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BILATERAL PERIPHERAL FACIAL NERVE PALSY: A RARE CLINICAL PICTURE

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ABSTRACT

Introduction: Bilateral peripheral facial palsy (BPFP) is a rare clinical entity often associated with systemic, infectious, or neurological diseases.

Case report: We present a 65-year-old male who developed a severe (House-Brackmann Scale grade VI) sequential BPFP within two weeks, accompanied by hoarseness. His history was remarkable for Crohn's disease in remission, a remote history of relapsing thrombotic thrombocytopenic purpura (TTP), nephrectomy for renal cell carcinoma and currently type 2 diabetes mellitus on insulin. Upon presentation, magnetic resonance imaging of the brain showed contrast enhancement of the facial nerves bilaterally, corresponding to a neuritis facialis, without any signs of stroke or demyelination. The basic laboratory blood tests as well as the cerebrospinal fluid analysis was unremarkable. Extensive laboratory testing showed no signs of infectious and autoimmune causes (Lyme disease, herpes infections, Epstein-Barr virus, human immunodeficiency virus) and no clinical signs of vasculitis, Guillain-Barré syndrome, sarcoidosis, a relapse of TTP or malignancy. Treatment with prednisolone (1 mg/kg body weight) led to clinical improvement (three weeks later House-Brackmann Scale II). The hoarseness disappeared after about three weeks.

Conclusions: BPFP is a rare condition requiring a broad differential diagnosis and systematic evaluation. In this patient, type 2 diabetes represented a recognized risk factor, while concomitant hoarseness suggested a possible viral aetiology. Although an association of BPFP with prior TTP has not been reported and with Crohn's disease is exceptional, a shared pathophysiological mechanism cannot be excluded..

KEYWORDS

Peripheral facial palsy, bilateral facial nerve palsy, diabetes mellitus, neuritis cranialis, Bells' palsy

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LEARNING POINTS

- We describe a case of bilateral facial nerve palsy in a patient with history of thrombotic thrombocytopenic purpura and Crohn' s disease.
- Bilateral facial nerve palsy is a rare neurological condition with a broad differential diagnosis.
- A detailed patient history and a stepwise diagnostic work-up are essential for identifying underlying systemic or neurological causes.

INTRODUCTION

Bilateral peripheral facial nerve palsy (BPFP) is a rare neurological presentation, accounting for less than 1% of all facial nerve palsies[1]. In contrast to the far more common unilateral variant, BPFP may signify an underlying systemic condition and warrants comprehensive investigation. Aetiologies include infectious diseases (e.g., Lyme disease, human immunodeficiency virus), autoimmune disorders (e.g., Guillain-Barré syndrome, sarcoidosis), neoplasms, and demyelinating conditions such as multiple sclerosis or neuromyelitis optica spectrum disorders[2,3].

In this report, we describe a patient with a complex medical background who developed a severe sequential BPFP due to bilateral neuritis facialis. After extensive diagnostic work-up, we concluded the aetiology was idiopathic, and the symptoms improved on glucocorticoid therapy.

CASE DESCRIPTION

A 65-year-old man presented initially on January 5, 2024, at an emergency department in Serbia with symptoms of left facial droop, eye closure difficulty, and numbness. A cranial computed tomography scan performed there showed no evidence of brain ischemia or haemorrhage. Upon return to Switzerland, he was evaluated on January 11, 2024 ,at the Emergency Department of our hospital. Complete left-sided idiopathic facial palsy was diagnosed (House-Brackmann Scale VI). Due to his presentation at our institution only 6 days after onset and concerns regarding his insulin-dependent type 2 diabetes mellitus, we refrained from glucocorticoid therapy at that stage, and the patient was dismissed with symptomatic treatment.

On re-presentation at our Emergency Department on January 19, 2024, symptoms had progressed, now involving the right side of the face, along with new-onset hoarseness. Neurological examination revealed bilateral peripheral facial palsy with House-Brackmann grade IV on the left (improvement since January 11) and new palsy grade IV on the right *(Video 1)*. Cranial nerves II–XII were otherwise intact. No limb weakness, cerebellar signs, or sensory levels were observed. Mild anisocoria without visual complaints had resolved by the time of evaluation.

The patient's past medical history included insulin-dependent type 2 diabetes mellitus with mild peripheral polyneuropathy as a complication, metabolic syndrome, arterial hypertension, presently inactive Crohn's disease (with ulcerative colitis pattern), a remote history of thrombotic thrombocytopenic purpura (TTP) with five previous episodes—the last in 2005—and status post right nephrectomy for renal cell carcinoma in 2002. The patient had a remote history of fully reversible neurological episodes (sensomotor hemiparesis in the early 1980s, vestibular symptoms in 1990 and 2012).

