



The bariatric route to Central Pontine Myelinolysis

Hypophosphatemia induced by refeeding syndrome causing CPM

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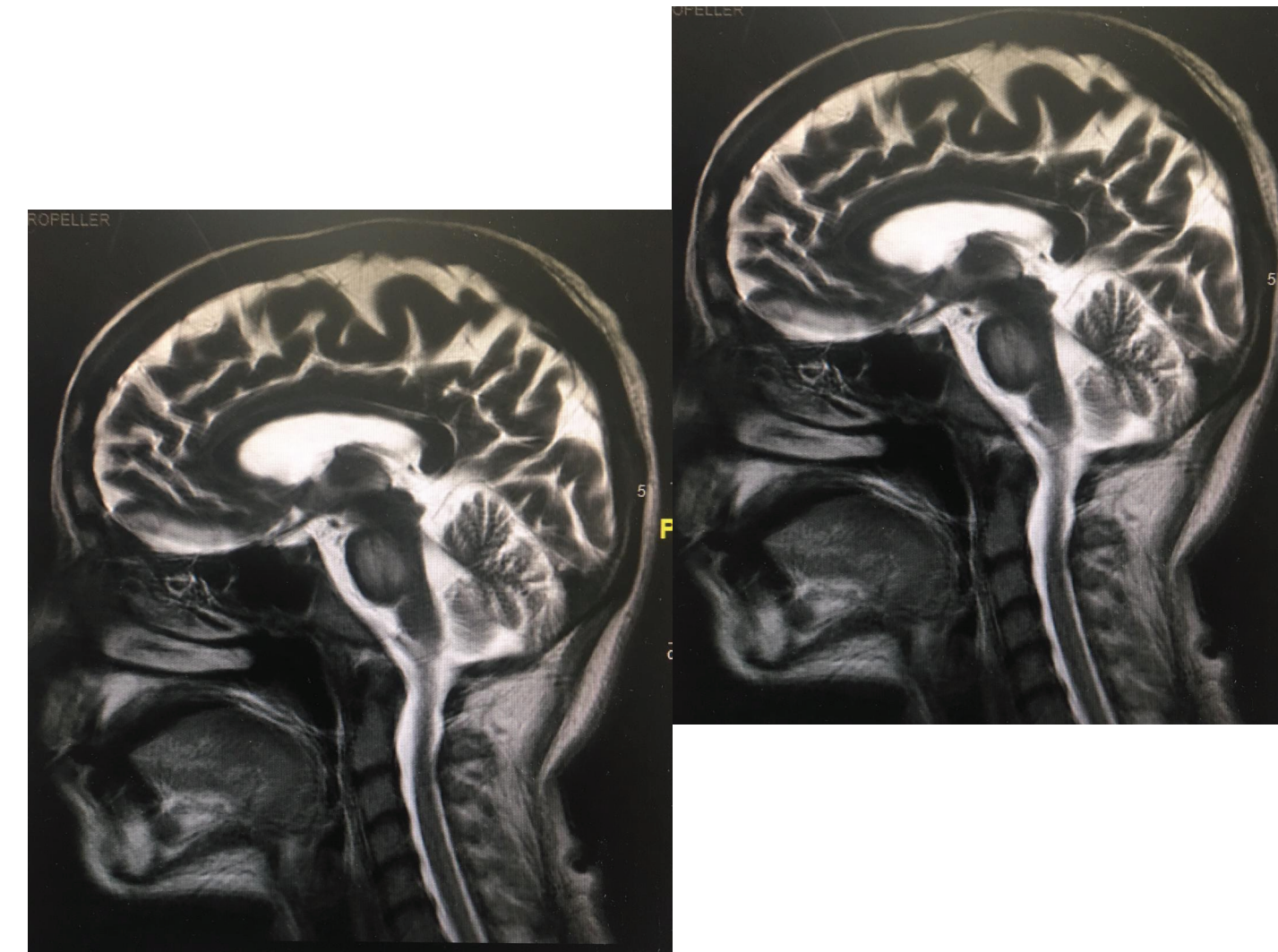
Introduction

- Central Pontine Myelinolysis (CPM) was originally considered to be the result of excessively rapid correction of slowly progressive hyponatremia in patients with chronic medical conditions, such as chronic alcoholism, malnutrition, and malignancy[1, 2]
- However, there have been reports of CPM not accompanied by hyponatremia or drastic changes in serum sodium level[1, 2]
- There has been cases in the literature which show hypophosphatemia, rather than hyponatremia, was suspected to have greatly contributed to CPM [3]
- Here we are presenting a case to showing hypophosphatemia induced by refeeding syndrome in the setting of malnourishment.

