AJNR

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AJNR Am J Neuroradiol 1995, 16 (6) 1331-1334
http://www.ajnr.org/content/16/6/1331

Imaging of Choroid Plexus Infection by *Stomatococcus mucilaginosus* in Neutropenic Patients

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Summary: We report two neutropenic patients with *Stomatococcus mucilaginosus* meningitis and choroid plexus involvement on neuroimaging studies. CT and MR showed abnormal enlargement of the choroid plexus in one lateral ventricle and intense enhancement. In one patient there was edema in the periventricular white matter adjacent to the involved choroid plexus; in both patients there was enhancement of the ependyma.

Index terms: Meningitis; Choroid plexus, infection

Infections of the choroid plexus (choroid plexitis) are not commonly encountered. The spectrum of pathogens causing choroid plexitis includes bacteria (*Nocardia asteroides*), fungi (*Cryptococcus neoformans*) and, presumably, viruses (aseptic) (1). We report two cases of choroid plexus involvement by *Stomatococcus mucilaginosus*, an emerging pathogen in neutropenic patients (2–6).

Case Reports

Case 1

A 14-year-old boy became febrile (40°C) 15 days after a second induction chemotherapy for acute myeloid leukemia. His peripheral white blood cell count was 480/mm^3, with 10 neutrophils per cubic millimeter. Signs of sepsis purpura fulminans subsequently developed. *S mucilaginosus* was isolated from blood cultures, and antibiotic therapy was started. Twelve days later, confusion developed with nausea, vomiting, and headache. Brain computer tomography (CT) showed an enlarged right lateral ventricular choroid plexus that enhanced markedly after administration of iodinated contrast. There was adjacent white matter edema (Fig 1A and B). Enhanced T1-weighted magnetic resonance (MR) images showed the enlargement and intense enhancement of the choroid plexus and pathologic enhancement of the ependyma (Fig 1C). Proton density–weighted images showed periventricular edema (Fig 1D). There was no abnormal meningeal enhancement.

Analysis of cerebrospinal fluid (CSF) obtained via lumbar puncture showed gram-positive cocci, and *S mucilaginosus* was cultured with a susceptibility pattern identical to that of the blood isolate.

Repeat CSF and blood cultures 3 days after initiation of vancomycin therapy yielded no organisms. The patient's condition improved, his neutrophil count normalized, and he was clinically stable with normal mental status.

A CT scan performed 20 days later showed that his choroid plexus was no longer enlarged, although there was slight enhancement. The antibiotic therapy was continued for 2 months, and CT scan obtained 3 months later showed the choroid plexus to be normal in size without abnormal enhancement.

Case 2

A 46-year-old man with acute myeloid leukemia was given chemotherapy for his second relapse in 12 months. Seven days after the initiation of this course of treatment, he became febrile (40°C) and neutropenic (10/mm^3). Staphylococci were isolated from blood cultures. Simultaneously, he had severe headaches, drowsiness, and confusion initially attributed to the febrile state. Altered mental status persisted in spite of antibiotic therapy.

Postcontrast brain CT on day 22 showed marked enhancement of an enlarged left choroid plexus (Fig 2A). MR imaging performed 3 days later demonstrated that there also was pathologic enhancement of the ependyma (Fig 2B and C). There was no abnormal meningeal enhancement, and T2-weighted images showed no periventricular edema.

The clinical features, neutropenia, and imaging findings allowed us to suggest the diagnosis of *S mucilaginosus* brain infection. CSF obtained by lumbar puncture and blood cultures revealed gram-positive cocci identified as *S mucilaginosus*. The fever resolved 2 days after initiation of intravenous vancomycin. Repeat lumbar puncture 5 days later yielded normal sterile CSF. The patient's condition slowly improved, and postcontrast CT performed 2 months later was normal.

Received April 27, 1994; accepted for publication June 23.

