IJMPR 2020, 4(4), 233-235

International Journal of Modern Pharmaceutical Research
www.ijmpronline.com

ISSN: 2319-5878
IJMPR
Case Report
SJIF Impact Factor: 5.273

ASYMPTOMATIC CENTRAL PONTINE MYELINOLYSIS IN HYPEREMESIS GRAVIDIUM

Maryam Jalali[1], Dr. Navid Kalani[2], Farshid Javdani[3] and Naser Hatami[4]*

[1]Department of Neurology, Jahrom University of Medical Sciences, Jahrom, Iran.
[2]Research Center for Social Determinants of Health, Jahrom University of Medical Sciences, Jahrom, Iran.
[3]Student Research Committee, Jahrom University of Medical Sciences, Jahrom, Iran.

Received on: 21/07/2020
Revised on: 11/08/2020
Accepted on: 01/09/2020

***Corresponding Author**
Naser Hatami
Student Research Committee, Jahrom University of Medical Sciences, Jahrom, Iran.

ABSTRACT

Central pontine myelinolysis occurs due to demyelination of the myelin sheath of the neurons of the Pons region of the brain stem, which may have different neurological symptoms. The case presented in our study was a 29-year-old pregnant woman who had no signs of neurological findings and was asymptomatic. The cause of the occurrence of Central pontine myelinolysis is mainly due to the incorrect correction of blood electrolytes. However, she did not show any clear electrolyte disturbances, which rejects that as an etiologic cause of this case. The similarity of this patient with other cases was malnutrition due to hyperemesis gravidium. However, Regarding to four weeks postpartum MRI, CPM complication was eliminated and normal MRI results were reported.

INTRODUCTION

Central pontine myelinolysis (CPM) is a neurological disorder caused by severe damage to the myelin sheath of the nerve cells in the region of the brain stem, called Pons. The causes of these lesions are largely uncertain or due to the incorrect treatment of electrolyte disorders. It is characterized by acute paralysis, dysphagia, dysarthria, and other neurological symptoms.[1] Central pontine myelinolysis was first described by Adams and colleagues in 1959. The authors deliberately used the term "demyelination" to describe the disease to isolate this condition from multiple sclerosis and other neurological disorders that became demyelinating because of inflammatory processes.[2]

Various factors, such as alcohol and malnutrition, and the incorrect treatment of electrolyte disturbances, can cause this disorder.[3] Vomiting in pregnancy is observed in 50% of pregnant women, but Hyperemesis gravidarum causes severe vomiting in pregnant women and is less common.[4] Changes in body electrolytes occur due to severe vomiting. If this condition is not treated, it will be dangerous to the mother and the baby. However, sometimes the inappropriate treatment of electrolyte disorders (Rapid correction of electrolytes disturbance) causes brain complications. Various studies have shown that Central Pontine Myelinolysis can occur due to the rapid correction of electrolytes after Hyperemesis gravidarum.[5]

CASE REPORT

A 29-year-old woman, with a gestational age of 34 weeks with complaints of headache, nausea, and vomiting, and oral intolerance was brought to the Emergency of Motahari Hospital in Jahrom. At the onset of the visit, the patient was totally conscious and aware of the time and place and the person, but she was lethargy and had headaches from about four days ago. The patient had worsened nausea and vomiting and had become cachectic.

Her headache was moderate holocephalic. Vital signs were normal at the referral time. She hadn't histories of fever. Histories were not preceded by the underlying condition or drug, alcohol or narcotics use. The neurological and general medical examinations of the patient were normal. The heart sounds of the fetus were normal. The patient had no problem in terms of strength and muscle tone, and the patient's gate was normal. The patient's sodium was 131 and the potassium was 3.8. The patient had normal U / A and normal renal function tests.

Brain MRI revealed a high signal change in T2 and flair in central of pons with no signal change in T1 and no restriction in DW. Other extrapontine structures were normal. The patient performed normal delivery at week 37 of gestational age. The mother and the baby were good and four weeks after giving birth, she again performed brain MRI, which was completely normal.

DISCUSSION

CPM disease has been seen in many patients, including diabetic patients,[6] Renal Filler,[7] pancreatic cancer, and pregnant women.[8] CPM can have different symptoms and ultimately causes coma or death. Various studies have shown that there is a higher incidence of CPM in alcoholic beverages. Alcoholics are more likely to suffer from malnutrition, which is itself a risk factor for CPM.

Weight loss has also been seen in most patients who have been diagnosed with CPM. In our patient, there was no severe hyponatremia, and no hyponatremia correction was conducted. Our patient was referred with headache manifestations that had not been reported before this study as a sign of CPM. Also, all neurological tests provided information on the individual's health, but CPM was detected in the MRI images. However, our patient acknowledges that she did not consume alcohol during pregnancy. The reason for weight loss in pregnancy was due to hyperemesis gravidium. Other hypotheses, including alcohol and anorexia, were also used to justify weight loss. If she had a cachexia due to chronic alcohol consumption, she would have had trouble in getting pregnant.

