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Peripheral Facial Paralysis in Granulomatosis with Polyangiitis: Two Case Reports

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Abstract

Granulomatosis with Polyangiitis (GPA) is a rare systemic autoimmune vasculitis characterized by necrotizing granulomatous inflammation involving small- and medium-sized blood vessels. Although the disease predominantly affects the upper and lower respiratory tracts and the kidneys, otorhinolaryngological manifestations represent a significant component of its clinical spectrum. Otologic symptoms, including chronic otitis media, persistent middle ear effusion, and hearing loss, may constitute early manifestations and, in some cases, precede systemic involvement. Peripheral facial nerve paralysis is an uncommon but clinically significant complication, typically arising in association with middle ear and mastoid inflammation or vasculitic ischemia of the facial nerve.

In this report, we present two patients in whom otologic complaints constituted the initial manifestations of GPA, with peripheral facial paralysis developing during the diagnostic course. Both patients exhibited middle ear effusion, progressive hearing loss, and persistent otologic inflammation prior to the onset of facial paralysis. Laboratory investigations revealed elevated acute-phase reactants and PR3-ANCA positivity, while imaging studies demonstrated mastoid and paranasal sinus involvement. Immunosuppressive therapy, including high-dose corticosteroids and rituximab, led to marked improvement in facial nerve function and overall clinical status.

These cases emphasize that GPA should be considered in the differential diagnosis of patients presenting with refractory serous otitis media, recurrent otologic infections, or unexplained peripheral facial paralysis. Early recognition and prompt initiation of immunosuppressive therapy are essential to prevent irreversible neurological damage and optimize clinical outcomes.

Case Presentations

Case 1: A 23-year-old female patient presented to the Department of Otorhinolaryngology and Head and Neck Surgery at Necmettin Erbakan University Hospital with complaints of frontal headache, nasal obstruction, and a sensation of fullness in both ears. She had previously received treatment for sinusitis at an external medical facility. Otoscopic examination revealed bilateral tympanic membrane bulging with decreased mobility and a dull appearance. Tympanometric evaluation demonstrated bilateral Type B tympanograms, prompting endoscopic nasopharyngeal and laryngeal examination, which revealed no pathological findings. The patient was diagnosed with acute sinusitis and serous otitis media, and medical therapy was initiated. A follow-

up visit was scheduled for three weeks later.

At follow-up, the patient reported progressive hearing deterioration. Otoscopic findings showed reduced otorrhea, while tympanic membrane abnormalities persisted. Pure-tone audiometry revealed airconduction thresholds of 85 dB in the left ear and 78 dB in the right ear, with bone-conduction thresholds of 41 dB and 34 dB, respectively. Bilateral myringotomy was performed, and the patient was hospitalized. Treatment with intravenous prednisolone (1 mg/kg), topical ciprofloxacin–steroid eardrops, ear irrigation with 4% boric acid solution, and oral ciprofloxacin (500 mg) was initiated.

After one week of therapy, repeated audiometric evaluation demonstrated improvement, with air conduction thresholds of 33 dB in the left ear and 55 dB in the right ear, and bone conduction thresholds of 11 dB and 12 dB, respectively. Tympanometry continued to show bilateral Type B curves with negative middle ear pressure (-400 daPa). Due to persistent middle ear effusion, bilateral ventilation tube insertion was performed. An immunodeficiency workup was conducted because of recurrent infections and yielded normal results.

One week later, the patient developed diplopia, fever, and arthralgia. Laboratory analysis revealed markedly elevated inflammatory markers: C-Reactive Protein (CRP) 117 mg/L, Erythrocyte Sedimentation Rate (ESR) 58 mm/h, and rheumatoid factor 593 IU/mL. Lumbar puncture was performed due to suspicion of meningitis; cerebrospinal fluid analysis was within normal limits. Intravenous piperacillin–tazobactam was added to the treatment regimen. Paranasal sinus Computed Tomography (CT) demonstrated erosion of the medial wall of the left maxillary sinus and effusion within the left ethmoid sinus (Figure 1). Temporal bone CT showed fluid accumulation in the middle ear cavity and mastoid air cells (Figure 2). Chest CT revealed cavitary lesions in the right lung.

