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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 fumigatus* 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.

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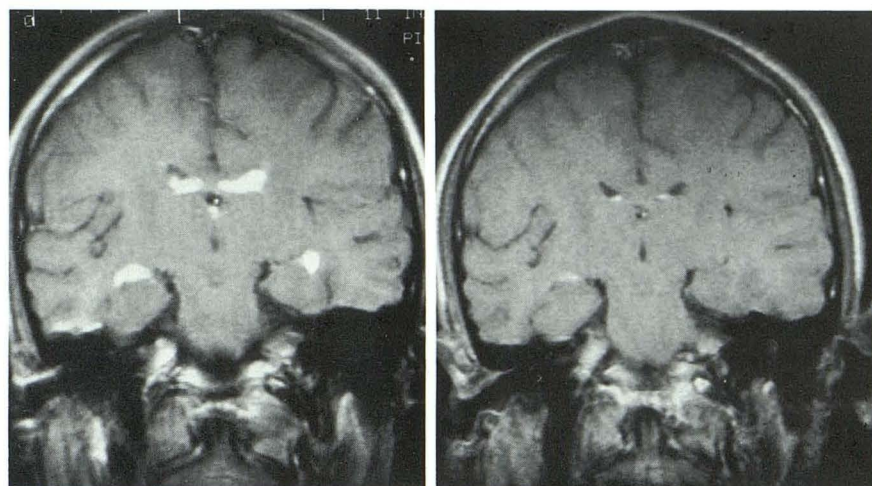


Fig. 1. Nineteen-year-old man with aseptic choroid plexitis.

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.

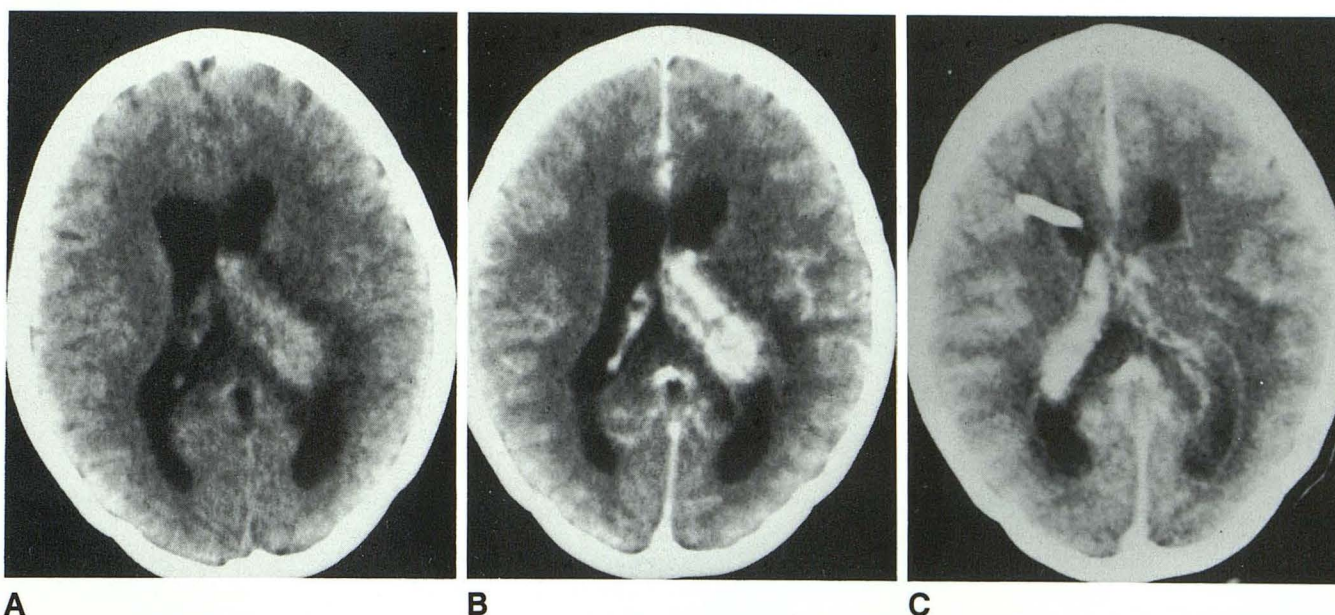


Fig. 2. Thirty-three-year-old woman with *Nocardia* choroid plexitis and ventriculitis.

