

Recurrent Peripheral Facial Paralysis Revealing Behçet's Disease: A Case Report

Abstract

We report the case of a 21-year-old female patient presenting with recurrent peripheral facial paralysis associated with oropharyngeal aphthosis and diffuse arthralgia. Immunological testing revealed HLA-B51 positivity, suggesting Behçet's disease. The patient's condition improved with a combination of corticosteroid therapy, colchicine, functional rehabilitation, and symptomatic measures.

Key Words

Behçet's disease – Recurrent facial paralysis – Neuro-Behçet – HLA-B51

Introduction

Behçet's disease (BD) is a chronic, relapsing, multisystem inflammatory disorder characterized by recurrent oral and genital ulcers, ocular involvement, and variable systemic manifestations. It is classified among the variable vessel vasculitides, as it can affect both arteries and veins of all calibers. Although the exact etiology remains unclear, an interaction between genetic predisposition particularly HLA-B51 positivity and environmental or infectious triggers is suspected. Neurological involvement, termed *neuro-Behçet's disease*, occurs in approximately 5–10% of cases and represents one of the most severe complications. It can manifest as parenchymal lesions involving the brainstem or as vascular complications such as cerebral venous thrombosis. Peripheral nervous system involvement, however, remains exceptional. Peripheral facial paralysis is a common neurological disorder, most often idiopathic or post-viral in origin. Its recurrence should prompt investigation for an underlying systemic disease. We report here an unusual presentation of Behçet's disease revealed by recurrent peripheral facial paralysis in a young woman, emphasizing the importance of multidisciplinary evaluation and early recognition of atypical forms.

Case Report

Ms. M. M., a 21-year-old woman with no significant medical history, experienced a right-sided peripheral facial paralysis three months earlier, which had been attributed to idiopathic (cold-induced) Bell's palsy. Her condition had improved under corticosteroid therapy and physical rehabilitation. Three months later, she presented again with a recurrence of the same facial deficit. Clinical examination revealed a complete right-sided peripheral facial paralysis (House-Brackmann grade IV), with no involvement of other cranial nerves. ENT examination identified a single aphthous ulcer located on the right tonsillar fossa. The patient

also reported migratory polyarthralgia, mainly affecting the knees and wrists, without swelling or local inflammatory signs.

Laboratory workup showed: normal CBC, erythrocyte sedimentation rate (ESR) of 28 mm in the first hour, nearly normal C-reactive protein (CRP), and positive HLA-B51. Brain MRI was strictly normal, showing no brainstem involvement or inflammatory lesions. Ophthalmologic examination revealed no uveitis or retinal involvement.

Discussion

Peripheral facial paralysis is one of the most common reasons for ENT consultations. It is usually idiopathic or post-viral. However, recurrence of such an episode within a short period in a young patient should prompt consideration of a systemic etiology. In this case, the presence of oropharyngeal aphthosis, polyarthralgia, and HLA-B51 positivity led to the diagnosis of Behçet's disease. This chronic systemic vasculitis predominantly affects young adults and is classically characterized by the triad of oral aphthosis, genital aphthosis, and uveitis. Neurological involvement in Behçet's disease is rare but severe, occurring in approximately 5–10% of cases, and may be either parenchymal or vascular. In our observation, the absence of brain MRI lesions ruled out central nervous system involvement but suggested inflammatory peripheral damage to the facial nerve. This case highlights the importance of a comprehensive approach and multidisciplinary follow-up in patients presenting with recurrent facial paralysis.

The patient was treated with colchicine (1 mg/day) to control systemic inflammation, low-dose oral corticosteroids to reduce facial nerve edema, vitamin B12 to support nerve regeneration, and artificial tears for ocular protection. Functional rehabilitation sessions were prescribed to accelerate recovery of facial motility. Clinical evolution was favorable after two weeks, with progressive recovery of facial movement and disappearance of joint pain. No recurrence was observed after three months of follow-up.

Conclusion

Behçet's disease should be considered in any case of recurrent facial paralysis associated with oral aphthosis. Early recognition of this etiology allows for targeted management and prevention of severe neurological or ocular complications. This case underscores the importance of a holistic clinical approach and close collaboration between ENT specialists, internists, and ophthalmologists in the management of such atypical presentations.

References

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