**CLINICAL HISTORY**

61-year-old male presenting with memory problems.

**FINDINGS**

Figure 21-1. Top row: Axial FLAIR MRI. There is mild bilateral temporoparietal volume loss. Second, third, and fourth rows: Axial and surface projection maps F-18 FDG PET. There is bilateral posterior temporal and parietal hypometabolism (right greater than left). Posterior temporal and parietal hypometabolism with preservation of precentral gyrus metabolism is a characteristic feature of Alzheimer disease (AD).

**DIFFERENTIAL DIAGNOSIS**

Vascular dementia, frontotemporal dementia, Lewy body dementia, corticobasal degeneration, normal pressure hydrocephalus.

**DIAGNOSIS**

AD.

**DISCUSSION**

AD is a progressive neurodegenerative disorder that manifests with gradual deterioration in cognition, behavior, and motor function. It is the most common dementia in the elderly followed by vascular and frontotemporal dementias. The definitive diagnosis is obtained through brain biopsy showing accumulation of extracellular senile β-amyloid plaques and intracellular neurofibrillary tangles formed by tau proteins.

The conventional CT and MRI are a part of the dementia workup primarily to rule out vascular dementia, normal pressure hydrocephalus, or secondary causes such as intracranial mass. Another common use of imaging is following the degree of brain atrophy. There are no early CT or MRI findings of AD. Functional MRI shows diminished intensity and/or delayed activation in the prefrontal cortex and medial temporal lobe, which are primary circuits of learning and memory. The magnitude of FDG temporoparietal metabolic deficits on FDG-PET correlate well with cognitive impairment. Basal ganglia, thalamus, cerebellum, and primary sensorimotor cortex are usually spared. A normal β-amyloid PET imaging with preserved gray–white differentiation excludes the possibility of dementia due to AD. Patients with mild cognitive impairment and a positive β-amyloid PET imaging have greater chance of progressing to AD (50% to 60%) than those with a negative amyloid PET study (less than 4% to 7%). However, amyloid PET study can be positive even in elderly patients without cognitive difficulties.

AD insidiously starts at the temporal lobe entorhinal cortex and gradually progresses to hippocampus and neocortex followed by association areas. Therefore, the earliest affected abilities are learning and short-term memory. Then cognitive loss is enhanced with loss of orientation, long-term memory, and personality. The inevitable outcome is behavioral changes (e.g., hallucinations), loss of language, visuospatial skills, and eventually motor function. There is no cure available, but slowing the disease progression or prevention in case of early diagnosis is extensively investigated. Thus, early diagnosis is crucial as atrophy translates into irreversable neuronal death. The functional imaging techniques
β-amyloid PET imaging sought to offer early recognition of the disease, potentially to slow devastating outcomes with antiamyloid therapies.

**Question for Further Thought**
1. What is the best follow-up imaging study for evaluating AD progression?

**Reporting Responsibilities**
Routine reporting is sufficient unless there are acute findings. The diagnosis of AD is clinical. Primary benefit of structural imaging (CT and MRI) is to rule out intracranial mass or acute infarct in an elderly patient. Secondary benefits are adjunctive providing areas of focal cerebral atrophy to support the diagnosis.

**What the Treating Physician Needs to Know**
- A negative β-amyloid PET imaging study is valuable to exclude AD
- A positive study in patients with mild cognitive impairment has prognostic implications
- The FDG PET is valuable in disease monitoring of AD and in the diagnosis of frontotemporal dementia and Lewy body dementia as the spatial pattern of FDG hypometabolism is characteristic

**Answer**
1. β-amyloid PET imaging is valuable in the early diagnosis or exclusion of AD. FDG PET/CT and MRI changes correlate best with disease progression and can be used for evaluating patients with AD in follow-up.
CLINICAL HISTORY  Motor vehicle accident with anterior and central skull base fractures 1 year ago. Patient is presenting with persistent headaches now.

FINDINGS  Figure 26-1. Axial CTA image, bone window through the ethmoid. There is comminuted ethmoidal fracture. Additionally, there is a fracture through the sphenoid roof at the skull base with pneumocephalus in the region of the suprasellar cistern (arrows). Figure 26-2. Coronal T2WI through the planum sphenoidale 1 year later. There is encephalomalacia and gliosis of the bilateral subfrontal lobes (vertical arrows). Additionally, there is hyperintense opacification in the region of the posterior ethmoid and sphenoid sinuses (star). There appears to be a tenuous tract through the planum on the left (transverse arrow). Figures 26-3 and 26-4. Axial heavily T2WI fast imaging employing steady state acquisition (FIESTA) and FLAIR, respectively, in a companion case. There is hyperintense collection anterior to the left middle cranial fossa (star) through a defect in the dura and the left greater wing of sphenoid. Fluid collection abuts and compresses the left lateral rectus (arrows). The fluid suppresses on FLAIR in Figure 26-4.
DIFFERENTIAL DIAGNOSIS Growing skull fractures, pseudomeningocele, leptomeningeal cyst, sinus retention cyst and infection.