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-

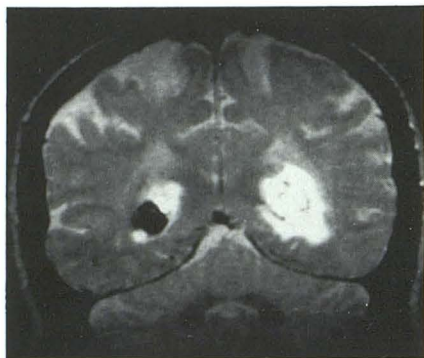


Fig. 3. Forty-eight-year-old man with choroid plexus cryptococcoma. Coronal SE 2000/80/1 (TR/TE/excitations) MR shows edema surrounding the left choroid plexus that is slightly larger and hyperintense relative to the contralateral plexus. Decreased signal of the right plexus is due to calcification that was seen bilaterally on CT. Inflammation and edema in the left choroid plexus presumably accounts for the hyperintensity seen on MR.

veloped fever that was attributed to a cutaneous infection at his gastric stoma and was treated with broad spectrum antibiotics. His fever persisted and a search for another source of infection was begun. Brain CT showed a ring enhancing lesion just above the left lateral ventricle. The choroid plexus on this side was slightly larger than the contralateral choroid plexus. MR performed 1 day later demonstrated that the ring enhancing lesion abutted the ependyma and that the ipsilateral choroid plexus was larger and enhanced more prominently than the plexus on the right (Fig. 4). The diagnosis of brain abscess was suggested by the imaging studies. CSF and blood cultures were negative but had not been obtained prior to the initiation of antibiotic therapy. After 28 days of antibiotic therapy the patient was clinically normal, and his CT scan was normal. Even though no specific pathogen was identified in this patient, the most likely explanation for his clinical course and imaging findings is a brain abscess secondary to bacteremia after an esophageal dilatation procedure.

Discussion

Typically when the choroid plexus is affected by infections, there is an associated encephalitis, meningitis, or ependymitis that is more severe than the choroid plexitis. Case 4, for example, demonstrates the coexistence of choroid plexitis with an adjacent brain abscess. In this case, the choroid plexus swelling and hyperemia may have been a reaction to the nearby abscess although, in the setting of septicemia, the choroid plexus may be the site of entry of bacteria into the CNS (2). Some bacterial infections, such as meningococcal and tuberculous meningitis, are commonly thought to start in the choroid plexus before becoming diffuse (2). Therefore, the choroid plexus may be the predominant site of abnor-

mality 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 systemically disseminated *Nocardia asteroides*. *Nocardia* brain abscesses are well known complications in organ transplant patients (4). Our patient was not a transplant patient but was immunocompromised by long-term steroid therapy for pulmonary fibrosis. The choroid plexus was the earliest site of abnormality in this patient's CNS, supporting the hypothesis that the organism spread hematogenously from the lungs to the choroid plexus. Our case of choroid plexus cryptococcoma (case 3) is unusual because this fungus usually is disseminated throughout the meninges or perivascular spaces and causes a chronic meningitis 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 demonstrates 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 organisms 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 disorders 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 another important diagnostic consideration when one evaluates a choroid plexus lesion. Patient age and location of the mass within the lateral ventricle are useful factors in differentiating tumors such as choroid plexus papillomas, ependymomas, subependymomas, subependymal giant cell astrocytomas, astrocytomas, glioblastomas, lymphoma, metastases, primitive neuroectodermal tumors, teratomas, and oligodendrogliomas (10).

