Sir,

Arterial ischemic stroke due to otomastoiditis is an uncommon complication that occurs when the locoregional inflammation spreads to the bony canals containing intracranial vessels, resulting in arterial thrombosis and brain ischemia or septic embolic showers to the vascular territory. The prevalence of intracranial complications is four times higher in chronic otitis media (OM) than acute OM owing to its indolent course, which causes a delay in diagnosis and initiating treatment. There is a paucity of literature describing brain arterial infarctions as first-hand complications of OM.

We report a case of a 47-year-old female who presented with intermittent left ear discharge for 2 years and had a history of recurrent left hemispheric strokes with focal neurological deficits. Her blood investigations showed mildly raised fasting blood sugar levels (126 mg/dl) and mildly raised glycosylated hemoglobin (5.8%). On physical examination, she had a foul-smelling left ear discharge, left aural fullness and mild left ear conductive hearing loss. Magnetic resonance imaging showed multiple scattered T2 and T2 FLAIR hyperintensities with no evidence of diffusion restriction [Figure 1a-c], suggestive of previous infarct/gliotic changes in the left high frontoparietal lobes that were limited to the left anterior cerebral/left middle cerebral arterial watershed territory. The lesions were T1 hypointense [Figure 1d] without evidence of blooming or any obvious postcontrast enhancement. MR angiography [Figure 1e and f] did not show flow-related signal in the left internal carotid artery (ICA). However, the anterior communicating artery (ACom) and bilateral posterior communicating arteries (PCom) showed normal flow-related signal (i.e., completing the circle of Willis). The T2 hyperintense signal was in the regions of the left middle ear and left mastoid, suggestive of left-sided OM with mastoiditis. A 5-month-old temporal bone computed tomography (CT) scan was reviewed [Figure 2a-c], which showed soft-tissue opacification of the left middle ear cleft and left mastoid air cells with locoregional bony sclerosis. Soft tissue in the left middle ear cavity was found to have eroded and extended in the posterolateral wall of the left carotid canal, thereby explaining the absence of flow-related signal in MR angiography, suggestive of

![Figure 1: T2-weighted image (a) showing few scattered T2 hyperintense foci in the left high frontal and left high parietal region with corresponding hyperintensity on T2 FLAIR images (b) without diffusion-weighted imaging restriction (c). The lesions appear hypointense on T1-weighted image (d). Magnetic resonance angiography (e and f) showing absence of flow-related signal in the left internal carotid artery; however, the circle of Willis is complete](image-url)
thrombosis/occlusion. Left aural pus culture revealed growth of *Pseudomonas aeruginosa*. At the time of reporting, the patient was on antihyperglycemics (metformin), lifestyle modification regime with proper antibiotic coverage (oral amoxicillin/clavulanic acid along with daily aural toilet) and was regularly followed up by the endocrinology and otolaryngology departments. The patient was free of aural symptoms and had mild weakness in the right upper limb, likely because of the old infarct.

Extracranial complications of OM comprise mastoiditis, subperiosteal abscess, facial paralysis and labyrinthitis, while intracranial complications include cerebral abscess, extradural abscess, lateral sinus thrombophlebitis, meningitis, otitic hydrocephalus and encephalitis.\(^2,3\) Chances of intracranial complications remain high even after an effective antibiotic therapy, with anaerobic bacteria being the most common causative agent. Intracranial complications usually develop quickly and are often diagnosed and dealt with promptly. However, early chronic OM often goes unnoticed, as it is difficult to diagnose and often life-threatening. Therefore, timely diagnosis is the key to prevent intra- and extracranial complications in chronic OM.\(^4\)

CT imaging of temporal bone is highly sensitive (97%) for evaluating the complications of OM.\(^6\) Arterial infarction in otitis externa of diabetic patients is a known complication,\(^6\) but recurrent ischemic stroke as a complication of chronic otomastoiditis has not been described previously. In our case, scattered areas of gliotic changes in the left anterior/middle cerebral watershed arterial territory could be explained by the septic embolic showers. MR angiography had shown absent flow-related signal in the left ICA (explained by spread of locoregional inflammation of OM to the left carotid canal resulting in occlusion), but due to the complete circle of Willis (ACom and bilateral PCom), the patient did not suffer a major episode of stroke. Recurrent ischemic arterial stroke in chronic otomastoiditis is a rare clinical entity. Here, the authors highlight the role of radiology in ruling out the cause of arterial complications and the extent of damage in OM that could help in containing and preventing further complications.

**Declaration of patient consent**
The authors certify that they have obtained all appropriate consent forms from the patient. The patient has given her consent for images and other clinical information to be reported in the Journal. The patient understands that her name and initials will not be published, and due efforts will be made to conceal the identity.

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There are no conflicts of interest.

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