Encephalopathy Due to Hypernatremic Dehydration: Computerized Tomography and Magnetic Resonance Imaging Findings: Case Report

HİPERNATREMİK DEHİDRATASYONA BAĞLI ENSEFALOPATİ: BİLGİSAYARLI TOMOGRAFİ VE MANYETİK REZONANS GÖRÜNTÜLEME BULGULARI

Ahmet Kemal FIRAT, MD,[a] Hakkı Muammer KARAKAŞ, MD,[a] Cengiz YAKINCI, MD[b]

Departments of [a]Radiology, [b]Pediatrics, Medical Faculty of İnönü University, MALATYA

Abstract

Magnetic resonance imaging (MRI) provides unique information about various pathological changes of the brain. We present CT and MRI findings of an infant with hypernatremic dehydration. CT imaging was performed at admission and MRI was obtained 4 weeks later. CT revealed hypodensity in the bilateral border zone of centrum semiovale and in the parasagital region of the left parietal lobe. These CT and MRI findings are compatible with vasogenic edema deteriorating to venous infarction. We present the early stage CT and late stage conventional MRI findings and discuss the effectiveness and priority of imaging techniques in this disease.

Key Words: Hypernatremia, brain diseases, venous infarction, Magnetic Resonance Imaging, computerized tomography

Özet

Magnetik rezonans görüntüleme (MRG), beyin patolojilerinin tanımlanmasında kullanılan önemli bir yöntemdir. Hipernatremik dehidratasyonu olan 1 olgunun BT ve MRG bulguları sunulmuştur. BT, hasta ilk başvurduğunda MRG ise takibin 4. haftasında elde edilmiştir. BT'de bilateral border zonu ve sol parasagital parietal zonu tutan hipodansite izlenmektedir. BT ve MRG bulguları bir arada değerlendirildiğinde vazojenik ödemden venöz infarkta ilerleyen süreç gözlenmektedir. Hipernatremik ensefalopatinin erken dönem BT ve geç ödem konvansiyonel MRG bulguları sunulmuş ve görüntüleme tekniklerinin etkinliği ve sıralaması tartışılmıştır.

Anahtar Kelimeler: Hipernatremik ensefalopati, venöz infarkt, MRG, bilgisayarlı tomografi

Turkiye Klinikleri J Med Sci 2006, 26:575-578

Hypernatremic encephalopathy (HE) has a wide spectrum of neurological signs and symptoms related to the serum sodium level and vary from mild lethargy to deep coma.

The medical literature contains only a few reports on the radiological findings of the central nervous system (CNS) in HE and its complications.[1-4]

Case Report

A 6 months-old boy was admitted to the pediatric critical care unit because of diarrhea, vomiting, lethargy and poor oral intake. He had a 7-day history of ten loose stools and five vomits per day, and two generalized convulsions in the last 12 hours. His body weight was 3.350 g. Since the mother was mentally retarded, maternal lactation was inadequate. In addition, he had been inadequately fed with an infant formula. Physical examination revealed a lethargic infant with signs of severe dehydration and hypovolemic shock. He was stuporous with generalized hypotonia. He had a heart rate of 205/min, respiratory rate of 40/min, and blood pressure of 50/15 mmHg. His skin was cool and dry. Serum electrolyte concentrations were as follows: Sodium 185 mmol/L, chloride 130 mmol/L, BUN 40 mg/dL, and creatinine 2 mg/dL. Arterial blood gas analysis showed pH 7.0, and HCO_3 7.8 mEq/L. Treatment for hypernatremic dehydra-

Geliş Tarihi/*Received:* 07.06.2005 **Kabul Tarihi/*Accepted:*** 02.01.2006

Yazışma Adresi/*Correspondence:* Ahmet Kemal FIRAT, MD
Turgut Özal Medical Center School of Medicine, Department of Radiology, 44069, MALATYA
ahmetfirat2@hotmail.com

tion, convulsions and acute renal failure with appropriate intravenous fluid and electrolyte therapy was initiated. After 48 h of treatment, serum sodium, BUN and creatinine concentrations returned to normal.

Initial CT images demonstrated diffuse hypodensity within the bilateral border zone of centrum semiovale and in the left paramedian parietal region (Figure 1). These findings support ischemia or vasogenic edema due to HE.

Four week after admission, MRI was performed. On MRI the left paramedian parietal lesion was hypointense on T1 and hyperintense on T2 weighted images (Figure 2A, 2B). T1-weighted images showed linear cortical T1 shortening and surrounding hypointensity in the same region representing laminar cortical necrosis (Figure 2A). FLAIR and T2-weighted imaging revealed diffuse T2 hyperintensity at the same region involving both the cortex and the subcortical white matter (Figure 2B). The venous sinuses were patent. These findings were compatible with venous infarction due to HE. On the follow up, there was no significant neurologic deficit except right hemianopia.

