Gradenigo Syndrome in a 14-Year-old Girl as a Consequence of Otitis Media With Effusion

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We report a case of successfully treated Gradenigo syndrome in a 14-year-old girl as a complication of left otitis media with bilateral effusion.

Our patient was admitted to the pediatric unit after review from a local ear nose and throat surgeon with the development of diplopia and intractable headache. This is after a 4-week history of topical antibiotic treatment for otitis media with bilateral effusions, positive for *Streptococcus pyogenes*. Her progressive left-sided unilateral headache with associated ear and maxilla distribution was not responsive analgesia. Her presentation follows a previous left perforated tympanic membrane at age 9 years after acute otitis media.

Clinical examination revealed left sixth cranial nerve palsy with bilateral tympanic perforation without mastoid tenderness. Her vital signs were all within normal limits, and the patient remained afebrile for the duration of her admission. No meningism or other cranial nerve pathology was identified. Audiometry on initial presentation demonstrated mild left-sided conductive hearing loss. MRI brain demonstrated a T2 hyperintense lesion 21 mm × 11 mm × 8 mm located in the left petrous apex with diffusion restriction and ring enhancement post gadolinium (Fig. 1), with loss of bony trabecular pattern and surrounding soft tissue opacification. Dural enhancement was identified around Meckel cave without brain involvement (Fig. 2). A diagnosis of Gradenigo syndrome secondary to *S. pyogenes* was made.

The abducens palsy was noted to resolve within 5 days after commencement of intravenous antibiotics. After 6 weeks of intravenous and oral antibiotic therapy, serial MRI brain imaging demonstrated radiological resolution. Repeat audiometry demonstrated resolution of conductive hearing loss. Our patient remains well after a 12-month clinical follow-up.

DISCUSSION

Gradenigo described the classic clinical triad of diplopia, trigeminal nerve distribution headache, and otorrhoea in 1904, the latter in reference to suppurative otitis media (1). McLaren et al define classic Gradenigo syndrome (GS) in the presence of otitis media, abducens nerve palsy, and trigeminal distribution facial pain, and incomplete GS in the presence of at least 2 of these clinical criteria (2). There must be radiological evidence of petrous apicitis (PA) to reach a diagnosis of either classic or incomplete GS. Based on these criteria, our patient meets the definition for classic GS.

![FIG. 1. Post gadolinium contrast T1 axial MRI-B image 2 —note the T1 hyperintense ring-enhancing lesion at the petrous apex.](image-url)
The clinical features of Gradenigo syndrome result from compression or irritation of the abducens and trigeminal nerves from inflammatory phlegmon. Infiltration may occur from mastoid or sphenoid air cells or lymphovascular tracts to then involve the mucosal lining of the apical petrous air cells of the temporal bone (1,3). The abducens nerve traverses the Dorello canal medial to the petrous apex, making it vulnerable to injury from surrounding edema, inflammation, and/or suppuration (3,4). The trigeminal nerve is also lateral to the petrous apex, and surrounding inflammation contributes to trigeminal nerve-mediated facial pain and headache.

Our patient described persistent and severe pain. Pain of such a character should serve as an indicator that further evaluation with imaging is urgently required to exclude deep-seated infection. Temporal views on contrast-enhanced CT may aid diagnosis (4). Gadolinium-enhanced T1 MRI brain imaging further characterizes ring-enhancing lesions at the petrous apex. Adjacent dural enhancement may further support cranial nerve involvement (4).

There is a predilection of GS for the pediatric population with a median age of 12 years. There seems to be no sex predilection (1,5). Mortality rates in both populations is between 2% and 2.6% (1,2,5). Causative agents are predominantly aerobic and include alpha and beta Hemolytic Streptococcus spp., Escherichia coli, and Staphylococcus (1,2).

Medical management is effective in most cases, with surgical intervention typically reserved for delayed response (1). Complete resolution of symptoms is expected on average after 4 to 6 weeks in the pediatric cohort, as demonstrated in this case (2).

STATEMENT OF AUTHORSHIP
Category 1: a. Conception and design: L. Bonavia and J. Jackson; b. Acquisition of data: L. Bonavia and J. Jackson; c. Analysis and interpretation of data: L. Bonavia and J. Jackson. Category 2: a. Drafting the manuscript: L. Bonavia; b. Revising it for intellectual content: L. Bonavia and J. Jackson. Category 3: a. Final approval of the completed manuscript: L. Bonavia and J. Jackson.

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