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Case report

A Case of Central Pontine Myelinolysis due to Severe Acute Prolonged Hyponatremia in Our County Hospital

Azhar Al-Shaibany [1], Shah Shabahat [1]; Moiz Shahid [2]

Department of Medicine/Endocrinology unit, Our Lady's Hospital, Navan, Co Meath, Ireland [1]
Department of General Medicine, Our Lady's Hospital, Navan, Co Meath, Ireland [2]

Corresponding author: Azhar Al-Shaibany
Email: azharalshaibany@gmail.com

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ABSTRACT

Background: Central pontine myelinolysis (CPM)is unique and rare clinical entity. It could be a neurological complication of prolonged hyponatraemia. Many predisposing risk factors (chronic alcoholism, malnutrition, malignancy) may aggravate the development of CPM. CPM usually have a poor or even fatal outcome. However, CPM does not inevitably have a bad prognosis, and could occur with proper restoration of normal serum sodium concentrations.

Case Summary: Here, we presented a case of a 48-year-oldfemale. She used to drink alcohol. She presented to Our Lady's Hospital/Navan with a history of four days feeling unwell, confused diarrhea, vomiting and by investigations, she had severe hyponatraemia. She was managed optimally in the ICU with close monitoring of her urea and electrolytes. Unfortunately, she got progressive neurological deficits (lower limbs weakness, and slurred speech) irrespective of gentle treatment of hyponatremia in intensive care unit. After Neurological and radiological input from Dublin institute, CPM confirmed under background of alcoholism and malnutrition. Following the diagnosis of CPM, she was discharged home after been rehabilitated with physiotherapy with ongoing recovery.

Keywords: Hyponatremia; Central Pontine Myelinolysis; Alcoholism; Malnutrition

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* Main subject and any subcategories have been classified according to the research topic.

INTRODUCTION

Electrolyte abnormalities are common cases daily encountered during hospitalizations. Hyponatremia one of major electrolytes deficit [the most common electrolyte abnormality] facing clinicians in emergency bases mainly in elderly patients [1]. The causes of hyponatremia are multiple and widely different [2]. CPM is a part from osmotic demyelination syndromes (ODS), which include central- and extra-pontine myelinolysis. ODS are neurological complications of abrupt osmotic fluctuations followed by aggressive serum low sodium restoration to normal levels [3]. ODS is a non-inflammatory demyelinating disorder affecting the pons and other regions of the central nervous system. Although most of the cases, earlier, were mainly attributed to rapid corrections of hyponatremias in certain predisposing factors such as chronic alcoholism and malnutrition, gentle correction of hyponatremia may be potentially associated with ODS. This is attributed to background of the patients, the severity and the duration of hyponatremia. The incidence of ODS has increased, probably due to better quality MRI as CT scan facilities hardly detect ODS in the initial stage [4]. The first case of CPM was reported by Adams et al.in 1959 in a report about four patients due to malnutrition and alcohol use disorder patient [5].

Subsequently, the disease was linked to the rapid correction of abnormal sodium levels, severe burns, after liver transplantation, toxicity, anorexia nervosa, hyper-emesis, bulimia, hyperglycemia, diabetic ketoacidosis [6-11]. The disease manifestations vary and the condition could be presented by manifestations of encephalopathy, coma or even death [12]. Here, we intended to represent a unique case report of Central Pontine Myelinolysis due to Severe Acute Prolonged Hyponatremia in Our County Hospital

The Description of the Case

The patient is a 48 years old female with fatty liver. She was not on regular medications, living with her husband and children. She is an ex-smoker and drinks around 18-22 unit of alcohol/week. She admitted with four days history of feeling unwell, lethargy, anorexia and feeling cold but no fever. Besides, there was a history of watery diarrhea for 3 days and vomiting for two days. On systematic review, she had no cardiopulmonary or urogenital symptoms. No history of seizure or altered mental status.

