Reversible CT changes in uremic encephalopathy.

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Reversible CT Changes in Uremic Encephalopathy

CNS involvement is observed occasionally in patients with renal failure. Its causes are thought to be metabolic abnormalities and organic changes [1–4]. Only a few reports have described cranial CT changes in uremic encephalopathy [3, 4]. We present a case of major uremic encephalopathy in which serial CT scans showed a return to normal.

Case Report

A 16-year-old girl who was in shock was admitted to the hospital after a traffic accident. The only obvious injury was a fracture of the left humerus. She had mild abdominal tenderness and gross hematuria and was semicomatose, but no focal neurologic deficit was present. Her WBC count, SGOT, SGPT, and serum levels of lactic dehydrogenase and fibrin degradation products were elevated slightly, but serum levels of blood urea nitrogen and creatinine were normal. Abdominal echography and CT showed injury to the right kidney, but a cranial CT scan was normal.
on the day after admission, the patient became alert and had no neurologic abnormalities, but mild renal dysfunction continued. Her blood pressure returned to normal. On the ninth day, her renal function deteriorated severely, and anuria developed. Subsequently, frequent generalized seizures occurred, and she became comatose. Peritoneal dialysis was carried out. Cranial CT scans at this time showed diffuse hypodense areas in the white matter, whereas the density of the gray matter was normal. However, papilledema was not observed (Fig. 1A). Electroencephalography showed diffuse slow waves. Serum levels of blood urea nitrogen and creatinine were elevated (92 and 9.6 mg/dl, respectively), but levels of fibrin degradation products were normal. Her renal failure and alteration in consciousness continued for 11 days.

With the recovery of renal function, her level of consciousness gradually improved, and finally she became alert and showed no neurologic deficits. On cranial CT scans, the density of the white matter returned to normal (Fig. 1B), and her electroencephalogram also became normal.

Discussion

A wide spectrum of CNS involvement has been observed in patients who have renal failure. Usually referred to as uremic encephalopathy, the major signs have included focal and generalized seizures, significant alterations of consciousness, hemiparesis, myoclonus, and decerebrate spasms. The causes are thought to be metabolic abnormalities (hyponatremia, hypocalcemia, acidosis) and organic changes (brain edema, cerebral infarction, microthrombosis) [1-4].

Cranial CT findings in uremic encephalopathy have been reported in only three cases of hemolytic uremic syndrome. Crisp et al. [3] described two cases in which CT scans showed hypodense lesions at first and hemorrhages that developed after 2 weeks. Mendelsohn et al. [4] described a case of extensive brain hypodensity that showed diffuse enhancement of the gray-white matter interface 10 days after the onset; the cause was thought to be cerebral microangiopathy. Some autopsy reports [1, 2] have noted evidence of hypoxic changes and microthrombi.

CT changes in our case were reversible. Previous published reports did not describe whether the CT changes returned to normal. Savazzi et al. [5] and Cusmano and Savazzi [6] described cortical atrophy, cerebral infarctions, and calcifications in cranial CT scans of patients who were on long-term hemodialysis. Those abnormalities were progressive and due to organic changes. Some aspects of the CT findings in our case were similar to those of cerebral sinus thrombosis [7, 8], but this diagnosis did not fit the findings, particularly the normal reversible CT changes, the benign clinical course, and the normal level of fibrin degradation products.

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Paraneoplastic Cerebellar Degeneration in a Patient with Ovarian Carcinoma

Paraneoplastic cerebellar degeneration (PCD) is a rare complication of human malignant diseases, particularly those involving the lung, ovary, breast, and lymphoid system [1-4]. It causes subacute and progressive atrophy of the cerebellum that may not be evident on CT until a few months after the onset of the disease [3]. This case is unique because ovarian carcinoma was diagnosed only after an intensive search for the cause of cerebellar atrophy found on CT.

Case Report

A previously well 55-year-old woman who was a nonsmoker was admitted to our hospital for evaluation of gait imbalance, dysarthria, and appendicular ataxia that was greater on the left than on the right. All had progressed over a 6-month period. Cranial CT (Fig. 1) showed diffuse cerebellar cortical atrophy and a normal cerebral cortex. The results of lumbar puncture showed an elevated protein level and a mild lymphocytosis. The results of hematologic tests were within normal limits except that titers of antibodies to Purkinje cells were high (1:4000) and migration of specific antigen on immunoblots oc-