

Central Pontine Myelinolysis and Localized Fluorodeoxyglucose Uptake Seen on ^{18}F -FDG PET/CT

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Abstract

Case report describing the finding of central pontine myelinolysis (CPM) using combined fluorine-18 (^{18}F)-fluorodeoxyglucose (FDG) positron emission tomography/computed tomography (PET/CT). The patient was a known alcoholic who, during admission was under treatment for hyponatremia, showed a significant decline in both motor and cognitive function. Combined ^{18}F -FDG PET/CT showed localized FDG uptake in the pons, consistent with the finding of CPM observed on magnetic resonance imaging (MRI). CPM is a demyelinating lesion of the pons, resulting in several neurological symptoms. The exact cause of CPM is not clear, but a strong relations between loss of myelin and osmotic stress exists, especially during rapid correction of hyponatremia. The osmotic stress is thought to induce disruption of the blood–brain barrier, allowing access for inflammatory mediators in extravascular brain tissue, which most likely attracts glial cells of the brain, attracts macrophages and activates astrocytes. We suggest that metabolism in these activated cells could be responsible for the localized FDG uptake during active CPM.

Keywords: Central pontine myelinolysis, demyelination, fluorine-18 (^{18}F)- fluorodeoxyglucose positron emission tomography/computed tomography

Introduction

Central pontine myelinolysis (CPM), initially described by Adams *et al.*^[1] in 1959, is a demyelinating lesion of the pons, resulting in a number of neurological symptoms, including weakness, impaired control of the limbs, gait instability, dysarthria, dysphagia, and altered mental status. All of these symptoms were seen in the present case report. The exact pathogenesis of CPM is not completely understood, but there are strong relations between loss of myelin and osmotic stress. In recognition of this, the term “osmotic demyelination syndrome”

has been suggested.^[2] Cases of CPM are associated with an overly rapid correction of chronic hyponatremia,^[3] which is a common condition in patients with chronic alcohol abuse.^[4] Review articles and case reports on CPM have been published based on clinical, computed tomography (CT), and magnetic resonance imaging (MRI)^[4,5] findings. Only one case report^[6] has described the hypermetabolism of the pons using ^{18}F -FDG PET, but, to our knowledge, the lesions of CPM have never been described using combined PET/CT before.

Case Report

A 51-year-old Caucasian female with a known history of alcohol abuse over several years was admitted on late

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Access this article online

Quick Response Code:



Website:
www.wjnm.org

DOI:
10.4103/1450-1147.174707

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How to cite this article: Rønne F, Tfelt-Hansen PC, Rørdam L. Central pontine myelinolysis and localized fluorodeoxyglucose uptake seen on ^{18}F -FDG PET/CT. World J Nucl Med 2017;16:56-8.

September 2012 due to severe intoxication and vomiting mixed with blood. She was found alcohol intoxicated, but otherwise without positive findings. Crude neurological examination was normal. Glasgow Coma Scale (GCS) score was 15. Electrolyte values showed decreased sodium (99 mmol/L) and potassium (3.2 mmol/L) levels. Initial treatment consisted of detoxification and medical treatment of withdrawal symptoms with simultaneous correction of electrolyte levels. Sodium was given intravenously as an isotonic saline solution at a rate of 50 mL/h and a maximum limit of 12 mmol per 24 h.

The following morning, the patient was difficult to wake (GCS: 6, Sodium: 107 mmol/L, potassium 3.3 mmol/L). CT of the cerebrum was normal, but CT of the thorax showed a total lower lobe atelectasis on the left side and a near closure of the left lower bronchus. Malignancy was suspected, and to elucidate on the CT findings, an ¹⁸F-FDG PET/CT was ordered.

Ten days after admission sodium and potassium levels were at 136 mmol/L and 3.9 mmol/L, respectively. However, the staff noticed a significant decline in the patient's overall function and mental capabilities. She had difficulty swallowing, required suction due to stagnation of secrete in her lungs, and vocal responses were limited to a few words only.

The ¹⁸F-FDG PET/CT performed 12 days after admission [Figure 1] had findings that could be compatible with CPM, and an MRI of the brain done two days after the

PET/CT was consistent with CPM and, possibly, an early stage of extra-pontine myelinolysis (EPM) [Figure 2].

Fourteen days after admission, the patient deteriorated further. She was unable to get up from supine to a sitting position unassisted, her self-maintained balance was poor, she had severe dysarthria, and she had no spontaneous swallowing.

At the time of discharge (approximately 2 months after admission), the patient could eat and drink independently. Her gait was wide-based gait, but without the use of aids. She was able to converse with both humor and irony and had only a minor degree of dysarthria.

