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Cat-Scratch Disease with an Extraaxial Mass

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Summary: CT and MR imaging of the brain and gallium-67 scintigraphy showed an enhancing, gallium-avid mass in the left middle cranial fossa of a 10-year-old girl. Craniotomy revealed an inflammatory mass related to the left trigeminal nerve. The lesion contained rodlike bacteria, and serologic tests were positive for cat-scratch disease. Neurologic involvement in cat-scratch disease is uncommon, and the presence of organisms in neural tissue has not been reported.

Cat-scratch disease is an infectious illness most commonly diagnosed in children. It usually presents with lymphadenopathy but may also cause cutaneous, ocular, hepatic, splenic, skeletal, and neurologic lesions (1). The illness is typically self-limiting. The causative agent of cat-scratch disease remained unidentified for many years. It now appears likely that most cases are due to a rickettsialike microorganism called *Bartonella* (formerly *Rochalimaea*) *henselae* (2–4). Neurologic involvement in cat-scratch disease is usually manifested by headache, decreased mental status, and seizures (5).

Case Report

A 10-year-old previously well girl had a 3-week history of pain in the left ear, pyrexia, and malaise. Physical examination revealed tender hepatomegaly, reduced sensation in the distribution of the maxillary division of the left trigeminal nerve, and no lymphadenopathy. Laboratory results included normal complete blood count and elevated levels of alanine aminotransferase (90 U/L; normal, less than 50) and γ-glutamyl transferase (265 U/L; normal, less than 20). Blood cultures and viral serologic tests were negative. Sonographic examination revealed diffusely abnormal hepatic echotexture and gallbladder wall thickening. A liver biopsy specimen showed fibrosis with no evidence of hepatitis or granuloma formation. Gallium scintigraphy showed increased uptake of tracer in the liver and in the left middle cranial fossa (Fig 1A). Cranial CT revealed the latter to be due to a hyperdense extraaxial lesion with homogeneous contrast enhancement. MR studies showed the mass to be isointense with cerebral cortex on T1-, proton density–, and T2-weighted images (Fig 1B and C). Enhancement with intravenous gadopentetate dimeglumine was intense and uniform, and extended to adjacent dura (dural tail sign) (Fig 1D). Imaging appearances were considered consistent with meningioma, other neoplastic lesions, such as lymphoma or granulocytic sarcoma, sarcoidosis, or infection.

At craniotomy, the lesion was found to arise from the left trigeminal nerve. A biopsy specimen showed chronic inflammatory changes in neural tissue, with infiltration of lymphocytes, plasmacytes, and histiocytes. The Warthin-Starry stain revealed rod-shaped bacteria. Serologic tests for cat-scratch disease showed titers rising to 1:1024 against *Bartonella henselae*. Antibiotic therapy was instituted with co-trimoxazole and rifampicin, and the patient's symptoms resolved over the next month. Repeat MR examination performed 2 months later showed complete resolution of the intracranial lesion.

Discussion

This case posed a diagnostic problem because of the extraaxial mass, the atypical biopsy findings, and the absence of enlarged lymph nodes. Lymphadenopathy is almost always detected in patients with cat-scratch disease (6, 7), and an extraaxial mass has not previously been reported. Although positive gallium scintigraphy has previously been described in extranodal lesions of cat-scratch disease (8, 9), and was present in this case (Fig 1), gallium uptake may also be seen in many other conditions, including other infections, tumors, and sarcoidosis.

The mass showed similar signal intensity to cerebral cortex on T1-, proton density–, and T2-weighted MR images, and there was intense homogeneous contrast enhancement with a dural tail sign. These are characteristic findings in meningioma, although similar imaging features have been reported in sarcoidosis (10, 11), granulocytic sarcoma and lymphoma (10), schwannoma (12), and sinus histiocytosis with massive lymphadenopathy (13). They have not been documented in cat-scratch disease.

The neuroimaging features of cat-scratch disease have been infrequently described. MR findings may be normal, even with severe cat-scratch disease encephalopathy (14, 15). There are single examples of abnormalities involving the deep white matter (16) and the pulvinar and occipital cortex (17). Another report (15) describes focal changes in the occipital area but gives no further details. In our patient, the cerebrum appeared normal.

