Mastoid Findings Secondary to Posterior Fossa Dural Venous Sinus Thrombosis

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OBJECTIVE. In the setting of posterior fossa dural venous sinus thrombosis, mastoid findings can potentially be mistaken as the cause rather than a secondary effect. Obstruction of the mastoid venous drainage can lead to transudation of fluid into the mastoid air cells. We hypothesize that a continuum of the mastoid manifestations secondary to dural venous sinus thrombosis can be seen on MRI and that the difference in venous drainage between the mastoid and middle ears can assist with this important imaging differential.

MATERIALS AND METHODS. A retrospective review of acute dural venous sinus thrombosis cases and their follow-up imaging were graded as follows: no involvement; grade 1, mucosal congestion; grade 2, film of fluid; or grade 3, complete opacification. Presence of middle ear fluid was also documented.

RESULTS. Of 22 posterior fossa dural venous sinus thrombosis cases without clinical infectious mastoiditis, 19 had mastoid findings: eight with grade 1, eight with grade 2, and three with grade 3. Middle ear fluid was seen in only one case. There was a statistically significant association between posterior fossa dural venous sinus thrombosis and mastoid findings (chi-square test ($n = 22$), $p < 0.04$). Fifteen of 18 cases with follow-up MRI examinations showed lessening of the mastoid findings with resolving dural venous sinus thrombosis.

CONCLUSION. Acute posterior fossa dural venous sinus thrombosis may present with a spectrum of mastoid findings that should not be misdiagnosed as the cause of the thrombosis. This study illustrates that in the absence of clinical findings of mastoiditis, mastoid fluid with a clear middle ear argues that the mastoid changes are the effect of the dural venous sinus thrombosis rather than the cause.

Keywords: dural venous sinus thrombosis, mastoid effusion, MRI, sigmoid sinus thrombosis, transverse sinus thrombosis

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Mastoid effusions can be seen incidentally or secondary to a variety of underlying pathologies. It is important not to misdiagnose mastoid fluid on MRI as mastoiditis, which should never be suggested without knowledge of the patient's clinical presentation. Evaluation of the surrounding structures is vital because several diseases can have associated mastoid effusions, such as nasopharyngeal carcinoma and dural venous sinus thrombosis. When a mastoid effusion is seen in the setting of dural venous sinus thrombosis, it is critical to recognize it as the possible sequela of the thrombosis—not the source.

Although mastoid mucosal thickening and fluid have previously been reported in the setting of intracranial dural sinus venous thrombosis [1, 2] in the nonradiologic literature, in this article, we explore the imaging perspective of this relationship through a larger series than has been previously reported. On MRI, dural venous sinus thrombosis–associated mastoid air cell changes can be mistaken for infectious mastoiditis with consequent thrombophlebitis, which has a different treatment regimen and could include unnecessary administration of antibiotics [3]. Mastoid effusion on MRI identified in the setting of posterior fossa dural venous sinus thrombosis is most likely a phenomenon of venous congestion [1, 2].

This is a retrospective study evaluating the association of mastoid findings and dural venous sinus thrombosis. It is hypothesized that a spectrum of mastoid findings can be seen in the setting of acute posterior fossa noninfectious dural venous sinus thrombosis and that these dural venous sinus thrombosis–related mastoid changes will resolve with the evolution of the dural venous sinus thrombosis. In addition, other imaging features contributing to the cause of dural venous sinus thrombosis–related mastoid changes may be helpful in avoiding misdiagnosis.

Materials and Methods

A retrospective review of the radiology information system (RIS) was performed from 2001 to 2011 after institutional review board approval. Reports were searched for the diagnoses of "intracranial dural venous thrombosis" and/or "cortical vein thrombosis." MRI, and, when available, CT, MR venography, and CT venography examinations were reviewed on a PACS by three senior neuroradiologists.