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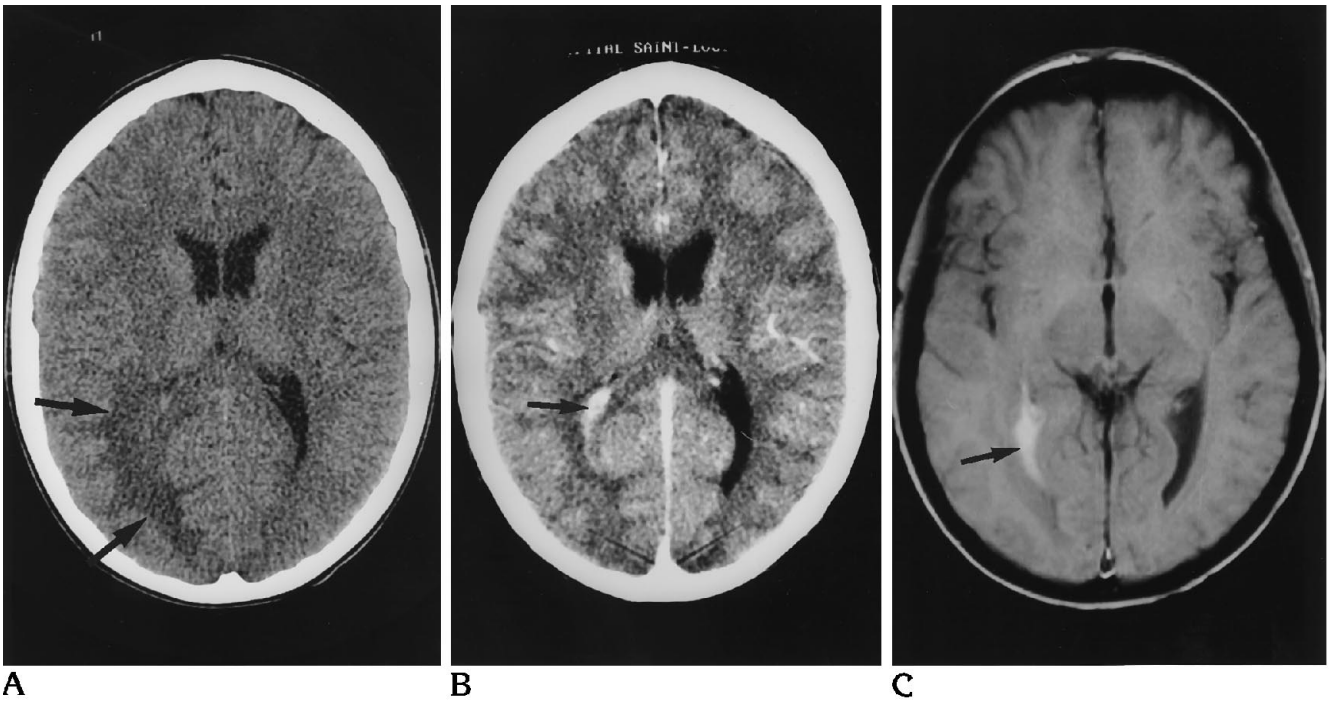
AJNR 16:1331–1334, Jun 1995 0195-6108/95/1606–1331



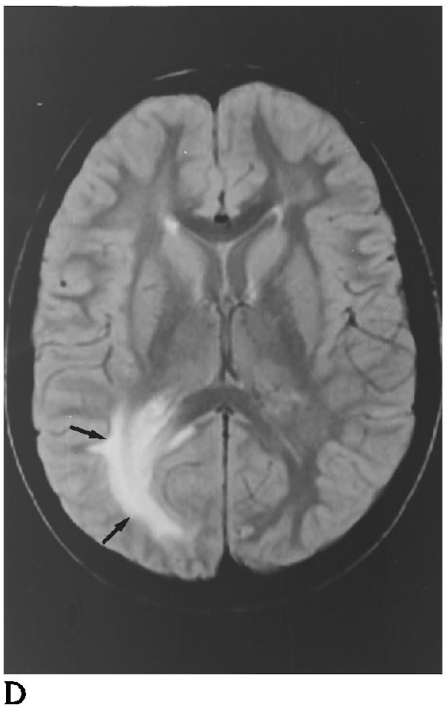


Fig 1. Fourteen-year-old boy with severe neutropenia.

