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Abstract:	The report described below is of an elderly patient who developed central pontine myelinolysis (CPM) during the course of her inpatient management for chronic electrolyte abnormalities in a resource-poor setting, with deterioration of her clinical status.

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Abstract: The report described below is of an elderly patient who developed central pontine myelinolysis (CPM) during the course of her inpatient management for chronic electrolyte abnormalities in a resource-poor setting, with deterioration of her clinical status.

Keywords demyelination; fluid and electrolyte management; hyponatremia; patient safety; stroke

Introduction

Management of electrolyte abnormalities follow standard protocols (1-5). However, care merits to be exercised for detection of these electrolyte abnormalities and correction of the conditions. For example, lack of judicious redressal of hyponatremia may lead to osmotic imbalance, which may cause extensive demyelination, including pontine/extrapontine myelinolysis (6-7). The report described below is of an elderly patient who developed central pontine myelinolysis (CPM) during the course of her inpatient management for electrolyte abnormalities, with deterioration of her clinical status.

Case Report

An online consultation was sought by the son of a 79 year old patient with multiple neurologic complaints that arose on an acute basis during the course of a short term nursing home stay. The initial presenting features were those of complete lower limb paresis, which prevented mobility and even getting out of bed. However, there were no complaints of cognitive deficits.

The week prior, the patient was admitted to a local nursing home with complaints of weakness. Upon admission, serum electrolytes were: Na 123.3 meq/L, K 2.4 meq/L, Ca 9.4 mg/dl; random blood sugar 105 mg/dl, blood urea 28.3 mg/dl, creatinine 1.1 mg/dl, total protein 6.9 g/dl, albumin 3.9 g/dl, globulin 3 g/dl, total bilirubin 1.2 mg/dl(unconjugated, 0.4 mg/dl, conjugated 0.8mg/dl), AST 22.2 IU/L, ALT 24.9 IU/L, alkaline phosphatase 115.9 IU/L. Lipid profile showed cholesterol 210 mg/dl, HDL 45 mg/dl, LDL 137 mg/dl and triglyceride 140 mg/dl. Routine urine examination could not detect sugar. 2D echocardiography revealed concentric LVH with left atrial dilation and normal other parameters, without any intra-chamber clot. CBC was normal, except lowered packed cell volume (35.4%). Abdominal USG was normal, except for two small cortical cysts detected in the left kidney.

The patient was “aggressively” replenished with fluid and electrolytes over the next two days. Repeat electrolyte assay performed after 3 days of admission showed serum NA 137.4 meq/L and K 3 meq/L. EKG obtained upon admission showed RBBB and T wave inversion in all leads, possible resulting from hypokalemia.

During the course of the hospital stay, her symptoms of weakness worsened, even preventing her from getting out of bed. She had additional complaints of vertigo and nausea and had falls. Previously, she was able to walk with assistance of cane. The emergent bilateral lower limb paresis prompted a MRI study of the brain. T2 amplified signal hyperintensity was observed in the central pons in a trident shaped fashion. FLAIR sequences revealed signal hyperintensity in the bilateral periventricular white matter. Ventricles were prominent in appearance, though no midline shifts were seen. Sulcal and cisternal spaces were also prominent. No occlusion, thrombosis or dissection were seen in the arterial system. The white matter changes were suggestive of Fazeka‘s grade I ischemia and also potentially age-related atrophic changes. Noteworthy was the image appearance of the pons, suggestive of central pontine myelinolysis (CPM). This likely could have resulted from the overenthusiastic replenishment of the electrolytes, especially sodium, soon after her admission.

Due to logistic reasons, the patients was shifted from the nursing home to her home. Over the course of the next three weeks, there was progressive deterioration of her status. The weakness worsened, completely making her bed-ridden. Around the end of the second week after her discharge, the patient developed marked anorexia and even refused her staple rice diet. She was put on semisoft food supplement and milk. Though specific complaint of dysphagia could not be obtained, she developed cough, which made swallowing difficult. She, however, had no complaints of bolus retention in the oral cavity. Antibiotics were prescribed for her cough.

Additionally, she developed diarrhea. Her cognition, including speech, was intact all throughout this period. The patient passed away peacefully in the next week.

