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Choroid Plexus Infections: Neuroimaging Appearances of Four Cases

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Summary: This report presents four cases in which CNS infections caused pathologic changes in the choroid plexus that were detected by neuroimaging studies, discusses the differential diagnosis of these lesions, and describes the anatomy and physiology of the choroid plexus.

Index terms: Choroid plexus, infection; Choroid plexus, magnetic resonance; Contrast media, paramagnetic

The choroid plexus is an important central nervous system (CNS) structure that is often neglected in pathologic and radiologic studies of intracranial diseases. The choroid plexus may serve as 1) a portal of entry for pathogens into the CNS, 2) a target for various systemic disorders, 3) a reflector of various diseases that affect the brain and meninges, or 4) a site of primary processes such as tumors or cysts (1). Whereas primary neoplasms of the choroid plexus are well known entities, primary infections of the choroid plexus (choroid plexitis) are not commonly encountered.

Case Reports

Case 1

A 19-year-old white man presented with a 1-day history of nausea, vomiting, headache, and photophobia. Cerebrospinal fluid (CSF) studies on the day of admission suggested a nonbacterial meningitis (white blood cell count = 58/ mm³ with 90% lymphocytes, protein = 76 mg/dL, glucose = 52 mg/dL). Bacterial cultures were negative. His headache resolved over 3 to 4 days, but he experienced prolonged nausea and vomiting requiring intravenous hydration. CSF examination 6 days after admission showed a further elevation in leukocytosis (1840/mm³ with 70% lymphocytes, 29% monocytes) and protein (418 mg/dL) and a further depression of glucose (30 mg/dL). India ink and gram stains, cryptococcal antigen, bacterial cultures, and fungal cultures were all negative. No viral cultures were obtained. Magnetic resonance (MR) imaging performed 9 days after admission showed symmetrically enlarged lateral ventricular choroid plexuses that enhanced markedly after Gd-DTPA administration (Fig. 1A). There were no other abnormalities, specifically none involving the meninges or ependyma. In view of the failure of CSF laboratory studies to identify a specific cause for the patient’s CNS inflammatory process, and in view of imaging studies that showed diffuse abnormality of the choroid plexus but no other structures, the patient was considered to have aseptic choroid plexitis. He gradually improved with supportive therapy over several weeks and resumed previous employment. MR obtained 10 months after his illness showed that his choroid plexuses were no longer enlarged (Fig. 1B) supporting the previous diagnosis of an inflammatory process affecting primarily the choroid plexus.

Case 2

A 33-year-old woman with a 3-year history of idiopathic pulmonary fibrosis treated with corticosteroids had been hospitalized for several days with a pulmonary infection diagnosed bronchoscopically as due to both Nocardia asteroides and Aspergillus fumigatis when she developed confusion that progressed to stupor and then coma. Brain computed tomography (CT) at that time showed hydrocephalus and an enlarged, radiodense, left lateral ventricular choroid plexus that enhanced markedly after administration of iodinated contrast (Figs. 2A and 2B). Based on the CT findings and the presence of a septic state, a hemorrhagic choroid plexitis was suspected. A ventricular drain was placed. Repeat CT 3 days later demonstrated extension of the infection to the other choroid plexus and the ependyma (Fig. 2C). Cultures from CT guided aspiration biopsy of the left temporal horn choroid plexus grew Nocardia asteroides. The patient continued to deteriorate despite antibiotic therapy and died 4 days later.
Fig. 1. Nineteen-year-old man with aseptic choroid plexitis.
A, Coronal SE 800/25 MR after Gd-DTPA injection (0.1 mmol/kg intravenously) shows abnormally enlarged and markedly enhancing choroid plexuses in the bodies and temporal horns of the lateral ventricles.
B, Coronal SE 800/25 MR after Gd-DTPA injection (0.1 mmol intravenously) performed 10 months later shows that the choroid plexuses are normal.

Fig. 2. Thirty-three-year-old woman with Nocardia choroid plexitis and ventriculitis.
A, Precontrast CT shows an enlarged dense left lateral ventricular choroid plexus.
B, Postcontrast CT demonstrates marked enhancement of the large left lateral ventricular choroid plexus.
C, Postcontrast CT 3 days later shows new enlargement of the right lateral ventricular choroid plexus and ependymal enhancement.

Case 3
A 48-year-old man had a history of cryptococcal meningitis diagnosed and treated at another institution. Six months following treatment with amphotericin B, he complained of persistent visual disturbance and progressive headache. CSF studies were consistent with continued cryptococcosis (cryptococcal antigen > 1:256, leukocytes = 7/mm³, protein = 50 mg/dL, glucose = 71 mg/dL). MR at that time showed a left lateral ventricular choroid plexus mass with adjacent white matter edema (Fig. 3). CT performed on the same day demonstrated bilaterally prominent calcified lateral ventricular choroid plexuses. The left choroid plexus was slightly larger and was associated with adjacent lucent parenchyma. The MR and CT findings suggested the presence of a cryptococcoma of the choroid plexus with inflammation and edema of the adjacent brain. The patient was retreated with intravenous amphotericin B and oral 5-flucytosine. Four months later both his neurologic examination and CSF studies were normal. Enlargement of the left choroid plexus and surrounding parenchymal lucency resolved on CT after several months, further supporting the diagnosis of an inflammatory process involving both the choroid plexus and adjacent ependymal and subependymal tissue.

