Mastoid Air Sinus Abnormalities Associated With Lateral Venous Sinus Thrombosis
Cause or Consequence?

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Background—Mastoiditis is a known cause of lateral venous sinus thrombosis (LST). We have encountered patients with LST associated with mastoid abnormality on MRI without any clinical signs of infection; the significance of these abnormalities is uncertain. This study examines the relationship of LST and mastoid air sinus abnormalities systematically.

Summary of Report—We performed a retrospective clinical and radiological review of a series of 26 patients with cerebral venous thrombosis. Mastoid abnormalities were detected ipsilateral to 9 of 23 thrombosed lateral sinuses (39%) and 0 of 29 unaffected lateral sinuses ($P<0.001$). No patient had clinical evidence of mastoiditis. Eight of 9 patients with mastoid abnormalities were treated without antibiotics; all made uneventful clinical recoveries. Repeated MRI in 1 patient revealed reversal of the mastoid changes.

Conclusions—The mastoid changes observed are likely to be due to venous congestion as a consequence of LST, not mastoiditis. (Stroke. 2002;33:290-292.)

Key Words: lateral sinus thrombosis • magnetic resonance imaging • mastoiditis • sinus thrombosis, intracranial

In the preantibiotic era, craniofacial infections were a frequent cause of cerebral venous sinus thrombosis (CVT). Infection is now an uncommon cause of CVT in general but remains the leading cause of cavernous sinus thrombosis. MRI with MR venography is now the investigation of first choice in the investigation of CVT. T2-weighted MRI is sensitive to mucosal changes and accumulation of fluid in the mastoid air sinus. We have encountered patients with lateral venous sinus thrombosis (LST) associated with mastoid abnormality on MRI without any clinical signs of infection. We hypothesized that mastoid air sinus abnormalities associated with LST could be due to venous congestion as a consequence of the LST and may not inevitably represent infective mastoiditis. This could have important implications for diagnosis and treatment of LST and has not been subject to systematic review previously.

Subjects and Methods
The records of all adult patients with CVT diagnosed or treated at Auckland Hospital (New Zealand) during 1990–1999 were reviewed retrospectively. Patients were identified by computerized search of discharge diagnosis codes and inpatient and outpatient neurological databases and by examination of the report of every MR venogram performed over this period. Patients with CVT associated with structural lesions such as cerebral tumors, skull fracture, or neurosurgical procedures and those with CVT involving only the cavernous sinus were excluded. The radiology findings were examined with the investigator blinded to clinical information.

Results
Twenty-six patients with CVT were identified. Thrombosis was present in 23 lateral sinuses in 20 patients. Mastoid abnormalities, as described below, were detected ipsilateral to 9 of 23 thrombosed lateral sinuses (39%; 5 left, 4 right) and 0 of 29 unaffected lateral sinuses ($P<0.001$, Fisher exact test). The clinical features of the patients with LST are summarized in the Table.

Radiological Appearance
The mastoid abnormalities consisted of increased T2-weighted MRI signal in the mastoid air spaces, often with a trabecular pattern consistent with mucosal thickening within the air cells. Fluid was seen filling cells in some cases. The mastoid abnormalities were described in the neuroradiologist’s initial report in 3 patients. The appearance of the mastoid abnormalities in 5 of the remaining patients was similar to that in the reported patients (Figure 1, Figure 2B). More minor mastoid abnormalities were identified in a final case. For 1 patient with a recognized MRI mastoid abnormality, the diagnosis of CVT was not made until several days later, after a clinical deterioration. In another patient who presented with left temporal lobe hemorrhage, both the mastoid abnormality and the diagnosis of CVT were initially overlooked (Figure 2).
Evidence of Infection
Evidence of systemic infection was present in only 1 patient with LST and mastoid abnormality: fever of 40°C, meningism, and a white blood cell count of 30×10⁹/L. This 18-year-old female patient had a remote history of maxillary sinus disease but had no recent sinus or otologic symptoms. She was taking high-dose prednisone for immune thrombocytopenia and was positive for lupus anticoagulant. An otorhinolaryngologist’s assessment and blood cultures were negative. Cerebrospinal fluid examination was prevented by anticoagulation. Empiric antibiotic treatment was added. Her recovery was uneventful.

None of the other 8 patients with LST and mastoid abnormality had symptoms or signs of mastoiditis or infection or a recorded history of otologic disease. None received antibiotic treatment; 6 received anticoagulants. All made uneventful recoveries.

Alternative etiologic factors were detected in 6 of the 9 patients with mastoid abnormality, including systemic lupus erythematosus or lupus anticoagulant in 3, inherited thrombophilia in 2, pregnancy in 1, and treatment with oral contraceptives and tranexamic acid (for menorrhagia) in 1.

Resolution of Mastoid Abnormalities
A repeated MRI was available for 1 patient with LST and mastoid abnormality 1 month after the initial image. Mastoid normalization and full clinical recovery occurred with anticoagulation only (Figure 2D).

Discussion
The diagnosis of mastoiditis in association with LST is important because septic cerebral thrombophlebitis requires a prolonged course of antibiotics and consideration of surgical treatment, whereas nonseptic CVT is generally treated with anticoagulants alone.¹ ² ⁵ ⁷ In developed countries, septic CVT has become rare in adults,¹ ² although it remains a more significant problem where access to antibiotic treatment is poor.⁵ In previous reports, the clinical signs of mastoiditis have been prominent when associated with LST.² ³ ⁵ In our series, by contrast, MRI abnormalities were common in the mastoid air sinus ipsilateral to LST (39%); however, there were no signs of otologic disease or mastoiditis in any patient. In 1 patient with clinical signs of infection, the diagnosis of mastoiditis was doubtful. The uneventful recovery of 8 patients with mastoid abnormalities and LST and the demonstration of reversal of mastoid changes in 1 patient, without antibiotic treatment, support our hypothesis that the mastoid abnormalities seen in association with LST may not be due to mastoiditis.

The sigmoid portion of the lateral venous sinus runs on the inner aspect of the mastoid process. While venous drainage of the mastoid air sinus itself receives little attention in anatomic texts, mastoid emissary veins connecting the transverse or sigmoid portion of the lateral venous sinus and the posterior auricular veins or occipital venous plexus are well de-
scribed. The direction of blood flow through these vessels is normally from extracranial to intracranial but can be reversed when the jugular vein or sigmoid sinus is occluded. Diploic veins and small veins from the middle ear also drain into the lateral sinus. Occlusion of the lateral venous sinus due to nonseptic thrombosis could therefore be expected to increase venous pressure in the vessels draining the mastoid air sinus mucosa, resulting in vascular congestion, interstitial edema, and transudation of fluid into the air spaces.

We conclude that the mastoid abnormalities observed ipsilateral to LST in our patients are probably due to mucosal edema resulting from venous congestion rather than infective mastoiditis. Individual venous anatomy and the extent of thrombosis may account for the variation in the appearance of mastoid congestion in association with LST. The MRI finding of mastoid congestion in the absence of otologic disease is a novel observation that can be an important clue to the presence of an unrecognized LST. Patients presenting with LST and mastoid congestion should have a thorough clinical assessment for the presence of mastoiditis. When there is no clinical evidence of infection, treatment should be directed at the underlying CVT. The evidence from this small case series suggests that additional treatment with antibiotics is not required. Confirmation from a randomized prospective study is needed.

References
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