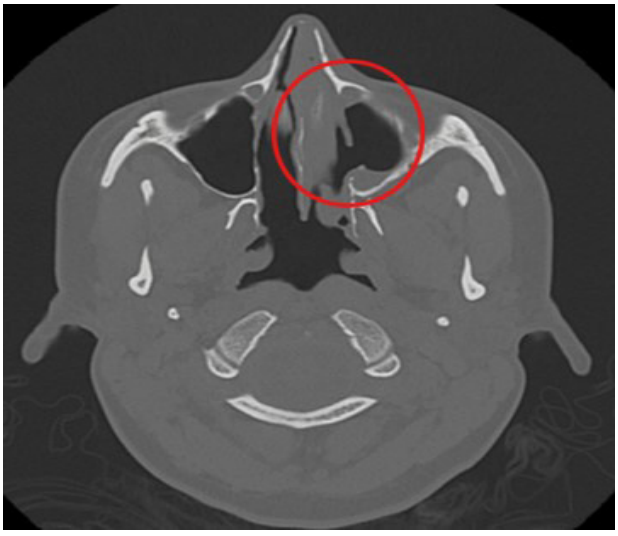

Figure 1: Paranasal sinus CT scan demonstrating left maxillary sinus wall erosion and ethmoid sinus effusion.

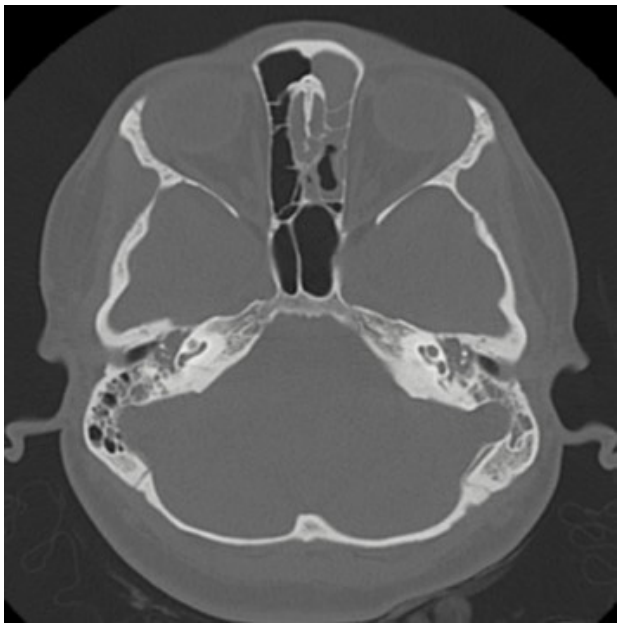

Figure 2: Axial temporal bone CT scan showing middle ear and mastoid effusion.

Following rheumatology consultation and serological testing, PR3-ANCA positivity was detected. Based on histopathological, immunohistochemical, and clinical findings, a diagnosis of Granulomatosis with Polyangiitis (GPA) was established. Renal function tests, including serum urea and creatinine levels, were within normal limits.

The patient was transferred to the Rheumatology Department, where treatment with rituximab and a 1 g pulse corticosteroid was initiated. One day after beginning immunosuppressive therapy, the patient developed left-sided peripheral facial paralysis, graded as House–Brackmann II. Intratympanic dexamethasone injections were administered every other day for a total of three doses.

Neuroimaging revealed no intracranial abnormalities. Inflammatory changes and effusion were observed in both mastoid air cells and middle ear cavities, more pronounced on the left side. Mucosal thickening related to inflammation was also noted in the left maxillary and sphenoid sinuses.

