CASE REPORT

Delayed lower cranial nerve palsy (Collet—Sicard syndrome) after head injury

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Introduction

The lower cranial nerves (IX—XII) originate from the brain stem and exit the intracranial cavity through the jugular foramen and hypoglossal canal, respectively. The jugular foramen and hypoglossal canal exist closely together. Therefore, multiple lower cranial nerve palsy tends to occur when this part is injured. The neurological symptoms vary according to the combination of damaged cranial nerves. The palsy of unilateral IX—XI nerves, which pass through the jugular foramen, is called 'Vernet syndrome' or 'Jugular foramen syndrome'. The combination of Vernet syndrome with palsy of the XII nerve, which passes through hypoglossal canal, is called 'Collet—Sicard syndrome'. Malignant lesions of the skull base and nasopharynx are more frequent causes of these syndromes. Lower cranial nerve palsy following blunt head injury is relatively rare. Here, we report a rare case of delayed lower cranial nerve palsy (Collet—Sicard syndrome) that developed 2 days after a head injury.

Case report

An 82-year-old woman fell from a bicycle, struck her forehead and lost consciousness. On admission to a regional hospital her consciousness was clear. She complained of severe occipitalgia, but neurological examinations did not reveal any abnormalities. No abnormality was detected on the plain X-ray films of her skull or plain computed tomography (CT) of her head. Two days later, the patient began to complain of hoarseness and difficulty swallowing. She was then transferred to our hospital for further investigation. Neurological examinations on admission revealed the absence of a gag reflex on her left, soft palate deviation to her right, mild weakness of her left trapezius muscle, and tongue deviation to her left (Fig. 1). Direct laryngoscopy revealed left vocal cord paralysis. The rest of the neurological examination was normal. Magnetic resonance imaging (MRI) of the brain stem and MR angiography revealed no abnormality. CT with the window level set for bone visualization revealed fractures of the left clivus and the right occipital bone including the occipital condyle (Fig. 2). Three-dimensional CT demonstrated these fractures more clearly (Fig. 3). We diagnosed the complaint as Collet—Sicard syndrome caused by the head injury and...
managed her conservatively without a collar. Nasogastric tube feeding was begun because of her persistent dysphagia. One week later, follow-up direct laryngoscopy showed not only left, but also right vocal cord paralysis (Fig. 4). Therefore, a tracheostomy was performed to secure the upper airways. Thereafter, her symptoms gradually improved. Six months after the accident, her gag reflex returned and the 11th and 12th cranial nerve function had completely improved. However, mild dysphagia and vocal cord paralysis remained.

Discussion

Collet—Sicard syndrome was initially reported in trauma patients by Collet and Sicard. Collet reported the first case of complete unilateral paralysis of the last four cranial nerves (IX—XII) following a bullet injury in the mastoid region and Sicard described cases with similar clinical features. Subsequently, some cases of lower cranial nerve palsy including Collet—Sicard syndrome have been reported. However, most cases are caused by a malignant lesion. Head or neck injury, internal carotid artery dissection, and ischemia were reported for other causes. Traumatic injury to these nerves is very uncommon. We found only seven cases of Collet—Sicard syndrome following a blunt head injury reported in the readily available literature (Table 1). Cranial nerve palsies after head injuries occurred immediately in two cases and after several days in two other cases. In three cases, initial neurological examination did not detect any cranial nerve palsy because of disturbance of consciousness. In most cases, lower cranial nerves palsies followed occipital condyle fracture.

Figure 1  Photo of the patient showing normal facial movements (A) and the tongue deviated to the left (B).

Figure 2  Head CT with the window level set for bone visualization showing fractures of the left clivus, right jugular tubercle and right occipital condyle (arrows).
fractures (OCF). A review of the literature regarding OCF revealed that cranial nerve deficits occurred in 31% of the patients with OCFs; among them, the deficits were delayed in 38%.

Also in our case, cranial nerves palsies followed OCF, and deficits were delayed for 2 days.

Several hypotheses have been proposed to explain lower cranial nerve palsies after head injuries. Grundy et al. suggested that cranial nerve palsies after head injuries are secondary to vertebral artery insufficiency. Bridgman and McNab suggested that the cranial nerves were damaged by traction injuries. Desai et al. suggested the possible role of a displaced bony fragment that could compress the nerves in the context of fracture extension to the posterior jugular foramen or to the hypoglossal canal. Orbay et al. reported a case in which hypoglossal nerve palsy occurred 3 months after a head injury and suggested that a scarring process and the formation of a callus at the level of the hypoglossal canal could progressively compress the main hypoglossal nerve. In the present case, cranial nerve palsy occurred 2 days after the head injury. There was no abnormality of the brain stem on MRI. The left clivus and right occipital bone including occipital condyle were fractured. However, a bony fragment that might compress cranial nerves was not detected. Therefore, the cranial nerve injury in our case appeared to be caused by neither brain ischemia nor direct injury by a bony fragment. We presume that the cranial nerves were injured by a small haematoma due to venous bleeding, or by soft-tissue edema associated with the fracture around the jugular foramen and hypoglossal canal.

