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Mastoid Pneumocele Causing Atlantooccipital Pneumatization

Michael L. Martin, Ravi Bhargava, Robert A. Ashforth, and Donald B. Russell

Summary: A 49-year-old woman had a palpable mass in her occipital region. Plain radiographs and CT examination revealed extensive atlantooccipital pneumatization with findings consistent with the diagnosis of mastoid pneumocele. Decompression was achieved with placement of a myringotomy tube, resulting in prompt symptomatic relief. On a follow-up CT examination, the pneumatized areas had become opacified and new bone formation was present.

Pneumoceles are uncommon entities defined as enlarged aerated paranasal sinuses or air cells with focal or diffuse thinning of the surrounding bony walls that have been described as affecting the mastoid air cells and all of the paranasal sinuses (1, 2). We present a case of extensive atlantooccipital pneumatization with findings consistent with the diagnosis of mastoid pneumocele, and discuss the successful management of this abnormality.

Case Report

A 49-year-old woman had a 2-day history of headache and a soft palpable mass in the occipital region. She had no history of trauma. She was a 30 pack-year smoker with chronic bronchitis and suffered from chronic headaches made worse by coughing. Physical examination revealed a boggy painless left-sided occipital mass and no neurologic abnormalities. Otoscopic examination disclosed a bulging right tympanic membrane that bulged further when the occipital mass was palpated. The left tympanic membrane was normal.

Skull radiographs showed radiolucency involving nearly the entire occipital bone, the right parietal bone, and the atlas (Fig 1A). A collection of air was present within the extracranial soft tissues near the occiput. A helical CT scan of the head and skull base was obtained with 1-mm collimation and 1.3:1 pitch. Scans were reformatted in multiple planes (Fig 1B-E) and showed extensive pneumatization of the occipital bone, the atlas, and the right temporal bone. Pneumatization involved the clivus immediately adjacent to the sphenoidal sinus. The left temporal bone was normal. There was mucoperiosteal thickening within the sphenoidal sinus and destruction of bone within and surrounding all areas of aeration. Air was present in both atlantooccipital joints (there was no atlantooccipital assimilation) and within the extracranial soft tissues in the occipital region, corresponding to the patient's palpable mass. Findings on a radionuclide bone scan were normal.

One month after radiologic evaluation the patient underwent a right myringotomy with placement of a grommet tube. The surgeon reported a rush of air released from the middle ear at the time of incision of the tympanic membrane. The patient's symptoms resolved promptly after surgery and she was asymptomatic at the time of the follow-up CT scan.

An axial CT study of the atlas and skull base was performed with 1-mm collimation 4 months after surgery. The abnormally pneumatized areas within the skull base and the atlas had become opacified. Newly formed bone trabeculae had developed within the opacified air cells, and the surrounding cortical bone had increased in thickness (Fig 1F-I).

Discussion

Three entities that cause enlargement of an aerated paranasal sinus have been described: hypersinus, pneumosinus dilatans, and pneumocele (3). A hypersinus is an enlarged paranasal sinus that does not expand the surrounding bone beyond its normal contours and has bony walls that are of normal thickness. Pneumosinus dilatans refers to a paranasal sinus with bony walls that are of normal thickness with focal or diffuse abnormal expansion of the sinus. Pneumoceles are hyperaerated paranasal sinuses or air cells associated with focal or generalized luminal enlargement and focal or diffuse thinning of the adjacent bony wall (1). Pneumoceles have been reported to affect the mastoid air cells and all the paranasal sinuses (1, 2). Persistently increased intraluminal pressure has been proposed as a mechanism of pneumocele formation, and symptomatic relief of maxillary pneumocele has been achieved with surgical decompression (2). Pneumoceles should be distinguished from pneumatocoles, which are extrasosseous gas collections that usually form after trauma, infection, or surgery (1).

Reports of abnormal pneumatization of the skull base published in the CT era are limited. Two cases of less extensive atlantooccipital pneumatization have been described (4, 5) and both articles proposed that the findings represented an unusual developmental variant, with the abnormal aeration caused by proliferation of accessory air cells said to arise from the mastoid air cells. Parks (6) described a case of occip-

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From the Departments of Radiology and Diagnostic Imaging (M.L.M., R.B., R.A.A., D.B.R.) and Pediatrics (R.B.), University of Alberta, Edmonton, Alberta, Canada.

