INTRODUCTION

Facial nerve paralysis is a debilitating condition. Patients with facial nerve disorders are often devastated due to the emotional and psychological impact of facial disfiguration and the subsequent physical limitations and difficulties associated with speaking, drinking, eating, and facial expression secondary to the disorder. Socialization and community participation is extraordinarily limited and difficult for many of these patients.2 Bilateral simultaneous facial paralysis is a rare clinical entity, its incidence being one per 5 million population per year,2 when compared to unilateral facial paralysis with 1,000 cases per 5 million population per year.3 Trauma is only one of the many aetiological factors causing bilateral facial paralysis.

CASE REPORT

A 23-year old university student of Kargystan nationality was referred for a neurological evaluation on the post-injury 10th day for evaluation of bilateral facial paralysis (House-Brackmann classification (HB) of grade V).4 Just after accident, he had loss of consciousness for about 2 hours. There were four bouts of vomiting on the day of accident with minimal headache. No history of dysphagia, aspiration or respiratory distress was obtained.

There was also bloody otorrhea from right ear. There was complete triad of vomiting, loss of consciousness and bloody otorrhea. The pure tone audiometry revealed mixed hearing loss in both ears (right side hearing loss more than left side). Hearing was improving with time. The laboratory examinations including complete blood cell counts, liver function tests, Erythrocyte Sedimentation Rate (ESR), C-Reactive Protein (CRP), Veneral Disease Research Laboratory (VDRL) and FTA-ABS, herjies simplex (HSV) titers, varicella-zoster virus titers, human immune deficiency (HIV) titers, chest X-rays etc. were within normal limits. Electrodiagnostic testing confirmed the diagnosis of severe degree of bilateral facial paralysis involving temporal, zygomatic, buccal and mandibular branches. Blink reflex tests showed bilateral peripheral conduction defects and electromyography (EMG) revealed increased insertional activity, fibrillations and positive sharp waves and severe denervation potentials.
Post-traumatic bilateral facial paralysis associated with temporal bone fractures in both sides. Electroneuronography (ENoG) evaluation was performed and revealed degeneration of 90% in facial nerves bilaterally.

A high-resolution computed tomography (CT) scans showed bilateral temporal bone fractures, along with hematoma fluid densities in both mastoid cavities (Figure 1). Furthermore, the transverse fracture of right petrous bone was extended from squamous portion, the medial aspect of epi tympanum to the vicinity of the geniculate ganglion, but, there was no involvement of the fallopian canal. The longitudinal fracture of left petrous bone fracture was not distinct, but, the fallopian canal was intact. Air density was observed in left internal acoustic meatus.

The patient was placed on corticosteroids. Surgery was not carried out because of delayed onset. Electro-stimulation of the facial muscles and physiotherapy along with a course of prednisolone was started. Appropriate measures for corneal protection were suggested. At 6th month of follow-up, the patient had partial recovery of facial nerve function bilaterally and improvement in HB classification to grade III and ENoG of 60% was observed.

DISCUSSION

Bilateral simultaneous facial paralysis is described as facial paralysis involving both sides of the face occurring within 4 weeks of each other, and is found in 0.3 - 2% of facial paralysis. The etiologies of bilateral facial paralysis range from infections, tumors, head injuries, degenerative diseases, vascular diseases, and idiopathic. A similar case was reported by Kumar and Gupta that presented with bilateral complete paralysis of facial nerve without loss of taste sensation. CT scan showed fractures of the mandibular symphysis and both condylar necks but did not show any fracture in the base of skull or temporal bones. Another report was of a 24-year old male with bilateral facial palsy and a right-sided abducens paralysis due to a motorcycle accident. The patient had bilateral temporal bone fracture, an anterior skull-base fracture, subarachnoid haemorrhage and pneumocephalus. Electrophysiological tests consisting of ENoG and spontaneous and voluntary EMG demonstrated 100% degeneration at the first examination. At one-year follow-up, the patient exhibited HB grade 1 recovery on the right and HB grade 2 recovery on the left side. Rarely bilateral facial palsy can be a presentation of lyse disease and sarcoidosis.

It is important that early detection, evaluation and intervention are carried out for optimal functional recovery after facial nerve injury. When the temporal bone is fractured, high suspicion for facial nerve injury, either unilateral or bilateral, is warranted.

REFERENCES