Rituximab therapy was continued during follow-up. After two weeks of treatment, a gradual improvement in facial nerve function was observed. The methylprednisolone dosage was progressively tapered in response to clinical improvement. The patient demonstrated overall clinical recovery and remains under regular follow-up in the Otorhinolaryngology and Rheumatology outpatient clinics.

Case 2: A 54-year-old male patient presented to an external medical center with a three-week history of fullness and pain in the right ear. He was diagnosed with serous otitis media and prescribed medical treatment. During the last ten days, he experienced progressive hearing deterioration in the right ear. One day prior

to admission, he developed right facial asymmetry and was subsequently admitted to the Department of Otorhinolaryngology and Head and Neck Surgery at Necmettin Erbakan University Hospital.

Anterior rhinoscopy and endoscopic examination revealed inflammatory crusting in the anterior portion of the right nasal cavity (Figure 3). Nasopharyngeal and laryngeal examinations were unremarkable. Otoscopic examination demonstrated a bulging, dull right tympanic membrane and a dull-appearing left tympanic membrane.

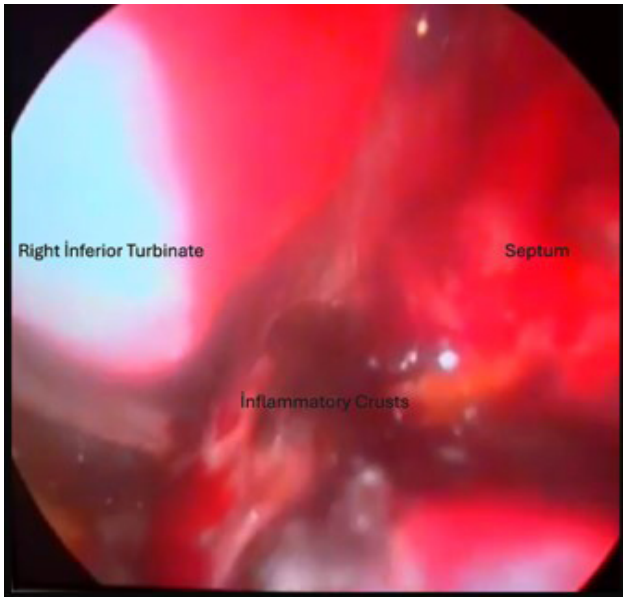

Figure 3: Endoscopic examination of the right nasal cavity, showing inflammatory crusts on the nasal septum.

The patient was diagnosed with right-sided peripheral facial paralysis and acute serous otitis media. Facial paralysis was graded as House–Brackmann III. Laboratory tests showed leukocytosis with a white blood cell count of 11.5 × 10^9/L and an elevated CRP level of 119 mg/L. Audiometric evaluation demonstrated air and bone conduction thresholds of 35 dB and 11 dB in the left ear and 37 dB and 12 dB in the right ear, respectively.

Treatment with intravenous prednisolone at a dosage of 1 mg/kg was initiated in conjunction with standard management for acute serous otitis media (Figure 4, Figure 5). Due to worsening otalgia, bilateral ventilation tube insertion was performed on the seventh day of hospitalization.

A biopsy obtained from the right nasal cavity revealed fibrotic tissue changes with granulomatous structures. Given the patient's history of recurrent otitis media and the current clinical findings, a rheumatology consultation was requested to evaluate possible autoimmune etiology. Serological testing demonstrated PR3-ANCA positivity. Based on clinical, histopathological, and serological findings, the Rheumatology Department established a diagnosis of Granulomatosis with Polyangiitis (GPA), and pulse corticosteroid therapy was initiated.