It seems likely that cat-scratch disease may involve the nervous system by more than one mechanism. In most patients with neurologic involvement, encephalopathy develops, for which immunologic phenomena and bacterial toxins have been suggested as possible causes (18). There is typically an interval of a few days

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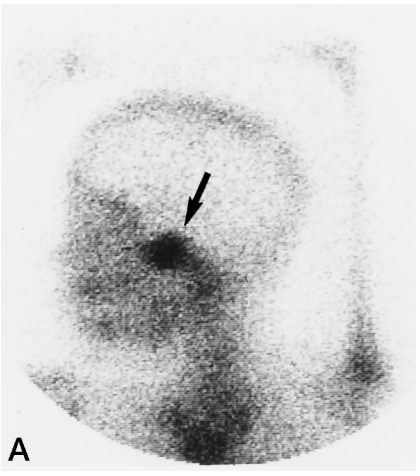

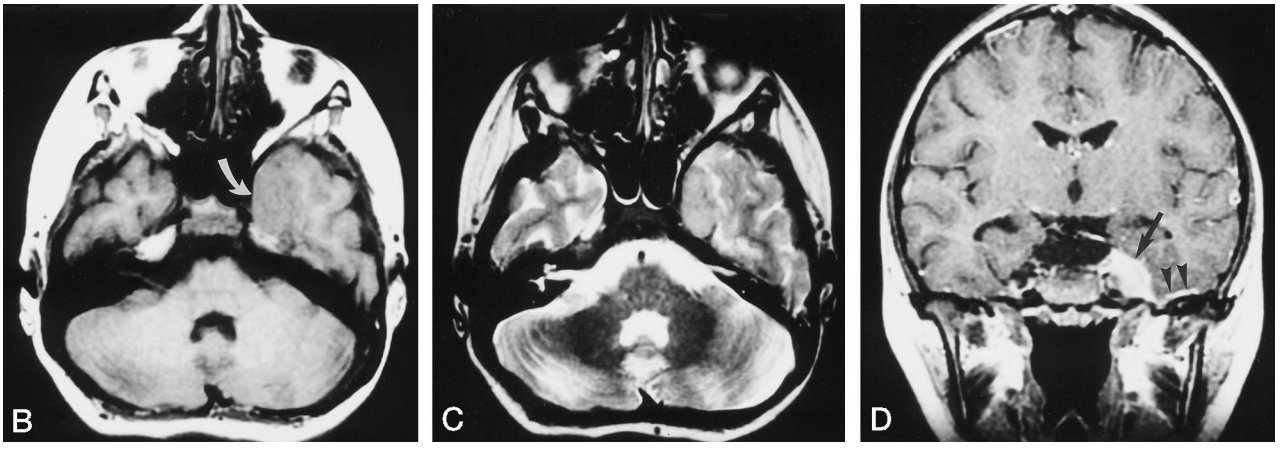

FIG 1. 10-year-old girl with 3-week history of pain in the left ear, pyrexia, and malaise.

A, Gallium-67 scan, lateral view, of the skull at 48 hours shows a gallium-avid lesion (*arrow*). The frontal image (*not shown*) confirmed that the lesion lay in the medial part of the left middle cranial fossa.

B, T1-weighted transverse MR image shows an extraaxial lesion (*arrow*), isointense with cerebral cortex.

C, The lesion is also isointense on a T2-weighted image.

D, Contrast-enhanced T1-weighted coronal image shows intense homogeneous enhancement of the lesion (*arrow*) with a dural tail sign (*arrowheads*).

to a few weeks from the onset of the illness to the development of neurologic symptoms (5, 15, 18). This seems to favor an immunologic mechanism. The demonstration of arteritis in one case (19) is consistent with this. Bacterial infection of the CNS has previously been thought unlikely to explain the encephalopathy, since CSF examination is often normal (5, 15, 18). In this case, however, organisms were demonstrated in neural tissue in a cranial nerve. Since only one bacterium, *Mycobacterium leprae* (20), is known to invade peripheral nerves directly, it seems likely that the organisms reached the trigeminal nerve by a hematogenous pathway.

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