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Central Pontine Myelinolysis Secondary to Hyperglycemia: A Case Report

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ABSTRACT

Centropontine myelinolysis (MCP) belongs to the osmotic demyelination syndrome, and occurs especially after the rapid correction of hyponatremia. In rare cases, it is caused by states of hyperosmolarity and it can be seen in hyperglycemia.

Here, we report the case of a 28-year-old diabetic patient, admitted to intensive care unit for a disorder of consciousness and seizures.

The physical examination found an apyretic patient with, Glasgow of 11, with quadriparesis and high blood pressure. Laboratory investigations showed hyperglycemia and metabolic ketoacidosis with chronic renal failure, natremia was normal, lumbar puncture was also normal. The diagnosis of MCP was retained on the MRI.

Insulin therapy and rehydration were started and the patient showed a clinical improvement. However, he died because of ventilator-associated pneumonia.

The aim of this work is to show that MCP can occur despite the absence of an abnormality of natremia and this diagnosis should be considered in diabetic patients with neurological disorders.

KEYWORDS: Central Pontine Myelinolysis, Diabetes Mellitus, Hyperglycemia

INTRODUCTION

Centropontin myelinolysis (CPM) belongs to osmotic demyelination syndrome. It is characterized by the destruction of myelin in the white matter of the protuberance. It occurs mainly after the rapid correction of hyponatremia (1). The incidence of CPM is difficult to quantify due to the lack of diagnosis (1,2). In rare cases, it can be found in case of hyperglycemia (3,4). In this observation, we report a CPM occurring in the absence of abnormalities of natremia.

OBSERVATION

We report a 28-year-old diabetic patient, on insulin for 14 years, at the stage of diabetic nephropathy, followed for chronic gastritis with *Helicobacter pylori* who was admitted to the emergency for a disorder of consciousness and seizure. He has been hospitalized several times for diabetic ketoacidosis secondary to bad compliance with his medication. The examination in the emergency room found an apyretic, polypneic patient with a Glasgow score at 11 (E = 2, V = 4, M = 5) with muscle weakness in all four limbs rated at 3/5, he had a grade 2 high blood pressure. Dextro was high, glucosuria and acetonuria were both positive at 3 crosses on the urine strip. Biological findings included metabolic acidosis with high anion gap, hyperglycemia at 30 mmol/l, natremia at 129 mmol/l, corrected natremia at 137 mmol/l and renal failure with urea at 1.35 g/l, creatinine at 40.3 mg/l and glomerular filtration rate at 18 ml / min. The lumbar puncture and the brain CT with contrast injection performed were both normal. The diagnosis of ketoacidosis was retained and the patient was rehydrated and put on insulin. The evolution was marked by the worsening of his neurological condition with deterioration of his vigilance requiring intubation and hospitalization in intensive care. A brain MRI was performed, showing centropontin myelinolysis (Figure 1). Rehydration and insulin therapy were maintained, the patient has improved neurologically, without sensory-motor deficits and without recurrence of

seizures. The evolution is marked by the death of the patient due to ventilator-associated pneumoniae.

