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Case Report Open Access

Iatrogenic Central Pontine Myelinolysis: A Case Report

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Abstract

Central pontine myelinolysis is disabling neurological disorder with diverse etiologies. Severe hyponatremia with rapid correction is one of the commonest causes described in literature. Involvement of extrapontine structures like thalamus, basal ganglia and cerebellum has also described. A slow correction of hyponatremia should be done in symptomatic patients to avoid potentially devastating neurological sequalae!

Keywords: Central pontine myelinolysis; Extra pontine myelinolysis; Hyponatremia correction

Introduction

Adams et al. first used the term "central pontine myelinolysis" as a symmetric, demyelinating disorder involving the central pons [1]. In their description of 4 patients the pons was the only region implicated and their all patients were chronic alcoholics. It was eventually recognized that demyelinating lesions can also be seen outside the pons, termed extratrapontine myelinolysis [2,3]. So, osmotic demyelination syndrome encompasses both Central Pontine Myelinolysis (CPM) and Extra Pontine Myelinolysis (EPM).

Case Report

A 54 year old hypertensive male presented in emergency department with generalized weakness, altered behavior, jerky movement involving upper limbs and trunk and slurred speech of 15 days duration. On asking, his relatives admitted that one month back he developed fever of a few days duration and multiple episodes of vomiting followed by altered sensorium. With these symptoms he was admitted at some private hospital where his cerebrospinal fluid examination was found to be normal. He was diagnosed as having a metabolic encephalopathy due to hyponatremia and 3% saline is infused leading to improvement in his sensorium. A week later when he was going to be discharged he again developed confusion and altered level of consciousness when he was referred to us. On examining his treatment records we found that his presenting serum sodium levels were 98 meq/L , which was rapidly corrected to 121 meq/L within 12 hours.

Patient was in altered sensorium, agitated with coarse jerky tremor like movement at rest involving the trunk and upper limbs with symmetrical cogwheel rigidity over both upper limbs and lower limbs. His vital were normal as was his routine blood investigations including serum sodium (134 meq/L). On MRI head there was a relatively well defined T2 hyperintense area involving the bilateral basal ganglia, genu of corpus callosum and pons sparing the peripheral corticospinal tract showing no evidence of restriction which was quite suggestive of central pontine and extrapontine myelinolysis (Figures 1-3). He was given symptomatic treatment for rigidly and tremor. His sensorium gradually improved but patient had quadripareisis with muteness, cogwheel rigidity and rest tremor predominantly involving upper limbs.

Discussion

(CPM/EPM) may occur in certain conditions such as alcoholism, malnutrition, after prolonged diuretic use, prolonged vomiting, burns, chronic psychogenic polydipsia, after liver transplant, and rarely, after pituitary surgery and after urological surgery/gynecological surgery, especially those requiring glycine infusions [2]. Studies show that (EPM) can occur with or without (CPM): in a necropsy series of 58 cases by Gocht and Colmant, isolated (CPM) was present in about half, (CPM) with (EPM) in about three fifths, and isolated (EPM) in about two fifths of cases [4]. They described cerebellum and lateral geniculate body as the most commonly affected sites in (EPM).

In 1976, pontine and extrapontine myelinolysis were first found to be associated with rapid correction of low serum sodium levels [1,2]. The sequelae of rapid correction of hyponatremia usually follow a biphasic clinical course [5], with initial encephalopathy or seizures from hyponatraemia, followed by a rapid recovery as normonatremia is restored, with deterioration several days later. In (CPM), the initial signs include dysarthria and dysphagia (secondary to corticobulbar fiber involvement), and flaccid quadriparesis (from pyramidal tract involvement) which later becomes spastic, all from involvement of the basis pontis. If the lesion extends into the tegmentum of the pons, pupillary and oculomotor abnormalities may occur. There may be changes in the level of consciousness [6]. EPM has identical pathological findings but the involved areas are extrapontine, so there are different

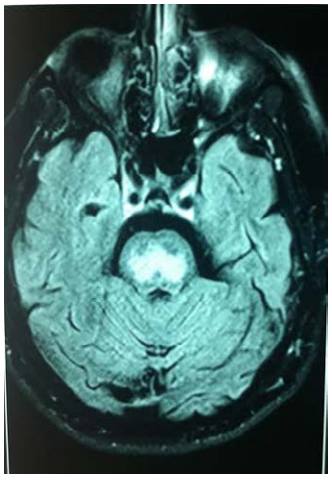

Figure 1: T2 Flair T2 hyperintisities pons.

