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Central Pontine Myelinolysis: About a Clinical Case in Niger and Literature Review

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Abstract

Introduction: Centro pontine myelinolysis or osmotic demyelination syndrome was described in 1959. It is defined as centro-protuberantial edema, two to three days after rapid restoration of hyponatremia. We report a clinical case of centro-pontine myelinolysis in a context of severe hyponatremia, diagnosed in September 2019 at National Hospital of Amirou Boubacar Diallo of Niamey, on the basis of cerebral magnetic resonance imaging (MRI). **Observation:** A 41-year-old man with no previous history consulted the emergency department for acute non-febrile gastroenteritis and altered consciousness. The clinical course was marked by the onset of flaccid tetraplegia and bilateral pyramidal syndrome. Biological tests revealed severe hyponatremia at 96.8 mmol/l, which was corrected to 149 mmol/l in less than 72 hours, and hypokalemia at 2.80 mmol/l. A cerebral MRI was used to diagnose Centro pontine myelinolysis, showing T2-weighted and Flair-weighted hyper signals in the white matter, periventricularly and in the bulb, which progressed favorably under treatment with dexamethasone 8 mg/8 h. **Conclusion:** Centro pontine myelinolysis is a serious pathology that is difficult to manage in our regions.

Keywords

Myelinolysis, Centro Pontine, Hyponatremia, Niger

1. Introduction

As water flows freely across the blood-brain barrier and cell membranes, a fall in serum sodium (in the absence of a compensatory rise in other osmoles) will cause

entry of water into brain cells and consequent brain swelling. Protective mechanisms come into play during the development of serum hypotonicity in all cell types to maintain cell volume, a process termed "regulatory volume decrease". In the brain, the first protective mechanism to act precedes this and is the forcing of interstitial sodium-rich fluid into cerebrospinal fluid (CSF) as a result of hydrostatic pressure [1] [2]. Described in 1959 by the team of Adam *et al.*, centropontine myelinolysis (MCP) is defined as centro-protuberantial (PCM) edema occurring two to three days after rapid restoration of hyponatremia [3]. The first cases were initially reported as a consequence of chronic ethylism and malnutrition, but it was not until 1976 that Tomlinson *et al.* suspected the role of hyponatremia [4]. Laureno R. confirmed the previous hypothesis, but emphasized the speed of correction of hyponatremia [5]. The pathophysiology is characterized by lesions combining edema and demyelination due to osmotic disorders associated with the release of myelinotoxic factors from the highly vascularized gray matter. The most common contributing factors are alcoholism, undernutrition, malnutrition, renal failure, liver disease and organ transplants. Clinical symptoms vary widely. The patient has usually gone through a biphasic clinical course, initially encephalopathic or presenting with seizures from hyponatraemia, then recovering rapidly as normonatraemia is restored, only to deteriorate several days later. The initial signs of the CPM, which reflect this second phase, include dysarthria and dysphagia (secondary to corticobulbar fibre involvement), a flaccid quadriparesis (from corticospinal tract involvement) which later becomes spastic, all from involvement of the basis points, accompanied by a pseudo-bulbar syndrome, and in severe forms, coma. If the lesion extends into the tegmentum of the pons pupillary, oculomotor abnormalities may occur. It is most often evoked by a T2/FLAIR centro-punctate hyper-signal on MRI, in the context of rapid correction of hyponatremia. In asymptomatic chronic hyponatremia, correction should be slow. If extracellular volume is high or normal, fluid restriction alone is often sufficient. When extracellular volume is reduced, sodium depletion is corrected by adding isotonic saline solution. In all cases, correction should not exceed 10 - 12 mmol/l in 12 hours, or 18 mmol/l in 48 hours. Active treatment should always be stopped as soon as the natraemia reaches 130 mmol/l. The treatment of centropontine myelinolysis is poorly codified, relying essentially on general resuscitation measures. However, non-specific therapeutic trials (corticosteroid therapy, Thyrotropin Releasing-Hormone, etc.) have been carried out, with uncertain results. Its prognosis is very guarded, due to the involvement of the brainstem [6]. We report a case of centropontine myelinolysis revealed by severe hyponatremia corrected in less than 72 hours, and a review of the literature.