Axial T2-weighted images with 5-mm slice thickness were evaluated for mastoid findings. Mastoid air cell involvement was graded on a scale from 0 to 3: 0, no mastoid involvement; 1, linear T2 hyperintensity along mastoid air cells representing mucosal congestion; 2, thickened crescentic T2 hyperintensity along mastoid air cells representing a thin film of fluid overlying the mucosa; and 3, T2 hyperintensity opacifying the mastoid air cells representing effusion (Fig. 1). In addition, the middle ear cavities were evaluated for the presence of fluid. After noting the MRI findings in the mastoid air cells for each case with dural venous sinus thrombosis, vascular imaging studies (CT venography, MR venography, or both) were reviewed to confirm the location of the dural venous sinus thrombosis.

In those cases with CT or CT venography available, the studies were assessed for possible osseous erosions of the sigmoid plate and mastoid septa. Axial CT slice thickness varied between 4.8 and 5.0 mm in collimation, depending on the CT scanner. The CT venography slice thickness was 1.0 mm. Follow-up examinations, including MRI, MR venography, CT venography, and CT, were reviewed for possible resolution of the dural venous sinus thrombosis as well as for persistence or decrease of the mastoid air cell findings. Statistical analysis was performed with an unpaired Fisher exact one-tailed Student *t* test.

Results

Of 291 cases from the RIS search for acute dural venous sinus thrombosis, 24 cases were positive for venous sinus (transverse and/or sigmoid sinus) and venous thrombosis (internal jugular vein and/or vein of Labbé). Two patients with clinical and laboratory findings suspicious for meningitis were excluded from the final analysis to avoid a confounding source of potential septic dural venous sinus thrombosis. The subjects included 14 women (64%) and eight men (36%) with an average age of 43 years (age range, 17–81 years).

Patients presented with myriad symptoms ranging from headache (73%) to aphasia (27%). Of the nine patients with leukocytosis (41%), four had negative blood and/or CSF cultures and five did not clinically warrant microbiologic tests. Fever was not documented in any of the cases at admission. The ear, nose, and throat surgery service performed dedicated otolaryngologic examinations, which were normal in all patients. All patients received anticoagulation therapy.

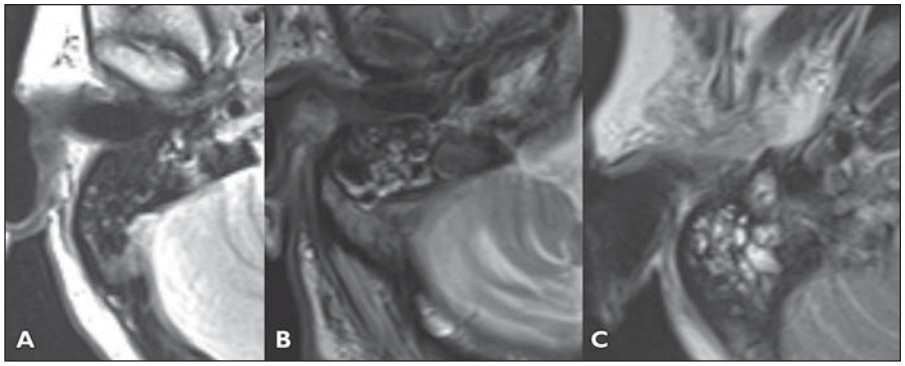

Fig. 1—MR images show grading system for dural venous sinus thrombosis.
A, Grade 1: curvilinear T2 hyperintensity is seen with mucosal congestion.
B, Grade 2: thickened crescentic T2 hyperintensity is due to film or fluid overlying mucosa.
C, Grade 3: complete T2 hyperintensity reflects fluid opacifying mastoid air cells.

The spectrum of mastoid involvement ranged from no findings to complete opacification. Of the 22 cases positive for dural venous sinus thrombosis, 19 (88%) had mastoid air cell findings. Eight subjects (36%) had grade 1 mastoid involvement, eight had (36%) grade 2 mucosal thickening, and three (14%) had grade 3 opacification. Of the 19 cases with mastoid air cell involvement, 16 (84%) had thrombosis of the ipsilateral transverse sinus, 17 (89.5%) had thrombosis of the ipsilateral sigmoid sinus, 15 (79%) had ipsilateral internal jugular vein thrombosis, and six (31.6%) showed thrombosis of the ipsilateral vein of Labbé. Bilateral dural venous sinus thrombosis was seen in one patient with distal sigmoid sinus and internal jugular vein involvement. Only one case showed a small amount of fluid in the middle ear cavity, which was ipsilateral to a thrombosed vein of Labbé. There was a statistically significant association between dural venous thrombosis and mastoid findings (chi-square test ($n = 22$), $p < 0.04$).