A, Precontrast CT shows right periventricular edema (*arrows*) and asymmetric size of trigones of lateral ventricles attributable to mass effect of edema.

B, Postcontrast CT demonstrates marked enhancement of an enlarged right choroid plexus (*arrow*).

C, Enhanced axial spin-echo T1-weighted (500/20) MR image (0.5 T). There is marked enhancement of the enlarged choroid plexus and adjacent ependyma (*arrow*).

D, Axial proton density–weighted (1940/40) MR image. High signal changes (*arrows*) in the periventricular brain parenchyma are attributable to edema.

Discussion

The choroid plexus is a central nervous system (CNS) structure rarely affected by infection (1). Normally, it is isodense to brain on CT and isointense to brain on MR images before contrast administration, unless the plexus is calcified, in which case it will be hyperdense on CT and hypointense on MR. The choroid plexus enhances homogeneously after intravenous contrast injection on both CT and MR imaging. The lateral ventricular plexuses usually are symmetric in size and shape. When there is an abnormally enlarged choroid plexus with marked enhancement after con-

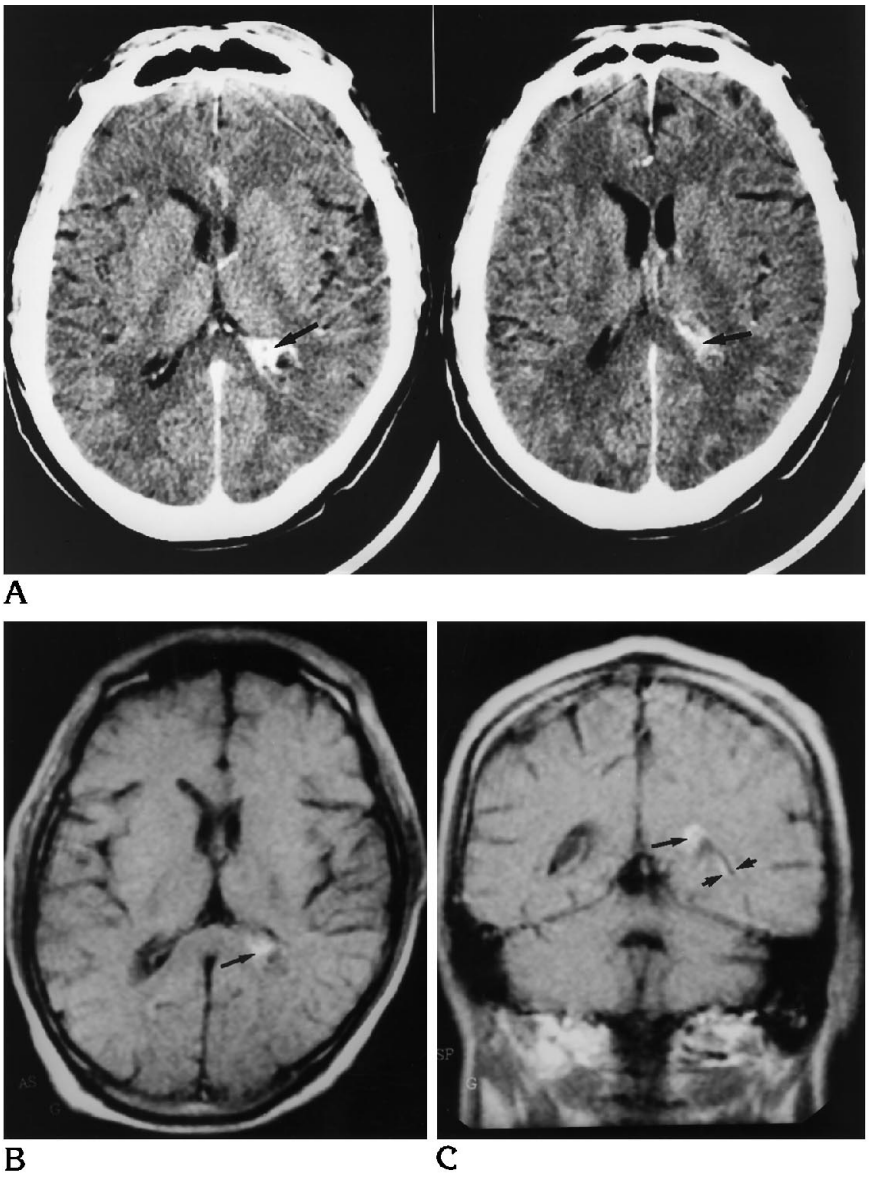


Fig 2. Forty-six-year-old man with severe neutropenia.

A, Postcontrast CT scans demonstrate marked enhancement of an enlarged left choroid plexus (*arrows*).

Enhanced axial (*B*) and coronal (*C*) spin-echo T1-weighted (500/30) MR images (0.1 T) performed 3 days later. Pathologic enhancement is seen in the left choroid plexus (*arrow*) and adjacent ependyma (*arrowheads*).

trast injection, disease should be suspected (1).

Mathews and Smith (1) presented a spectrum of pathogens causing primary choroid plexitis including bacteria (*N asteroides*), fungi (*C neoformans*), and, presumably, viruses (aseptic). Besides the pathogens, the differential diagnosis of choroid plexus lesions must include noninfectious inflammatory disorders (xanthogranulomas, neurosarcoid granulomas), congenital abnormalities such as angiomatous malformations in Sturge-Weber syndrome, and neoplasms (1).

Typically, when the choroid plexus is involved by infection, there is an associated ependymitis, encephalitis, or meningitis. Patronas and Makariou (7) suggest that the intense inflammatory reaction of the choroid plexus can spread to the ventricular wall and can produce edema in the adjacent brain parenchyma. In our two cases, there was choroid plexitis with adjacent ependymitis, and in case 1, there was associated periventricular edema.

