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Case Report / *Olgu Sunumu*

Cerebellar Infarction Complicated with Acute Hydrocephalus: A Case Report

Akut Hidrosefali Komplikasyonlu Serebellar Enfarkt: Olgu Sunumu

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Summary

Acute hydrocephalus is a rare manifestation of posterior circulation strokes. We present a patient with cerebellar infarct and secondary obstructive hydrocephalus. The patient's brain computed tomography scan showed severe third and lateral ventricular dilation suggestive of obstructive hydrocephalus. Following shunt placement, the patient recovered and was able to walk without assistance. *(Turkish Journal of Neurology 2012; 18:175-176)*

Key Words: Cerebellar infarction, acute hydrocephalus

Özet

Akut hidrosefali posterior dolaşım iskemik inmelerinde nadiren meydana gelir. Bu yazıda serebellar infarkt ve sekonder obstrüktif hidrosefalisi olan bir hasta sunulmuştur. Hastanın bilgisayarlı tomografi (BT) incelemesinde üçüncü ve lateral ventriküllerde akut obstrüktif hidrosefali düşündüren ciddi dilatasyon görülmüştür. Erken şant uygulaması ile hasta nörolojik olarak hızla düzelme göstermiş ve sonrasında desteksiz yürüyebilmiştir. *(Türk Nöroloji Dergisi 2012; 18:175-176)*

Anahtar Kelimeler: Serebellar enfakt, akut hidrosefali

Introduction

The main complaints and findings seen in cerebellar infarcts are ataxia, vertigo, dysarthria, nausea, vomiting and headache. Clinical worsening and coma may occur in addition to these symptoms of cerebellar dysfunction. This may occasionally result from reversible causes including development of hydrocephalus, brain stem compression. Timely and careful approach will certainly prove to be life-saving when deciding for a shunt procedure in a patient developing obstructive hydrocephalus following cerebellar infarct. The case presented here is a reminder for both this rare complication, and the treatment approach.

Case

Forty year old male patient was brought to the emergency room in our hospital with suddenly developing dizziness, loss of balance and vomiting. His neurological examination showed that he was conscious, orientation (spatial, temporal

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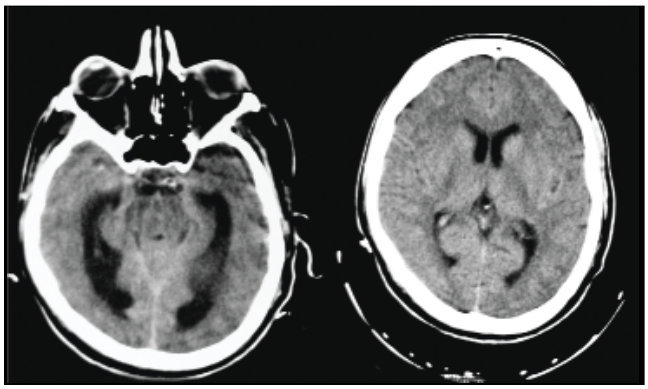

Image 1. The patient's brain CT scan taken after 24 hours

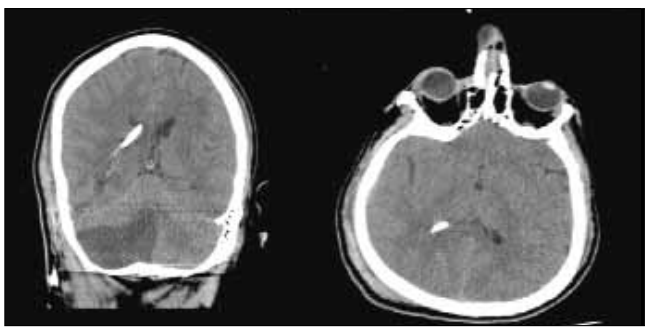

Image 2. The patient's brain CT scan taken post-surgery

and personal) and cooperation was assessed as normal. He had horizontal nistagmus with right gaze, dysarthric speech and ataxia of the rgunk with tendency to fall towards the right. Muscle strength was normal in four extremities. Babinsky response was positive on the right. There was nothing of interest in his medical history or his family history. His computerized brain tomography (CT) taken in the emergency room at admission was normal. Signal increase in the right hemisphere of the cerebellum and hypointensity in ADC mapping was demonstrated in diffusion weighted magnetic resonance imaging (DWI). Blood chemistry (AST, ALT, GGT, ALP, urea, creatinine, creatine kinase, electrolites, cholesterol levels, triglicerides, complete blood count, erythrocyte sedimentation rate, HbA1c) and urinalysis were found to be within normal range. Treatment was initiated with enoxaparin (4000 Anti-XA IU/4 ml 2x1 SC), acetylsalicylic acid 300 mg/day, piracetam 2400 mg/day. Subacute infarct area was seen in the right cerebellar hemisphere in his brain CT scan taken 24 hour later. The patient's consciousness deteriorated progressively after 48 hours, and he was able to localize painful stimuli. Brain CT scan was repeated when the Glascow coma scale score decreased to 7/15, and subacute infarct and hydrocephalus was detected (Image 1). As the patient did not have hydrocephalus in his previous neuroimaging investigations, an urgent decision was made as a result of the neurosurgical consultation to place a ventriculoperitoneal shunt. Following the procedure, CT scan showed that hydrocephalus had disappeared, there was a subacute-chronic infarct area in the right cerebellar hemisphere and the catheter in the right lateral ventricle could be seen (Image 2). The patient's neurological examination performed on Day 5 following the procedure showed that he was conscious, and muscle strength was complete in all four extremities. His gait was ataxic to the right, and his speech was dysarthric. He had horizontal nistagmus with right gaze.

Discussion

Cerebellar infarcts may cause death as a result of pressure increase in the posterior fossa and pressure on the brain stem due to edema. Moreover, the aquaductus or the fourth ventricle may close because of edema and cause obstructive hydrocephalus and acute intracranial pressure increase (1). Mortality is reported to be 5-23% in cerebellar infarct cases (2, 3, 4). Death occurs as a result of pressure on the brain stem and cardiac arrest (3). As shown in the repeated brain CT scans of our patient, acutely developing hydrocephalus is the cause of worsening in the clinical picture. Therefore, patients with cerebellar infarct should be followed closely for neurologic signs, patients with consciousness impairment should be assessed with a neurosurgical team for surgical intervention in a timely fashion. The surgical treatment indications in ischemic stroke are quite limited. Temporary external ventricular drainage or permanent shunt systems may be considered to prevent progressive neurologic worsening (5, 6). In conclusion, we wished to point out that a timely surgical procedure in a cerebellar infarct case where acute hydrocephalus developed could be life saving.

References

1. Lehrich JR, Winkler GF, Ojermann RG. Cerebellar infarction with brainstem compression. Arch Neurol 1970;22:490-8.
2. Amarenco P. The spectrum of cerebellar infarction. Neurol 1991;41:973-9.
3. Macdonell RA, Kalnins RM, Donnan GA. Cerebellar infarction: Natural history, prognosis and pathology. Stroke 1987;18:849-55.
4. Shenkin HA, Zavala M. Cerebellar strokes: Mortality, surgical indications and result of ventricular drainage. Lancet 1982;12:429-31.
5. Gurol ME, St Louis EK, Treatment of Cerebellar Mases. Current treatment options in Neurology 2008;10:128-50.
6. Baldauf J, Oertel J, Gaab MR, Schroeder HW. Endoscopic third ventriculostomy for occlusive hydrocephalus caused by cerebellar infarction. Neurosurgery 2006;59:539-44.