Bilateral simultaneous facial palsy is a rare clinical entity that can result from various etiologies and thus representing a diagnostic challenge. Myasthenia gravis (MG) is an autoimmune disorder usually caused by antibodies directed towards the skeletal muscle nicotinic acetylcholine receptor that lead to a compromise of neuromuscular transmission. Facial palsy is common in MG, but usually occurs together with ocular involvement. The involvement of facial muscles as a presenting feature without ocular weakness is extremely rare.

Case Report

A 36-year-old woman presented with a 1-year history of difficulty in facial expression. Her medical history was unremarkable. Neurological examination showed bilateral facial weakness with lagophthalmos. The patient could not whistle or blow her cheeks. Mild dysarthria was present. Limitation of extraocular muscle and ptosis were not observed. The patient had no problems with tearing, taste, and hearing. Limb muscle strength, sensory examination, and deep tendon reflexes were normal. Pathologic reflexes, cerebellar ataxia, and autonomic dysfunction were not observed. Brain MRI and cerebrospinal fluid examination were normal. There were neither clinical nor laboratory findings of systemic disease. Blink reflex test showed no response, but facial nerve stimulation demonstrated normal distal latency and bilateral reduced amplitude of compound muscle action potential. Repetitive nerve stimulation test revealed a decremental response in the orbicularis oculi and trapezius muscles (Fig. 1). Pharmacologic test with neostigmine showed improvement in facial weakness. Serum acetylcholine receptor antibody titer was significantly increased at 7.905 nmol/L (normal < 0.2). A diagnosis of MG was made based on serological, pharmacological and electrophysiological results. Chest CT was normal. The patient had moderate improvement in bilateral facial weakness and speech on oral pyridostigmine. Diplopia and ptosis were absent on consecutive examination for a year follow-up with pyridostigmine therapy.
Figure 1. The results of repetitive nerve stimulation test and facial nerve conduction study. The repetitive nerve stimulation at 2 Hz recording over the orbicularis oculi (left) and trapezius (right) muscles showed decremental response (A). Facial nerve conduction study showed normal distal latency and bilateral reduced amplitude of compound muscle action potentials (B).

Discussion

Bilateral simultaneous facial palsy is a rare neurological manifestation of diverse causes such as Lyme disease, Guillain-Barré syndrome, trauma, and sarcoidosis.\textsuperscript{5} Diagnostic workup for a patient presenting with bilateral facial palsy depends on the history. The history should include time sequence of onset, prior history of facial palsy, recent viral or upper respiratory infection, otological symptoms, change in taste, or recent immunization. The first priority in the workup is to rule out a life-threatening disease such as leukemia or Guillain-Barré syndrome. However, to our knowledge, there are a few reports of facial diplegia as the only manifestation of MG.

More than 75% of MG patients present with visual complaints of droopy eyelids or double vision. Differences in acetylcholine receptor isoform expression, safety factor, firing frequency and sensitivity to complement deposition have been suggested to explain the increased vulnerability of extraocular muscles in MG compared to other skeletal muscles. The facial and bulbar muscles are frequently affected in generalized MG and their weakness occurs in about 5-10% of patients at the onset of the disease.\textsuperscript{5} However, the involvement of facial muscles without ocular weakness is extremely rare at the presentation of MG. Three similar cases have been previously reported in literature.\textsuperscript{2,4} Two of the reported patients were not serologically confirmed, and MG was associated with thymoma and clinical remission followed thymectomy.\textsuperscript{3,4} Jeannet et al. reported isolated facial and bulbar paresis in a neonatal MG patient.\textsuperscript{7} However, this case was different in that the patient neither had acetylcholine receptor antibody, nor responded to any treatment. Prominent facial and bulbar involvement is also encountered in MG patients with anti-MuSK antibody. Although we did not evaluate the anti-MuSK antibody in our patient, it was unlikely that our patient had anti-MuSK antibody considering the favorable response to oral pyridostigmine.

The present case described a patient with MG presenting as bilateral facial weakness and dysarthria without ocular signs. We suggest that the patient with bilateral simultaneous facial palsy may need to be screened for MG even in the absence of ocular muscle involvement.

REFERENCES

2. Cucurachi L, Cattaneo L, Gemignani F, Pavesi G. Late onset


