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CRANIAL COMPUTED TOMOGRAPHIC FINDINGS IN A PATIENT WITH HYPERTENSIVE ENCEPHALOPATHY IN ACUTE POSTSTREPTOCOCCAL GLOMERULONEPHRITIS*

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SUMMARY: Saatçi I, Topaloğlu R (Departments of Radiology and Pediatrics, Hacettepe University Faculty of Medicine, Ankara, Turkey). Cranial computed tomographic findings in a patients with hypertensive encephalopathy in acute poststreptococcal glomerulonephritis. Turk J Pediatr 1994; 325-328.

A 13-year-old girl with a severe headache, blurred vision, altered mental status, seizures, high blood pressure, edema and hematuria is presented. With a previous history of upper respiratory tract infection, acute onset of edema, gross hematuria, high ASO and low C_3 levels, she was diagnosed with acute poststreptococcal glomerulonephritis (APSGN) and hypertensive encephalopathy. Computed tomography (CT) revealed symmetric hypodense areas representing edema in the parieto-occipital regions. As noted in previous reports, these CT findings are of value in establishing the diagnosis of hypertensive encephalopathy. In this particular case the CT appearance and the subsequent clinical improvement without any neurological deficit supported the diagnosis of hypertensve encephalopathy due to APSGN. We emphasize that awareness of the CT findings of hypertensive encephalopathy may facilitate in making the correct diagnosis in symptomatic hypertensive patients, especially in cases with an unusual presentation or clinical course. *Key words: CT findings, hypertensive encephalopathy, acute poststreptococcal glomerulonephritis.*

Encephalopathy refers to a non-infective cause of brain dysfunction. A wide spectrum of conditions including hypertension can cause encephalopathy[1,2].

Hypertension is found in over 75 percent of acute poststreptococcal glomerulonephritis (APSGN) cases, but hypertensive encephalopathy due to APSGN has been reported rarely in children[3].

We present a 13-year-old girl with hypertensive encephalopathy due to APSGN and the cranial computed tomographic (CT) findings.

Case Report

A 13-year-old girl was admitted to the hospital with altered mental status. Her past history revealed a sore throat and fever about 20 days prior, and after a latent period of ten days, she developed edema, nausea, vomiting and gross

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hematuria. She was referred to Hacettepe Children's Hospital when she developed a severe headache, blurred vision, altered mental status, seizures and high blood pressure (180/110 mmHg) one week after the onset of edema and hematuria.

Physical examination revealed a blood pressure of 150/110 mmHg and a stuporous state with (+) Babinsky sign on the left side. The fundoscopic examination showed bilateral minimal papilledema.

Laboratory findings revealed a hemoglobin of 15 g/dl, hematocrit 44% and white blood cell count of 8500/mm^3 with 54% neutrophils and 46% lymphocytes. Urine analysis showed a specific gravity of 1025, 1+ proteinuria, numerous erythrocytes and 5-6 leukocytes per high power field in urinary sediment. BUN, serum creatinine, electrolytes, total protein, albumin, SGOT, SGPT and alkaline phosphatase levels were within normal limits. Urine and throat cultures were negative. The chest and abdominal ultrasonography were unremarkable. Antistreptolysine-O (ASO) titer was 500 U, C-reactive protein (CRP) + erythrocyte sedimentation rate 50 mm/hr, C_3 30 mg/dl, C_4 20 mg/dl, and antinuclear antibody (ANA) was negative. The evaluation of cerebrospinal fluid (CSF) was normal. EEG demonstrated paroxysmal activity in the right posterior hemisphere with a slow background rhythm. CT examination performed four days after the onset of acute signs and symptoms demonstrated symmetric hypodense areas in the parieto-occipital regions bilaterally (Fig. 1).