In those 10 patients with positive mastoid findings in whom contrast-enhanced MRI was performed, seven (70%) showed mastoid mucosal enhancement (Fig. 2). Parenchymal or subarachnoid hemorrhage was noted in five patients (22.7%). Increased parenchymal T2 hyperintensity, likely representing edema, was observed in seven (31.8%), four of which showed hemorrhage. CT examinations were performed in 19 patients and CT venography examinations with higher spatial resolution were obtained in nine patients. None of these studies revealed osseous erosions. Follow-up MRI was performed in 14 of the 22 patients, 11 of whom underwent MR venography studies as well. Repeat MR examinations were performed from 5 days to 1 year after the initial study. Eleven cases showed a decrease or resolution of the mastoid findings (Figs. 3A and 3B). One of these 14 patients exhibited no change in mastoid findings and two showed an increase in grade of mastoid air cell involvement. Five patients underwent follow-up CT venography examinations and one patient underwent follow-up CT only. Table 1 shows the distribution of the dural venous sinus thrombosis and mastoid air cell findings at presentation and at follow-up.

Discussion

When attempting to elucidate the cause of mastoid air cell fluid, the radiologist must consider the possibility of the mastoid findings being primary, secondary, or incidental and remember to evaluate the surrounding structures. This study found a statistically significant correlation between dural venous sinus thrombosis and varying grades of mastoid mucosal thickening or fluid. Various case reports in the literature have also described this as "mastoid air cell abnormality" [1], "mastoid effusion" [2], and "pseudomastoiditis" [4], in association with a nonseptic lateral sinus thrombosis (composed of the sigmoid and transverse sinuses) [4–6]. This imaging finding of curvilinear T2 hyperintensity on MRI is likely due to mucosal edema [1, 5, 7]. The mechanism of this fluid transudate is likely related to the vascular congestion from venous occlusion. On follow-up imaging, most cases with dural venous sinus recanalization showed commensurate resolution or decrease in the mastoid findings.

