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BILATERAL PERIPHERAL FACIAL PARALYSIS IN GRANULOMATOSIS WITH POLYNGITIS: CASE REPORT

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BACKGROUND

Granulomatosis with polyangiitis (GPA) or Wegener's granulomatosis is a systemic, immune-mediated disease with anti-neutrophil cytoplasmic antibody (cANCA) in approximately 90% of cases. It is characterized by necrotizing granulomatous vasculitis, of small and medium vessels, mainly affects the upper and lower airways and the kidneys, generating numerous spectra of clinical presentation according to the affected site. Unilateral peripheral facial paralysis secondary to inflammatory otomastoiditis occurs in up to 10% of the patients; however, the cases described in the literature of simultaneous bilateral involvement in an activation of the disease are rare.

CASE REPORT

Patient, female, 34 years old, previously healthy, started in July 2018 with otalgia and bilateral otorrhea with progressive worsening, refractory to several antibiotic schemes, evolving after 4 months with hypoacusis, bilateral intense pulsatile otalgia and peripheral facial paralysis, being hospitalized for investigation on May 17, 2019. At admission, the patient was in regular general condition, eupneic, febrile (38.5 °C), acyanotic and anicteric. Physical examination: two deep hard palate ulcers, nasal obstruction, bilateral peripheral facial paralysis, pain on palpation of the mastoid bilaterally. The exams showed leukocytosis, VHS 1st hour (80 mm); PCR (235 mg/L) and, in summary, proteinuria, cylindruria and erythrocyte dysmorphism; without elevation of nitrogenous slags or reduction in glomerular filtration rate, in addition to c-ANCA: 1:40. Computed tomography of the skull: bilateral acute inflammatory otomastoiditis and acute sphenoid sinusitis (Fig. 1), and chest CT: pulmonary nodule with attenuation of soft tissues in the left hemithorax. Electroneuromyography demonstrated motor axonal lesion of the bilateral facial nerve, without involvement of other nerves, confirming the existence of bilateral peripheral facial paralysis (Fig. 2). The patient met GPA criteria for presenting 3 of the 4 American College of Rheumatology criteria for GPA. She received treatment with prednisone 80 mg/day, followed by pulse therapy with methylprednisolone (1 g/day, 3 days) followed by cyclophosphamide 15 mg/kg, according to the guidelines of the Consensus of Vasculitis of the Brazilian Society of Rheumatology. She presented total improvement of otalgia, and in progressive improvement of hearing loss. The patient remains in follow up and in continuity of treatment.

Figure 1. Skull tomography, bilateral otomastoiditis (green arrows) in the coronal (A) and axial (B) sections.

Figure 2. Facial nerve conduction study with bilateral absent potential. Chronic denervation and reinnervation potentials were seen without spontaneous activity at electromyography.

CONCLUSION

Facial nerve involvement usually occurs through vasculitis of the vasa nervorum or by contiguity of the inflammatory process of the mastoid, generating lesion ipsilateral to the inflammatory process. However, bilateral peripheral facial paralysis, during the same flare of vasculitis, is a rare event, with few cases described in the literature, therefore of relevance for medical knowledge.