On Admission, she Looked unwell, confusion/ drowsiness with cold extremities and dehydrated. Blood pressure was 123/61 mmHg, heart rate was 79 beat per minute, temperature was 35.8^{o}C respiratory rate [RR] 20 cycle/minute, oxygen saturation 100.0%, and random blood sugar 7.2 mmol/L.

No lower limb edema or signs of fluid overload. No abdominal or other neurological abnormalities were detected after exhaustive examination.

Table [1] represented the results laboratory and radiological investigations on admission. She was admitted to the intensive care unit and been treated as gastroenteritis. The hyponatraemia was mostly due to regular alcohol consumption and gastrointestinal [GIT] loss. The treatment started by supportive treatment and thromboprophylaxis. Arterial line and urinary catheter inserted. In view of hypovolemic severe acute hyponatremia with confusion/drowsiness, hypertonic saline 3% was initiated in a dose 150 ml/bolus (single dose), then followed normal saline 0.9 % [50-100 ml/hr] during her stay. The treatment aimed to increase Na 0.5 mmol/hr, not more than 5 mmol in the first 5 hours and not more than 10-12 mmol/24 hours. Her Na was monitored during her four days in ICU [Table 2].

In the second day, her blood pressure dropped to 80/50 in ICU, improved with boluses of IV fluids and antibiotics were added as CRP raised and bacterial pathogen suspected after discussion with micro-biologist. Additionally, adrenal insufficiency was suspected clinically (Hypotension, high normal K and low Na levels). Thus, a stress dose hydrocortisone was initiated as short Syanacthen test was not reliable in acute setting. She remained on normal saline 0.9 %, antibiotics and other supportive treatments. Her inflammatory factors improved, her diarrhoea and vomiting were gone. The CT of adrenals revealed normal size, no adenoma and the patient was prepared for MRI brain) after stabilization. The Na level improved from day 5-10 [Table 3, figure 1]. Patient was clinically improved, and medically fit to go home but she had intermittent confusion in in last few days. However, on day 10, patient discharged home upon her request. On discharge day (day 10) her serum levels of urea were

2.3 mmol/L, creatinine [60umol/L], K [4.1 mmol/L], Na [138 mmol/L] and CRP was 8mg/dl. After discharge, she returned to the hospital within 48 hours with generalised weakness, lethargy, and inability to walk without assistance. Clinically, she was not in distress, well hydrated, no signs of encephalopathy, stable vital sings; her neurological examination showed reduced power on both lower limbs grade 4/5 on both with mild scanning speech. Her blood was stable (same as the discharge day). CT brain and angiogram were done, with no abnormality identified. The MRI brain shows two cm symmetrical pontine lesion 2 with no significant enhancement after IV contrast, Figure 2 and 3]. the MRI spine revealed mild degenerative findings which considered normal with her age. The case discussed thoroughly at Dublin Neurology unit (Neurology and Radiology). The conscience diagnosis was CPM secondary to sever prolong hyponatraemia under background chronic alcoholism. Requested for physiotherapy, Speech therapy/ Occupational therapy. During the second admission, short syanacthen test ordered with 48 hrs off hydrocortisone [table 4]. Short Syanacthen test showed good response results. Thus, steroid was stopped and her follow up Na remained with in normal levels after stopping steroid. After physiotherapy and speech therapy, she shifted to rehabilitation unit and she got prolonged ongoing physiotherapy. Finally, she got fair improvement on discharge from hospital.