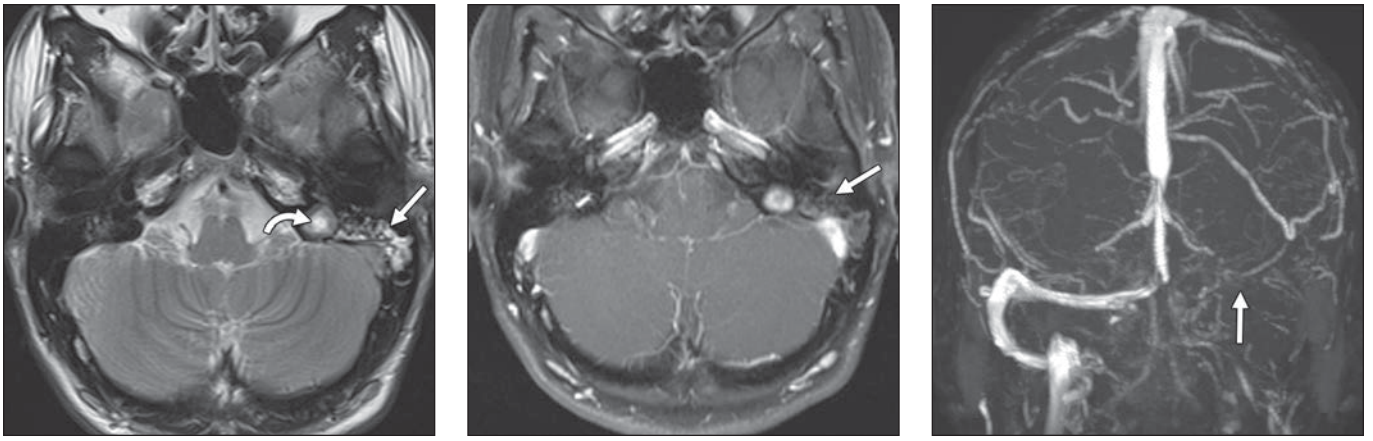

Fig. 2—25-year-old man with left-sided headache, pain behind left ear, photophobia, and left eye pain.
A, Axial T2-weighted MR image shows grade 3 mastoid findings with fluid in left mastoid air cells (*white arrow*). There is abnormal hyperintense signal within left sigmoid sinus-internal jugular vein junction, consistent with thrombus (*curved arrow*).
B, Contrast-enhanced axial T1-weighted MR image with fat saturation shows curvilinear enhancement of mucosa of left mastoid air cells (*arrow*).
C, Maximum-intensity-projection image from 2D MR venography reveals absence of left transverse, sigmoid, and internal jugular vein flow–related enhancement (*arrow*).

Anatomy and Pathophysiology

The skull base venous anatomy plays a fundamental role in explaining the pathophysiology of this MRI finding. The venous drainage of the skull base consists of the internal jugular veins and a complex internal jugular vein–independent pathway. This internal jugular vein–independent system is composed of a superficial network, which drains the occipital scalp, the suboccipital region, and the posterior neck muscles, and a deep network, which is composed of the vertebral venous plexus and epidural venous plexus [8, 9]. Cerebral venous outflow alters with posture: With the patient supine, the internal jugular veins are the main drainage pathway, but when the patient is upright, the venous outflow diverts to the internal jugular vein–independent pathway [9–11]. Such diversion is achieved via the noncollapsible emissary veins of the posterior fossa [5, 7]. These emissary veins represent anastomosing channels between the scalp and cervical veins and the dural sinuses [12, 13]. Myriad small vascular channels within the bony mastoid septa draining directly into the sigmoid sinus or indirectly via the mastoid emissary veins provide mucosal venous drainage of the mastoid air cells [14]. In these valveless emissary veins, blood flows generally from the external to internal venous systems, although it can be bidirectional [1, 15]. Such flow dynamics serve as a safety valve diverting blood away from the brain in conditions leading to cerebral congestion, such as bilateral internal jugular venous obstruction [1, 16].

In addition to evaluation of the spectrum of mastoid findings on CT and MRI, involvement of the middle ear cavity was noted and observed in only one patient. This is likely due to the different venous drainage of the middle ear cavity compared with that of mastoid air cells. The middle ear cavity venous drainage is to the pterygoid venous plexus and to the superior petrosal sinus [17, 18]. As mentioned, the mucosal venous drainage of the mastoid air cells is via transosseous vascular channels in the mastoid septa, with drainage directly into the sigmoid sinus or indirectly through mastoid emissary veins [14]. This patient had grade 3 mastoid effusion involving the aditus ad antrum, which may have extended into the epitympanum. In this study of posterior fossa dural venous sinus thrombosis, the pathophysiology of the mastoid air cell abnormalities may be related to the complex mastoid venous anatomy. The lack of mastoid findings in three patients may be related to venous collateral pathways. With venous occlusion, the vascular congestion leads to interstitial edema and progressive fluid transudation [1, 5, 7]. The middle ear cavity mucosa is spared because its venous drainage empties separately and anteriorly into the pterygoid venous plexus.

The morphohistologic separation of the middle ear cavity from the mastoid air cells [19] may also help explain the lack of middle ear involvement in our cases. An anteroinferior compartment, lined by mucus-secreting ciliated pseudostratified columnar epithelium with thick and relatively dense submucosal connective tissue, is similar to the nasopharyngeal and eustachian tube mucosa and has a similar mucociliary clearance function [19]. The epitympanum, mastoid antrum, and mastoid air cells constitute the posterosuperior compartment, which is covered by nonciliated richly vascularized cuboidal epithelium, with loose submucosal connective tissue performing mainly gas exchange [20]. In addition to being highly vascularized, the posterosuperior compartment has loose lamina propria and a shorter distance between submucosal vessels and the epithelial basement membrane [19], which makes it more responsive to increases in hydrostatic pressure from venous occlusion, as in our dural venous sinus thrombosis cases. This may also explain the small amount of fluid in the epitympanum seen in one patient. Circumstances that increase the hydrostatic capillary pressure in the mucosa, such as venous congestion, can cause interstitial transudate in the mucosa [20].

