Case report

Indian Journal of Neurotrauma (IJNT)
2009, Vol. 6, No. 1, pp. 67-68

Delayed lower motor neuron facial palsy contralateral to surgical decompression in patients with head injury: Report of two cases

Dhandapani SS M Ch, **Thiagarajan G** M Ch, **Cherian TA** M S*
Department of Neurosurgery & ENT*
Pondicherry Institute of Medical Sciences, Pondicherry-14, India

INTRODUCTION

The facial nerve is the most commonly affected motor cranial nerve in closed head injuries[1]. Delayed facial palsy is a well-known entity after head injury, and is commonly seen in association with fractures involving petrous part of temporal bone[2]. Though many neurosurgeons would have had the experience of seeing delayed LMN facial palsy developing after craniotomy for various causes, it had not been reported much. We report two such cases in which delayed LMN facial palsy developed contralateral to surgical decompression in patients with head injury.

CASE 1

A 26 years old male was brought in an unconscious state after RTA. There was 5 cm right frontal laceration with no ear bleed. GCS was 6/15 (E1V1M4). CT brain revealed right frontotemporoparietal acute SDH with right temporal contusion and midline shift (Fig 1a); there was no evidence of petrous fracture (Fig 1b). He immediately underwent Right frontotemporoparietal craniotomy, evacuation of SDH, and partial temporal lobectomy. Intra-operatively, brain was tense on opening and lax on closure. In the postoperative period on day 1, GCS improved to 15/15 with no focal deficits. On third postoperative day, patient developed left LMN facial palsy (House & Braackman H & B Grade 4), with no evidence of associated hearing deficit. Left eyelid strapping was done for 5 days, and patient was put on oral prednisolone 1 mg/Kg/d for 2 weeks along with physiotherapy. At 2 weeks follow-up, his facial palsy had improved to H & B Grade 3. At 3 months, it had further improved to H & B Grade 2.

Address for correspondence:
Cherian TA
Department of Neurosurgery & ENT
Pondicherry Institute of Medical Sciences,
Pondicherry-14, India

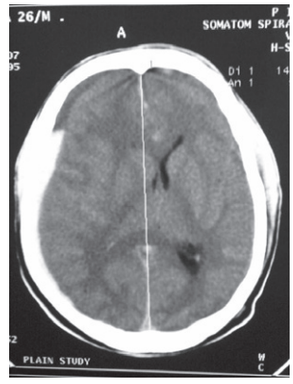

Fig 1a: CT showing right frontoparietal subdural hematoma

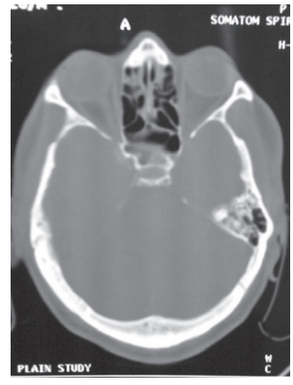

Fig 1b: CT (bone window) showing no evidnce of petrous fracture

CASE 2

A 45 years old male, known case of diabetes, presented after RTA with H/O LOC for 20 min. There was some right ear bleed. GCS was E4 V4 M6. CT brain revealed small left temporal contusion with no significant mass effect (Fig 2); there was no evidence of petrous fracture. He was initially managed conservatively. Thirty six hours

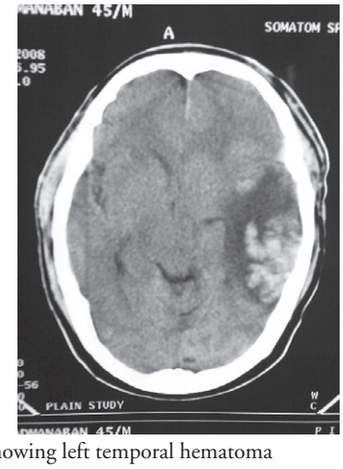

Fig 2: CT showing left temporal hematoma

later, patient worsened in neurological status with increase of mass effect as seen on follow-up CT. He underwent left frontotemporoparietal craniotomy, and partial temporal lobectomy. Intra-operatively, brain was tense on opening and lax on closure. GCS improved to 15/15 with no focal deficits on the first postoperative day. On fourth postoperative day, he developed right LMN facial palsy (H & B Grade 5), with no associated significant hearing deficit. Right eyelid strapping was done for 5 days, and patient was put on oral prednisolone 1 mg/Kg/d for 2 weeks along with physiotherapy. Two weeks later, his facial palsy had not improved, and EMG revealed no signs of reinnervation. He underwent transmastoid facial nerve decompression, and facial nerve was found to be edematous. At 3 months follow-up, his facial palsy had improved to H & B Grade 2.

DISCUSSION

The facial nerve is the most commonly affected motor cranial nerve in closed head injuries[1]. The onset of facial palsy can be early, delayed or mixed[2]. It is usually seen in association with fractures involving petrous part of temporal bone and does not occur without either a demonstrable petrous fracture, or a bleeding ear (or haemotympanum) on the affected side[2]. Both the patients reported here had no fracture involving petrous part of the temporal bone. Case 1 had no ear bleed or hemotympanum whereas, Case 2 had some right ear bleed. Both patients had significant mass effect with early evidence of transtentorial herniation at the time of surgery, and both improved dramatically with surgical decompression indicating quick reduction of intracranial pressure.

The possible reasons for development of delayed LMN facial palsy contralateral to surgical decompression in these patients with head injury are:

1. Injury to the facial canal (coup injury) without fracture, with slow expanding hematoma, swelling of loose fibrous tissue or delayed vascular spasm or thrombosis resulting in delayed facial palsy, with contre coup injury causing intracranial bleed (surgery being coincidental)
2. Traction of facial nerve fibers due to brainstem displacement, as a result of sudden intra cranial decompression
3. Impact activation of latent viral infection of facial nerve
4. Idiopathic (Bell's palsy)

Though the first hypothesis is likely in Case 2, it is improbable in Case 1 as the external wound was on the same side as acute SDH, and there was no ear bleed. The second hypothesis is possible in both cases. Similar development of contralateral facial palsy was also noted in two children after surgical evacuation of subdural hygroma[3]. With respect to the outcome, first patient had spontaneous improvement, while the second patient improved after facial nerve decompression.

To conclude, delayed facial palsy after head injury is likely even without obvious petrous fracture and management has to be decided based on clinical course.

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