Case 4
A 2-year-old boy with an esophageal stricture due to lye ingestion underwent multiple dilatation procedures. He de-
thought to start in the choroid plexus before (2). Some bacterial infections, such as meningo-
coccal and tuberculous meningitis, are commonly
plexus may be the predominant site of abnor-
mally if imaging studies are performed early in
the course of these diseases. However, choroid
plexitis as an isolated focus of infection is rare
(3).

The cases presented here illustrate a spectrum
of pathogens causing primary choroid plexitis
including bacterial (Nocardia asteroides), fungal
(Cryptococcus neoformans), and presumed viral
(aseptic) etiologies. After pulmonary involvement
the CNS is the next most frequent site of system-
ically disseminated Nocardia asteroides. Nocardia
brain abscesses are well known complications in
organ transplant patients (4). Our patient was not
a transplant patient but was immunocompro-
mised by long-term steroid therapy for pulmo-
mary fibrosis. The choroid plexus was the earliest
site of abnormality in this patient’s CNS, sup-
porting the hypothesis that the organism spread
hematogenously from the lungs to the choroid
plexus. Our case of choroid plexus cryptococco-
coma (case 3) is unusual because this fungus
usually is disseminated throughout the meninges
or perivascular spaces and causes a chronic men-
ingitis or cystic lesions within dilated perivascular
spaces or brain parenchyma (5). However, there
have been previous reports of cryptococcosis
presenting as unilateral or bilateral choroid plexus
masses (2). Case 1 is unusual because it demon-
strates that a primary inflammatory process of
the choroid plexus can produce symptoms and
laboratory results that mimic aseptic meningitis.
Enhanced MR may allow the determination of
the frequency of choroid plexus inflammation in
patients suspected to have meningitis.

In addition to the bacterial and fungal organ-
isms reported here, the differential diagnosis of
choroid plexus lesions includes a variety of other
purulent and granulomatous infections as well as
parasitic infestations described by Netsky and
Shuangshoti (2). Noninfectious inflammatory dis-
orders that may also result in choroid plexus
abnormalities include xanthogranulomas of the
choroid plexus (6, 7), neurosarcoid granulomas
(8), and rheumatoid nodules (9).

Neoplasms within the lateral ventricles are an-
other important diagnostic consideration when
one evaluates a choroid plexus lesion. Patient age
and location of the mass within the lateral ventri-
cle are useful factors in differentiating tumors
such as choroid plexus papillomas, ependymo-
mas, subependymomas, subependymal giant cell
astrocytomas, astrocytomas, glioblastomas, lym-
phoma, metastases, primitive neuroectodermal
tumors, teratomas, and oligodendrogliomas (10).
Clinical history and CSF studies are helpful in distinguishing tumors from infections in many cases.

Congenital abnormalities such as angiomatous malformations in Sturge-Weber syndrome (11) and in Klippel-Trenaunay-Weber syndrome (12) and megachoroid plexuses in meningomyelocele patients (13) must also be considered in the differential diagnosis of choroid plexus lesions.

Some systemic toxic-metabolic disorders may also result in choroid plexus abnormalities that potentially could be seen with MR or CT. Experimentally, cyclophosphamide, various tertiary amines, and heavy metals such as mercuric chloride can produce severe, often hemorrhagic, choroid plexitis (1). Disseminated intravascular coagulopathy (DIC) has been reported to result in choroid plexus hemorrhage (14). Either DIC or these toxins could result in a CT appearance similar to the case of Nocardia choroid plexitis described here.

Since the imaging findings in CNS infections are often not specific, these studies should be interpreted in the appropriate clinical context. The determination of specific pathogens in CNS infections is often based on CSF studies or the presence of systemic infection. Patients are treated empirically for the suspected pathogen or treated with broad spectrum antibiotics. Consequently, biopsy or surgical intervention is not performed unless absolutely necessary. This limits reports such as ours since only one of four cases was biopsy proven.
The normal lateral ventricular choroid plexus extends from the choroidal fissure of the medial temporal lobe posteriorly through the temporal horn to the atrium of the lateral ventricle and then forward through the body of the ventricle to the foramen of Monro where it joins the contralateral plexus. Prior to contrast administration, the choroid plexus is isodense to brain on CT and isointense to brain on MR images unless the plexus is calcified, in which case it will be hypodense on CT and hypointense on MR. The choroid plexus enhances homogeneously after intravenous contrast injection on both CT and MR. The function of the choroid plexus capillary epithelium and choroidal epithelium is not, however, identical to that of the blood-brain barrier. Tight junctions at this level prevent passive exchange of proteins and other solutes (17). This explains the diffusion of other solutes (17). This explains the diffusion of substances from blood into CSF and brain. This difference in function has led to the use of the term "blood-CSF barrier" to refer to the choroidal epithelium (18).

The differential permeability of the choroid plexus capillary epithelium and choroidal epithelium accounts for the roles of the choroid plexus as an entry site into the CNS for various pathogens and as a primary target for toxins and systemic disorders. Since the choroid plexus develops as an invagination of neuroepithelium, it has the unique property of being continuous with the meninges yet located within brain substance. Given this, it is not surprising that the choroid plexus can also reflect changes within both the meninges and the brain parenchyma (1). Even though the choroid plexus is susceptible to the effects of pathogens, the frequency of choroid plexus lesions that can be identified on imaging studies of the CNS is surprisingly low. With increased awareness of choroid plexus infections and wider use of Gd-DTPA-enhanced MR in patients with CNS infections, perhaps more of these lesions will be reported.

References

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