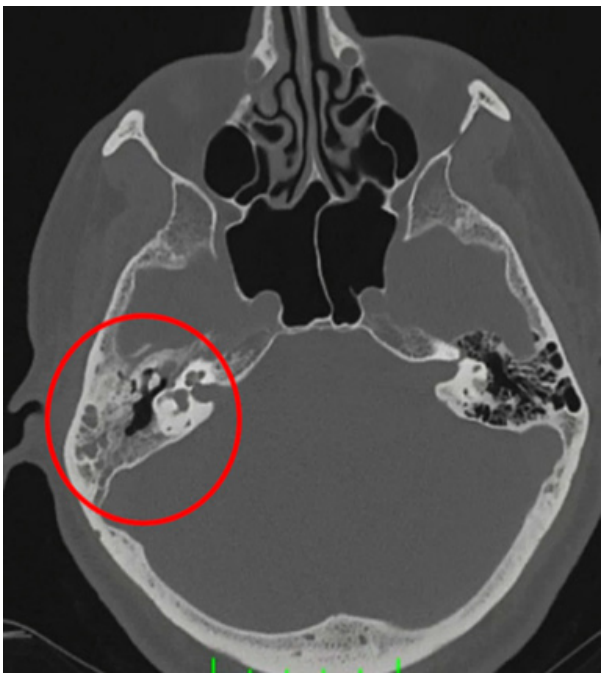

Figure 4: CT imaging showed significantly reduced aeration in the right mastoid air cells and middle ear. Increased density was observed due to mucosal thickening and effusion. No destruction was noted along the facial nerve canal.

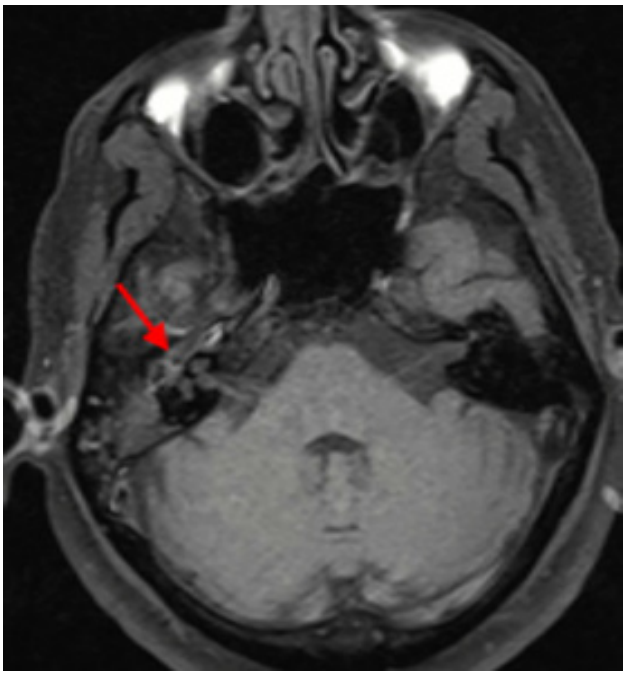

Figure 5: Temporal MRI demonstrated a heterogeneously enhancing lesion in the right middle ear and mastoid region. The lesion appeared to involve the facial nerve pathway at the mastoid segment.

Discussion

Peripheral facial paralysis is an extremely rare neurological manifestation of Granulomatosis with Polyangiitis (GPA), with a reported incidence ranging from 2.5% to 5%, and occasionally reaching 8%–10% in large case series [1,2]. This underscores the heterogeneous and complex clinical presentation of GPA, as

well as the rarity of neurological involvement. In this context, the occurrence of facial paralysis in the presented cases represents an uncommon but clinically relevant complication of GPA.

The Ear, Nose, and Throat (ENT) region is frequently involved in GPA, with otologic manifestations such as serous otitis media, chronic otitis media, and sinusitis reported in 20%–60% of cases. However, progression to facial nerve paralysis remains rare and is mainly documented through isolated case reports [3]. Proposed mechanisms of facial nerve involvement in GPA include: (1) compression due to granulomatous inflammation in the middle ear and mastoid region, (2) ischemic neuropathy resulting from vasculitis of the vasanervorum, and (3) hypertrophic pachymeningitis independent of middle ear pathology [4].

