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An unusual CT appearance of lupus cerebritis.

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Pseudofoamina of the Skull Base: A Normal Variant

Radiologic evaluation of basal foramina of the skull is frequently a critical aspect in the diagnosis of patients with deficits referable to cranial nerves. Commonly encountered normal asymmetries and individual variations often make interpretation difficult. A pseudofoamina in the skull base was initially observed in skull radiographs of several patients and correlated with images of a dried skull. We stress the importance of recognizing pseudofoamina to avoid diagnostic confusion and error.

Bilateral, rounded lucent areas with sclerotic margins may be visualized on submentovertex or Water's views of the skull base. Located anteromedially to the hypoglossal canals and directly medial to the jugular foramen, these structures may be mistaken for the hypoglossal canal, erosion of the jugular foramen, or other normal structures (Fig. 1). The posterior margins are well defined while the anterior margins of pseudofoamina are indistinct, suggestive of bony erosion. Asymmetry between the two sides is frequent. Pseudofoamina may also be seen on CT scans of the skull base.

Using a dried skull for anatomic correlation, a pointer was placed adjacent to the medial border of the hypoglossal canal outlining the posterior margin of the pseudofoamena while the tip of a second pointer in the anterior condylar fossa lay in the center of the pseudofoamena (Fig. 2). The hypoglossal canal begins on the anteromedial aspect of the foramen magnum and runs anterolaterally and inferiorly, exiting the skull base lateral to the occipital condyles. The thicker medial wall of the hypoglossal canal forms the posterior wall of the pseudofoamena. The longus capitis muscle originates at the C6 vertebral level and inserts on a bony ridge just anterior to the anterior condylar fossa [1]. This ridge forms the anterior border of the foramen. The size and thickness of this ridge is subject to considerable individual variation, presumably reflecting muscle bulk and use patterns. This may also account for the variation in clarity with which the anterior margin of the foramen is seen. The anterior condylar fossa is located posterior to the bony insertion of the longus capitis muscle;

its relative lucency accounts for the apparent opening of the pseudofoamena.

CT of the region of the foramen magnum in a dried skull also demonstrates the presence of the pseudofoamena (Fig. 3), but only at certain gantry angulations, which vary for each patient.

Correlation of radiographic features of the pseudofoamena in both dried skull and clinical case material demonstrates the anatomic basis for this normal variant. Recognition of its benign nature is vital to avoid diagnostic error in evaluation of the skull base.

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An Unusual CT Appearance of Lupus Cerebritis

Cerebritis is a common complication of systemic lupus erythematosus (SLE). It affects 14-75% of SLE patients and is a leading cause of death [1]. Diagnosis of this disease remains difficult because of its nonspecific and varied presentation. Persistent headache, alteration in mental status, seizures, psychiatric symptoms, and stroke syndromes among others may represent lupus cerebritis. Patients with such symptoms require prompt corticosteroid therapy.

CT plays an important role in diagnosing lupus cerebritis, and its patterns have been the subject of several recent reviews [2-6]. Two general CT patterns have been described, one associated with an acute clinical presentation, e.g., infarction or focal hemorrhage, and another with more chronic symptoms and signs. CT may demonstrate single or multiple infarctions or hemorrhages in areas unusual for hypertensive bleeding. Patients with a chronic or insidious presenta-

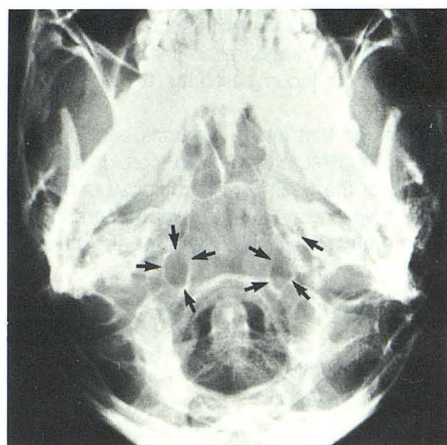


Fig. 1.—Plain film demonstration of bilateral pseudofoamina, more prominent on right (arrows).

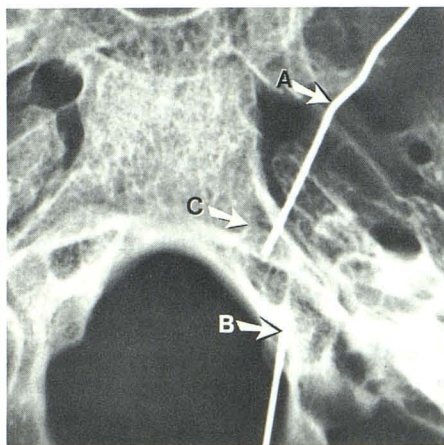


Fig. 2.—Dried skull radiograph. A = tip in anterior condylar fossa appears inside pseudofoamena; B = tip adjacent to medial border of hypoglossal canal; C = bony ridge for insertion of longus capitis muscle.

