Delayed Facial Nerve Palsy after Seat Belt Induced Injury

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Blunt trauma causing facial nerve palsy and facial nerve trauma due to seat belt injury is rarely reported. We describe a 63 year old female who was involved in a road traffic accident without any imaging evidence of temporal bone fracture developing left facial nerve palsy on the second day. We discuss the pathophysiology of this condition and the need to be aware of this unusual complication when evaluating patient with blunt trauma to the face.

Key words: Facial, nerve, blunt, injury, Palsy

INTRODUCTION

Facial nerve is crucial to the movement of muscles, which are required for facial expression and compared to other cranial nerves, it is the most commonly injured [1].

Due to the location of the facial nerve cell bodies in the pontine region of the brainstem, most traumatic incidents to the facial nerve typically involves injury to its axons. Most facial nerve palsy (FNP) due to trauma is usually seen with high intensity trauma that is typically associated temporal bone fractures [2]. FNP due to blunt trauma without imaging evidence of bony fracture has been reported in a few case reports while FNP due to seat belt is rather rare [3].

We report delayed FNP in an adult patient without imaging evidence of bony fracture who had seat belt induced trauma to the temporal region of her face during a road traffic accident.

CASE DESCRIPTION

A 63-year-old Caucasian female was involved in a road traffic accident when her vehicle was hit on the side during a case of road rage on 08/09/2014. Patient was wearing a seat belt and she felt the left side of her face hit the seat belt strap and she was taken to the emergency room. Essential findings on examination were a slight swelling at the peri-zygomatic arch area. Vital signs were within normal limits and neurologic examination did not reveal any abnormalities. Patient was given pain medications and sent home.

The next morning, her husband noticed that her face was asymmetric with left sided facial droop (Figure). There was associated inability to close the left eye. Patient stated that she was concerned that she might be having a stroke thus she returned to the emergency room where neurological examination revealed left facial nerve weakness and it mainly involved the lower face. Patient hearing was normal and rest of the neurological examination did not reveal any abnormality. Patient was in the hospital for three days and was also worked up for stroke. She had a CT scan, MRI of the brain, cervical spine and lumbosacral spine, 2D Echo, carotid Doppler which did not reveal any soft abnormality.

Patient was informed that she did not have a stroke and discharged home.

Patient was seen by our neurologists DVP on 9/25/14 and neurological examination revealed that left facial weakness persists and mainly involved the lower face. The rest of the neurological examination was unchanged. An impression of contusion of her left facial nerve at the exit, below the left ear lobe involving predominantly the lower branch was made. She was started her on methylprednisolone 4 mg (Medrol dosepak) followed by physical therapy including electrical stimulation to the VII nerve. She was also informed of exercises she could do for the face and facial exercises with her fingers to improve weakness.

DISCUSSION

FNP due to blunt trauma without imaging evidence of bony fracture is uncommon [1]. Review of literature suggests that most common FNP are associated with temporal bone fracture and the paralysis occurs on the day of the injury.

Seddon classified nerve injury as neuropraxia, axonotmesis and neurotmesis in order of increasing severity [4].

Neurapraxia involves selective nerve sheath demyelination due to compression ischemia with axon continuity retained. Although conduction may be altered in the nerve, the prognosis for recovery is usually good. Axonotmesis represents a more severe spectrum of nerve injury with disruption of myelin sheath and the axon while the perineurium and epineurium remains

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Figure Left facial nerve palsy.

intact ensuring some form of continuity in the nerve. This form of nerve injury typically accompanies crush injuries and fractures. The disruption in the myelin sheath and axon means that wallerian degeneration is common sequelae of this form of nerve injury and prognosis for recovery is fair requiring many months. The most severe nerve injury called Neurotmesis occurs when there is complete nerve transection involving the myelin sheath, connective tissue and disruption of the endometrium. This is commonly seen in lacerations, wallerian degeneration occurs after the injury and nerve regeneration is rather poor or almost non-existent [4].

Our patient presentation was rather unique since there was no high velocity injury. Immediate or complete FNP has been reported in disruption of facial nerve axon continuity with poor resolution of the facial nerve palsy. Our patient facial nerve injury occurred on the second day suggesting that secondary compression of the nerve may have occurred due to swelling after the blunt trauma. We speculate that these may have led to compression ischemia leading to a demyelinating structural change in the axon that altered nerve conduction causing Neurapraxia [5].

Patient FNP led to her been worked up as a case of potential stroke and imaging done did not reveal any abnormality and more importantly there were no pontine lesion detected.

The fact that the patient's FNP recovery has been poor after 6 months might be attributed to the fact that the initial diagnosis was not made until roughly 8 weeks after when she presented to our Neurologists (DVP) and there was no evidence that patient was availed the opportunity of anti-inflammatory agents like steroids during the critical 24 hours after her injury. The management of FNP has been subject of debate in recent years with most favoring steroids at the expense of surgical decompression since majority of patients have spontaneous resolution of their symptoms [6].

Our patient was not offered the option of surgical decompression as we expected spontaneous resolution of her symptoms and also she presented much later to our team.

CONCLUSION

In conclusion we report a case of facial blunt trauma induced by seat belt leading to delayed FNP. This rare presentation makes it pertinent that patient with blunt trauma to the face with no radiological evidence of fracture be made aware that delayed onset of FNP might be a secondary sequelae of facial trauma.

CONFLICTS OF INTEREST DISCLOSURE

The authors do not have any conflict of Interest to Disclose.

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