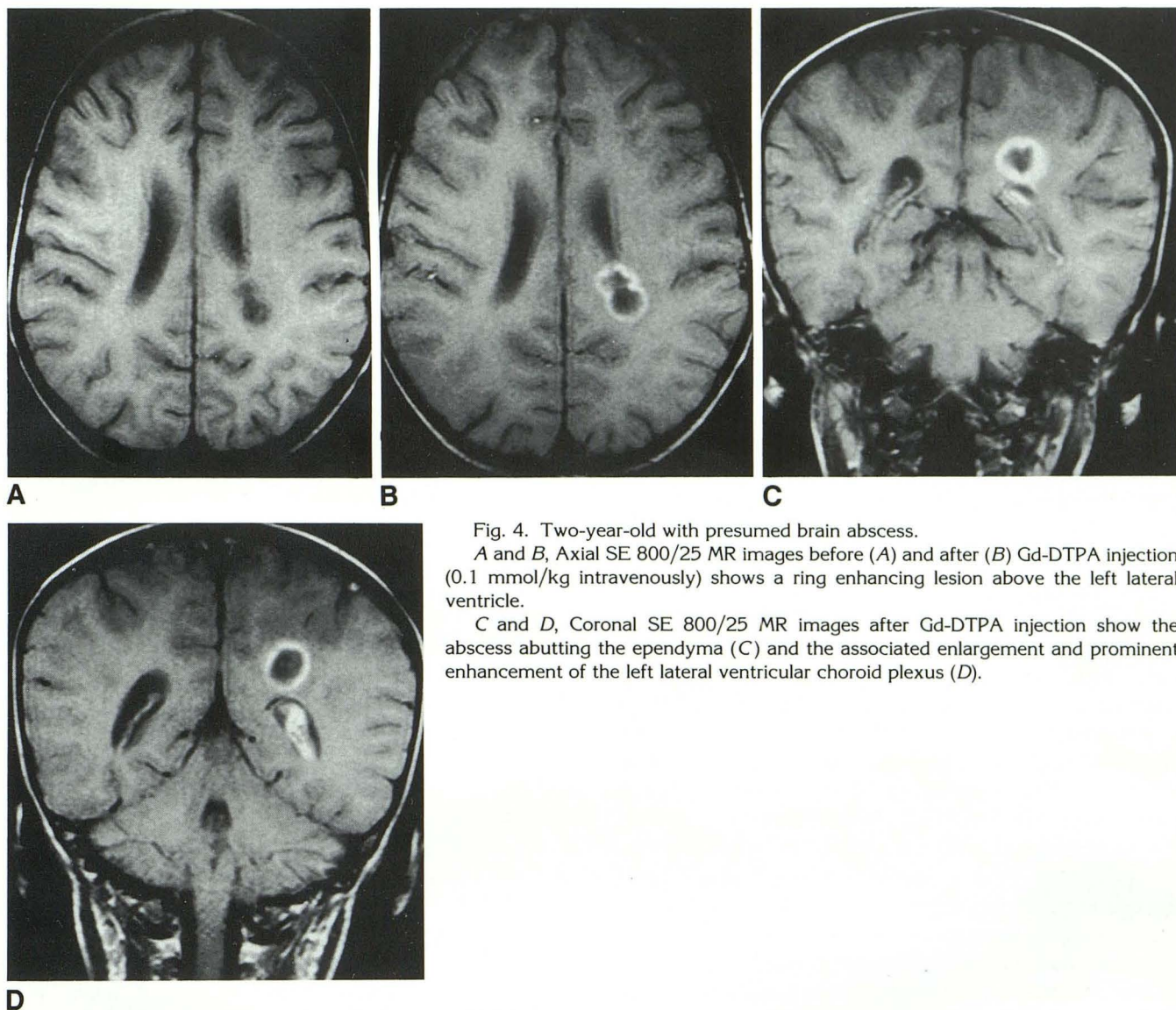


Fig. 4. Two-year-old with presumed brain abscess.

A and B, Axial SE 800/25 MR images before (A) and after (B) Gd-DTPA injection (0.1 mmol/kg intravenously) shows a ring enhancing lesion above the left lateral ventricle.

C and D, Coronal SE 800/25 MR images after Gd-DTPA injection show the abscess abutting the ependyma (C) and the associated enlargement and prominent enhancement of the left lateral ventricular choroid plexus (D).