With sustained increased hydrostatic capillary pressure, a thin film of fluid may form over the mucosal surface [20]. Most patients showed grade 1 or 2 mastoid findings, corresponding with interstitial edema and film of fluid, respectively. Those patients with grade 3 mastoid findings had factors contributing to increased vascular congestion. One of the patients with grade 3 mastoid findings had bilateral distal sigmoid sinus and internal jugular vein thrombosis. Another patient had ipsilateral intracranial hemorrhage, which may have contributed to increased intracranial pressure. The third patient with grade 3 mastoid findings presented 2–3 weeks after onset of headache symptoms.

Theoretically, the sustained vascular congestion may have gradually developed into frank

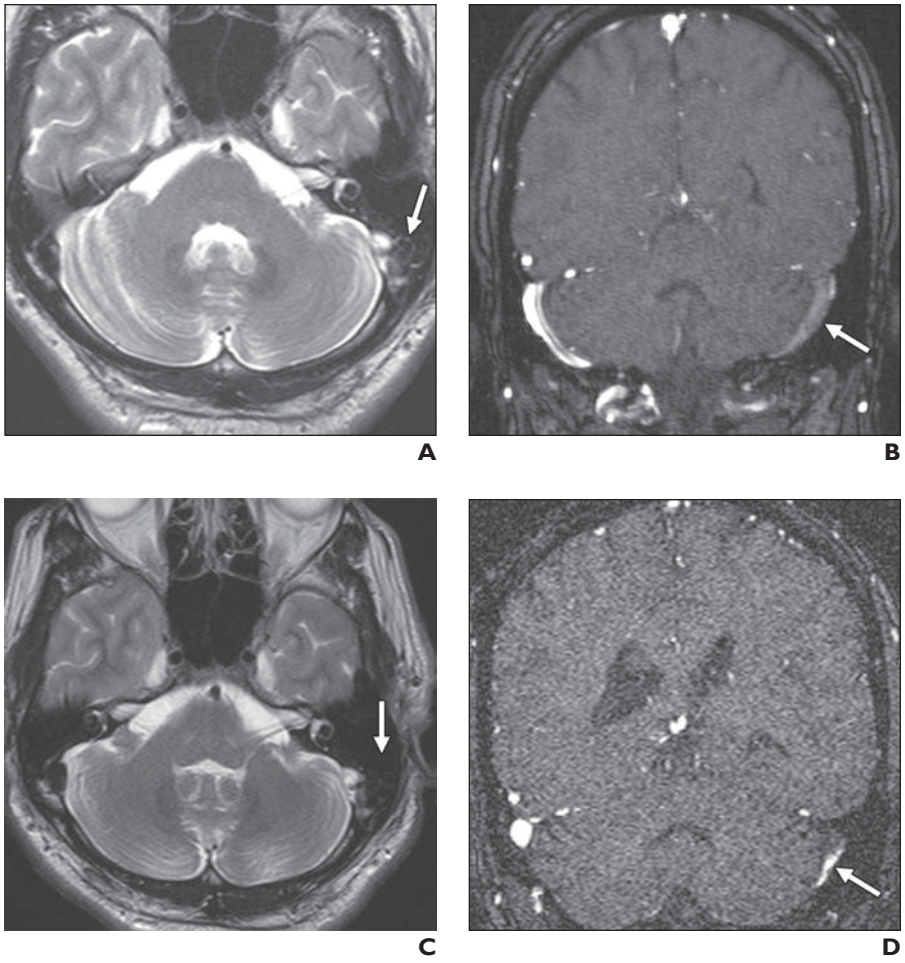

Fig. 3—46-year-old man with headaches and numbness and tingling on left side of face.
A, Axial T2-weighted MR image shows grade 1 crescentic hyperintensity in left mastoid air cells (*arrow*) corresponding with thin film of fluid.
B, Coronal 2D MR venogram displays abnormal lack of flow-related enhancement in left sigmoid sinus, consistent with thrombus (*arrow*).
C, At 5-month follow-up, patient had improvement in headaches as well as left facial paresthesias. Axial T2-weighted MR image shows resolution of mastoid mucosal fluid with diminished hyperintensity (*arrow*).
D, Coronal 2D MR venogram at slightly different angle obtained at 5-month follow-up shows partial recanalization with irregular diminutive flow-related enhancement in left sigmoid sinus (*arrow*).