Histopathological studies have demonstrated vasculitic changes in the vasanervorum of the tympanic and mastoid segments of the facial nerve, which may or may not correlate with over clinical symptoms [5]. Chronic otitis media, mastoiditis, and granuloma formation may result in bony erosion and mechanical nerve compression, thereby precipitating facial paralysis [6]. In both presented cases, the response to immunosuppressive therapy was rapid and substantial, suggesting that ischemic injury secondary to vasculitis played a dominant role in the pathogenesis. This observation is consistent with previous reports emphasizing the inflammatory–ischemic mechanism in similar clinical scenarios [4]. Although rare, surgical facial nerve decompression has been described in cases involving significant granulomatous compression unresponsive to medical therapy.

Facial paralysis in GPA typically occurs in conjunction with ENT manifestations and only rarely represents the initial presentation. In our cases, a history of chronic otitis media, sinusitis, and hearing loss preceded the onset of facial paralysis, mirroring diagnostic challenges reported in the literature. For example, Lee et al. described a case of GPA initially presenting with acute otitis media followed by bilateral facial paralysis, while Roszkowska et al. reported a misdiagnosed case requiring surgical intervention before the correct diagnosis was established.

Bilateral facial nerve involvement is exceptionally rare in GPA, with an incidence of approximately 2% even in large patient series [4]. One of our patients exhibited bilateral clinical features, further supporting this observation. Immunosuppressive therapy remains the cornerstone of GPA management. High-dose corticosteroids in combination with cyclophosphamide or rituximab represent standard induction therapy [6]. Rituximab has demonstrated comparable efficacy to cyclophosphamide, particularly in cases with renal involvement or refractory disease [7]. Both of our patients responded favorably to high-dose corticosteroids and rituximab, achieving substantial recovery of facial nerve function. This highlights the importance of early and aggressive immunosuppressive treatment in preventing permanent neurological sequelae.

Adjunctive otorhinolaryngological interventions may also be required. Ventilation tube placement is commonly employed to alleviate middle ear effusion and to prevent conductive hearing loss or secondary nerve compression. In our second case, bilateral ventilation tube insertion contributed to symptomatic control. Although surgical intervention may be indicated in selected cases with extensive mastoid destruction, most authors emphasize that immunosuppressive therapy should remain the primary treatment strategy [4,6]. Our cases further support this perspective, as both patients achieved clinical resolution without the need for surgical decompression.

Conclusion

The two presented cases demonstrate clinical characteristics, treatment approaches, and therapeutic responses that are largely consistent with those reported in the existing literature. This case series highlights that, although peripheral facial paralysis is a rare manifestation of Granulomatosis with Polyangiitis (GPA), it represents a clinically significant condition that should be promptly recognized. With appropriate and timely management, substantial recovery of facial nerve function can be achieved. Furthermore, these cases underscore the importance of considering GPA in patients presenting with persistent middle ear infections and unexplained facial paralysis.

In patients with peripheral facial paralysis accompanied by a typical or unexpected otorhinolaryngological symptoms, underlying rheumatologic diseases should also be suspected. These observations contribute valuable insights to the current literature.

Authors' Contributions

Concept – Hamdi Arbag, Rukiye Ozcelik Erdem; Design – Ahmet Talha Demir, Rukiye Ozcelik Erdem; Supervision – Hamdi Arbag, Mehmet Akif Dundar; Materials – Hamdi Arbag, Rukiye Ozcelik Erdem; Data Collection and/or Processing – Ahmet Talha Demir; Analysis and/or Interpretation – Hamdi Arbag, Mehmet Akif Dundar; Literature Search – Ahmet Talha Demir; Writing – Ahmet Talha Demir; Critical Review – Rukiye Ozcelik Erdem, Mehmet Akif Dundar.

Financial Disclosure

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Informed Consent

Written informed consent was obtained from all patients.

Conflict of interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article. Informed consent was obtained for this publication.

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