AJNR

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AJNR Am J Neuroradiol 1996, 17 (7) 1398-1399
http://www.ajnr.org/content/17/7/1398.citation

LETTER

Choroid Plexitis Caused by Cytomegalovirus in a Patient with AIDS

Infections of the choroid plexus (choroid plexitis) are not common. The spectrum of pathogens causing choroid plexitis includes bacteria (*Nocardia asteroides*), fungi (*Cryptococcus neoformans*) and, presumably, viruses (aseptic) (1, 2, 3). We have reported two cases of choroid plexus infection by *Stomatococcus mucilaginosus* in neutropenic patients (4). Recently, we saw a case of choroid plexitis caused by cytomegalovirus in a patient with the acquired immunodeficiency syndrome (AIDS).

This 48-year-old man was admitted for evaluation of fever, vertigo, confusion, and lethargy of 2 weeks' duration. Neurologic findings included lower-extremity weakness associated with diminished patellar reflexes. Upper-extremity strength and reflexes were normal. A CD4 cell count was 42/μL. Contrast-enhanced computed tomography showed abnormally marked enhancement of a slightly enlarged right plexus (Fig 1A). Magnetic resonance imaging 2 days later confirmed the findings and the absence of enhancement of the ependyma (Fig 1B). There was cerebral atrophy but no abnormal meningeal enhancement. T2-weighted images showed no periventricular edema. Analysis of cerebrospinal fluid obtained via lumbar puncture revealed 12 white blood cells per microliter (80% neutrophils, 20% lymphocytes), total protein 1.20 g/L, and glucose 1.38 mmol/L. Pleocytosis with elevated protein and hypoglycorrhachia were highly suggestive of infection with cytomegalovirus. Cryptococcal antigen, nocardial, viral, and *S mucilaginosus* cultures of the cerebrospinal fluid were negative. A blood culture grew cytomegalovirus. Ganciclovir therapy was initiated, with an induction course of 10 mg/kg per day. Confusion and lethargy progressed despite this therapy, and the patient died on the 15th hospital day.

Central nervous system infection by cytomegalovirus in patients with AIDS is commonly described as ventriculoencephalitis. Cerebrospinal fluid often demonstrates pleocytosis with a neutrophilic predominance, elevated total protein, and occasional hypoglycorrhachia. Culture of cerebrospinal fluid for cytomegalovirus is thought to be diagnostic but is insensitive, with successful isolation of the virus reported in only 30% of cases. Magnetic resonance imaging may show progressive ventriculomegaly and increased periventricular signal intensities on T2-weighted images, sometimes associated with nonspecific periventricular enhancement after intravenous administration of contrast material (5).

We conclude that cytomegalovirus may be included in the causes of choroid plexitis.

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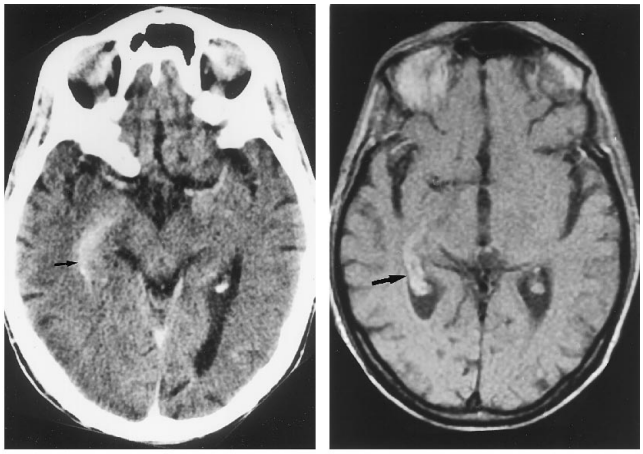
Comment

Dr Guermazi and colleagues have presented an interesting patient. The fact that choroid plexus infections are infrequently encountered, as these authors point out, is perplexing because the choroid plexus is highly vascular and serves as a portal of entry into the central nervous system for hematogenously borne pathogens (1). The uncommon observation of choroid plexitis on imaging studies of patients with infection by cytomegalovirus is particularly puzzling, because this virus has a propensity to infect endothelial cells of microvessels such as those in the choroid plexus. Pathologic studies have shown severe necrotizing ventriculitis, encephalitis, and choroid plexitis to be the major causes of neurologic morbidity in patients with AIDS and cytomegaloviral infection. Even though the choroid plexus may contain focal cytomegalic inclusions and mononuclear inflammatory infiltrates, choroid plexitis is usually much less severe than the accompanying ventriculitis and encephalitis (2), which probably accounts for the emphasis on the latter two components of cytomegaloviral infection in imaging studies.



A B

Fig 1. *A*, Postcontrast computed tomogram shows marked enhancement of a slightly enlarged right choroid plexus (*arrow*).

B, Enhanced axial spin-echo T1-weighted (500/20) magnetic resonance image (0.5 T) 2 days later. Pathologic enhancement is seen in the right choroid plexus (*arrow*) without enhancement of ependyma or periventricular edema.

In patients with AIDS, cytomegalovirus is also known to cause a rapidly ascending polyneuropathy that corresponds pathologically to severe radiculomyelitis (2). Abnormal swelling and enhancement of the cauda equina and conus medullaris have been seen on MR imaging of the lumbar spine (3, 4). The patient described by Guermazi and colleagues had weakness and abnormal reflexes of the lower extremities but normally functioning upper extremities. Although no imaging evaluation of the lumbar spine was reported in this patient, the clinical examination suggests that the patient might also have had cytomegaloviral polyradiculopathy.

Early recognition of possible involvement of the brain, spinal cord, or nerve roots by cytomegalovirus is important so that appropriate antiviral therapy can be instituted. The patient presented by Guermazi and colleagues did not receive medical attention for 2 weeks after the onset of neurologic symptoms. Subsequent antiviral therapy was unsuccessful in preventing fatal progression of the disease.

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