According to T Sugimoto et al, CPM can be due to hypokalemia which is caused by anorexia nervosa,[9] Anorexia nervosa can be seen during pregnancy.[10] On the other hand, this hypothesis is interesting in the view that people with nutritional problems and anorexia nervosa during pregnancy may also come across headaches. Perhaps it can be concluded that the main reason for the occurrence of CPM in alcoholic patients is not alcohol consumption itself. It's due to malnutrition due to high alcohol consumption and cachexia.

Although much research has acknowledged the rapid blood sodium correction in hyponatremia as one of the causes of CPM, in a 26-year-old patient suffering from pancreas cancer, doctors were aware of this issue and tried to slowly correct the sodium status. But in MRI findings, it was observed that CPM also occurred in this patient. He was an alcoholic, suffering from malnutrition and weight loss. After confirmation of CPM in the MRI, neurological examinations were performed and all examinations informed that patient's neurological health That patient's similarity with the case presented in our study was being asymptomatic. Our patient, despite the diagnosis of CPM in MRI findings, showed no neurological symptoms in the neurological exam.

Hyponatremia has been reported less frequently in patients who do not show symptoms of CPM or have a normonatremia.[8,7] In our patient, there was no clear hypernatremia. Recovery or remyelination is seen in asymptomatic CPM cases.[12] In the patient we studied, there was also a remyelination and the patient was treated after delivery.

CONCLUSION

The presented patient in our study, despite the CPM conflict, did not have a neurological symptom and was asymptomatic. Her blood electrolytes did not show any clear hyponatremia or electrolyte imbalance. Shortly after delivery, CPM lesions were eliminated. The similarity of CPM cases is the sharp drop in weight, which in our case was considered the malnutrition caused by Hyperemesis gravidarum.

ACKNOWLEDGEMENT

We would like to thank the Clinical Research Development Unit of Peymanieh Educational and Research and Therapeutic Center of Jahrom University of Medical Sciences for providing facilities to this work.

Authors Contributions

All the authors met the criteria of authorship based on the recommendations of the international Committee of Medical Journal Editors.

Conflict of interest

There are no conflicts of interest in this study.

REFERENCES

1. Bose, P; Kunnacherry, A; Maliakal, P (19 September). "Central pontine myelinolysis without hyponatraemia". The Journal of the Royal College of Physicians of Edinburgh, 2011; 41(3): 211–214.
2. Adams RD, VICTOR M, MANCALL EL. Central pontine myelinolysis: a hitherto undescribed disease occurring in alcoholic and malnourished patients. AMA Archives of Neurology & Psychiatry, 1959 Feb 1; 81(2): 154-72.
3. Shah SO, Wang A, Mudambi L, Ghuznavi N, Fekete R. Asymptomatic central pontine myelinolysis: a case report. Case reports in neurology, 2012; 4(3): 167-72.
4. Sheehan, P (September). "Hyperemesis gravidarum assessment and management" (PDF). Australian Family Physician, 2007; 36(9): 698–701.
5. Anand, Keertana & rout R, ranjan & Subbaiah, Murali & Dorairajan, Gowri & sagili, haritha. Rare Complication of Hyperemesis Gravidarum - Central Pontine Myelinolysis, A Case Report. J Gynecol. 2. 138. 10.23880/OAJG-16000138, 2017.
6. Kote SS, Khandelwal A, Pathak DG, Nath R. An unusual case of osmotic demyelination syndrome without electrolyte changes in a patient with diabetes. Journal of Neuroanaesthesiology and Critical Care, 2016 May 1; 3(2): 145.
7. Yadav RK, Das CJ, Bagchi S, Agarwal S. Asymptomatic pontine and extra-pontine lesions in a patient with end-stage renal disease. Saudi Journal of Kidney Diseases and Transplantation, 2016 Mar 1; 27(2): 395.
8. Bergin PS, Harvey P. Wernicke's encephalopathy and central pontine myelinolysis associated with hyperemesis gravidarum. BMJ: British Medical Journal, 1992 Aug 29; 305(6852): 517.
9. Sugimoto T, Murata T, Omori M, Wada Y. Central pontine myelinolysis associated with hypokalaemia in anorexia nervosa. Journal of Neurology, Neurosurgery & Psychiatry, 2003 Mar 1; 74(3): 353-5.
10. Goldman RD, Koren G. Anorexia nervosa during pregnancy. Canadian family physician, 2003 Apr 1; 49(4): 425-6.
11. Asymptomatic central pontine myelinolysis

Neurology, Sep 1999; 53(5): 914. DOI: 10.1212/WNL.53.5.914.

12. Haynes HR, Gallagher PJ, Cordaro A, Likeman M, Love S. A case of chronic asymptomatic central pontine myelinolysis with histological evidence of remyelination. Forensic Science, Medicine and Pathology, 2018 Mar 1; 14(1): 106-8.