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Fibrous Dysplasia of the Temporal Bone Presenting with Chronic Suppurative Otitis Media: Not All Chronic Ear Discharge is CSOM

Dear Editor,

We read with interest the grand rounds presentation of Alvarez and Uy describing a patient with fibrous dysplasia (FD) of the temporal bone.[1] While the authors provide valuable insights into a rare presentation, we wish to highlight significant concerns regarding the diagnostic framework used in this case.

Chronic suppurative otitis media (CSOM) is described as a chronic inflammation of the middle ear and mastoid that presents with recurrent ear discharge or otorrhea through a tympanic membrane perforation. The disease usually begins in childhood as a spontaneous tympanic perforation due to an acute infection of the middle ear, known as acute otitis media (AOM), or as a sequelae of less severe forms of otitis media.[2] It is clear that the infection begins in the middle ear, and is associated with a perforation of the tympanic membrane. One cannot diagnose CSOM in the absence of evidence of a tympanic membrane perforation.

In the case presented in the article, we note that the diagnosis of chronic suppurative otitis media was first made simply based on the presence of a 1-year history of clear, non-foul-smelling discharge from his left ear associated with intermittent otalgia. This suggests that the physician evaluating the patient thinks that all patients presenting with a long history of otorrhea and otalgia have CSOM. That is an obvious fallacy, as conditions such as (but not limited to) chronic otitis externa, temporal bone carcinoma, a long-standing foreign body in the ear canal, and external canal cholesteatoma can also present with the exact same symptoms. We would like to point this out as a demonstration of the availability bias or availability heuristic. This diagnosis led to the patient being treated for 3 months with management specific to CSOM. It is only later that we learn that there is a hard mass in the external auditory canal. This apparently blocks the external auditory canal, as there is no mention whatsoever of the status of the tympanic membrane. The CT scan, done after the patient did not respond to their medical therapy, shows the presence of a bony lesion whose description ("ground glass") indicates a fibro-osseous lesion such as fibrous dysplasia. It must be noted that this lesion obliterates the external auditory canal and middle ear.

The medical literature on fibrous dysplasia of the temporal bone describes a sequelae that can explain the patient's symptoms without resorting to a diagnosis of CSOM. Fandino *et al.*[3] state:

> Fibrous dysplasia involving the temporal bone is an unusual presentation, with the most common associated finding being conductive hearing loss and stenosis of the external auditory canal (EAC), seen in approximately 8% of patients. Stenosis of the ear canal can result in secondary canal cholesteatoma with extension into the middle ear, leading to conductive hearing loss because of either canal obstruction or erosion/fixation of the ossicular chain.

Likewise, Zhang *et al.*[4] state:

> The most common clinical manifestations of the temporal bone FD were progressive stenosis of the EAC and conductive hearing loss followed by tinnitus and otorrhea. There was no obvious clinical symptom in the early stage of the disease, and the median time delay between the first symptom onset and diagnosis was 54.2 months in this report. The lesion mainly leads to stenosis of the external auditory meatus especially at the osteochondral

junction. Cholesteatoma was the main complication of this disease, which was secondary to occlusion of the EAC with the growth of the lesion. All of the patients we reported have cholesteatoma including 7 cases were limited to the EAC and 4 cases invaded the middle ear.

As such, the medical literature identifies a common sequelae - external auditory canal stenosis, and a SECONDARY cholesteatoma due to occlusion of the ear canal. This type of cholesteatoma is also called an external canal cholesteatoma, albeit secondary to canal obstruction. This same condition can be seen in post-traumatic ear canal stenosis or congenital stenosis (< 4 mm) of the ear canal. It must be noted from the references that erosion of the ossicular chain and invasion of the middle ear can occur from the cholesteatoma. It must also be noted that these references make no mention whatsoever of the development of, or association with chronic suppurative otitis media.

We feel that it is important to make this distinction, not only because of the differences in their pathophysiologic mechanism, but also because of the management implications. Classifying this case as CSOM risks delaying appropriate definitive treatment, as management initially focuses on prolonged antibiotic therapy prior to the acquisition of imaging studies. Surgical management that focuses on addressing infection (mastoidectomy) may fail to address obstruction (meatoplasty, canalplasty), leading to symptom relapse.

In conclusion, accurate diagnosis hinges on recognizing that obstruction of the external auditory canal with keratin accumulation is pathologically distinct from CSOM. Misclassification delays interventions targeting the primary pathology. We commend the authors for reporting this rare case but urge reconsideration of the diagnostic label to guide optimal management.

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Sincerely,

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