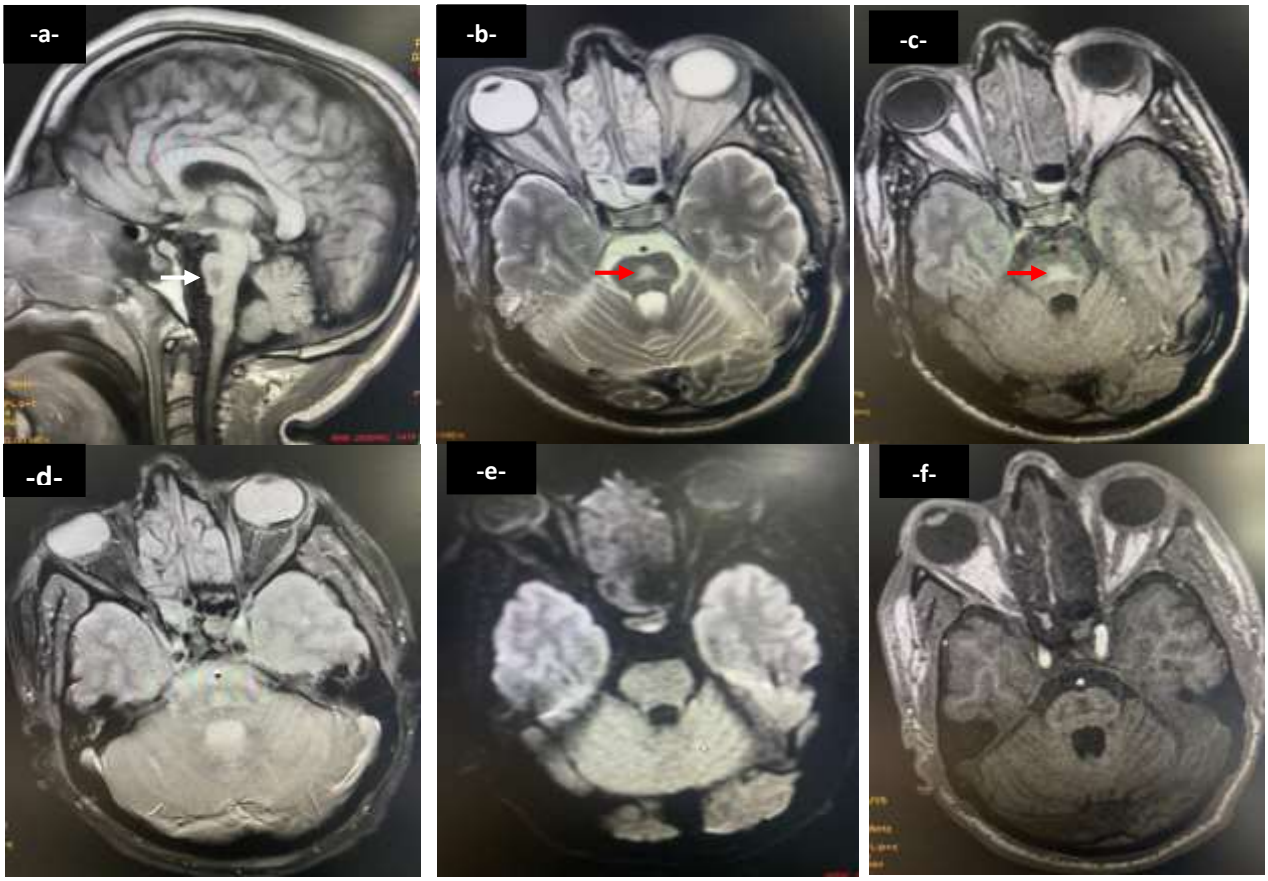

Figure 1: Brain MRI showing a triangular-shaped, with posterior base, centropontine signal anomaly, hyposignal on the sagittal T1 sequence (a, white arrow), hypersignal on the axial T2 (b) and T2 FLAIR (c) (red arrow), without hemorrhagic remodeling on the T2* sequence (d), without restriction of diffusion (e) and not enhanced after injection of Gadolinium (f).

DISCUSSION

CPM was first described in 1959 by Adams and the risk factors are mainly chronic alcoholism, malnutrition (5) but also liver transplantation (6). The leading role of hyponatremia and especially its rate of correction were only confirmed later (7). Together with extrapontin myelinolysis, it constitutes osmotic demyelination syndrome (8).

Its frequency is still unknown but remains rare (8.9), an incidence of 0.5% is reported by some authors (10), others find an incidence of 2.5% (11). Data on gender predominance are contradictory, with some authors finding a predominance in men (6,8), while others have noted a female predominance (12).

In histology, there is a demyelination of the white matter due to macrophagic and lymphocyte infiltration (13) and a decrease in oligodendrocytes whose role is the synthesis of myelin. There are no inflammatory lesions, vessels as well as neurons and axons are preserved (5,14).

CPM results from an abrupt change in osmolarity compromising the adaptation mechanisms already initiated by the brain to fight cerebral edema, leading to intracellular dehydration and also promotes apoptosis of oligodendrocytes (1,8,15). A disruption of the blood-brain barrier resulting in vasogenic oedema may also be a mechanism involved in the formation of CPM (15).