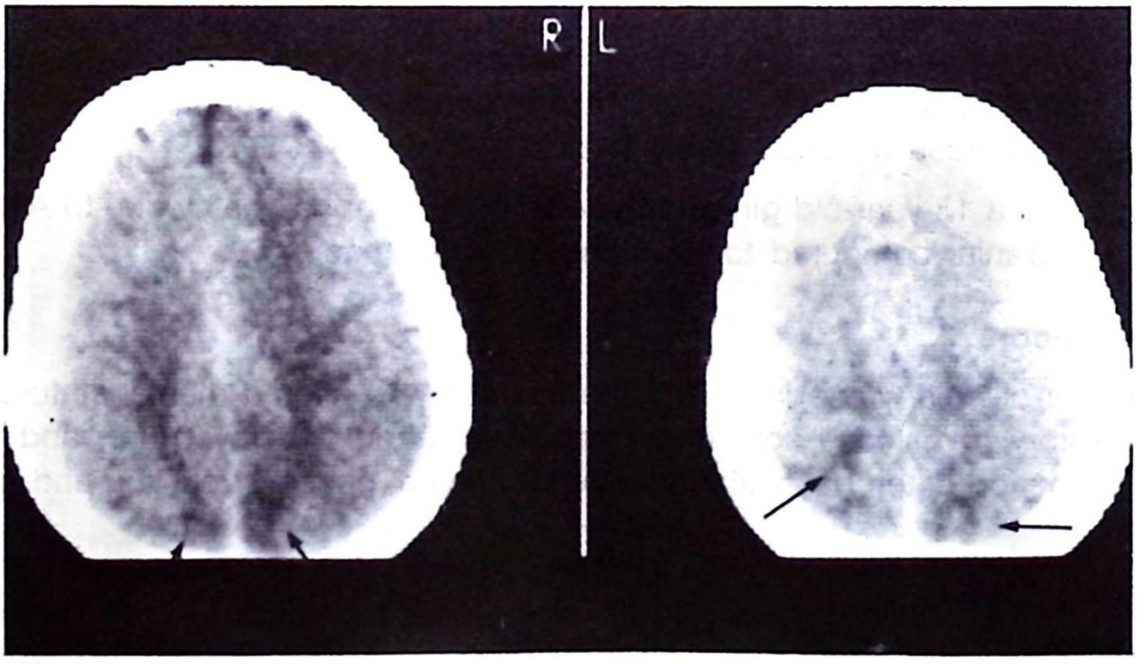

Fig. 1: Contiguous axial non-contrast CT images reveal bilateral parieto-occipital areas with decreased attenuation, more obvious on the right side.

Discussion

Hypertensive encephalopathy is a neurologic syndrome characterized by headache, visual disturbances, seizures, altered mental status and focal neurologic signs[1,2]. Various neurologic conditions such as stroke, intracranial hemorrhage, venous thrombosis and encephalitis can mimic hypertensive encephalopathy[4]. These were excluded with the CSF analysis and CT in the present case. Even after controlling the patient's blood pressure, her stuporous state and agitation persisted, arousing the suspicion of systemic lupus erythematosis (SLE) or other vasculitic syndromes. SLE and other vasculitic syndromes were ruled out with the normal C_4 level, negative ANA and lack of signs and symptoms related to vasculitis. With the typical history and classical clinical presentation of APSGN of acute onset of edema, hematuria, high ASO titer and low C_3 level, the patient was diagnosed with APSGN and hypertensive encephalopathy.

Hypertension is found in over 75 percent of APSGN cases. Hypertension is usually mild to moderate and not accompanied by the retinal alterations of malignant hypertension[5]. Hypertension is usually more evident at or near the onset of the renal disease and subsides promptly with the onset of diuresis. Encephalopathy is occasionally a complication of APSGN in children[3].

Hypertensive encephalopathy is a subacute phenomenon requiring several days of increasing pressure to manifest. The susceptibility of the posterior circulation to the lesions of hypertensive encephalopathy is well-known[6]. The theory of the regional heterogeneity of the sympathetic vascular innervation has been proposed for the posterior predilection of the lesions in hypertensive encephalopathy[7]. Schwartz et al.[8] noted that the imaging findings in hypertensive encephalopathy are distinct from those in acute as well as chronic hypertension. Acute hypertensive crises may result in cerebral hemorrhage or infarction, whereas in chronic hypertension multiple small infarctions and focal hemorrhages are frequently seen in the basal ganglia, thalamus, centrum semiovale and, less commonly, the pons and cerebellum. In their series of 14 patients with hypertensive encephalopathy, all displayed edema in the cortex and subcortical white matter of the occipital lobes.

In consistency with the previous reports, bilateral hypodensity representing edema in the parieto-occipital area as well as the subsequent clinical improvement of the patient without any neurological deficit supported the diagnosis of hypertensive encephalopathy due to APSGN in this particular case.

The present case report is intended to emphasize that awareness of the CT appearance of hypertensive encephalopathy may facilitate in making the correct diagnosis in symptomatic hypertensive patients, particularly those who have an atypical presentation or clinical course.

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