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 meningocele 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 hyperdense on CT and hypointense on MR. The choroid plexus enhances homogeneously after intravenous contrast injection on both CT and MR (15, 16). Although the amount of choroid plexus present is variable, the lateral ventricular plexuses are usually symmetric in size and shape. When there is asymmetry of the plexuses as in cases 2, 3, and 4, pathology should be suspected. When symmetric lateral ventricular choroid plexuses fill the ventricles as in case 1, pathologic enlargement should be suspected in the appropriate clinical circumstances. However, verification of the diagnosis of choroid plexitis with symmetrically enlarged or slightly asymmetric plexuses may only be possible with follow-up imaging.

Understanding images of the choroid plexus requires understanding the physiology of this structure. The capillaries of the choroid plexus have fenestrated epithelium that does not restrict the exchange of solutes, unlike the capillary endothelium in most areas of the brain where tight junctions prevent unrestricted solute exchange, thus forming the blood-brain barrier. The barrier between blood and CSF in the choroid plexus lies at the choroidal epithelium. Tight junctions at this level prevent passive exchange of proteins and other solutes (17). This explains the diffusion of contrast agents into the extracellular space of the choroid plexus, resulting in choroidal enhancement on both CT and MR. The function of the choroidal epithelium is not, however, identical to that of the blood-brain barrier as evidenced by the different rates of entry 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 de-

velops 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

1. Levine S. Choroid plexus: target for systemic disease and pathway to the brain. *Lab Invest* 1987;56:231-233
2. Netsky MG, Shuangshoti S. *The choroid plexus in health and disease*. Charlottesville, VA: University of Virginia Press, 1975:249-264
3. Enzmann DR. *Imaging of infections and inflammations of the central nervous system: computed tomography, ultrasound, and nuclear magnetic resonance*. New York: Raven Press, 1984:176-177
4. Hall WA, Martinez AJ, Dummer JS, et al. Central nervous system infections in heart and heart-lung transplant recipients. *Arch Neurol* 1989;46:173-177
5. Wehn SM, Heinz ER, Burger PC, Boyko OB. Dilated Virchow-Robin spaces in cryptococcal meningitis associated with AIDS: CT and MR findings. *J Comput Assist Tomogr* 1989;13:756-762
6. Pear BL. Xanthogranuloma of the choroid plexus. *AJR* 1984;143:401-402
7. Hinshaw DB, Fahmy JL, Peckham N, et al. The bright choroid plexus on MR: CT and pathologic correlation. *AJNR* 1988;483-486
8. Strefling AM, Summerville JW, Urich H. Involvement of the choroid plexuses in neurosarcoidosis. *Acta Neuropathol (Berl)* 1987;74:402-404
9. Kim RC, Collins GH, Parisi JE. Rheumatoid nodule formation in the choroid plexus. *Arch Pathol Lab Med* 1982;106:83-84
10. Jelinek J, Smirniotopoulos JG, Parisi JE, Kanzer M. Lateral ventricular neoplasms of the brain: differential diagnosis based on clinical, CT, and MR findings. *AJNR* 1990;11:567-574
11. Stimac GK, Soloman MA, Newton TH. CT and MR of angiomatous malformations of the choroid plexus in patients with Sturge-Weber disease. *AJNR* 1986;7:623-627
12. Williams DW, Elster AD. Cranial CT and MRI in the Klippel-Trenaunay-Weber syndrome. *AJNR* 1992;13:000-000
13. Netanyahu I, Grant EG. Prominent choroid plexus in meningomyelocele: sonographic findings. *AJNR* 1986;7:317-321
14. Deshpande V, Levine S. Choroid plexus thrombosis and hemorrhage caused by disseminated intravascular coagulation in Down's syndrome. *Hum Pathol* 1984;15:195-197
15. Naidich TP, Pudlowski RM, Leeds NE, Naidich JB, Chisolm AL, Rifkin MD. The normal contrast-enhanced computed axial tomogram of the brain. *J Comput Assist Tomogr* 1977;1:16-29
16. Kilgore DP, Breger RK, Daniels DL, Pojunas KW, Williams AL, Haughton VM. Cranial tissues: normal appearance after intravenous injection of Gd-DTPA. *Radiology* 1986;160:757-761
17. Rapoport SI. *Blood-brain barrier in physiology and medicine*. New York: Raven Press, 1976:43-86
18. Sage MR. Blood-brain barrier: phenomenon of increasing importance to the imaging clinician. *AJNR* 1982;3:127-138