DIAGNOSIS Posttraumatic cerebrospinal fluid (CSF) collection (pseudomeningocele).

DISCUSSION High-resolution CT at the level of skull base is highly sensitive for demonstrating skull base fractures acutely, and the lack of healing, bony resorption or “growing fractures” may be identified on follow-up imaging.

Advanced MR techniques such as T2 FIESTA, FLAIR, T1, and T2 sequences can be extremely helpful in determining the extent of posttraumatic fluid collections and oftentimes permit identification of the direct communication with the subarachnoid space (as shown in our companion case). The fluid collection in pseudomeningoceles should be the same as the CSF within the ventricles and the subarachnoid space on all MRI sequences.

For definitive documentation of communication of the fluid collection with the subarachnoid space and identification of the exact point of communication, radionuclide cisternography or CT cisternography is required. For radionuclide cisternography, indium is instilled into the thecal sac via lumbar puncture and the patient tipped head down on the fluoroscopy table in order to advance the radionuclide into the intracranial subarachnoid space. Imaging is obtained immediately and delayed in order to define the location of the communication. Nasal and external auditory canal pledgets may be employed and counted for radioactivity when active CSF leak is suspected. For CT cisternography, myelographic nonionic iodinated contrast is instilled into the thecal sac via lumbar puncture and the patient tipped head down on the fluoroscopy table in order to advance the contrast into the intracranial subarachnoid space. Direct coronal CT imaging is extremely useful in the depiction of contrast leaking into the dependent fluid collection in the setting of a skull base fracture.

Asymptomatic or minimally symptomatic cases of post-traumatic fluid collection without active CSF leak may be identified and subacutely treated surgically. When active leak is present, the increased risk of meningitis, empyema, or infectious complications warrants emergent surgical treatment.

A growing skull fracture (leptomeningeal cyst) is a rare but well-documented complication of craniofacial trauma less commonly seen following fractures of the skull base. A well-defined fracture of the skull base in association with traumatic dural injury results in exposure of the fracture to the pulsations of the cerebrospinal fluid within the subarachnoid space. Pulsatile pressure begins to gradually widen the fracture line permitting the dura/meninges to prolapse through the defect. The content may be slightly hyperintense on T1WI reflecting slightly more proteinaceous contents within the leptomeningeal cyst as compared with a pseudomeningocele.

With infection, abscess will present with single or multiple fluid pockets with poorly defined margins associated with overlying soft tissue swelling and edema. The exact constellation of findings is related to the path of spread of the infection. The fluid collections associated with infection do not follow the imaging characteristics of CSF.

Questions for Further Thought
1. What is the natural history of this disease?
2. What treatment options are there?

Reporting Responsibilities
Direct reporting is essential in view of the possible complications. Location and density or signal intensity of the fluid collections and their proximity to the location of the skull base fracture and the presence of complications such as pneumocephalus, empyema, or other infection must be urgently reported.

What the Treating Physician Needs to Know
• Location, size, and relationship to adjacent structures
• Presence/absence of complications as assessed by imaging
• Usefulness of cisternography for identification of the tract

Answers
1. Increasing fracture diastasis over time may lead to prolapse of meninges and meningocele formation. Progressive neurologic deficits may develop from low CSF pressure states and from infection in cases of active CSF leak.
2. All of these complications of skull base fractures are treated surgically with the addition of antibiotic therapy in those patients with active CSF leak and/or meningitis.
CLINICAL HISTORY

62-year-old female for stroke evaluation.