fluid levels within the air cells. Additionally, a majority (70%) of patients with higher grades of mastoid findings also showed mucosal enhancement, likely due to venous congestion. Follow-up MRIs showed decreased or resolved mastoid findings, with recanalization of the dural venous sinus in most cases (84.6%), likely due to decreased venous congestion with a secondary decrease in hydrostatic capillary pressure [20]. The role of venous congestion and increased hydrostatic capillary pressure also explains the lack of resolution of mastoid findings in the three patients in whom the dural venous sinus thrombosis did not recanalize.

In infectious otomastoiditis, there is often opacification of the middle ear cavity and mastoid air cells with preservation of the mastoid cortical bone, trabeculae, and ossicular chain [21, 22]. Although dedicated high-resolution temporal bone CT was not available for these cases, none of the patients who underwent CT or CT venography had osseous erosions. CT venography, in particular, with higher spatial resolution because of the 1-mm slice thickness showed preservation of the mastoid septa and sigmoid plate in all patients for whom it was performed. In classic acute otomastoiditis, the aditus ad antrum becomes obstructed by the edema and swollen mucosa. Subsequently, there is development of acute mastoiditis with trapping of secretions in the antrum and mastoid air cells. In the appropriate clinical context, the presence of middle ear effusion and soft-tissue attenuation in the mastoid air cells without osseous resorption or periostitis is considered incipient mastoiditis [21]. Dural venous sinus thrombosis accounts for 6% of intracranial complications of infectious otomastoiditis in the era of antibiotic treatment [23, 24].

Clinical Findings

The clinical manifestations of these patients are attributable to this complex anatomy and pathophysiology. Generalized headache is usually the initial symptom in dural venous sinus thrombosis without a distinct identifiable pattern. However, with transverse or sigmoid sinus thrombosis there may be localized neck or occipital pain [25]. Similarly, in this study, most (73%) patients presented with headache. Some of the neurologic manifestations, such as seizure, may have been secondary to parenchymal involvement related to the dural venous sinus thrombosis. Leukocytosis was documented at presentation in less than half (41%) of our cases. This may be due to a generalized inflammatory response, possibly along the dural sinus walls. The inflammatory response as well as the possible increased intracranial pressure, intracranial hemorrhage, and stretching of nerves along dural venous sinus walls may be causes for the patients' headaches [6]. Fever is not a typical clinical feature of nonseptic dural venous sinus thrombosis [5, 6] and was not a presenting symptom in any of our cases at admission.

Nonseptic dural venous sinus thrombosis, which was the cause of the mastoid findings in our cases, affects the adult age group, the majority being women taking oral contraceptives [26] or puerperium [5]. Over half of our patients were women (14/22, 64%), and two patients were 3–5 months postpartum. Other causative factors included dehydration from vomiting and diarrhea, which was reported before presentation in seven cases (32%). Immune disorders have also been reported as possible causes [27]. Two patients in our series were later diagnosed with Behçet disease, one of whom was also a heterozygous carrier of factor V Leiden deficiency. One patient was heterozygous for a prothrombin gene mutation and another was a thrombophilia gene mutation carrier.

