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Why No Unilateral Vestibular Atelectasis?

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Editor:

It was with great interest I read the recently published paper, “Patients with Vestibular Loss, Tullio Phenomenon, and Pressure-induced Nystagmus: Vestibular Atelectasis?”(1) All of us who see patients with vestibular disorders occasionally see patients with symptoms and findings that cannot be explained by an established diagnosis. The recent report of 4 patients by Wenzel, et al. explains how the combination of bilateral vestibular hypofunction and pressure-induced nystagmus may be due to vestibular atelectasis (VA) a histopathologic finding previously described by Merchant and Schuknecht in 1988(2). The prospect of linking clinical symptoms with histopathology is not only exciting but of potentially great clinical importance.

Merchant and Schuknecht reported VA in 426 temporal bones from 213 patients(2). They initially excluded patients with known pathology such as Meniere's, tumors, labyrinthitis, and trauma. Care was also taken to avoid cases related to post-mortem artifact. Eight of these 213 patients had VA on histopathology. Review of the medical record in these patients revealed that 5 (63%) had a history of vertigo, a much higher rate than the 12% of those who did not have VA. Three of these cases were sudden onsets of vertigo which likely resolved similar to what could be diagnosed as vestibular neuritis in a living patient. In the other two cases there were reports of chronic symptoms. One of these patients had a cholesteatoma and chronically draining ear. The other had dizziness associated with head motion which began at age 75. These symptoms are consistent with bilateral vestibular hypofunction and this patient was most like those reported in the recent clinical series(1). This last patient was also the only one that could be considered bilateral VA, although the finding was limited on one side. Merchant and Schuknecht also included 6 additional cases of secondary vestibular atelectasis which they believed to be due to other causes such as aging, hydrops, surgery, or trauma.

When comparing the original report of VA based on histopathology(2) with the recent clinical series(1) several issues arise: First, in the temporal bone series VA was relatively

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common (1 in 27 people) but the proposed clinically associate symptoms are clearly much more rare. Second, all the cases in the Wenzel et al. series were bilateral but histologic VA is almost always a unilateral finding. Third, it is likely that VA is frequently secondary to other pathology.

Some of these issues can be reconciled for instance, most unilateral cases of VA may be asymptomatic due to compensation from the contralateral side, and it may only be the relatively rare bilateral case that causes persistent and clinically relevant symptoms. Although it is possible that there is a genetic origin to bilateral VA as Wenzel et al. speculated it is also possible that VA is secondary to some other cause which occurred bilaterally in a fraction of patients. But, if unilateral VA is relatively common as the histopathology suggests and a cause of sound evoked nystagmus as Wenzel et al. proposed then why don't we see this clinical presentation? Is it possible that unilateral VA could be a source of sound evoked nystagmus in a subset of patients with conditions such as the near-dehiscence of the superior semicircular canal recently described by the same group(3)?

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