Other abnormalities have been described as being responsible for CPM such as hyperosmolarity states, hypernatremia (12) but also other metabolic disturbances such as hypophosphoremia (16). It may be associated in rare cases with persistent hyperglycemia in type 1 or type 2 diabetes. Concomitant ketoacidosis can be observed (3,4,12,17) as in the case of our patient but is not systematic (17,18). Some factors have been described as being at risk for CPM in diabetes, such as diabetic nephropathy and high blood pressure (3,18,19). The imbalance of diabetes was strongly found (3,4,12,18). This is the case of our patient who has already been hospitalized for diabetic ketoacidosis secondary to a poor adherence to treatment twice in six months. However, osmotic demyelination syndrome can also occur in well-controlled diabetes. Indeed, Ichikawa found that three out of four patients had balanced diabetes (19).

CPM manifests clinically by impaired consciousness, cognitive impairment, quadriplegia, pseudobulbar involvement and sometimes seizures (18,20), which explains

the patient's symptomatology. Oculomotor disorders and pupillary abnormalities can also be observed or even a locked in syndrome (15). Asymptomatic forms and others associated with psychiatric manifestations have also been described (20).

The diagnosis of CPM used to be confirmed by autopsy, but with advances in imaging, the diagnosis is now made antemortem. CT scans show pontine hypodensity not enhanced by the injection of contrast medium, but most often the CT scan is normal (9). MRI is the best choice for making the diagnosis of CPM because it is more sensitive (8,14). The main abnormalities described are symmetrical lesions in hyposignal in T1 sequence weighting, hypersignal in T2 sequence and the absence of contrast enhancement (3,15,19). The CT without abnormalities found in our patient is consistent with the literature.

Several conditions are differential diagnoses with CPM. They can be of autoimmune origin such as myasthenic crisis, Guillain Barré syndrome and autoimmune encephalitis (17), toxic (Gayet-Wernicke encephalopathy), infectious such as encephalitis, inflammatory including multiple sclerosis, not to mention vascular and tumor ologies. Lumbar puncture and MRI are in these conditions a great contribution to invalidate some of these diagnoses (9).

Treatment is mainly symptomatic as there is currently no specific treatment (9,17). Management also involves treating aggravating factors such as hypoxia and preventing or treating neurological and general complications such as those related to mechanical ventilation and decubitus (9). Some treatments have been proposed such as corticosteroid therapy, immunoglobulins and supplementation with thyrotropin-releasing hormone leading to an approvement (21–23). However, their real efficiency remain to be demonstrated by multicenter studies and they are not currently recommended for the treatment of CPM (11).

The prognosis has changed at the present time since the advent of MRI which allows an antemortem diagnosis, the evolution can be towards restitution ad integrum, survival with sequelae or towards death (6). The decrease in mortality can also be explained by the better understanding of the pathophysiology but also by the progress of resuscitation (8). There are no clinical and radiological criteria that can predict outcome (15). There was an improvement in the neurological condition of our patient but he still died by ventilator-associated pneumoniae.

CONCLUSION

CPM is a rare pathology that can also be observed apart from the rapid correction of hyponatremia as in diabetes especially when it is poorly balanced with chronic uncontrolled hyperglycemia. The existence of associated nephropathy could promote the occurrence of CPM given the hyperosmolarity that this generates, the presence of high blood pressure is also described as a precipitating factor. A brain MRI should be performed for any neurological abnormality responsible for impaired consciousness, quadriparesis, pseudobulbar signs and seizures in a diabetic patient even in the absence of hyponatremia looking for osmotic demyelination syndrome.

Conflict of interest

The authors are contributed equally and declare no competing interest

Contributions

All authors contributed equally in this work

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REFERENCES

1. Danyalian A, Heller D. Central Pontine Myelinolysis. 2022 Aug 8. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan–. PMID: 31869161.
2. Renjen PN, Chaudhari D, Namala V. Central pontine and extrapontine myelinolysis. Apollo Med. déc 2016;13(4):220-3.
3. Talluri S, Charumathi R, Khan M, Kissell K. Atypical presentation of central pontine myelinolysis in hyperglycemia. Endocrinol Diabetes Metab Case Rep. 2017 Sep 4;2017:17-0064. doi: 10.1530/EDM-17-0064. PMID: 28924482; PMCID: PMC5592705.
4. Donnelly H, Connor S, Quirk J. Central pontine myelinolysis secondary to hyperglycaemia. Pract Neurol. déc 2016;16(6):493-5.
5. ADAMS RD, VICTOR M, MANCALL EL. Central Pontine Myelinolysis: A Hitherto Undescribed Disease Occurring in Alcoholic and Malnourished Patients. AMA Arch Neurol Psychiatry. 1 févr 1959;81(2):154-72.
6. Singh TD, Fugate JE, Rabinstein AA. Central pontine and extrapontine myelinolysis: a systematic review. Eur J Neurol. déc 2014;21(12):1443-50.
7. Laureno R, Illowsky Karp B. PONTINE AND EXTRAPONTINE MYELINOLYSIS FOLLOWING RAPID CORRECTION OF HYPONATRAEMIA. The Lancet. juin 1988;331(8600):1439-41.
8. Lambeck J, Hieber M, Dreßing A, Niesen WD. Central Pontine Myelinosis and Osmotic Demyelination Syndrome. Dtsch Arztebl Int. 2019 Sep 2;116(35-36):600-606. doi: 10.3238/arztebl.2019.0600. PMID: 31587708; PMCID: PMC6804268.
9. Louis G, Bollaert PE. Centro- and extrapontin myelinolysis. Current data and specificities in intensive care. Resuscitation. 2012 Sep;21(5):563-71.