Although this study is limited by its relatively small sample size, it is the largest series reported in the imaging literature, and a statically significant correlation between mastoid findings and dural venous sinus thrombosis was shown. Further supporting this correlation is the resolution of the mastoid findings with resolution of dural venous sinus throm-

TABLE 1: Imaging Findings on Presentation and Follow-Up

Patient No.	Venous Structure Involved	Side	Mastoid Grade	Follow-Up Mastoid Grade	Dural Venous Sinus Thrombosis Resolution
1	Transverse, sigmoid	Left	2	Yes	No
2	Transverse, sigmoid, internal jugular vein	Right	2	Yes	Partial recanalization
3	Transverse, sigmoid, internal jugular vein	Left	3	NA	NA
4	Transverse, sigmoid	Right	0	0	NA
5	Transverse, sigmoid, internal jugular vein	Left	2	Yes	Partial recanalization
6	Transverse, sigmoid, internal jugular vein	Right	2	Yes	Partial recanalization
7	Straight, torcula, transverse	Left	0	0	Yes
8	Straight, transverse	Right	0	3	No
9	Transverse, sigmoid, internal jugular vein	Bilateral	3	Yes	Yes
10	Transverse, internal jugular vein	Left	1	Yes	Yes
11	Transverse, sigmoid, internal jugular vein	Left	1	Yes	Partial recanalization
12	Transverse, sigmoid, internal jugular vein	Left	2	NA	NA
13	Transverse, sigmoid, internal jugular vein	Right	2	Yes	Partial recanalization
14	Transverse, sigmoid	Right	1	Yes	Yes
15	Vein of Labbé	Right	3	NA	NA
16	Transverse, sigmoid, internal jugular vein	Right	2	NA	NA
17	Distal sigmoid, internal jugular vein	Left	2	Yes	Partial recanalization
18	Transverse	Right	1	2	No
19	Transverse, sigmoid, internal jugular vein	Right	1	Yes	Partial recanalization
20	Transverse, sigmoid, internal jugular vein	Left	1	Yes	Partial recanalization
21	Transverse, sigmoid, internal jugular vein	Right	1	Yes	Partial recanalization
22	Distal sigmoid, internal jugular vein	Left	1	1	No

Note—NA = not available (lost to follow-up).

bosis on follow-up imaging studies. Although subtle osseous detail could not be examined with the CT studies available, this supplementary imaging information did support the absence of osseous destruction, particularly preservation of the sigmoid plate. The cases were selected by search of the RIS reports for dural venous sinus thrombosis, which may have excluded patients with infectious thrombophlebitis. The retrospective nature of this study creates a slight bias in the interpretation of the images. Though the readers were not blinded to the clinical status of the patients, mastoiditis is a clinical diagnosis and should not be made by the radiologist in isolation. Obviously, direct pathologic correlation would be not feasible in this clinical scenario.

Conclusion

In the setting of posterior fossa dural venous sinus thrombosis without supportive clinical features of acute mastoiditis, it is important to recognize that the mastoid findings are often a secondary phenomenon rather than a primary infectious process. The mastoid air cell findings in these cases are likely related to a cascade of venous occlusion leading to mucosal congestion and subsequent interstitial edema, typically sparing of the middle ear. The resolution of the mastoid findings with dural venous sinus recanalization in our cases strengthens the diagnosis of a noninfectious cause of the mastoid air cell abnormality. This important imaging finding can guide the radiologist in the important differential diagnosis of either clinically evident mastoiditis leading to dural venous sinus thrombosis with middle ear involvement or noninfectious dural venous sinus thrombosis leading to mastoid findings without middle ear involvement.

References

1. Fink JN, McAuley DL. Mastoid air sinus abnormalities associated with lateral venous sinus thrombosis: cause or consequence? *Stroke* 2002; 33:290–292
2. Agid R, Farb RI. Mastoid effusion associated with dural sinus thrombosis. *Eur Radiol* 2005; 15:755–758
3. Ropposch T, Nemetz U, Braun EM, Lackner A, Tomazic PV, Walch C. Management of otogenic sigmoid sinus thrombosis. *Otol Neurotol* 2011; 32:1120–1123
4. Thota R, Narayanan N, Mathuram D. Pseudomastoiditis in lateral sinus thrombosis: a rare presentation with review of literature. *Indian J Otolaryngol Head Neck Surg.* 2011; 63(suppl 1):S135–S139
5. Rosen A, Scher N. Nonseptic lateral sinus thrombosis: the otolaryngologic perspective. *Laryngoscope* 1997; 107:680–683
6. Viswanatha B. Nonseptic lateral sinus thrombosis: the role of the otolaryngologist. *Ear Nose Throat J* 2009; 88:731–733
7. Hawkins DB. Lateral sinus thrombosis: a sometimes unexpected diagnosis. *Laryngoscope* 1985; 95:674–677
8. Louis RG Jr, Loukas M, Wartmann CT, et al. Clinical anatomy of the mastoid and occipital emissary veins in a large series. *Surg Radiol Anat* 2009; 31:139–144
9. Boyd GI. The emissary foramina of the cranium in man and the anthropoids. *J Anat* 1930; 65(Pt 1):108–121
10. Gisolf J, van Lieshout JJ, van Heusden K, Pott F, Stok WJ, Karemaker JM. Human cerebral venous