Table [2]: Investigations on admission

Test	Value	Test	Value
HB	11.6 gm/dl	**PTH**	64 pg/ml
WBCs	11.3 x 10^9/L	**TSH**	2.00 miu/l
Platelets	242 x $10^{\wedge 9}$	**FT4**	12.4 pmol/l
CRP	18 mg/l	**Cortisol**	348nmol/l
Urea	5.4 mmol/l	**Urine spot Na**	<20mmol/l
Creatinine	87 unmol/l	**Urine osmolality**	418
Sodium	101 mmol/l	**Serum osmalarity**	222
Potassium	4.6 mmol/l	**Blood and urine cultures**	No growth after 5 days
ALT	24 u/l	**Stool analysis**	Negative for C difficile, salmonella, shigella, Campylobacter & E coli
GGT	74 u/l		
ALP	94 u/l		
Albumin	29 g/l	**COVID 19 PCR**	Negative
Protein	55 g/l	**ECG**	NSR, nil acute
Total cholesterol	4.1 mmol/l		
Triglyceride	1.8 mmol/l		
Amylase	100ul	**Chest X ray**	reported as normal
Corrected calcium	2.28mmol/l		
Magnesium	0.74 mmol/l		
Troponin	<5.1 --→<5.1 ng/l		
Arterial PH	7.53		
Arterial Co2	1.90 kPa		
Arterial PO2	17.9 kPa		
Arterial O2sat	99.9%		
Arterial Potasium	4.4 mmol/l		
Arterial Sodium	102 mmol/l		
Arterial Cl-	83 mmol/l		
Lactate	5.8 mmol/l		

Table [2]: Urea, creatinine, K and Na flow chart during the first 3 days

	0hrs	2hrs	4hrs	12hrs	24hrs	36hrs	48hrs	60 hrs	72hrs	96hrs
Urea [mmol/L]	5.4	5.3	5.5	5.5	4.5	4.4	4.4	2.8	2.2	2.1
Creatinine [umol/L]	87	83	82	73	67	63	67	64	67	61
K[mmol/L]	4.6	5.2	4.7	4.6	4.6	4.4	4.2	4.1	4.0	3.9
Na[mmol/L]	101	103	102	109	111	115	117	119	120	124

Table [3]: Mean serum sodium levels at the fifth to ninths days

Day 5 Na level	126mmol/l
Day 6 Na level	131mmol/l
Day 7 Na level	133 mmol/l
Day 8 Na level	130 mmol/l
Day 9 Na level	138 mmol/l
Day 10 Na level	138 mmol/l

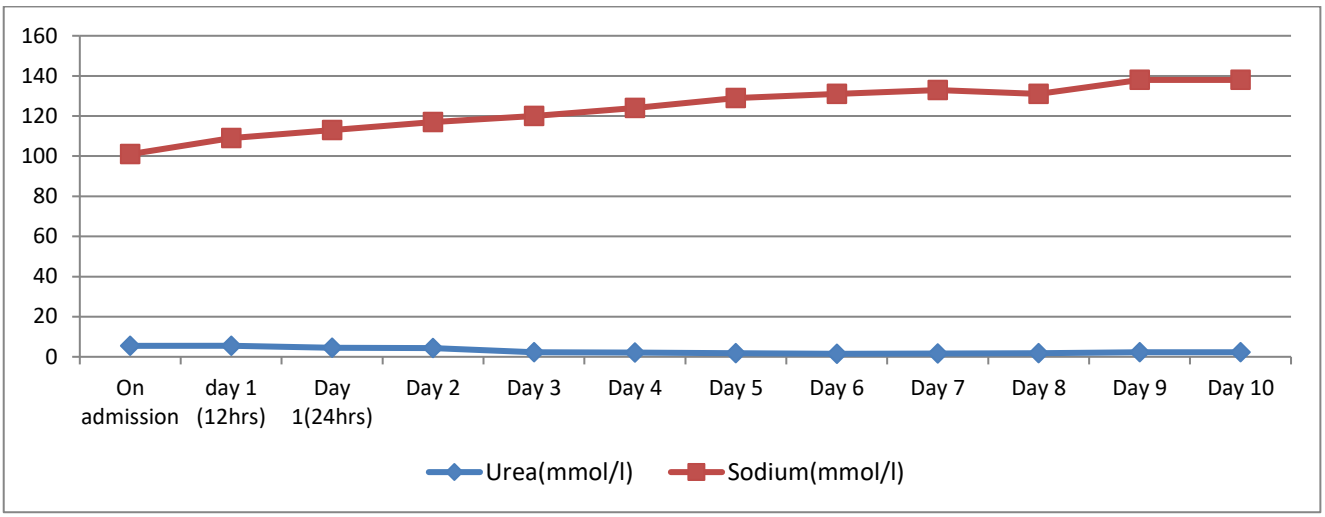

Figure [1]: The trend of Na during her stay.

