Bilateral Peripheral Facial Paralysis in a Pregnant Patient Admitted to Emergency Service: A case of Guillain-Barre Syndrome

Acil Servise Başvuran Gebede Bilateral Periferik Fasiyal Paralizi: Guillain Barre Sendromu

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Abstract
Unilateral facial paralysis is frequently seen in emergency services but bilateral facial paralysis (BFP) is rare. The most common causes of bilateral facial paralysis are Guillain Barre syndrome, Lyme disease, Bell paralysis, skull fracture, moebius, multiple sclerosis, and infectious mononucleosis. Guillain Barre Syndrome is a demyelinating polyneuropathy which is associated with acute infection. The majority of cases present with symmetric muscle weakness and areflexia. Guillain Barre Syndrome can be diagnosed by clinical findings together with lumbar puncture and/or electrodiagnostic results. It is a rare disease in pregnancy. Plasma exchange or gamma globulin treatment is the preferred treatment. In this report, we present the case of a 20-week pregnant woman who presented with bilateral facial paralysis and was eventually diagnosed with Guillain Barre syndrome.

Keywords: Guillain Barre Syndrome; Pregnant; Bilateral Facial Nervous Paralysis.
INTRODUCTION

Guillain-Barre syndrome is an acute inflammatory demyelinating nerve disease with an incidence rate of 1-2 in 100,000 in the population. It is more common in men than females by 1.5-2 times (1). Retrospective studies have shown that it has increased incidence within the first 4 weeks of the the postpartum period although it has a similar incidence rate in the general population and pregnant women (2).

Cases with GBS usually show symptoms around 1-3 weeks after gastrointestinal tract or respiratory tract infections. Lower limbs are usually more affected than upper extremities. The disease usually begins with numbness in both lower extremities and progresses with growing loss of strength in the following hours and days later (3).

CASE REPORT

A 30-year-old 5-month pregnant patient was admitted to the emergency department with complaints of weakness and inability to close both eyelids in the last 2 days. We learnt that the patient had had diarrhea for a few days about one month ago. Two weeks before her admission, she had suffered from the onset of back pain and numbness in both hands and feet. She had body-wide weakness and could not walk the stairs. She consulted another physicians for her inability to close both eyelids two days ago and was referred to our hospital with normal audiometric test results for BFP etiology. On admission, she did not have any infection-related complaints.

In neurologic examination, she was conscious, oriented, and cooperative. She had isochoric pupils with a light reflex of + / + and corneal reflex of + / +, respectively. Both upper extremity had a muscle strength of 5/5 while both lower limbs had a distal muscle strength of 4/5 and a proximal muscle strength of 4/5. Deep tendon reflexes in the upper extremities were normoactive; these reflexes were hypoactive in the lower extremities. The patient had ataxic gait. Apart from this, other system examination results were normal. The complete blood count, C-reactive protein, liver and kidney function tests were within the normal range except for a 14000/mm³ value of white blood cell count in the urinalysis.

The patient was suggested to undergo a diagnostic lumbar puncture for central nervous system infection but the patient refrained from this application. The electroneuromyography (ENMG) showed extension along the distal latency as observed in the bilateral median and ulnar nerves in both upper extremities and in the tibial and peroneal nerves in the right lower extremity and decrease in the amplitude of the combined floor action potential. The motor conduction velocity of the patient was normal. Sensory conduction velocity and amplitude of sensory nerve action potentials were normal in the bilateral median and ulnar nerves in both upper extremities and the sural nerves in the right lower extremity. We detected prolongation in the F latency of the nerves in both upper limbs and the right lower extremity. The ENMG conducted also revealed signs of motor polyneuropathy accompanied by axonal degeneration. The patient was given 25 g/day intravenous immunoglobulin (IVIG) therapy. She was discharged after a five-day follow-up period due to the decline of her symptoms.

DISCUSSION

0.3-2% of facial paralysis cases have bilateral facial nerve paralysis (4). Viral infections, hyper-coagulability, hypertension, and immunosuppression factors are among the causes of facial paralysis during pregnancy (5). Guillain-Barre syndrome is an acute autoimmune-induced polyneuropathy often rooted in a bacterial or viral infection. The earliest and most common symptom is tingling sensation. The most important finding of the syndrome is usually symmetrical power loss developing in the early stages. Cranial nerve involvement is less common at the beginning but bilateral facial nerve paralysis (5-5%) and oculomotor palsy (5-13%) usually accompany the symptoms (6). 40% of the patients have a history of respiratory tract infection while 20% of the patients share a history of gastroenteritis about a month before the onset of the symptoms (7). The most common infectious agents are campylobacter jejuni by 26% and cytomegalovirus by 13%. Our patient, too, had diarrhoea about 1 month ago and numbness in both hands and feet as well as back pain 2 weeks after the diarrhea. These were followed by loss of strength inability to walk the stairs, and, for the last 2 days, inability to close both eyelids.

Early diagnosis is very important for the treatment. IVIG and plasmapheresis are among the treatment methods used in GBS. IVIG and plasmapheresis administration have been shown to be equally effective in the prevention of progress of neurological symptoms (8). With its easy applicability, IVIG comes to the forefront as the better option. Affecting all components of the immune system, IVIG provides Fc receptor blockade, inhibition of complement activation, suppression of cytokines, adhesion molecules and chemokines, T cell activation and differentiation, and regulation of effector functions (9).

Bilateral facial nerve paralysis is very rare. An accurate and detailed patient history, complete physical examination, and laboratory investigations are required in order to elucidate the etiologies. In conclusion, GBS should be kept in mind while evaluating patients presenting with bilateral facial nerve paralysis.

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REFERENCES