Magnetic resonance imaging (MRI) of the brain performed on January 15, 2024, showed contrast enhancement of the bilateral facial nerves, consistent with neuritis *(Fig. 1)*, along with punctate microangiopathic changes in the frontal lobes *(Fig. 2)*. There was no evidence of mass effect, ischemia, or hydrocephalus or demyelination. No other cranial nerves were contrast enhanced. The new

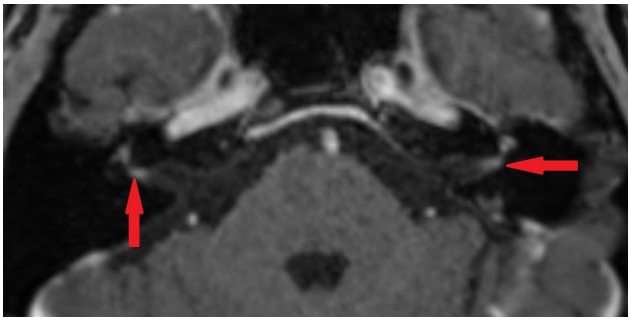


Figure 1. *Brain magnetic resonance imaging (T1 VIBE post-contrast): Bilateral enhancement of the facial nerve within the fundus of the internal auditory canal and the labyrinthine segment (arrows), consistent with bilateral facial neuritis.*

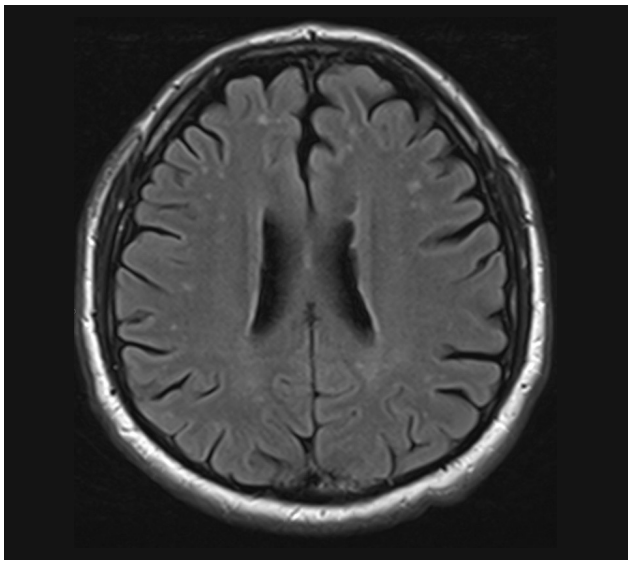
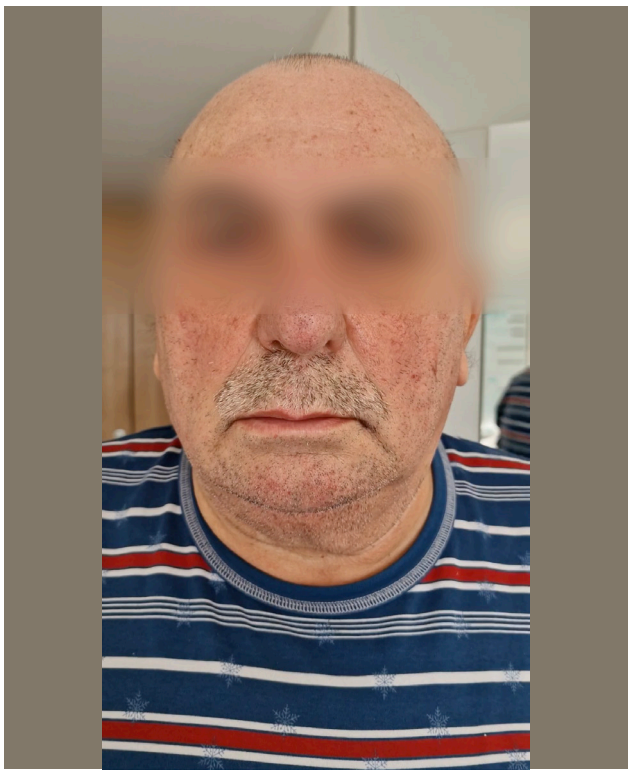


