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Reversible CT changes in uremic encephalopathy.

Y Komatsu, A Shinohara, C Kukita, T Nose and Y Maki

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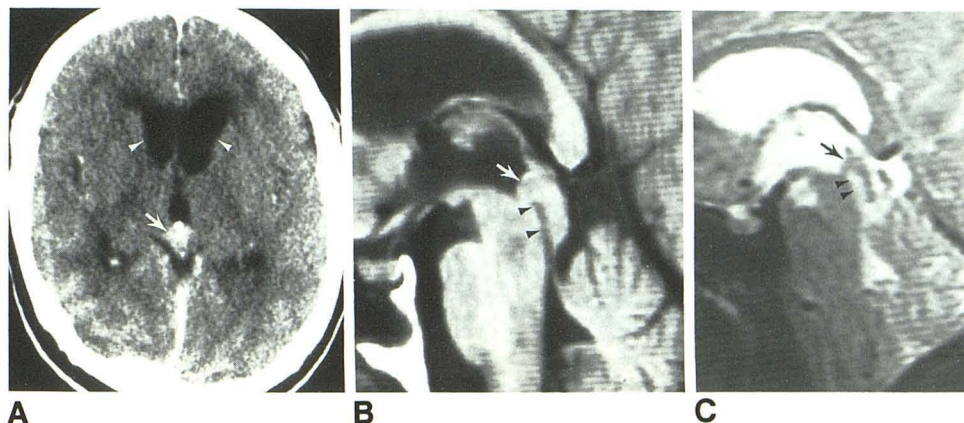
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Fig. 1.—Primary melanoma of the quadrigeminal plate.

A, Postcontrast axial CT scan shows small contrast-enhancing mass posterior to third ventricle (arrow), causing obstructive hydrocephalus (arrowheads).

B and C, Midline sagittal MR images show isointense mass located within superior aspect of quadrigeminal plate (arrows), causing obstruction of proximal aqueduct of Sylvius (arrowheads). B = T1-weighted (TR 600 msec/TE 20 msec, 5 mm thick, 2.5-mm gap); C = T2-weighted (TR 2500 msec/TE 80 msec, 3 mm thick, 1.5-mm gap).



benign melanosis of the leptomeninges and primary CNS melanomas [1–3]. Primary meningeal melanomas have a peak incidence in the fourth decade, are relatively radioresistant, and carry a poor prognosis; most patients die within 1 year of diagnosis. Hydrocephalus is often present, principally because of preponderant involvement of the basal leptomeninges. In the few previously described cases of primary melanoma of the pineal region, noncommunicating hydrocephalus was due to obstruction of the aqueduct [1], as was seen in our patient.

The MR characteristics of primary CNS melanoma have not been described previously. The relatively isointense signal on T1- and T2-weighted images of this melanotic melanoma of the quadrigeminal plate differs from most CNS tumors, which appear isointense to hypointense on T1-weighted images and hyperintense on T2-weighted images [9].

The cause of the peculiar MR characteristics of melanoma within the CNS [7, 8] and within the globe [4–7] is a subject of debate. A preliminary report [7] described the signal intensities of melanoma metastatic to the brain as “highly variable.” Five lesions had high signal intensity on T1-weighted images; five other lesions had low signal intensity on T2-weighted images. When six lesions were evaluated histologically, the MR signal intensities correlated best with the presence of acute or chronic hemorrhage within the lesion [7]. Gomori et al. [5] correlated the melanin content of six choroidal melanomas with their MR appearance. A trend toward shorter T1 and T2 with increasing melanin content was attributed to the paramagnetic free radicals in melanin. Melanotic choroidal melanomas appeared hyperintense on T1-weighted images and hypointense on T2-weighted images in their study [5].

MR characteristics of the normal and abnormal mesencephalic tectum have been described recently [10]. On midline sagittal T1-weighted images, the average anteroposterior diameter of the superior and inferior colliculi was 5 mm (range, 2–7 mm). The superior aspect of the quadrigeminal plate measured in this manner in our patient was 9 mm, exceeding the upper limits of normal.