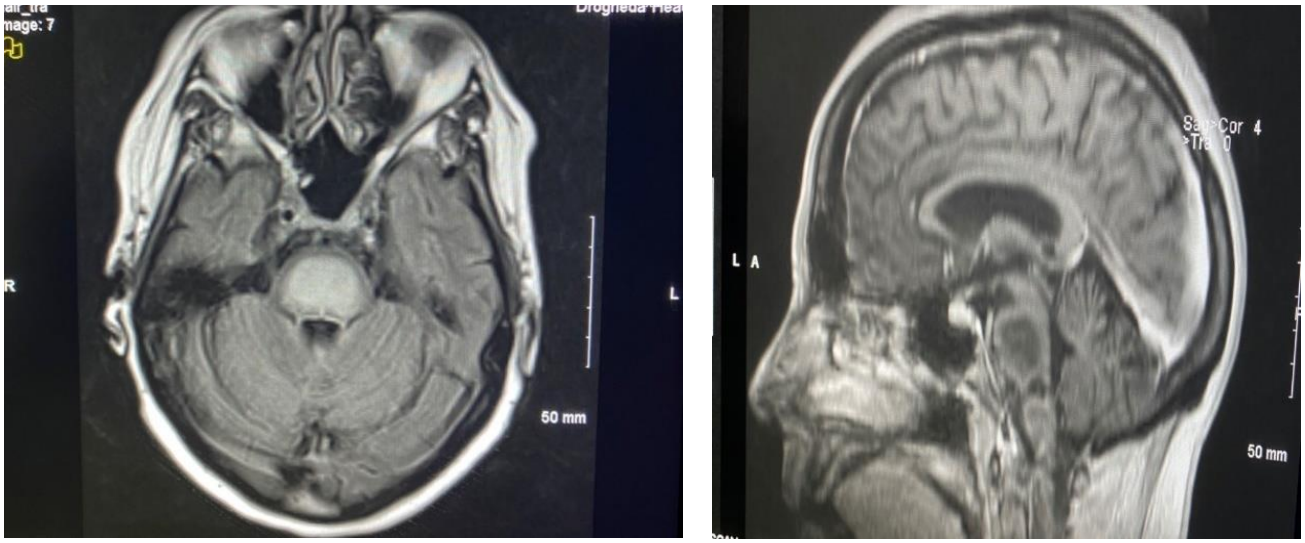

Figure [2]: MRI brain T2 pre contrast

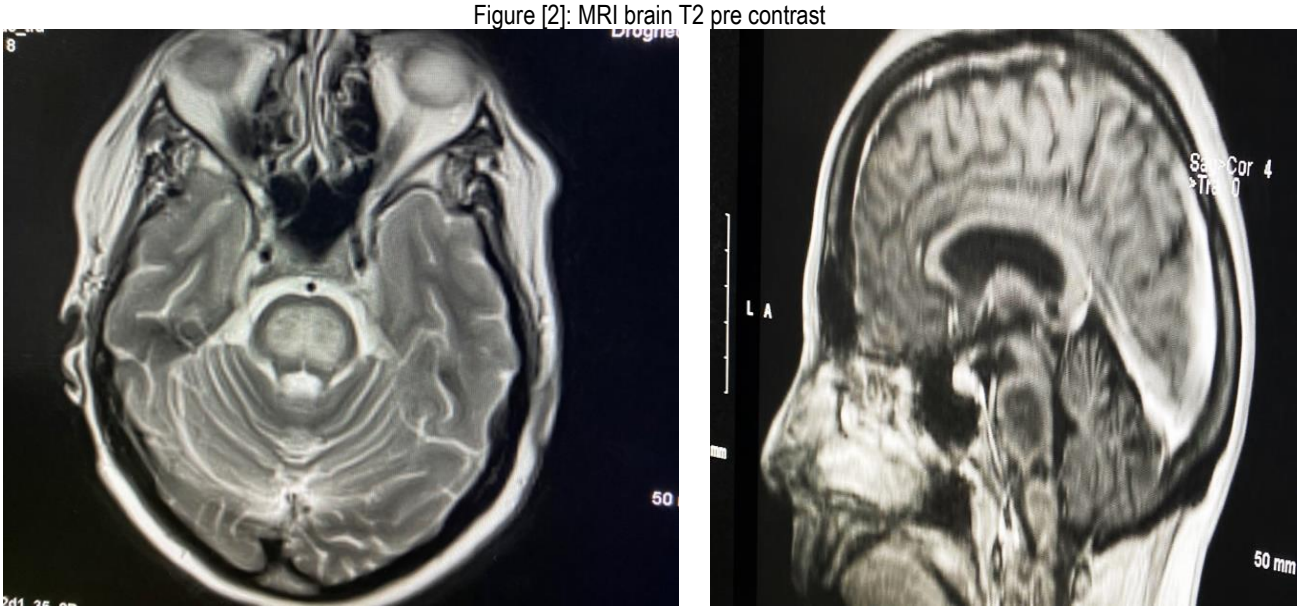

Figure [3]: MRI brain T2 post contrast

Table [4]: Short syanacthen test

Cortisol at 9 AM	334 nmol/l
ACTH	54nmol/l
Cortisol after 30 mins	546 nmol/l
Cortisol after 60 mins	656 nmol/l

DISCUSSION

CPM is an uncommon symmetric demyelination of the central pons. Its prognosis is extremely poor. However, favourable outcome described as survival without significant neurological deficit has been reported in individual cases and ascribed to recent advances in imaging and intensive care tools and technique [13].

CPM was initially reported after fast correction of hyponatraemia in alcoholic patients and was thought to be a complication of alcoholism. However, "there have been occasional reports of CPM not accompanied by over-correction of hyponatremia or extreme alterations in the serum sodium levels during acute phase of treatment [14-16].

In hyponatremia, there is a reduction in serum osmolality, accompanied by entry of water inside the cells with subsequent cellular edema. The neurons are protected from damage by the rich interostial fluid [rich in osmoles]. However, with failure of such protective mechanism, the neurons are exposed to damage especially oligodendrocytes. Pathologically, neural injury is in the form of myelin loss, severe loss of axons and necrosis in severe cases [17]. In addition, rapid fluctuations of osmotic pressure are associated with the damage of the underlying endothelium lining of the blood-brain barrier [BBB]. This exposes the brain tissues to myelinotoxic agents, which are responsible for myelinolysis and vasogenic edema, which leads to compression