Isolated Extrapontine Myelinolysis of Osmotic Demyelination Syndrome

Yılmaz Ö.[1], Armağn H. H.[2], Turan A.[3], Duymuş M.[4]
[1]Department of Radiology, Faculty of Medicine, Süleyman Demirel University, Isparta, Turkey;
[2]Department of Emergency, Isparta State Hospital, Isparta, Turkey;
[3]Department of Radiology, Etlik Training and Research Hospital, Ankara, Turkey;
[4]Department of Radiology, Faculty of Medicine, Kafkas Kars University, Kars, Turkey

Received April 5, 2012; Accepted January 15, 2013.

Key words: Extrapontine myelinolysis – MR – Osmotic demyelination syndrome

Abstract: The osmotic demyelination syndrome (ODS) has been identified as a complication of the rapid correction of hyponatremia for decades (King and Rosner, 2010). However, in recent years, a variety of other medical conditions have been associated with the development of ODS, independent of changes in serum sodium which cause a rapid changes in osmolality of the interstitial (extracellular) compartment of the brain leading to dehydration of energy-depleted cells with subsequent axonal damage that occurs in characteristic areas (King and Rosner, 2010). Slow correction of the serum sodium concentration and additional administration of corticosteroids seems to be a major prevention step in ODS patients. In the current report we aimed to share a rare case which we observed in our clinic.

Mailing Address: Dr. Ömer Yılmaz, Department of Radiology, Faculty of Medicine, Süleyman Demirel University, Isparta, Turkey; Phone: +90 246 211 29 01; Fax: +90 246 237 11 65; e-mail: droyilmazrad@hotmail.com

Introduction

The human demyelination disorder myelinolysis may be a pathological condition caused by a rapid rise in serum sodium, usually when hyponatremia is trying to be corrected (Kleinschmidt-DeMasters and Norenberg, 1981). However, in recent years, a variety of other medical conditions have been associated with the development of ODS, independent of changes in serum sodium this will cause a rapid changes in osmolality of the interstitial (extracellular) compartment of the brain which leads to dehydration of energy-depleted cells with subsequent axonal damage that occurs in characteristic areas (King and Rosner, 2010). Slow correction of the serum sodium concentration and additional administration of corticosteroids seems to be a major prevention step in ODS patients.

Case report

We would like to present a case of isolated extrapontine myelinolysis of the osmotic demyelination syndrome which has been reported only few times in the literature (Sajith et al., 2006). A 28-year-old man was referred to emergency department after fainting at home with vomiting and confusion who had a history of a months-long diarrhoea resulting from antibiotic usage for upper respiratory tract infection. After 2 days of hydration with low sodium concentration, his neurologic condition started to progress and tetraparesis developed in a single day. Magnetic resonance imaging (MRI) showed high signal intensity on T2-weighted, FLAIR (fluid attenuated inversion recovery) and DWI (diffusion weighted imaging) images in the bilateral symmetric putamina, caudate nuclei and external capsule (Figures 1–3). There were no changes within the pons in T2-weighted images (Figure 4).

Discussion

The serum sodium concentration is the primary determinant of the serum osmolality. Chronic alcoholism, electrolyte disturbances especially hyponatremia-hypernatremia and their corrections, liver transplant patients, pulmonary infections and malignant tumors are the most common major etiological factors (Lampl and Yazdi, 2002). The fall in serum osmolality in patients with true hyponatremia promotes water movement into the brain and, if the hyponatremia is acute and severe, can lead to cerebral edema, neurologic symptoms and osmotic stress. In response to hyponatremia, the brain makes adaptations that lower the cerebral volume toward normal and reduces the likelihood of these complications (Pekic et al., 2011). The osmotic demyelination syndrome (ODS) has been a recognized complication of the rapid correction of hyponatremia (Khositseth et al., 2010). The term osmotic myelinolysis is used for demyelinating disease of the pons that are often associated with demyelization of other areas of the central nervous system and characterized by acute paralysis, dysphagia (difficulty swallowing), dysarthria (difficulty speaking), and other neurological symptoms. It occurs as a

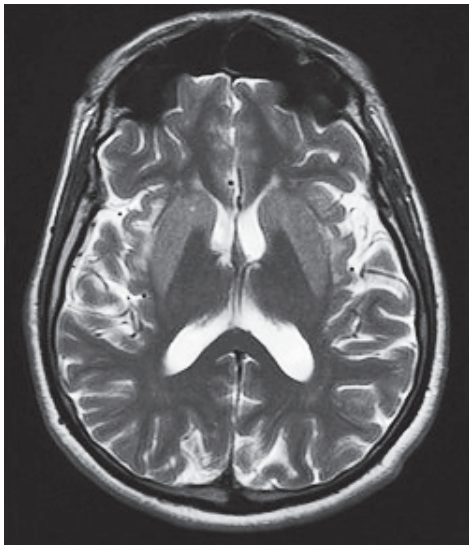

Figure 1 – T2-weighted image showing bilateral symmetrical hyperintensities in the putamen, caudate nucleus and external capsula.