The imaging findings of choroid plexus involvement should be interpreted in the appropriate clinical context. The determination of specific pathogens in choroid plexitis is based on CSF studies or the presence of systemic infections and positive blood cultures (1).

S mucilaginosus (formerly *Micrococcus mucilaginosus* or *Staphylococcus salivarius*) is a member of the family Micrococcaceae. This

slime-producing, catalase-variable, gram-positive coccus is a component of the normal oral flora of humans and also may colonize the respiratory tract (4). The portal of entry for the organism usually is an indwelling venous catheter or, sometimes, an oral mucositis attributable to current chemotherapy (2, 3, 6). *S mucilaginosus* is recognized as a cause of potentially fatal sepsis and meningitis in immunocompromised patients, especially those with profound neutropenia (3, 4). *S mucilaginosus* meningitis (3, 4, 6, 8–10) and subdural empyema (*S mucilaginosus* was isolated from fluid draining from a subdural hematoma) (11) have been reported. CT examinations performed in four cases of meningitis were normal in three cases (4, 6, 10) and showed only slight enlargement of the CSF spaces secondary to chemotherapy in one case (3).

Infections attributable to *S mucilaginosus* are likely to be underreported (2, 5), because *S mucilaginosus* may be mistaken for other gram-positive cocci. In our patient 2, the isolate from the blood cultures was initially identified as the staphylococcus. *S mucilaginosus* can be differentiated from catalase-positive staphylococci and micrococci, because *S mucilaginosus* characteristically fails to grow in the presence of 5% NaCl (2, 5). In our neutropenic patients with bacteremia and meningitis attributable to *S mucilaginosus*, the indwelling venous catheter was probably the portal of entry for the organism. The choroid plexus was the early site of infection in the CNS, supporting the hypothesis that in the setting of bacteremia, the choroid plexus may be the portal of entry for pathogens to the CNS (1).

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