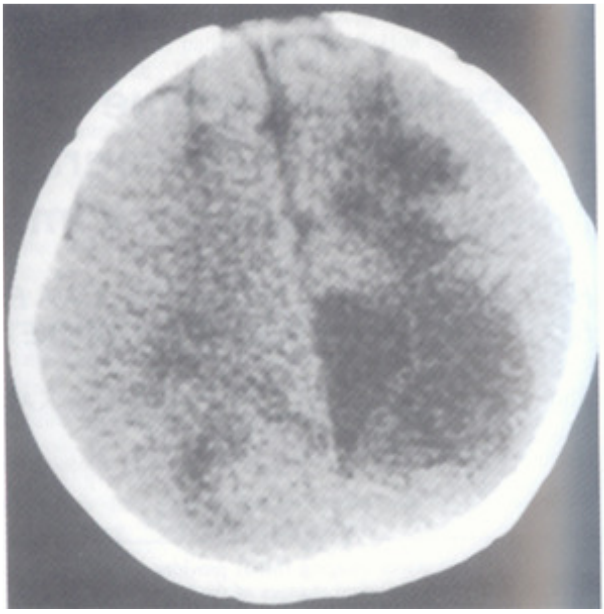

Figure 1. Axial CT image shows diffuse hypodensity in the bilateral border zone and in the left posterior parietal region.

Discussion

Hypernatremia may cause acute CNS dysfunction and even lead to permanent CNS sequelae. It may also lead to the development of multiple cerebral infarctions and extrapontine myelinolysis. Several reports on humans and animals describe pontine and extrapontine myelinolysis secondary to hypernatremia following a normonatremic state.[5,6]

The mechanism of HE is thought to be cellular dehydration, causing shrinkage of the cells and tearing of the fragile bridging veins, which results in bleeding.[7,8] Intracranial hemorrhage may be complicated by thrombosis of the small veins or the dural sinuses causing cerebral edema and venous infarction.[8-10]

The initial CT in our case revealed hypodensity in bilateral border zone and left paramedian parietal region. These finding could represent edema or ischemic changes. The differential diagnosis is possible by DWI in this situation.[11] Owing to locations and radiological features of the lesion, a complication of hypernatremic dehydration-venous infarction located in the left paramedian parietal region-was diagnosed. The bilateral hypodens border zone areas did not reveal any abnormal signal on MRI. The interval changes noted with the comparison of both imaging findings represent the pathway between venous congestion and venous infarction despite appropriate therapy. Edema due to congestion resolved at the border zones, however infarction appeared in the left parasagital parietal region.

Neuroimaging findings in HE have recently been described in the radiological literature.[2,3] The cranial MRI findings in severe hypernatremia were reported in a few cases.[5,7] They reported multiple cortical and subcortical hemorrhagic lesions, sagital and transverse sinus thrombosis, signal intensity alterations on thalamus and edema. Additionally, osmotic myelinolysis, especially at pons is a well