The diagnosis of OCF is difficult because conventional X-ray films of the skull are not sufficiently sensitive to detect it. The sensitivity of plain X-rays calculated from various reports of the diagnosis of OCF is 3.2% (2 of 62 patients). Legros et al. suggested that OCF should always be considered in cases of persistent neck pain after trauma, regardless of the presence of cranial nerve palsies. CT with the window level set for bone visualization and three-dimensional CT have been reported to be the most useful modalities for demonstrating these fractures.

The mechanisms proposed for these fractures include hyperextension associated with a vertical compression force. Anderson and Montesano classified OCF into Type I to Type III fractures depending on the fracture morphology and mechanism of injury (Table 2). Types I and II OCF are stable fractures. Type III OCF is unstable because the alar ligament is loaded. Most OCFs are managed conservatively by immobilization of the cervical spine with a collar. A review of the literature regarding OCF revealed that 12 of 15 patients who had developed delayed neurological symptoms or cranial nerve deficits were not initially treated for OCF.
### Table 1  Review of the literature on Collet—Sicard syndrome after blunt head trauma

<table>
<thead>
<tr>
<th>Reference</th>
<th>Cause of injury</th>
<th>Associated fracture</th>
<th>LOC</th>
<th>Onset of cranial nerve palsies</th>
<th>Treatment for fracture</th>
<th>Remained nerve palsies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bolender et al. 2</td>
<td>Traffic accident</td>
<td>Condyle fracture Type III</td>
<td>+</td>
<td>Unknown</td>
<td>Observation</td>
<td>Deficit of N.X</td>
</tr>
<tr>
<td>Hashimoto et al. 8</td>
<td>Traffic accident</td>
<td>Condyle fracture Type II</td>
<td>–</td>
<td>Immediately</td>
<td>Observation</td>
<td>Mild deficits of N.X, XI</td>
</tr>
<tr>
<td>Wani et al. 20</td>
<td>Falling down</td>
<td>Condyle fracture Type II</td>
<td>+</td>
<td>1 day delayed</td>
<td>Observation</td>
<td>Deficit of N.X, XI</td>
</tr>
<tr>
<td>Sharma et al. 17</td>
<td>Traffic accident</td>
<td>Condyle fracture Type II</td>
<td>+</td>
<td>Unknown</td>
<td>Removal of bony fragment</td>
<td>None</td>
</tr>
<tr>
<td>Young et al. 21</td>
<td>Traffic accident</td>
<td>Condyle fracture Type III</td>
<td>+</td>
<td>Unknown</td>
<td>Halo vest for 12 weeks</td>
<td>Mild deficits of N.IX—N.XII</td>
</tr>
<tr>
<td>Legros et al. 11</td>
<td>Traffic accident</td>
<td>Condyle fracture Type III</td>
<td>–</td>
<td>2 days delayed</td>
<td>Collar for 45 days</td>
<td>Mild deficit of N.X</td>
</tr>
<tr>
<td>Connolly et al. 5</td>
<td>Traffic accident</td>
<td>Jefferson fracture</td>
<td>–</td>
<td>Immediately</td>
<td>Halo vest for 10 weeks</td>
<td>Mild deficit of N.XII</td>
</tr>
<tr>
<td>Present case</td>
<td>Traffic accident</td>
<td>Condyle fracture Type II</td>
<td>+</td>
<td>2 days delayed</td>
<td>Observation</td>
<td>Deficit of N.X</td>
</tr>
</tbody>
</table>

LOC: loss of consciousness on admission to the hospital. Unknown: onset of cranial nerve palsies was unknown because of disturbance of consciousness.

### Table 2  Anderson and Montesano’s classification of occipital condyle fractures

<table>
<thead>
<tr>
<th>Fracture type</th>
<th>Morphology</th>
<th>Trauma mechanism</th>
<th>Stability</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Comminution of the occipital condyle</td>
<td>Axial compression between atlas and skull base</td>
<td>Stable</td>
</tr>
<tr>
<td>II</td>
<td>Extension of a linear basilar skull fracture to the occipital condyle</td>
<td>Axial distension between atlas and skull base</td>
<td>Stable</td>
</tr>
<tr>
<td>III</td>
<td>Avulsion fracture of the occipital condyle by the alar ligament</td>
<td>Rotation or lateral flexion with avulsion of the condyle and involvement the alar ligament</td>
<td>Unstable</td>
</tr>
</tbody>
</table>

*See Table 2.*
and suggested that patients with OCF should be treated with external immobilization, especially in the case of Type III OCF. Most authors advocate 3 months’ treatment with a collar for OCF. Sharma et al. reported that surgical relief of bony fragment compression in the jugular foramen led to favorable results. Surgical treatment for OCF may be indicated for decompression or stabilization.

In most cases of OCF with cranial nerve palsies, neurological deficits partially improved, but residual neurological deficits may persist for a long time. In our case, OCF was classified as Type II. We managed the patient conservatively, and delayed cranial nerve palsies occurred. Vocal cord paralysis became worse after a further time.

Conclusions

OCF should be considered in case of persistent neck pain or occipitalgia after trauma. CT with the window level set for bone visualization and three-dimensional CT are useful modalities for demonstrating OCF. In the event of OCF, even when a cranial nerve deficit is not recognized on initial examination, immobilization of the neck with a collar for 3 months is recommended to prevent delayed cranial nerve palsies.

References