2. Observation

A 41-year-old man, with no previous history of illness, had been admitted to the medical ward of the Regional Hospitalier Center of Poudrière for a hypertensive attack and an ionic disorder following an acute non-febrile gastroenteritis. The

evolution was marked by the development of altered consciousness, tonic-clonic convulsions and psychomotor agitation, which led to his transfer to the emergency department of the National Hospital of Niamey. Clinical examination on admission revealed an altered general condition with a Glasgow Coma Score (GCS) of 10/15, conjunctivae and mucous membranes well stained, blood pressure 128/95 mim Hg, pulse 94 beats/min, core temperature 36.4˚C, ambient air saturation 93%. Apart from these neurological abnormalities, the physical examination was normal. Given this picture, the hypotheses of encephalitis (bacterial, viral and parasitic) and metabolic disorders were evoked.

Paraclinical examinations revealed uraemia at 0.26 g/l, creatininaemia at 9.13 mg/l, a positive thick drop at 80/µl of parasitic density, glycaemia at 1.15 g/l, severe hyponatremia at 96.8 mmol/l, hypokalaemia at 2.80 mmol/l and hypochloremia at 60.11 mmol/l. Blood count and PL lumbar puncture were normal. Treatment to correct the hyponatremia was instituted, with natraemia rising from 96.8 mmol/l to 149 mmol/l in less than 72 hours. His condition worsened with the onset of tetraplegia. Neurological examination revealed bilateral pyramidal syndrome, locked in syndrome and a Glasgow score of 7, subject to motor scoring. Pulmonary auscultation was normal, apart from bronchial congestion. Cardiac rhythm was regular, with no murmurs or additional noises. Given this symptomatology, the hypotheses of multiple sclerosis (first attack) and centro pontine myelinolysis were put forward. Brain MRI was used to diagnose MCP in the context of hyponatremia, with T2-weighted and Flair-weighted hypersignal lesions in the white matter and T1 hypo signals (Figure 1).

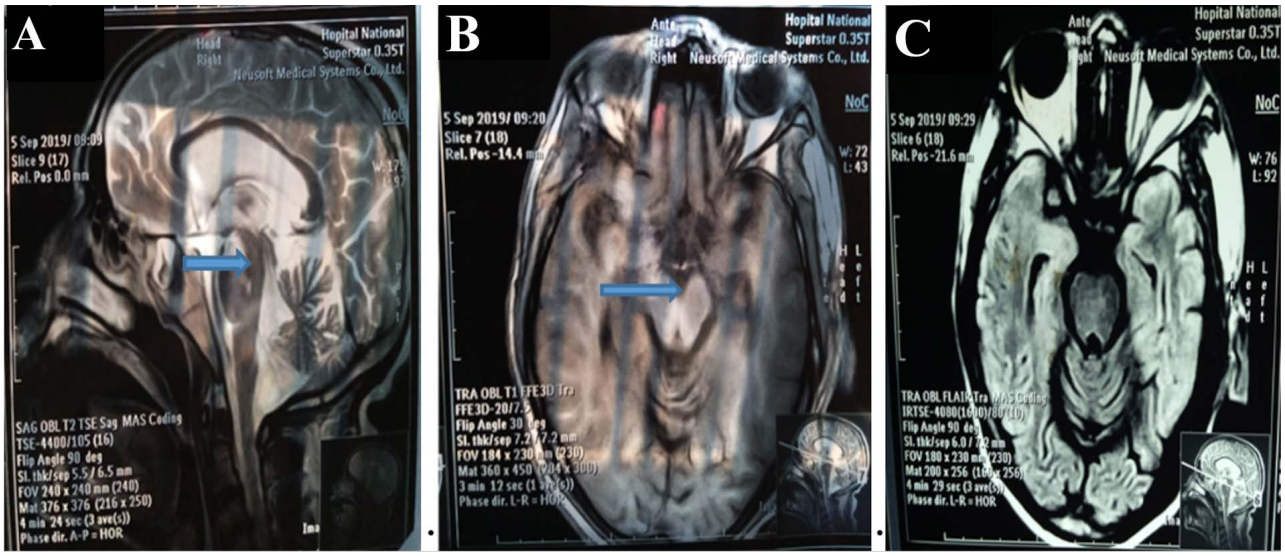

Figure 1. Brain MRI showing a protuberant hypersignal in T2-weighted sequence (A), Axial slice in T1 sequence showing a hyposignal at the level of the pons (B), Axial slice in FLAIR sequence (C).