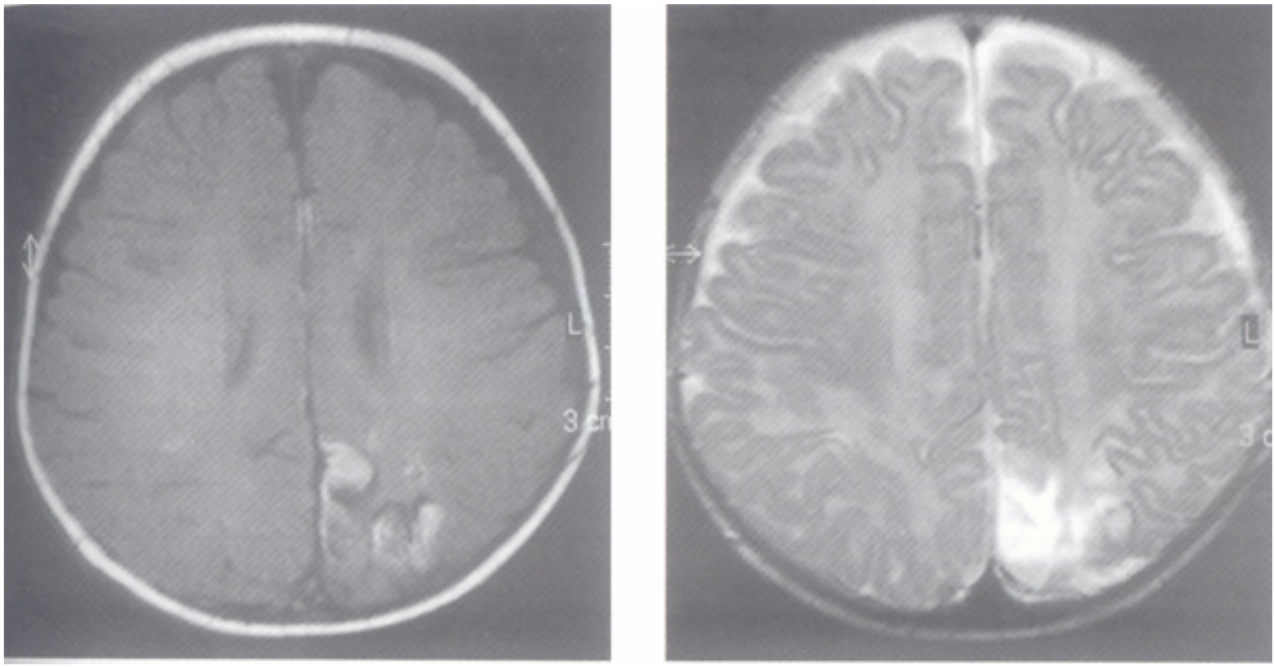

Figure 2. Axial T1 weighted image (TR: 560, TE: 15) shows multifocal linear cortical T1 shortenings and surrounding edema in the left parietal region **(A)**. Axial T2 weighted image (TR: 4530, TE: 100) reveals marked hyperintensity involving the cortex and the subcortical white matter **(B)**.

known entity due to rapid correction of hypernatremia. On MRI in the acute phase, hemorrhagic infarcts may be observed secondary to sinus thrombosis. These hemorrhages are hyperintense both on T1-T2 weighted images and are located adjacent to venous sinuses. US and CT are widely used techniques with well-established findings in this entity. On US these lesions are hyperintense and additionally obliterated sulci, fissures, cisterns and compressed ventricles are seen. On CT these hyperdense hemorrhagic lesions, hypodensity and obliteration of sulci due to edema may be observed.[5,7]

The primary goal in HE treatment is early diagnosis for rehydration, correction of electrolyte disturbances and control of seizures, and early detection of possible complications. DWI allows early recognition of acute ischemic changes prior to T1-T2 weighted images and differentiation of ischemic disease from resembling lesions. On DWI, infarcts show increased signal intensity representing restricted diffusion and they may be differentiated from vasogenic edema, which is not possible by conventional MRI and CT in the early stage. Besides, DWI may determine vasogenic edema representing venous congestion in HE, which may be reversible with early recognition.

US, CT and conventional MRI are the first choice of clinicians in the evaluation of HE since they are widely used imaging modalities. The radiologist needs to know the neurologic complications of hypernatremia and their imaging findings to avoid the major cause of morbidity and mortality. Further studies investigating the imaging findings of DWI in HE are needed to figure out its role in the early diagnosis.

REFERENCES

1. Schaefer PW, Grant PE, Gonzalez RG. Diffusion-weighted MR imaging of the brain. Radiology 2000; 217:331-45.
2. Han BK, Lee M, Yoon HK. Cranial ultrasound and CT findings in infants with hypernatremic dehydration. Pediatr Radiol 1997;27:739-42.

3. Mocharla R, Schexnayder SM, Glasier CM. Fatal cerebral edema and intracranial hemorrhage associated with hypernatremic dehydration. Pediatr Radiol 1997;27: 785-7.
4. Grunwald I, Reith W. Non-traumatic neurological emergencies: Imaging of cerebral ischemia. Eur Radiol 2002;12:1632-47.
5. AlOrainy IA, O'Gorman AM, Decell MK. Cerebral bleeding, infarcts, and presumed extrapontine myelinolysis in hypernatraemic dehydration. Neuroradiology 1999;41:144-6.
6. McKee AC, Winkelman MD, Banker BQ. Central pontine myelinolysis in severely burned patients: Relationship to serum hyperosmolality. Neurology 1988;38:1211-7.
7. Korkmaz A, Yigit S, Fırat M, Oran O. Cranial MRI in neonatal hypernatraemic dehydration. Pediatr Radiol 2000;30:323-5.
8. Votey SR, Peters AL, Hoffmann JR. Disorders of water metabolism: Hyponatremia and hypernatremia. Emerg Med Clin North Am 1989;7:749-69.
9. Finberg L. Hypernatremic (hypertonic) dehydration in infants. N Engl J Med 1973;289:196-8.
10. Conley SB. Hypernatremia. Pediatr Clin North Am 1990; 37:365-72.
11. Moritani T, Shrier DA, Numaguchi Y, et al. Diffusion-weighted echo-planar MR imaging: Clinical applications and pitfalls-a pictorial essay. Clin Imaging 2000;24: 181-92.