Case

- Ms X, is a 31 year old female, 5 months pregnant who presented as a case of malnutrition secondary to multiple bariatric surgeries and complicated by an unplanned pregnancy. She had a Gastric bypass in 2005 and distalization in 2018.
- She was transferred from the Maternity Hospital to Mubarak alKabeer hospital on March 2019 due to severe malnutrition following admission under their care for a month.
- The patient was initially admitted under our medical team in January 2019 due to symptomatic Anaemia and severe Hypoalbuminemia while she was 3 months pregnant. At the time; She was managed with multiple blood transfusions and iron replacement and transferred to maternity hospital for worsening lower limb edema and weakness, as well as multiple skin lesions.
- During her stay at the maternity hospital, she had an unremakable LL doppler and a MRI brain, and fetal U/S showed a showed a single living fetus, 17 weeks in size
- Ms X, was transferred to our care on the 19/3/2019 for multiple active issues: severe malnourishment, shock, fever in a setting of a bariatric patient.
- On examination at the time of transfer the patient was Tachycardic and hypotensive with an altered level of consciousness with a generalised rash all over her body and was admitted to the ICU for management and close observation
- Once she was stable and transferred to the ward. She was then started on TPN (20kcal/kg (1000kcal/day)
- She was also given:
 - Thiamine 100mg IV
 - Evaton D 1 ampule
 - Neurobion Ampule
 - Vitamin A 50 000
 - Albumin 20% 100ml
- A repeat US was done a week later, showed an Intra-uterine fetal death, and the gynecological team carried out evacuation of products of conception on the 6th of April 2019.
- Prior to the evacuation she began experiencing difficulties with articulating words and sentences on multiple occasions associated with bouts of disorientation.
- CT brain showed no acute ischemic brain insult.
- Following the evacuation, her symptoms progressed as she began experiencing lower limb weakness, persistent dysarthria and difficulties with swallowing.
- MRI, as requested by a neurologist, showed Central Pontine Myelinolysis, despite the patient not receiving any sodium corrections since the time of her admission.
- An EMG was done following this diagnosis, which showed; Diffuse axonal demyelination with evidence of denervation in the proximal muscle.
- The cause was as per the neurology team contributed to severe malnutrition.

MRI ad Serum biochemistry parameters



Figures 1 & 2: MRI T2W image showing a hyperintense lesion in the pons

Serum biochemistry parameters

Hospital day	1	2	3	4	5	6	7	8
Sodium (136-149mmol/L)	141	143	144	141	142	141	142	140
Potassium (3.5-5.1mmol/L)	4.0	3.56	4.49	5.00	3.75	3.46	3.10	3.73
Phosphate (0.8-1.45mmol/L)	0.78	0.63	0.54	0.63	0.35	0.71	0.68	0.73
Magnesium (0.77-1.03mmol/L)	0.76	0.86	0.99	0.86	0.82	0.76	0.78	0.76
Albumin (35-52g/L)	21	22	26	24	32	37	38	41

Discussion

- This patient developed CPM without rapid change of serum osmolality
- Although this is not fully understood in the literature. The pathophysiology around what happens can be explained by 2 of the following hypothesis:
 - 1- Hypophosphatemia leads to reduction of ATP production = Lack of energy supply to glial cells = widespread dysfunction of Na⁺ /K⁺- ATPase pumps resulting in apoptosis[4]
 - 2- Cell protective organic osmolytes such as: Phosphocreatine and Glycerophosphorylcholine, are not being produced as phosphate is required for their synthesis[5]
- Hypophosphatemia, caused by redistribution of phosphate, which is a hallmark feature of refeeding syndrome[8]
 - Eventually leading to CPM
- Pre existing Hypokalemia in our patient may also have contributed to her diagnosis
 - The correlation of hypokalemia causing CPM, is due to the fact that it results in an increase in the potential gradient against which the ATPase must operate [11]
- She also had a severe Thiamine deficiency which was over-corrected in our case
 - Possible risk factor for CPM [12, 13]

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