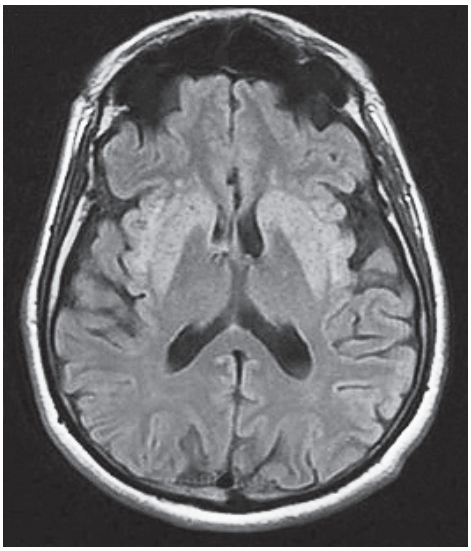

Figure 2 – FLAIR image showing bilateral symmetrical hyperintensities in the putamen, caudate nucleus and external capsula.

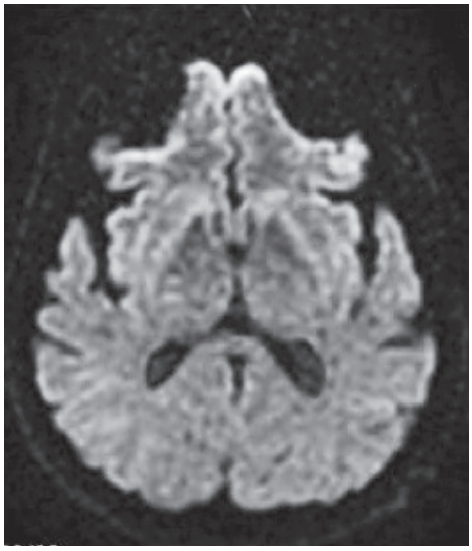

Figure 3 – DWI image showing bilateral symmetrical hyperintensities in the putamen, caudate nucleus and external capsula.

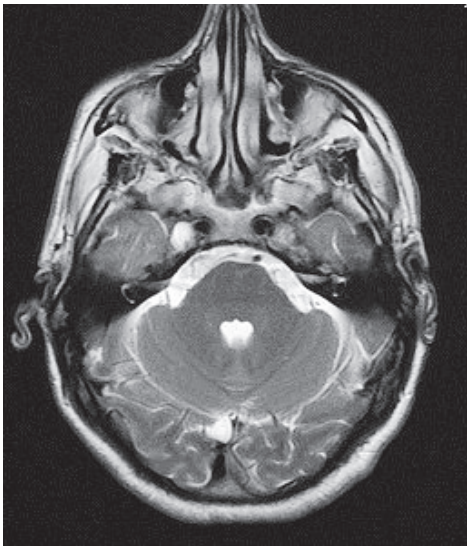

Figure 4 – T2-weighted magnetic resonance scan image shows no changes within the pons.

consequence of a rapid rise in serum tonicity following treatment for chronic, severe hyponatremia in condition of intracellular adaptation to the prevailing hypotonicity. Hyponatremia should be corrected at a rate of no more than 8–10 mmol/l of sodium per day to prevent central pontine myelinolysis. The diagnosis is usually done with MRI and prognosis depends on the underlying reason (Iwama et al., 2011). As far as we know, a case with isolated extrapontine myelinolysis after rapid correction of severe hyponatremia associated with advanced diarrhea by antibiotherapy has not been reported previously. According to the hypothesis of Norenberg, rapid rise in serum sodium may cause central pontine myelinolysis (CPM).

Conclusion

This case indicates that osmotic demyelination syndrome should always be considered as an emergency condition and correction of electrolyte disturbances should be made carefully.

References

Iwama, S., Sugimura, Y., Suzuki, H., Suzuki, H., Murase, T., Ozaki, N., Nagasaki, H., Arima, H., Murata, Y., Sawada, M., Oiso, Y. (2011) Time-dependent changes in proinflammatory and neurotrophic responses of microglia and astrocytes in a rat model of osmotic demyelination syndrome. *Glia* **3**, 452–462.

Khositseth, S., Intrakao, S., Pao-in, W., Visudtibhan, A. (2010) A fluctuation of serum osmolality inducing osmotic demyelination syndrome. *J. Med. Assoc. Thai*. **93**, S299–S302 (Suppl. 7).

King, J. D., Rosner, M. H. (2010) Osmotic demyelination syndrome. *Am. J. Med. Sci*. **6**, 561–567.

Kleinschmidt-DeMasters, B. K., Norenberg, M. D. (1981) Rapid correction of hyponatremia causes demyelination: relation to central pontine myelinolysis. *Science* **4486**, 1068–1070.

Lampl, C., Yazdi, K. (2002) Central pontine myelinolysis. *Eur. Neurol*. **47**, 3–10.

Pekic, S., Doknic, M., Miljic, D., Saveanu, A., Reynaud, R., Barlier, A., Brue, T., Popovic, V. (2011) Case seminar: a young female with acute hyponatremia and a sellar mass. *Endocrine* **3**, 325–331.

Sajith, J., Ditchfield, A., Katifi, H. A. (2006) Extrapontine myelinolysis presenting as acute parkinsonism. *BMC Neurol*. **6**, 33.