of the fibre tract areas of both white and grey matter [18-20]. CMP is presented clinical with a wide array of symptoms, included neuropsychiatric disturbances, flaccid paralysis, cognitive abnormalities, confusion, dysarthria, dysphagia, spasticity, looked in syndrome, and movement disorders with extrapontine involvement [12]. The diagnosis of CMP is usually based on clinical suspicion. However, magnetic resonance imaging is the diagnostic modality of choice; it has a higher sensitivity and could detect the CPM lesions early. The classical findings are hypodensity of central pons, evident on a T2- weighted images. It is advised to repeat MRI after two weeks of the onset of suspicious clinical manifestations with absence of specific changes on initial MRI [21].

Although the prognosis of CMP is usually poor, the following factors were associated with a relatively good prognosis: absence of coexisting hypokalemia, mild to moderate hyponatremia, higher coma score at admission and at discharge, and short duration of hyponatremia [22].

In short, we presented our case, where favourable outcome was achieved by gentle and gradual correction of hyponatremia; the probable cause could be attributed to chronic alcoholism with was aggravated by vomiting and diarrhea with malnutrition. The clinical manifestations are among those reported previously in literature and favourable outcome could be attributed to early diagnosis and gentle correction of hyponatremia. Finally, the MRI is the radiological modality of choice for early detection of CMP, that could make a difference in the overall prognosis.

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None

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