He was treated with: Phenobarbital 50 mg 2 times/day, Dexamethasone 2 ampules/12 h, Glucose serum 5% 500 ml/24 h, Cimetidine injection 200 mg 2 times/day. The evolution was favorable with partial regression of neurological

signs: Glasgow score from 7 to 15, muscle strength from 0 to 4, without any agitation. Sodium levels are normal.

3. Discussion

This condition is much more common in adults than in children. Several publications [3] [7]-[9] consulted had reported cases in adults such as our patient. The prevalence of this pathology is low [10]. Gautam D. *et al.* [11], reported in 2015 that osmotic myelination syndrome accounts for 0.4% - 0.56% of patients admitted to the neurology department and 0.05% of hospitalizations in general. They also found 0.3% - 1.1% of patients suspected of MCP during autopsies. To our knowledge, no prevalence studies have been carried out in Niger. In a study carried out in Nigeria in 2019, Alkali *et al* found a prevalence rate of 0.5% in certain autopsy series [9]. The course of the disease is highly variable, with the patient's condition stabilizing, improving with or without persistence of a minimal residual deficit syndrome (cognitive and memory disorders, ataxia, spasticity, diplopia) or, in most cases, worsening towards major disorders of consciousness and death [12]. The most recent studies show significant survival and neurological recovery rates, and run counter to an older literature that put forward the notion of appalling mortality, or even a catastrophic neurological prognosis [13]. Mortality rates vary widely, from 6 to 90% according to a study carried out in Nigeria by Alkali *et al.* [9]. In our case, the evolution was favorable under treatment, with partial regression of neurological signs (Glasgow score from 7 to 15, muscle strength from 0 to 4). Jonathan G.R. *et al.* [14], in 2011 reported on 24 patients over an almost identical period, noting a favorable prognosis in 60% of cases and an initial mortality of 8%. In France in 2015 on 36 patients with MCP/MEP, mechanically ventilated in intensive care units, no prognostic factors could be identified. Morbimortality was associated neither with neurological severity, nor with general complications due to neurological severity, nor with certain factors that could increase neurological severity [13]. Diffusion tensor and fiber tracking is a new tool for qualitative assessment of white matter bundles. It can be used to assess the precise location and extent of lesions in the initial workup of centropontic myelinolysis, as well as to monitor the evolution of the disease [12]. According to the various studies carried out, MCP has a variable course, which is favorable in the majority of cases, despite the persistence of certain neurological signs. There are currently no precise prognostic factors. The incidence after correction of severe hyponatremia varies widely in the literature, ranging from 25% to 60% in retrospective studies [13].

In our case, the patient presented with severe hyponatremia at 96.8 mmol/l, which was corrected to 149 mmol/l in less than 72 hours. Experimental studies suggest that chronic hyponatremia in itself does not induce MCP, and that MCP occurs mainly when correction is too rapid. After years of controversy, recent animal studies have confirmed that it is rather the amplitude of correction in the first 24 - 48 hours that is responsible for the occurrence of PCM [14] [15]. Ruiz S. *et*

al. [16] in France in 2009 reported a case of MCP in a context of severe hyponatremia at 107 mmol/l on a background of chronic alcoholism and severe undernutrition (BMI 15.5), and Traverta B. *et al.* [17] reported a case of alcoholism and hyponatremia in 2015, demonstrating that alcoholism and undernutrition are non-negligible cofactors in the occurrence of MCP in hyponatremia. Inadequate correction of severe hyponatremia: speed and amplitude of natremia correction are the suspect factors most often analyzed [16]. It is not uncommon to find cases of MCP due to the combination of hyponatremia and hypokalemia. This was the case in our patient with a hypokalemia of 2.80 mmol/l.

Maulin *et al.* [18], in 2018 had published a clinical case of osmotic demyelination unrelated to hyponatremia, leading them to conclude that a rapid increase in serum sodium concentration and plasma tone even in the absence of hyponatremia can exceed the brain's ability to adapt to hypertonia and lead to osmotic demyelination in predisposed individuals.