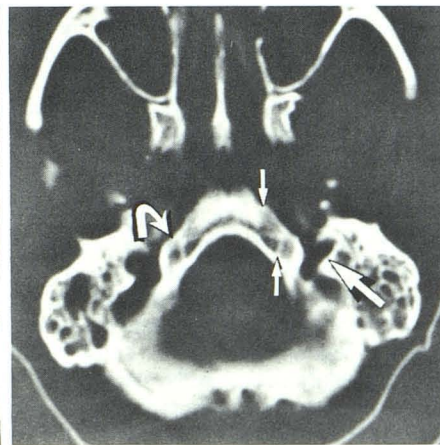


Fig. 3.—CT of dried skull demonstrates anterior and posterior borders of pseudofoamena (small arrows), showing relationship to hypoglossal canal (curved arrow) and jugular foramen (large arrow).

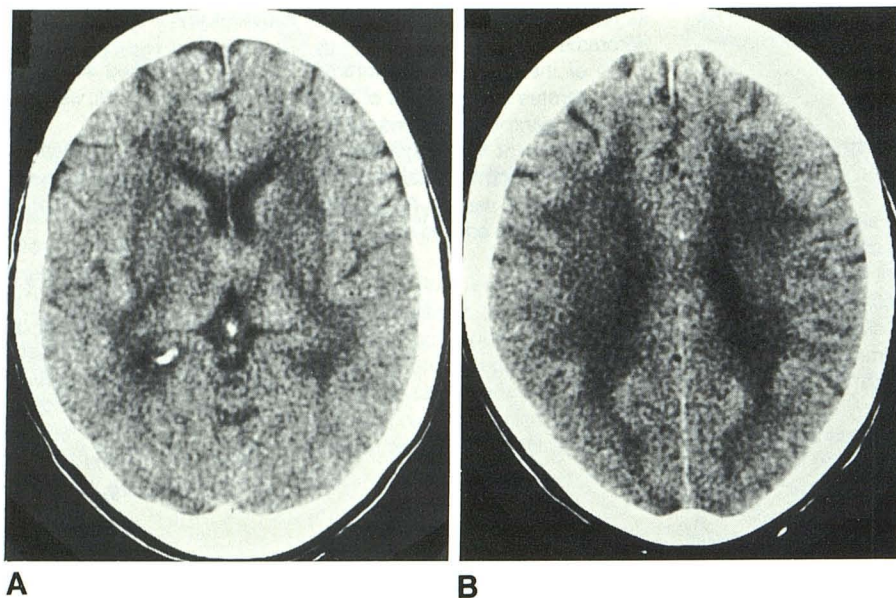


Fig. 1.—Cranial CT scan showing extensive white matter low-density areas with (A) and without (B) contrast enhancement.



Fig. 2.—Leptomeningeal vessel showing vasculitic changes.

tion show enlarged ventricles and subarachnoid spaces that are considered unrelated to chronic steroid use.

We encountered a patient with pathologically proven cerebritis whose CT, rather than conforming to established patterns, demonstrated extensive nonenhancing white matter low density simulating progressive multifocal leukoencephalopathy (PML) [7]. This pattern has not been previously reported.

Case Report

The patient is a 64-year-old woman with a 2-year history of SLE diagnosed in line with the 1982 American Rheumatism Association revised criteria (arthritis, serositis, anti-DNA antibody of 50.5 [0–20 normal], ANA of 1:320, and questionably related thrombocytopenia 6 years prior) [8].

She presented in December 1985 with a 10–12-day history of headache, reduced alertness, fever, and periodic urinary incontinence. Medications had included maintenance Prednisone 5 mg/day, which was then increased to 40 mg/day for presumed cerebritis. Upon admission to our hospital she was oriented to her name only, was dyscalculic, neglected her left side, and showed marked bifrontal release. Additionally, she would not open her eyes to command while being otherwise cooperative. There were no other focal findings.

Cranial CT scan (Fig. 1) revealed extensive white matter low density without contrast enhancement. At the time of brain biopsy, the neurosurgeon noted multiple small cortical infarctions. Microscopic examination (Fig. 2) of the cerebral biopsy revealed scattered foci of fibrinoid necrosis involving leptomeningeal arterioles and venules. The adjacent subarachnoid space contained dense aggregates of inflammatory cells, mostly macrophages and lymphocytes with smaller numbers of neutrophils. There were also multiple small areas of edema and early gliosis within the cortex and white matter. However, no abnormalities of intraparenchymal blood vessels were found.

Accordingly, the patient was treated with 100 mg of Prednisone per day and made impressive improvement in cognition and mental

status. She remains improved on maintenance Prednisone 30 mg/day 1 year later.

Discussion

To our knowledge, this is the first case of lupus cerebritis displaying bilateral nonenhancing deep white matter low-density lesions reminiscent of PML. PML was initially suspected in this case on the basis of the white matter low densities, the lack of mass effect, and the lack of contrast enhancement. However, lupus cerebritis was confirmed subsequently with brain biopsy and response to corticosteroids.

We believe the extensive white matter low density in this case represents multiple areas of infarction secondary to small vessel vasculitic occlusions.

From a clinical standpoint, this case is important as the therapy is radically different for these two diseases. High-dose corticosteroid treatment is required for lupus cerebritis while such therapy would be contraindicated in PML. This CT appearance needs to be recognized as an unusual pattern for lupus cerebritis to allow proper diagnosis and therapy.

Finally, note should be made that lupus cerebritis and PML can coexist in the same patient, the PML attributed to steroids or immunosuppression in treatment of the SLE [9]. Brain biopsy is the only clear means of determining which one, PML or lupus cerebritis or both, may be operative in a patient with SLE and a CT picture like that seen in this case.

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