

Guillain Barre syndrome with peripheral facial paralysis: case report

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ABSTRACT

Facial nerve paralysis is a common clinical condition. It is mostly located unilaterally. A 51 year old patient who presented with unilateral peripheral facial nerve paralysis due to the Guillain-Barré Syndrome is presented.

Keywords: Unilateral peripheral facial nerve paralysis, Guillain-Barre Sendrome.

INTRODUCTION

Facial nerve paralysis is one of the clinical manifestations that is accepted to develop due to unilateral ischemic, infectious or inflammatory causes of the facial nerve or as a result of compression of the facial nerve in the fallopian canal. Etiology of facial nerve paralysis can often be determined and it is a symptom of a serious underlying disease and therefore requires detailed examination and immediate treatment [1]. In this case report, we present a patient who applied to us with symptoms of peripheral facial paralysis (PFP) and was subsequently diagnosed with Guillain Barre syndrome (GBS) with EMG and CSF protein elevation.

CASE PRESENTATION

A 51-year-old female patient complained of common body pains. The patient's body pains started 3 weeks ago and she had antibiotic treatment considering an infection. but the patient did not show clinical improvement. Then 5 days later PFP occurred in the patient. Prednisolone treatment was started at an external center. After ten days of prednisolone, minimal improvement

in complaints was observed. It has been referred to us for further examination. When we examined the patient, no pathology was observed except for the right PFP in the neurological examination and the patient's electromyography (EMG) was found to be compatible with sensorimotor mixed type polyneuropathic involvement. CSF protein resulted as 74. At the next control examination, the patient had difficulty walking, paresthesia in the hands and feet, and the Achilles reflex could not be obtained bilaterally. The patient was admitted to the neurology service ward with a preliminary diagnosis of GBS. A positive response was obtained with early initiation of intravenous immunoglobulin infusion (IVIG) treatment.

DISCUSSION

Peripheral facial nerve paralysis is one of the most common neuropathies. Guillain-Barre Syndrome is an autoimmune, acute and often severe and fulminant polyradiculopathy triggered by a previous bacterial or viral infection. The most common and earliest symptom of GBS is paresthesia. But the main finding is the symmetrical weakness that occurs in

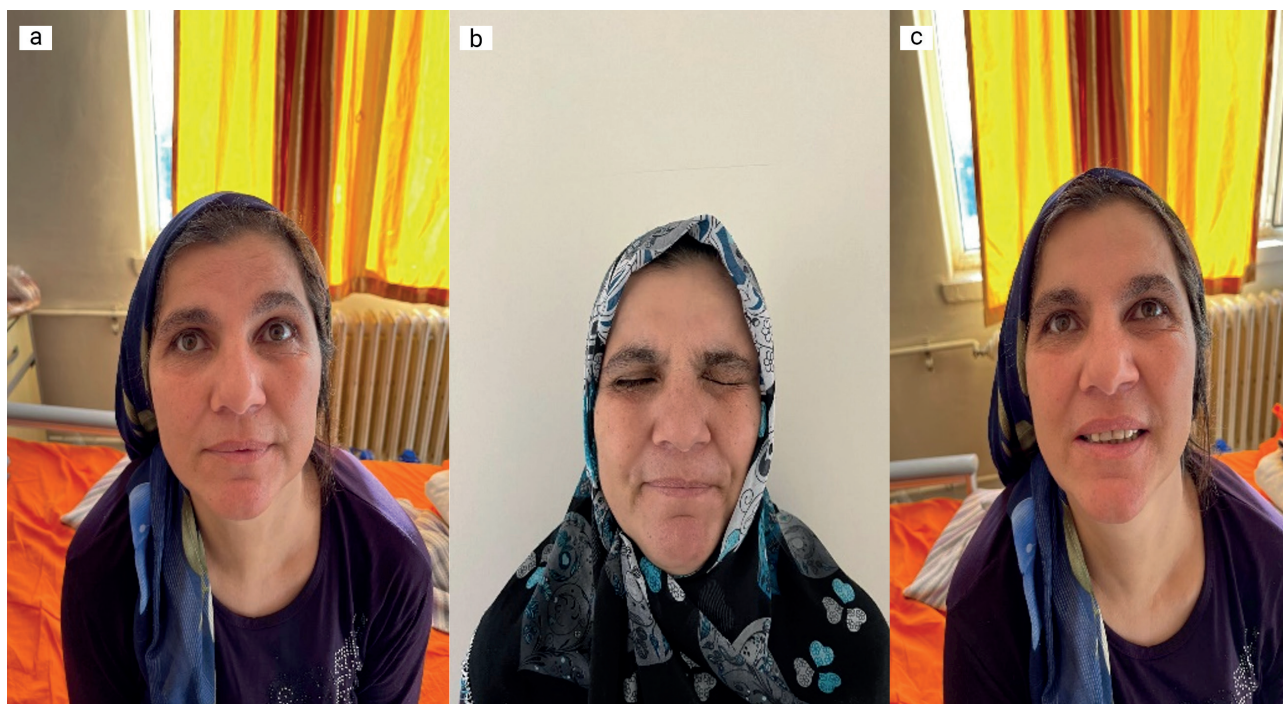


Figure 1. a) The patient cannot raise his right eyebrow. b) In this photo, the patient cannot close his right eye completely. c) The right nasolabial groove is faintly observed.

the early stages of the syndrome. Both distal and proximal muscles may be involved, usually distal. After involvement of the distal muscles in the lower extremity, the involvement may progress to the intercostal muscles, leading to respiratory failure and the need for mechanical ventilation. Although cranial nerve involvement is less common in the syndrome, the majority are bilateral. The frequency of facial nerve paralysis varies between 25-55% and the frequency of oculomotor paresis varies between 5-13% [1]. Other cranial nerve involvements may be seen, although rare, and may also be accompanied by dysarthria and dysphagia. Some patients have distinct and persistent clinical variants of GBS that do not progress to the classic pattern of sensory loss and weakness [2]. These variants include:

- Weakness without sensory symptoms (pure motor variant)
- Weakness limited to cranial nerves (bilateral facial paralysis with paresthesia)
- Upper extremities (pharyngeal-cervical-brachial weakness)
- Lower extremities (paraparetic variant)
- Miller Fisher syndrome with ophthalmoplegia, areflexia, and ataxia [3].

In general, GBS variants are rarely isolated, often partially overlapping with the classical syndrome or showing features characteristic of other variant forms. Symptoms usually begin 3-4 weeks after the viral infection. As in our patient, in addition to electrophysiological tests, CSF analysis shows protein increase without specific cell increase for GBS. It is usually treated with IVIG and plasmapheresis. The prognosis is generally good. Our patient was considered to have GBS, which is a weakness group limited to cranial nerves. The diagnosis of our patient was confirmed with the positive response to the treatment that was started early and the patient healed clinically [2].

CONCLUSION

In patients with spontaneous peripheral facial nerve paralysis taking accurate anamnesis and performing physical examination with wide range of laboratory and radiological imaging are necessary to explained the etiology, and it is necessary to keep the diagnosis of GBS in mind in patients presenting with peripheral facial nerve paralysis.

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Conflict of interest

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