Figure 2. *Brain magnetic resonance imaging (T2 FLAIR): Punctate microangiopathic white matter changes, subcortical, bilaterally.*



Video 1. Video demonstrating the facial movements of the patient with bilateral facial nerve palsy. The examiner asks first to smile, then to whistle, then to raise the eyebrows and finally to close the eyes. The voice of the patient exhibits a newly onset hoarseness. https://youtu.be/bJwSmy7xfdk

hoarseness could indicate involvement of the vagus nerve, however, the ear-nose-throat examination revealed physiological findings and there was no sign of inflammation of the vagus nerve on the cranial MRI. A lumbar puncture revealed clear cerebrospinal fluid with normal glucose and protein levels and no pleocytosis. Cultures and polymerase chain reaction for neurotropic pathogens were negative. Extensive serological tests, including Borrelia, human immunodeficiency virus, Epstein-Barr virus, and syphilis, were negative. Autoimmune screening including antinuclear- and anti-neutrophil cytoplasmic antibodies, angiotensin-converting enzyme (ACE), and lupus anticoagulants were also unremarkable.

A 5-day course of high-dose oral methylprednisolone (80 mg/day) was initiated. The patient noted gradual improvement in eye closure and speech. On discharge, he was referred to speech therapy and received ophthalmological instructions, including eye protection with vitamin A ointment and a moisture chamber.

At follow-up neurological examinations on January 30 and February 15, 2024, further improvements were noted with eventually a House-Brackmann grade II facial weakness on the left and minimal residual upper lip weakness on the right.

DISCUSSION

BPFP is a rare but clinically significant condition[1]. In contrast to unilateral facial palsy, which is idiopathic in the majority of cases, BPFP usually mandates a comprehensive evaluation due to its association with underlying systemic, infectious, or inflammatory disorders[1,3].

Idiopathic facial palsy (Bell's palsy) is generally considered an immune-mediated inflammatory neuritis. The prevailing hypothesis suggests that a reactivation of a latent virus—most commonly herpes simplex virus type 1 —triggers an inflammatory response, resulting in nerve oedema and ischemia within the narrow confines of the bony facial canal[2,3].

While this mechanism is common in unilateral cases, the frequency of idiopathic bilateral involvement is extraordinarily low, occurring in only about 1% of patients diagnosed with Bell's palsy. In our patient, the sequential involvement was accompanied by hoarseness. Although this clinically suggested potential involvement of the vagus nerve, contrast-enhanced MRI showed neuritis of the facial nerves exclusively. In the absence of radiological evidence for vagus neuritis, the hoarseness may have been a manifestation of a concurrent viral upper respiratory infection, further supporting a viral trigger for the facial nerve inflammation.

Guillain–Barré syndrome (GBS), particularly the facial diplegia variant, represents a critical differential diagnosis in BPFP[4,5]. In this case, the absence of limb weakness, sensory deficits or areflexia, and a normal cerebrospinal fluid profile argued strongly against GBS. Furthermore, the prompt response to corticosteroids is untypical for GBS, where steroids are generally considered ineffective[4]. Similarly, neurosarcoidosis and Melkersson–Rosenthal syndrome seemed improbable due to normal ACE levels, chest imaging, and the absence of orofacial oedema. The absence of acute lesions on MRI and the normal cerebrospinal fluid profile argue against an acute central demyelinating event[6].

Significant comorbidities of our patient include the remote history of TTP, Crohn' s disease in remission, state after renal carcinoma and type 2 diabetes mellitus. Of these, only diabetes mellitus is a recognized risk factor for cranial mononeuropathies and BPFP; here, it may have precipitated the development of BPFP by impairing microvascular repair or amplifying inflammatory nerve injury due to a potential viral insult. There are no known cases of BPFP in patients with history of TTP or renal carcinoma. In Crohn's disease, one case of BPFP due to chronic inflammatory demyelinating polyneuropathy in a patient treated with adalimumab was described[7].

The clinical presentation is thus most consistent with a primary peripheral inflammatory event of unknown origin. The marked improvement of both facial palsy and hoarseness following high-dose corticosteroid therapy reinforces the diagnosis of an immune-mediated neuritis[2,3].

In conclusion, BPFP represents a diagnostically challenging clinical situation. A structured diagnostic pathway is essential to exclude underlying systemic diseases and guide effective anti-inflammatory treatment. In our

case, a combination of microvascular and immunological predisposition (diabetes, Crohn's disease and history of TTP) and a presumed viral trigger might have been involved in the development of the bilateral neuritis facialis leading to BPFP.

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