Discussion

The general consensus in previously published literature^[2,7] is that osmotic stress from a rapid increase in serum tonicity in patients with chronic hyponatremia is the most important factor causing CPM. During this correction, the intracellular/extracellular osmolality imbalance will cause a net movement of water out of the cells, which is thought to cause a series of damaging factors leading to demyelination.^[2] One such factor is shrinkage of the glial cells, which may lead to axonal shear damage.^[2] Other factors include cellular damage and induction of apoptosis resulting in disruption/impairment of the blood-brain barrier, which in turn exposes glial cells to "myelinolytic factors" such as serum complement, cytokines/chemokines, and other inflammatory mediators.^[2,3,7]

In contrast, there is no inflammatory reaction with perivascular infiltrates of lymphocytes in likeness active

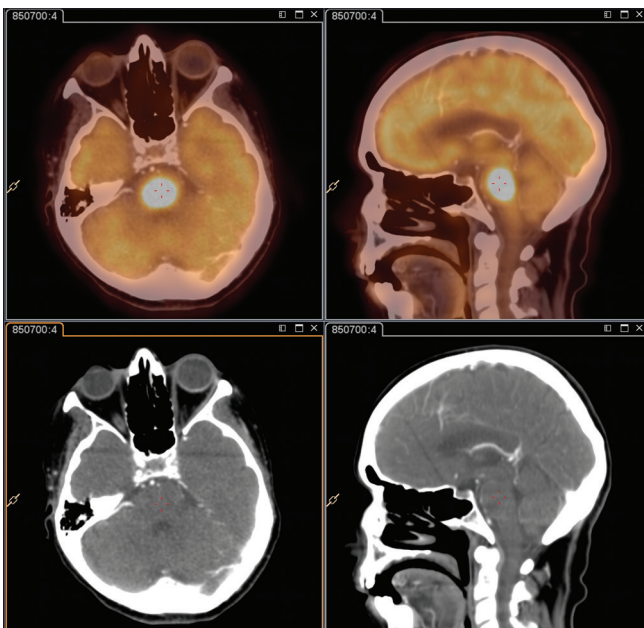


Figure 1: ¹⁸F-FDG PET/CT scan of a 51-year-old Caucasian female with symptoms of central pontine myelinolysis. Scan is showing localized FDG uptake in the pons, with normal and symmetrical activity in the rest of the brain

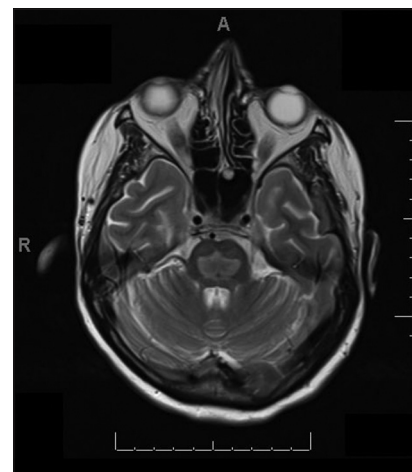


Figure 2: MR-scan T2-weighted image showing a hyperintense region in the central part of pons. In addition, hyperintense region was present symmetrically in the caput nucleus caudatus, putamen, and the lateral part of thalamus

plaques in multiple sclerosis for example. The lesions in CPM are usually symmetrical^[5] and contain sheets of lipid-laden macrophages and a large number of reactive astrocytes during active disease.^[8] The increased metabolism of these accumulated cells is probably responsible for the localized uptake of FDG seen on the PET scanning. In addition, it has been suggested that the structural property of intermixed white and gray matter in the central pons compared to other parts of the brain, where white is isolated from the capillary-rich gray matter, can account for the vulnerability of this area to osmotic injury.^[7]

One other case report^[6] from 1998 has described hypermetabolism in the pons in the form of focal uptake of ¹⁸F-FDG in a patient with suspected CPM. The ¹⁸F-FDG-PET scanning was done one week after the onset of decreased sensorium. The authors suggested that the increased glucose metabolism was caused by activity of microglial cells and/or astrocytes.

The ¹⁸F-FDG PET/CT from the present case report was done ten days after the onset of decreased sensorium and, therefore, temporally quite comparable to the previously mentioned scanning done by Roh *et al.*^[6] The present ¹⁸F-FDG-PET showed normal and symmetrical ¹⁸F-FDG uptakes in both hemispheres, but with a focus of abnormally increased ¹⁸F-FDG uptake in the central pons [Figure 1]. Standard uptake values (SUV) of this region of interest (ROI) had a maximum of 13.8. The scan was done using a Philips Gemini PET/CT TF 64 (with Time-of-Flight capability), with a CT exposure of 107 mAs, PET time per bed position (frame duration) 120 s, and an injected dose of 301 MBq ¹⁸F-FDG.

Conclusion

Regarding CPM, the consensus is that osmotic stress from overly rapid correction of chronic hyponatremia plays an important role in demyelination and associated symptoms. Some reports describe CPM as a demyelination without an inflammatory reaction.^[9,10] However, the osmotic stress

is thought to induce disruption of the blood-brain barrier, and thereby allow access for inflammatory mediators in extravascular brain tissue, which most likely activates the astrocytes as well as attract macrophages and glial cells of the brain. We suggest that metabolism in these activated cells could be the mechanism responsible for the localized FDG-uptake seen on PET/CT during active CPM.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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