Symptomatic intracranial pneumatocele from mastoid sinus of spontaneous origin

Case report

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A case of a large intracranial pneumatocele arising from mastoid air cells is reported. The patient became symptomatic after multiple coughing spells and required craniotomy for repair of the dural hiatus. The possible etiology of such a large spontaneous pneumatocele is discussed in the context of a review of the pertinent literature.

KEY WORDS • pneumatocele • mastoid sinus

Pneumatoceles stemming from the mastoid air cells are much less common than those arising in the frontal ethmoid region. Although frequently the cause is not obvious, such pneumatoceles may be the result of mastoiditis. Traumatic and congenital varieties of pneumatoceles have also been described, especially in the frontal region. This case report describes a large spontaneous pneumatocele in the left temporal region, the mass effect of which led to aphasia. Prompt surgical obliteration of the air and closure of the dural defect ameliorated the symptoms completely.

Case Report

This 64-year-old man was admitted with a 3-day history of worsening word-finding difficulties and comprehension of speech. Except for severe fluent aphasia, the neurological examination was unremarkable. The patient’s medical history was significant for the diagnosis of an unresectable small-cell carcinoma of the lung, diagnosed 30 months prior to admission and complicated by a left recurrent nerve paralysis. This tumor was treated by chest irradiation and chemotherapy at the time of diagnosis. Cranial computed tomography (CT) was unremarkable at that time, but the patient underwent prophylactic whole-brain irradiation because of the high probability of disseminated seeding of the tumor. The cranial irradiation consisted of 3000 rads to the entire cranial vault, in a treatment protocol of 300 rads/day over 10 days. There were no previous episodes of neurological dysfunction and no apparent sequelae from the irradiation.

Following chest irradiation, the patient experienced bouts of coughing, up to 10 to 12 episodes per day. He also required multiple tooth extractions for periodontitis 2 months prior to admission. Neither he nor his wife could describe any specific etiological factors preceding the onset of the aphasia, except for the frequent protracted bouts of coughing. There was no history of acute or chronic otitis or any evidence of trauma to the cranial vault.

Examination. At the time of admission, cranial CT demonstrated a large air-containing structure in the left temporal region which appeared to exert a mass effect on the left temporal lobe (Fig. 1). The mass originated at the base of the left temporal fossa near the junction of the left petrous ridge and lateral temporal bone, and invaginated into the left temporal lobe from the basal aspect. The structure extended superiorly to the level of the left trigone, but there was no air in the lateral ventricle and no evident communication with the subarachnoid space. A high-resolution CT scan through the temporal bone was normal, with no evidence of fracture or inflammatory disease (Fig. 2). The CT studies revealed a large air-containing structure invaginating into the left temporal lobe from the base, probably in
FIG. 1. Left: Computerized tomography (CT) scan demonstrating a large air-containing structure in the left temporal region directly adjacent to the left petrous ridge posteriorly and laterally. The temporal horn appears displaced slightly medially. Right: Scan showing a higher transverse image, at the level of the pineal body. This CT scan clearly demonstrates the extensive size of the intracranial pneumatocele, and its delimitation into a roughly circular size. There was no free air in either the ventricular system or the subarachnoid space.

FIG. 2. High-resolution computerized tomography scan through the temporal bone showing the left petrous ridge and the thinness of the tegmen tympani and the lateral mastoid air cells. The mastoid air cells demonstrate no cloudiness of fluid. There is no overt site for entry of the air into the intracranial space.

communication with lateral mastoid air cells at the base. However, there was no evidence of fluid in the structure or inflammatory changes in the temporal views, suggesting that a chronic mucocele might be unlikely.

Operation. A left temporal craniotomy was undertaken to seal off the intracranial communication and to decompress the mass. The squamous skull was opened over the temporal area revealing a clear communication from a far lateral mastoid air cell; this was found to penetrate the dura on an initially epidural exploration. When the dura was opened a thin mucocele-lined cyst could be followed into the left temporal lobe, which was continuous with the dural rent. A small amount of liquid was evacuated from the interior of the cavity, which was mostly collapsed after the initial decompression. On further exploration of the cavity, a small communication with the left trigone of the lateral ventricle could be visualized. The lining of the cavity was thin and could not be removed, but the dural rent was closed with a patch graft. The mastoid air cells were packed after removal of the mucous lining, and the communication to the intracranial space was completely sealed. Pathological examination of the intracranial cyst lining demonstrated minimal gliosis; neither malignancy nor active mucous tissue was found.