Steven M. Weindling
Gary A. Press
John R. Hesselink

University of California, San Diego, Medical Center
San Diego, CA 92103

REFERENCES

1. Gibson JB, Burrows D, Weir WP. Primary melanoma of the meninges. *J Pathol Bacteriol* 1957;74:419–438
2. Rubinstein LJ. Tumors of the central nervous system. in: *Atlas of tumor pathology*, 2nd series, fasc. G. Washington, DC: Armed Forces Institute

of Pathology, 1972:309–311

3. Copeland DD, Sink JD, Seigler HF. Primary intracranial melanoma presenting as a suprasellar tumor. *Neurosurgery* 1980;6:542–545
4. Edwards JH, Hyman RA, Vacirca SJ, et al. 0.6T magnetic resonance imaging of the orbit. *AJNR* 1985;6:253–258, *AJR* 1985;144:1015–1020
5. Gomori JM, Grossman RI, Shields JA, et al. Choroidal melanomas: correlation of NMR spectroscopy and MR imaging. *Radiology* 1986;158:443–445
6. Mafee MF, Peyman GA, Grisolan JE, et al. Malignant uveal melanoma and simulating lesions: MR imaging evaluation. *Radiology* 1986;160:773–780
7. Woodruff WW Jr, Djang WT, McLendon RE, Heinz RE, Voorhees DR. High-field MR imaging of intracerebral melanoma. Presented at the annual meeting of the Radiological Society of North America, Chicago, December 1986
8. Atlas SW, Grossman RI, Hackney DB, Goldberg HI, Bilaniuk LT, Zimmerman RA. MR imaging of intracranial metastatic melanoma. Presented at the annual meeting of the Radiological Society of North America, Chicago, December 1986
9. Lee BCP, Kneeland JB, Cahill PT, Deck MDF. MR recognition of supratentorial tumors. *AJNR* 1985;6:871–878
10. Sherman JL, Citrin CM, Barkovich AJ, Bowen BJ. MR imaging of the mesencephalic tectum: normal and pathologic variations. *AJNR* 1987;8:59–64

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.

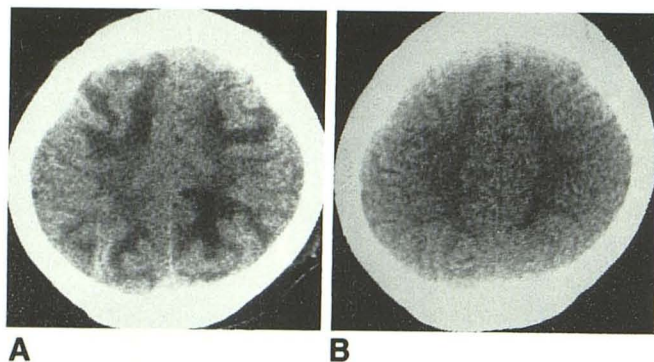


Fig. 1.—Cranial CT scans of 16-year-old girl with uremic encephalopathy.

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.

Yoji Komatsu
Akira Shinohara
Chikashige Kukita
Hata General Hospital
Ayukawa, Hitachi, Ibaraki 316, Japan
Tadao Nose
Yutaka Maki
University of Tsukuba
Tsukuba Science City, Ibaraki 305, Japan

REFERENCES

1. Rooney JR, Anderson RM, Hopkins IJ. Clinical and pathological aspects of central nervous system in the haemolytic uremic syndrome. *Aust Paediatr J* 1971;7:28–33
2. Upadhyaya K, Barwick K, Fishaut M, Kashgarian M. The importance of non renal involvement in hemolytic uremic syndrome. *Pediatrics* 1980;1:115–120
3. Crisp DE, Siegler RL, Bale JF, Thompson JA. Hemorrhagic cerebral infarction in the hemolytic-uremic syndrome. *J Pediatr* 1981;99:273–276
4. Mendelsohn DB, Hertanu Y, Chaitowitz B, Cartwright JD. Cranial CT in the haemolytic uremic syndrome. *J Neurol Neurosurg Psychiatry* 1984;47:876–878
5. Savazzi GM, Cusmano F, Degasperis T. Cerebral atrophy in patients on long-term regular hemodialysis treatment. *Clin Nephrol* 1985;23:89–95
6. Cusmano F, Savazzi GM. Cerebral computed tomography in uremic and hemodialyzed patients. *J Comput Assist Tomogr* 1986;10:567–570
7. Buonanno FS, Moody DM, Ball MR, Laster DW. Computed cranial tomographic findings in cerebral sinovenous occlusion. *J Comput Assist Tomogr* 1978;2:281–290
8. Kingsley DPE, Kendall BE, Moseley IF. Superior sagittal sinus thrombosis: an evaluation of the changes demonstrated on computed tomograph. *J Neurol Neurosurg Psychiatry* 1978;41:1065–1068

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-