10. Newell KL, Kleinschmidt-DeMasters BK. Central pontine myelinolysis at autopsy; a twelve years retrospective analysis. J Neurol Sci. oct 1996;142(1-2):134-9.
11. Rao PB, Azim A, Singh N, Baronia AK, Kumar A, Poddar B. Osmotic demyelination syndrome in Intensive Care Unit. Indian J Crit Care Med Peer-Rev Off Publ Indian Soc Crit Care Med. mars 2015;19(3):166-9.
12. Ranger AM, Chaudhary N, Avery M, Fraser D. Central pontine and extrapontine myelinolysis in children: a review of 76 patients. J Child Neurol. août 2012;27(8):1027-37.
13. Haynes HR, Gallagher PJ, Cordaro A, Likeman M, Love S. A case of chronic asymptomatic central pontine myelinolysis with histological evidence of remyelination. Forensic Sci Med Pathol. 2018;14(1):106-8.
14. Alleman AM. Osmotic Demyelination Syndrome: Central Pontine Myelinolysis and Extrapontine Myelinolysis. Semin Ultrasound CT MRI. 1 avr 2014;35(2):153-9.
15. Martin R. CENTRAL PONTINE AND EXTRAPONTINE MYELINOLYSIS: THE OSMOTIC DEMYELINATION SYNDROMES. J Neurol Neurosurg Psychiatry. sept 2004;75(Suppl 3):iii22-8.
16. Qadir F, Hasan A, Masood M. Extra Pontine Myelinolysis associated with Hypophosphatemia. :3.
17. Mir WAY, Shrestha DB, Aryal BB, Reddy VK, Yadullahi MAA. Central Pontine Myelinolysis Secondary to Hyperglycemia in a Young Patient. Cureus. 2021 Oct 5;13(10):e18495. doi: 10.7759/cureus.18495. PMID: 34754656; PMCID: PMC8569644.
18. Saini M, Mamauag M, Singh R. Central pontine myelinolysis: a rare presentation secondary to hyperglycaemia. Singapore Med J. avr 2015;56(04):e71-3.
19. Ichikawa H, Murakami H, Katoh H, Hieda S, Kawamura M. Central Pontine Lesions Observed with MRI in Four Diabetic Patients. Intern Med. 2008;47(15):1425-30.
20. Odier C, Nguyen DK, Panisset M. Central pontine and extrapontine myelinolysis: from epileptic and other manifestations to cognitive prognosis. J Neurol. 1 juill 2010;257(7):1176-80.
21. Sakamoto E, Hagiwara D, Morishita Y, Tsukiyama K, Kondo K, Yamamoto M. [Complete recovery of central pontine myelinolysis by high dose pulse therapy with methylprednisolone]. Nihon Naika Gakkai Zasshi J Jpn Soc Intern Med. 10 oct 2007;96(10):2291-3.
22. Escribano-Gascón AB, Casanova-Peño LI, Bartolomé-Puras M, Porta-Etessam J. [Efficacy of intravenous immunoglobulins in central pontine myelinolysis]. Neurol Barc Spain. août 2008;23(6):392-4.
23. Zein EF, Karaa SE, Rollot F, Blanche P, Chemaly RE. Treatment of central pontine myelinolysis with thyrotropin-releasing hormone. Presse Medicale Paris Fr 1983. avr 2006;35(4 Pt 1):618-20.