Hyperazotemia is the only situation in which rapid correction of hyponatremia is inconsequential for the occurrence of PCM [19]. Murtaza F.D. *et al.* [19], in Pakistan in 2014 on 52 patients with hyperazotemia, who had undergone hemodialysis and hyponatremia treatment, despite the known assumption that rapid correction will cause PCM, no cases of PCM were detected in these patients on MRI. Ben A chour T. *et al.* in Tunisia, in 2018 reported the case of a patient who presented with PCM occurring in a context of major hyperglycemia [20]. Kinda B. *et al.* in Burkina Faso in 2014, reported a case of MCP during quinine treatment by billet of severe hypoglycemia [21]. A case of adrenal insufficiency with hyponatremia complicated by MCP was published in 2018 by Robert *et al.* [22].

In our case, the diagnosis of MCP was made clinically with flaccid tetraplegia, pyramidal syndrome and locked in syndrome, confirmed by MRI showing T2-weighted and Flair-weighted hyper signals in the white matter, periventricular and bulbar areas, and T1 hyposignal. Diagnosis remains essentially clinical and is confirmed by cerebral nuclear magnetic resonance [8]. However, radiological signs may be delayed by up to 4 weeks compared with clinical signs [23]. Cerebral MRI reveals a T2-weighted hypersignal centropontine lesion in the acute phase, corresponding to demyelination that progresses to a T1 hyposignal in the chronic phase. Cases of incidental discovery of centropontine lesions on MRI have been described [24]. Meyer P. *et al.*, reported a case of pauci symptomatic centropontine myelinolysis [24]. However, these abnormalities have a delayed onset and a normal MRI does not rule out the diagnosis [25] [26]. MRI may fail to detect lesions revealed by autopsy. Nevertheless, Cuvellier *et al.* [27], and Okeda *et al.* [28] have described cases of MCP/MEP diagnosed exclusively on pathology. Treatment with steroids, intravenous immunoglobulins, Thyrotropin-releasing hormone (TRH) supplementation or plasma exchange has been proposed in small series or even isolated cases. To date, in the absence of a validated curative treatment, the only option is symptomatic therapy. Recognized aggravating factors need to be counteracted: correction of hypokalemia, vitamin substitution, coun-

teracting hypoxia [13]. A recent animal model study (rat) shows that after rapid correction of chronic hyponatremia, the occurrence of MCP lesions and mortality are reduced by reinducing hyponatremia with hypotonic solutions and vasopressin [15]. Correcting hyponatremia to no more than 0.55 mmol/h would prevent the onset of PCM. Multiple published recommendations, including that of Horacio *et al*, have suggested that the rate of correction should not exceed 8 mmol/L/24 h in chronic hyponatremia [29].

Experimental work suggests that urea or myo-inositol could be interesting therapeutic adjuvants to prevent neurological damage induced by correction of chronic hyponatremia [13]. We can say that PCM has no specific treatment, but everything depends on etiological management, especially hyponatremia, which is the most frequent risk factor. In an experimental study, rats not treated with dexamethasone were exposed to severe neurological disorders, and 77% of them died within 5 days of correction. When dexamethasone (2 mg/kg) was administered intraperitoneally within 6 hours of rapid correction, all rats survived. Dexamethasone treatment was most effective when administered shortly after rapid correction. Dexamethasone administration at 0 and 3 hours, or 3 and 6 hours after correction, had a clearly beneficial effect on preventing demyelination, whereas administration at 6 and 12 hours after correction had no apparent beneficial effect [30].

Our case was treated with dexamethasone 8 mg/8 h, which led to a favorable clinical outcome. Given these experimental results and our own, we can say that dexamethasone is part of the symptomatic treatment that has shown its effectiveness in the management of MCP.

4. Conclusions

MRI is the key examination for the diagnosis of centropontine myelinolysis, as it reveals the protuberant lesion. It is the consequence of the rapid rise in natraemia in patients with chronic hyponatremia and the cellular adaptations associated with plasma hypotonicity.

Correction of deep, chronic hyponatremia requires a degree of caution. It must be carried out slowly, with particular attention paid to the patient's level of consciousness, in order to detect early symptoms of MCP. To this end, an information sheet on the management of hyponatremia has been designed and distributed to health facilities.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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