AJNR

Paraneoplastic cerebellar degeneration in a patient with ovarian carcinoma.

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AJNR Am J Neuroradiol 1988, 9 (1) 216-217
http://www.ajnr.org/content/9/1/216.citation

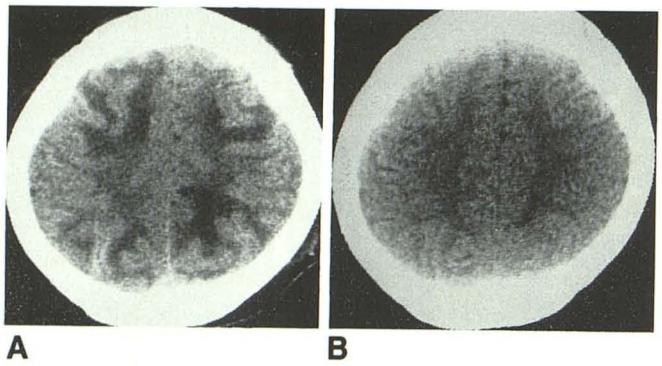

Fig. 1.—Cranial CT scans of 16-year-old girl with uremic encephalopathy.

***A*, Plain scan obtained when uremic encephalopathy was present shows diffuse hypodensity in the white matter but not in gray matter. Cerebral swelling was not observed.**

***B*, Follow-up scan obtained after recovery of renal failure shows a return to 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 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-

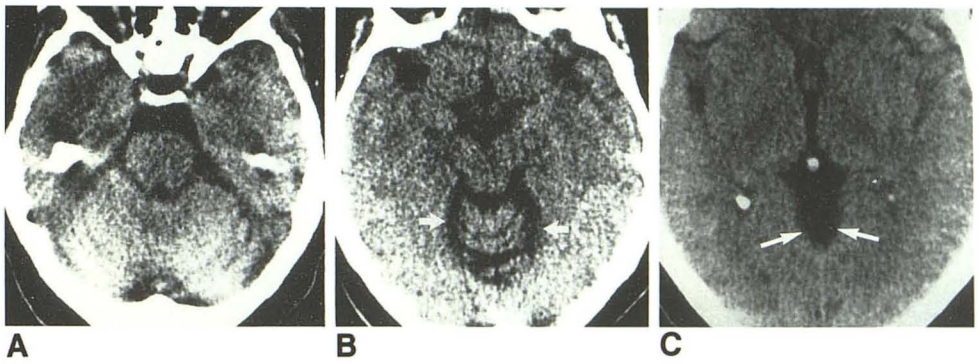

Fig. 1.—*A–C*, Plain cranial CT scans at levels of fourth ventricle (*A*), midbrain (*B*), and third ventricle (*C*) show widening of supracerebellar cistern (*long arrows*) and prominent cerebellar folia cisterns (*short arrows*). Fourth ventricle is normal.

curred in a pattern previously reported as consistent with PCD [4]. An intensive search for malignancy was carried out, but the findings on chest film, mammograms, thyroid scan, and chest and abdominal CT scans were all within normal limits.

The results of a pelvic examination suggested a uterine leiomyoma but no abnormalities of the ovarian structures. These findings were confirmed by pelvic CT scan.

The patient's condition continued to deteriorate, and selective laparoscopy and laparotomy were performed 2 months after her first admission to the hospital. A 1.5-cm mass that was discovered in the right ovary proved to be a poorly differentiated carcinoma. No metastases were found. The results of a serum assay for antibodies to Purkinje cells were positive (1:1000). The patient's clinical condition stabilized after the surgery.

Discussion

PCD is characterized pathologically by a widespread loss of Purkinje cells and thinning of the granular layers of the cerebellum. Jaeckle et al. [4] reported that the presence of antibodies to Purkinje cells is specific for PCD, though not all patients who had this syndrome tested positive for the antibody. In addition, Greenlee and Brashear [2] detected antibodies in two of 14 patients who had ovarian carcinoma and no neurologic symptoms. The antigenic stimulus that leads to the production of these antibodies is unclear, and several hypotheses have been suggested [4].

The CT findings of PCD are nonspecific. The differential diagnosis includes hereditary cerebellar degenerative disorders and alcohol abuse. In the absence of either alcohol abuse or a family history of PCD, a patient who has a progressive cerebellar ataxia and a CT scan that shows generalized cerebellar cortical atrophy should be evaluated for the presence of an occult malignancy.

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A Mimicker of a Postoperative Spinal Mass: Gelfoam in a Laminectomy Site

After a laminectomy, absorbable gelatin sponges (Gelfoam) may be placed in the dorsal epidural space. The Gelfoam mixes with blood and plasma and on MR may have an appearance that simulates a compressive postoperative epidural mass. We report two such cases.

Case Reports

Case 1

A 55-year-old man underwent a laminectomy at T8–T11 for drainage of a posttraumatic spinal cord cyst. The patient complained of postoperative back pain and had fluctuating weakness in his right leg. An MR scan obtained 11 days after surgery showed an abnormality that simulated an epidural mass (Fig. 1 A–C). Because he had no significant change in his neurologic symptoms, the patient was followed clinically, and his pain and weakness improved. On the follow-up MR obtained 10 days later, the effect of the epidural mass was diminished (Fig. 1D).

Case 2

A 42-year-old woman had a laminectomy from T6–T7 to T9 for resection of a cord hematoma. On MR 9 days later, an inhomogeneous mass with a similar effect and signal intensity as that seen in case 1 was noted at the laminectomy site (Fig. 2). The patient experienced no postoperative neurologic deterioration.

Discussion

When intradural spinal surgery is performed, Gelfoam strips or particles are commonly placed in the dorsal spinal canal at the levels of laminectomy after the dura is closed. Historically, Gelfoam was used to prevent epidural scarring [1], but because recent studies have shown that it induces scarring, our surgeons use Gelfoam in