Discussion

Though it is common knowledge, care should be exercised regarding the correction of hyponatremia (7-9). In all likelihood, the rapid correction of hyponatremia led to the development of CPM, and consequent pyramidal and bulbar involvements. This necessarily may not have contributed to the ultimate mortality.

Though pieces of her history is incompletely known, her current medication list included glimepiride, with a chronic history of diabetes mellitus. We could also elicit information from her past history of several episodes of occurrence of delirium and convulsions, which were incompletely followed-up and for which she was then advised psychiatric consultation. Though hyponatremia was detected during all those past episodes (since the last seven years), the root cause was never endeavored to be determined. The hyperosmolar state of diabetes is associated with hyponatremia (8). The chronic diabetes could have resulted in frequent serum electrolyte abnormalities, with resultant deliria episodes (9). Whether there was a lung lesion or hypoadrenalism is not known. The patient belonged to a semiurban town in the eastern part of India, where resources for assessment of serum electrolytes are not always available. Additionally, blood samples are sent to distant metropolis, without adequate care to sample storage, often resulting in erroneous reads. During replenishment of serum electrolytes, the requirement to obtain serial measurements cannot be overemphasized.

The hypokalemia may have potentially resulted from chronic diarrhea. The reasons for her chronic diarrhea remains unexplored, though diabetes, fecal incontinence, antibiotics, infections from her nursing home stay or a combination of all of these may have contributed. The

exacerbation of vertigo and nausea during the terminal illness may have resulted from vertebrobasilar insufficiency, either arising from the acute brainstem lesion (visual image of brainstem lesion involving NTS/area postrema could not be documented on the MRI) or chronic ischemic changes as evidenced from the brain MRI findings. The nausea could have contributed to the deep anorexia during the terminal phase of illness, impairing food intake and creating a vicious cycle of electrolyte deficiencies.

The presented case highlights several learning points: the need for comprehensive metabolic assessment upfront, careful inpatient management of serum electrolytes, providing high quality chronic care for diabetes patients, optimizing care for the elderly populations, ruling out organic causes of delirium, the options for tele-healthcare and taking into account complex social and logistic issues that are involved in care of elderly patients.

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References

1. Czoski-Murray C, Lloyd Jones M, McCabe C, et al. What is the value of routinely testing full blood count, electrolytes and urea, and pulmonary function tests before elective surgery in patients with no apparent clinical indication and in subgroups of patients with common comorbidities: a systematic review of the clinical and cost-effective literature. Health Technol Assess. 2012 Dec;16(50):i-xvi, 1-159.

2. Pfennig CL, Slovis CM. Sodium disorders in the emergency department: a review of hyponatremia and hypernatremia. Emerg Med Pract. 2012 Oct;14(10):1-26.

3. Ganguli A, Mascarenhas RC, Jamshed N, Tefera E, Veis JH. Hyponatremia: incidence, risk factors, and consequences in the elderly in a home-based primary care program. Clin Nephrol. 2015 Jun 3.

4. Lunøe M, Overgaard-Steensen C. Prevention of hospital-acquired hyponatraemia: individualised fluid therapy. Acta Anaesthesiol Scand. 2015 May 9.

5. Corona G, Giuliani C, Verbalis JG, Forti G, Maggi M, Peri A. Hyponatremia improvement is associated with a reduced risk of mortality: evidence from a meta-analysis. PLoS One. 2015 Apr 23;10(4):e0124105.

6. Hepp P, Jüttner T, Beyer I, Fehm T, Janni W, Monaca E. Rapid correction of severe hyponatremia after hysteroscopic surgery – a case report. BMC Anesthesiol. 2015 Jun 9;15:85.

7. Podestà MA, Faravelli I, Cucchiari D, et al. Neurological counterparts of hyponatremia: pathological mechanisms and clinical manifestations. Curr Neurol Neurosci Rep. 2015 Apr;15(4):18.

8. Liamis G, Liberopoulos E, Barkas F, Elisaf M. Diabetes mellitus and electrolyte disorders. World J Clin Cases. 2014 Oct 16;2(10):488-96.

9. Rai D, Garg RK, Malhotra HS, et al. Acute confusional state/delirium: An etiological and prognostic evaluation. Ann Indian Acad Neurol. 2014 Jan;17(1):30-4.