hyponatremia for more than 48 hours[2]. As part of the adaptive response of the brain to the hypoosmolar state, water will move into the brain tissue, along the osmotic gradient, through the aquaporin-4 channels expressed on the foot processes of the astrocytes, in an attempt to limit osmotic stress injury to the neurons[2]. A rapid correction of chronic hyponatremia, that exceeds the brain's ability to recapture the lost osmolytes, causes an inverse osmotic gradient with a consequent dehydration of brain tissue and possible demyelination of the white matter[2,3].

In the initial description of the condition by Adams and others, the diagnosis was common among alcoholics and those suffering from malnutrition[4]. However, the clinical condition has been described in many situations where severe prolonged hyponatremia is corrected too rapidly, including those with a history of malnutrition, chronic liver disease, liver transplantation and hyperemesis gravidarum[5,6,7,8]. An extensive overview of the disorder has been done by Aunie Danyalian and Daniel Heller[1].

Natural history of the central pontine myelinolysis: Many of the original cases were described at autopsy. Recent data suggest that more than 90% of those with this diagnosis recover and up to 40% make a full recovery[9].

Why this case is important to report?

Our aim is not to describe the pathophysiology of central pontine myelinolysis. There are many good reviews on the this subject in the literature. From our knowledge, this is the first case of central pontine myelinolysis that has been described after correction of dehydration following severe cholera. Malawi in 2022 is experiencing an outbreak of cholera that is unusual in that it has come after the rainy season. At the time of writing this case report, the case fatality rate stands at approximately 3%; much higher than what is expected (less than 1%) when the epidemic is well managed[10] .

Our patient presented to a treatment center after prolonged diarrhea and vomiting. She was diagnosed with cholera; a disease when severe results in profound hypokalemia and hyponatremia[11]. Cholera in Malawi is usually managed at cholera camps. There are no baseline electrolyte assessments at these camps before instituting treatment to correct dehydration. This scenario is not restricted to cholera camps alone. At health centers and many hospitals in Malawi, baseline serum electrolytes are rarely checked before instituting fluid replacement therapy. The health center treating her would not have carried out these baseline tests to determine the level of serum sodium. When she came into our care, her sodium had returned to normal (138 mmol/L) although she still had mild to moderate hypokalemia (3.2 mmol/L) that required correction.

Learning Points

This case reminds us to think about potential complications our cholera patients may develop as a result of the treatment we provide in addition to those complications due to the disease itself. During this epidemic we have seen many patients being admitted to hospital with acute tubular necrosis even after what was thought to be successful treatment at cholera treatment centers. Central pontine myelinolysis is a rare complication of rapid correction of hyponatremia. However, in times of cholera epidemics, this rare complication is likely to be encountered more than usual.

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Therefore, as we treat our patients at the cholera treatment centers, we should consider this complication especially if our patients present with diarrhoea and vomiting that may have resulted in hyponatremia lasting more that 48 hours. In centers where electrolytes can be checked before initiation of fluid replacement, a baseline sodium concentration of less than 20 mmol/L is a major risk factor for this complication. In such situations, it is recommended that correction of serum sodium concentration should not exceed 10 mmol/L/day.

Conclusion

This patient has taught us that we need to provide capacity for baseline laboratory testing in unique circumstances even when there is an epidemic. Our guidelines for managing the current cholera epidemic should include taking a brief history about the duration of the illness to make sure the treatment we provide does not cause harm.

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