outflow pathway depends on posture and central venous pressure. *J Physiol* 2004; 560(Pt 1):317–327

11. Schreiber SJ, Lurtzing F, Gotze R, Doepp F, Klingebiel R, Valdueza JM. Extrajugular pathways of human cerebral venous blood drainage assessed by duplex ultrasound. *J Appl Physiol* 2003; 94:1802–1805
12. Streeter G. The development of alterations in the vascular system of the embryo. *Am J Anat* 1918; 24:7–38
13. Marsot-Dupuch K, Gayet-Delacroix M, Elmaleh-Berges M, Bonneville F, Lasjaunias P. The petrosquamosal sinus: CT and MR findings of a rare emissary vein. *AJNR* 2001; 22:1186–1193
14. Ars B, Ars-Piret N. Morpho-functional partition of the middle ear cleft. *Acta Otorhinolaryngol Belg* 1997; 51:181–184
15. Valdueza JM, von Munster T, Hoffman O, Schreiber S, Einhaupl KM. Postural dependency of the cerebral venous outflow. *Lancet* 2000; 355:200–201
16. Hadeishi H, Yasui N, Suzuki A. Mastoid canal and migrated bone wax in the sigmoid sinus: technical report. *Neurosurgery* 1995; 36:1220–1223; discussion, 1223–1224
17. Csillag AL. *The organ of hearing and equilibrium: atlas of the sensory organs: functional and clinical anatomy*. Totowa, NJ: Humana Press, 2005:7
18. Baker EW, Schuenke M, Schulte E, Schumacher U. Temporal bone and ear. In: *Head and neck anatomy for dental medicine*. New York, NY: Thieme, 2010:166
19. Ars B, Wuyts F, Van de Heyning P, Miled I, Bogers J, Van Marck E. Histomorphometric study of the normal middle ear mucosa: preliminary results supporting the gas-exchange function in the postero-superior part of the middle ear cleft. *Acta Otolaryngol* 1997; 117:704–707
20. Magnuson B. Functions of the mastoid cell system: auto-regulation of temperature and gas pressure. *J Laryngol Otol* 2003; 117:99–103
21. Vazquez E, Castellote A, Piqueras J, et al. Imaging of complications of acute mastoiditis in children. *RadioGraphics* 2003; 23:359–372
22. Dhooge IJ, Vandenbussche T, Lemmerling M. Value of computed tomography of the temporal bone in acute otomastoiditis. *Rev Laryngol Otol Rhinol (Bord)* 1998; 119:91–94
23. Manolidis S, Kutz JW Jr. Diagnosis and management of lateral sinus thrombosis. *Otol Neurotol* 2005; 26:1045–1051
24. Dentali F, Gianni M, Crowther MA, Ageno W. Natural history of cerebral vein thrombosis: a systematic review. *Blood* 2006; 108:1129–1134
25. Wasay M, Kojan S, Dai AI, Bobustuc G, Sheikh Z. Headache in cerebral venous thrombosis: incidence, pattern and location in 200 consecutive patients. *J Headache Pain* 2010; 11:137–139
26. Samsioe G. Coagulation and anticoagulation effects of contraceptive steroids. *Am J Obstet Gynecol* 1994; 170:1523–1527
27. Barnett HJM, Mohr JP, Stein BM, Yatsu FM. *Stroke: pathophysiology, diagnosis, and management*, 3rd ed. New York, NY: Churchill Livingstone; 1997