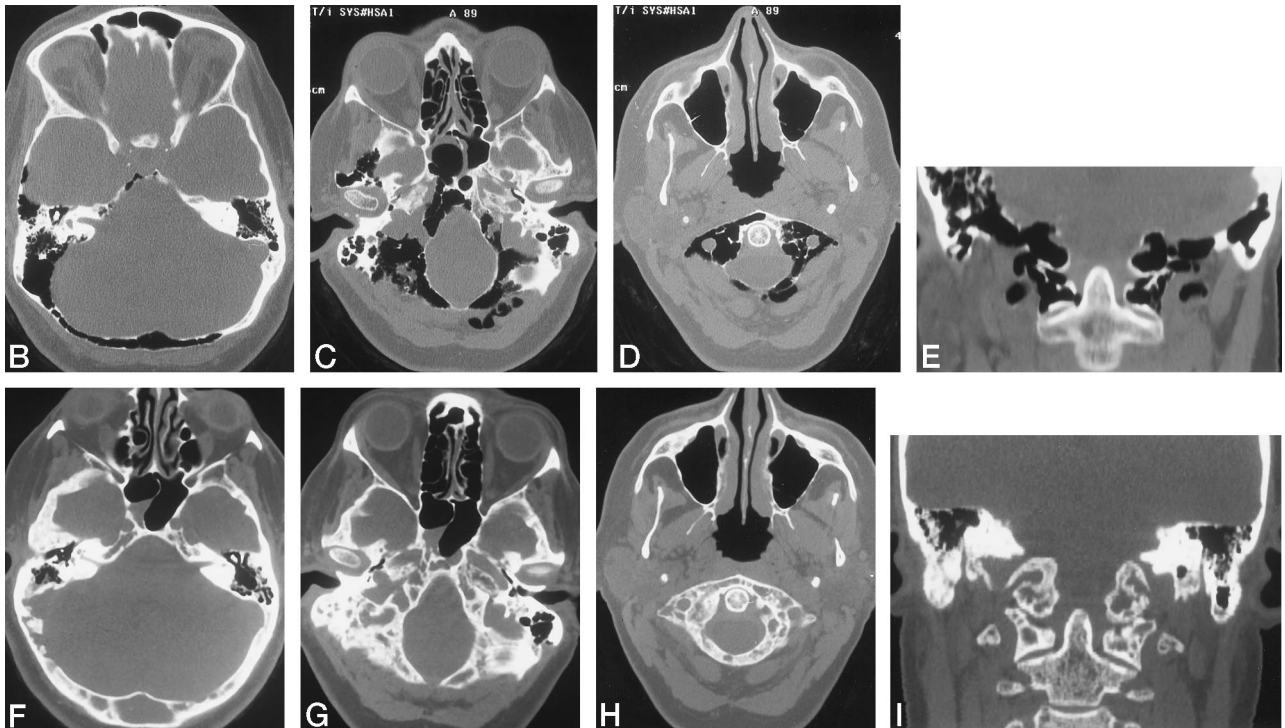
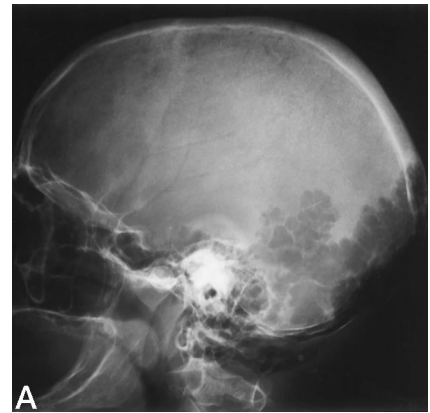
Address reprint requests to Michael L. Martin, MD, Department of Radiology and Diagnostic Imaging, University of Alberta Hospitals, 8440 112 St, Edmonton, Alberta, Canada T6G 2B7.

FIG 1. 49-year-old woman with mastoid pneumocele causing atlantooccipital pneumatization.

A, Lateral skull radiograph shows radiolucency of the skull in the occipital region with thinning of the inner and outer tables of the occipital bone. A collection of air is visible in the soft tissues posterior to the atlas.

B-E, Axial (B-D) and reformatted coronal (E) CT scans at the time of presentation show extensive pneumatization of the occipital bone, atlas, and right temporal bone. The sphenoidal portion of the clivus is pneumatized immediately adjacent to the sphenoidal sinus. Mucoperiosteal thickening is present within the sphenoidal sinus. Air is present within the atlantooccipital joints, best seen on the coronal reformatted image, and within the soft tissues posterior to the occiput.

F-I, At 4 months' follow-up, reformatted paraxial and coronal CT scans from an axial data set show opacification of previously pneumatized areas. Bony trabeculae have formed in areas previously devoid of medullary bone, and cortical bone has increased in thickness. The mucoperiosteal thickening in the sphenoidal sinus has become slightly more extensive.



ital and parietal pneumatization and also speculated that the findings were related to a developmental variant. Levenson et al (7) reported a case of extracranial pneumocele that was associated with mastoid hyperpneumatization in which there was dehiscence of the outer cortex of the temporal bone. The bone dehiscence was thought to be of developmental origin. Surgical intervention was undertaken in the case reported by Levenson et al, but radiologic follow-up was apparently not performed. More recently, Brown et al (8) described three patients with abnormal pneumatization within the skull base or the spine in association with intraosseous lymphangiomatosis. Two of these patients had involvement of the skull base and upper cervical spine, but in only one of these patients was there a definitive pathologic diagnosis of lymphangiomatosis within the pneumatized areas.

In our case, all areas of pneumatization within the skull base appeared to intercommunicate and there

was diffuse thinning of the surrounding bony structures. The atlantal pneumatization appeared to communicate via the atlantooccipital joints with the pneumatized occipital condyles. Otologic examination showed that the diploic air within the occipital bone was in continuity with the middle ear and that the intraluminal pressure was increased. The mechanism by which the increased intraluminal pressure arose in this case is not clear. A ball valve mechanism may have occurred at the eustachian tube, whereby air was forced into the middle ear more rapidly than it could escape. The patient's chronic bronchitis may have exacerbated this process, as repeated coughing episodes could have increased the rate and the pressure of air entering the middle ear.

Conclusion

The findings in our case of a hyperaerated air cell with surrounding bony destruction was more consis-

tent with the diagnosis of mastoid pneumocele than of hypersinus, in which bone destruction is not a feature. The air cell opacification and new bone growth seen after decompression supports this diagnosis, as these changes are not seen after myringotomy in patients with enlarged but otherwise normal mastoid air cells. We believe that the mastoid pneumocele seen in our case was an acquired lesion produced by the destructive effect of persistently elevated intraluminal pressure that caused the mastoid air cells to expand throughout the skull base and into the atlas. It was a destructive and aggressive process capable of crossing suture lines and synovial joints. The simple treatment performed in our case resulted in both immediate symptomatic relief and marked radiologic improvement of a destructive lesion.

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