Postoperative Course. The patient's speech immediately improved after operation and there were no sequelae. On follow-up examination 6 months after surgery his speech had returned to normal and the craniotomy was well healed.

Discussion

In 1971, Nomura, et al., described a patient with a mastoid air cyst, and reviewed nine previous case reports. Later, a mastoid pneumatocele was demonstrated on an early-generation CT scanner. These few examples in the literature emphasize the rarity of this condition. Numerous etiologies have been proposed for the development of intracranial pneumatoceles, including infection, trauma, and congenital or anatomical anomaly. Patients may also present with no predisposing cause.

Regardless of the inciting event, the common findings are the violation of the dura and an intracranial air-filled communication. Air is propelled into the cavity, usually from an air sinus, by a variety of methods such as coughing or sneezing. Air can also be drawn into the cavity if there is a cerebrospinal fluid leak or communication with the cyst, due to the negative intracranial pressure that may develop. A ball-valve phenomenon, with brain or dura plugging the hole, prevents air egress. This has been described in cases of traumatic frontal pneumatoceles. Either method of air entry and trapping is possible with our patient's pneumatocele. As in this example, pneumatoceles can reach a large size due to the nonirritating effect of air on the brain and the lack of formation of a thick capsule wall.

Of the 11 patients reported in the English literature, four had a history of otitis media. When the mastoid mucoperiosteum becomes infected, serum exudation can coalesce and cause decalcification and osteoclasis followed by bone erosion and dural or intracranial extension. This is a severe complication of mastoiditis and usually results in bone changes in a poorly pneumatized mastoid that can be appreciated on CT. Chronic mastoiditis is often accompanied by keratoma or cholesteatoma formation. These findings were
not a feature of this patient's history, pathology, or CT scan.

Mastoid pneumatoceles are occasionally due to petrous bone fracture, and otorrhea, facial paralysis, and hearing impairment have been reported as common sequelae. The reason for this is speculative but may be related to the presence of thicker dura mater over the petrous bone or to associated mastoid hemorrhage and mucosal edema preventing an ingress of air at the time of injury if there is a dural tear.

Iatrogenic causes of cerebral pneumatoceles are uncommon, with only a few cases reported, and none involving the mastoid region. Ionizing radiation in the 4000- to 6000-rad range can cause transient otic inflammation and vasculitis. A spontaneous frontal pneumatocele has been described following tumor irradiation with 5500 rads. Our patient received 3000 rads. One remote possibility in this case is that radiation may have caused tegmental or attic necrosis leading to pneumatocele origin, although violation of the dura is somewhat harder to explain. The enlarging size of the air mass in the dominant hemisphere caused the patient's aphasia. Prompt surgical removal prevented further enlargement of the mass and the potential danger of uncal herniation and superinfection of the air space.

Since the CT scan showed bilateral symmetry, it may be suggested that this patient's tegmen tympani and mastoid air cells were limited intracranially only by very thin bone or merely by the apposition of mucous membranes to the dura. Such absence of the basilar temporal bone has been described in approximately 25% of unselected autopsies and would anatomically predispose to intracranial consequences following increased pressure in the middle ear. Bone covering the tegmen and superior air cells is commonly so thin that it is not visualized on high-resolution CT. In this case, chronic partial obstruction of eustachian tube drainage of the middle ear might have led to increasing pressure within this space, particularly exacerbated by the patient's chronic coughing bouts. Thus, the rupture of the thin or nonexistent bone covering the mastoid air cells may have relieved pressure building within the middle ear. The lack of a significant cerebral response to the pneumatocele may also point to the short history of this cyst, clearly unaccompanied by mastoid sinus infection or bone injury. Thus, the combination of acquired thinness of the floor of the middle fossa and increased air pressure within the middle ear may have been the most significant circumstance contributing to the development of this patient's pneumatocele.

References


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