FIGURE 30-1

FIGURE 30-2

FIGURE 30-3

FIGURE 30-4
**FINDINGS** Figure 30-1 Axial post-contrast CT. There is a right frontal extraaxial cerebrospinal fluid (CSF) density collection with compression of the right frontal lobe (vertical arrow). There is mild thinning and remodeling of the overlying right frontal bone (transverse arrow). Figures 30-2 to 30-4. Axial DWI, FLAIR, and T2WI, respectively, through the lesion. The collection follows CSF intensity on all sequences. There is compression of the frontal lobe (vertical arrows). Thinning of the right frontal bone is again demonstrated (transverse arrows). Figures 30-5 and 30-6. Axial NCCT and axial T2W MRI, respectively, through the right middle cranial fossa in a 3-year-old boy. These demonstrate an arachnoid cyst (AC) in a more typical location in the right middle cranial fossa. There is smooth compression of the frontal and temporal lobes (arrows) with overlying right temporal bone remodeling and thinning seen on the CT. In these two cases like in all cases, the mass is extraaxial to cortical gray matter (GM).

**DIFFERENTIAL DIAGNOSIS** AC, epidermoid cyst, neurocysticercosis, porencephalic cyst (PC), pilocytic astrocytoma, hemangioblastoma.

**DIAGNOSIS** Arachnoid cyst (AC).

**DISCUSSION** Both CT and MRI are capable of demonstrating the changes of AC, but MRI is the preferred method of choice. AC is an extra axial CSF containing mass located in the subarachnoid space compressing the underlying brain with remodeling and thinning of the overlying calvarium. It is homogeneously CSF hypodense on CT and follows CSF intensity on all MRI sequences. There is usually no vascular structure within it, and the wall can rarely be identified being composed of a very thin arachnoid membrane. The immediate surrounding brain is usually cortical GM which could be slightly thickened and flattened due to mass effect. PC on the other hand is surrounded by gliotic tissue since it is a result of parenchymal destruction. PC also communicates freely with the CSF space. The majority of ACs are noncommunicating, while a small percentage may communicate via a ball valve mechanism which could be demonstrated by CT cisternogram or phase contrast cine MR. Epidermoid tumor follows CSF intensity on all spin echo sequences but restricts diffusion which distinguishes it from AC. However, some ACs may contain proteinaceous fluid or become hemorrhagic making differentiation difficult. Hemorrhage into ACs could be spontaneous, traumatic, or due to a ruptured aneurysm. Racemose neurocysticercosis is usually multiloculated with septations. Pilocytic astrocytoma and hemangioblastoma are parenchymal lesions with solid mostly contrast-enhancing components.

AC accounts for less than 1% of all intracranial masses and is widely accepted as developmental anomalies in which splitting or duplication of the primitive arachnoid membrane allows subarachnoid fluid collection. It is a common benign lesion occurring in the CNS both within the intracranial compartment (most common) and within the spinal canal. Common intracranial locations include the middle cranial fossa/sylvian fissure (50% to 60%), suprasellar (10%), quadrigeminal plate cistern (10%), cerebellopontine angle (5% to 10%), supracerebellar cistern (<5%), and cisterna magna (<5%).

Most cases begin during infancy; however, onset may be delayed until adolescence. Although the vast majority is sporadic, they are seen with increased frequency in mucopolysaccharidoses. The majority of ACs is small and asymptomatic. Headache and seizures are the most com-
mon symptoms. When symptoms occur, they are usually the result of gradual enlargement resulting in mass effect. This results in either direct neurologic dysfunction, or distortion of normal CSF pathways resulting in obstructive hydrocephalus. Surgical treatment when necessary includes surgical excision, surgical fenestration, or cyst shunting into the CSF space or peritoneum.

Questions for Further Thought
1. Does AC cause seizure?
2. Is there any difference between communicating and noncommunicating AC?

Reporting Responsibilities
This is a benign usually incidental finding and requires routine reporting. It is important to report the size, presence of bone remodeling, and associated hydrocephalus if any.

What the Treating Physician Needs to Know
- Size and location
- Presence of adjacent compressed or dysplastic tissue
- Communicating or noncommunicating in symptomatic lesions at CT cisternogram or phase contrast cine MR

Answers
1. No. Most ACs are incidental, asymptomatic, and do not cause seizures. However, since ACs are more common in the middle cranial fossa than elsewhere, there is the suggestion that underlying temporal lobe compression, dysplasia, or hypogenesis could lead to seizures.
2. It has been suggested that symptomatic ACs are usually the growing or enlarging ACs and that these symptomatic ACs tend to communicate with the arachnoid space via a ball valve mechanism that allows CSF to enter but not exit. CT cisternography and/or phase contrast cine MR have been used to demonstrate presence of such communication. There is a consensus that symptomatic cysts causing seizures, hydrocephalus, focal neurologic deficits, or raised intracranial pressure may benefit from surgical management and demonstration of communication between the ACs and the subarachnoid space is important in the preoperative evaluation.