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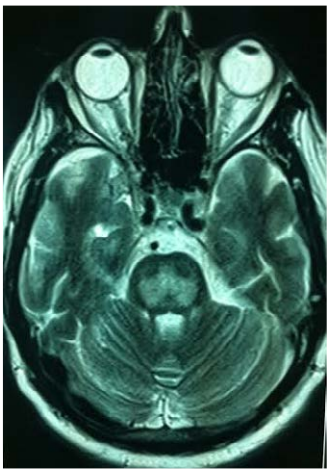

Figure 2: T2 hyperintensities pons.

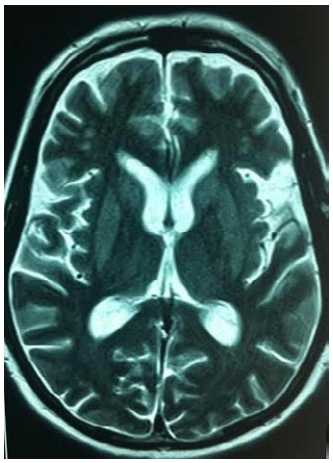

Figure 3: T2 hyperintensities basal ganglia.

clinical manifestations [6]. Because (EPM) is rare, its manifestations continue to attract study. Different types of movement disorders have also been described in (EPM), including mutism, parkinsonism, catatonia, dystonia, and tremors [2]. The clinical diagnosis can be challenging if (CPM) occurs in conjunction with (EPM) like it did in our patient.

Conventional imaging findings (MR and CT) typically lag clinical manifestations, limiting the utility of imaging in early diagnosis of CPM. Because myelinolytic lesions may be not demonstrated within the first 2 weeks by using conventional MR imaging pulse sequences, later imaging has been advocated to confirm the diagnosis [5,7]. MRI findings in CPM usually shows symmetric signal hyperintensity in the central pons on T2 weighted and FLAIR imaging. Diffusion weighted imaging can detect changes of (CPM) before FLAIR and T2 sequences [8]. The mechanism of myelinolysis is characterized by intramyelinitic splitting, vacuolization, and rupture of myelin sheaths presumably because of osmotic effects. Macrophages with cytoplasm filled by myelin debris appear after several days [9]. Pathologically, (ODS) is characterized by sparing of axons and neurons, sparse or absent infiltration by lymphocytes, and degeneration or loss of oligodendrocytes [2,10].

Hyponatremia can be acute or chronic. When hyponatremia develops in <48 hours it is called as acute hyponatremia while its chronic if develops over >48 hours. In patients with acute hyponatremia, a 4- to 6-mmol/L increase in serum [Na^+] is sufficient to reverse the most serious manifestations of acute hyponatremia, after which corrective guidelines for chronic hyponatremia are appropriate.

In chronic hyponatremia slower correction is warranted. A 1-day increase of 12 mmol/L/d was initially proposed based on a literature review and observational studies of outcomes in patients with severe hyponatremia [10]. A prospective cohort study of 184 consecutive patients with serum [Na^+] <120 mmol/L confirmed that sequelae were associated with more rapid correction; but, of the 9 patients with sequelae whose serum [Na^+] was measured during the first 24 hours of correction, 3 had been corrected by 12 mmol/L, 2 by 11 mmol/L, and 1 by 10 mmol/L [11]. Similarly, case reports and case series of patients with osmotic demyelination syndrome (ODS) have included a few patients corrected by <12 mmol/L/d. Although there is some evidence that correction by <3 to 4 mmol/L/24 h may be associated with excess mortality in patients with acute or postoperative hyponatremia [12,13], there is no evidence that correction by >6 mmol/L/24 h improves outcomes in acute or chronic hyponatremia. So the current recommendation for the minimum correction of chronic hyponatremia should be 4-8 mmol/L/d for those at low risk of (ODS), with an even lower goal of 4-6 mmol/L/d if the risk of ODS is high [14]. Patients with high risk are those with serum sodium <105 mmol/L, hypokalemia, alcoholism, malnutrition and advanced liver disease. In high-risk patients, correction by more than 8 mmol/L/d should be actively avoided; whereas in patients without major risk factors for osmotic demyelination, correction should not exceed 10-12 mmol/L in any 24- hour period and 18 mmol/L in any 48-hour period [5]. For patients with severe symptoms, the first day's increase can be accomplished during the first 6 hours of therapy, with subsequent increases postponed until the next day. In our patient rate of correction was 23 mmol/L in just 12 hours which is way beyond the safe limit.

Conclusion

Thus, if hyponatremia correction is done carefully osmotic myelinolysis can be prevented. It is recommended that hyponatremia correction should not exceed a rate of 1-2 mmol/L/hour and no more than 8 mmol/L/day. Regular monitoring of serum sodium should be done while infusing 3% saline.

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