Two pediatric cases of post-traumatic facial paralysis with delayed onset

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ABSTRACT
Posttraumatic facial paralysis with delayed onset generally has a good outcome. This is especially true in pediatric cases in which incomplete paralysis always suggests a favorable prognosis. We describe two children with posttraumatic facial paralysis treated with surgical facial nerve decompression. Both patients had longitudinal, otic capsule-sparing fractures with ossicles displacement. Fracture line reached the vertical segment of facial canal for one case, while enhanced MRI revealed the abnormal signal intensity around geniculate ganglion for the other case. They showed asymmetrical facial movement after recovering from coma, which evoked delayed-onset paralysis. Electroneurography showed more than 90% nerve degeneration, suggesting an unfavorable prognosis in both. However, while one had permanent moderate facial paralysis, the other had complete recovery after surgery. These cases reconfirmed the difficulty of estimating prognosis for post-traumatic pediatric facial paralysis and suggested the earlier examination of ENoG and enhanced MRI, especially in comatose cases.

1. Introduction
Post-traumatic facial paralysis with delayed onset generally suggests a favorable prognosis, especially in pediatric cases with incomplete paralysis which always have a good outcome. However, it is sometimes difficult to determine correctly the time of onset and the severity in unconscious children because facial asymmetry might be obvious only when the patients are conscious. In addition, surgical indications for pediatric facial paralysis remain controversial and poorly defined. Here, we describe two children with post-traumatic facial paralysis treated with surgical facial nerve decompression, resulting in different outcomes. This case report is approved by the Tsuchiura Kyodo General Hospital institutional review board.

2. Cases
Case 1 is a 9-year-old boy who fell off his bicycle and was admitted to our emergency department. On admission, he was unconscious with a Glasgow Coma Scale score of E1V2M4. He had cerebrospinal otorrhoea from the right ear. Facial paralysis was not apparent. Routine laboratory examination and chest radiography showed no remarkable findings. Brain computed tomography (CT) revealed hemorrhage and multiple temporal bone fractures (Figure 1(A)). He was carefully observed in the intensive care unit (ICU). On day 2, follow-up CT demonstrated reduction of the hemorrhage and he had recovered consciousness to the point of reacting to his parents speaking to him. There was no obvious abnormality in motion of limbs. On day 3, as his level of consciousness further improved, right facial paralysis and hearing loss were recognized. On referral to the otolaryngology division, he had grade III House–Brackmann (H–B) facial paralysis. He was given methylprednisolone 20 mg intravenously which was then tapered over 10 days. However, his facial paralysis worsened to H–B grade V and 2/40 on the Yanagihara grading system. High-resolution CT of the temporal bone revealed an otic capsule-sparing longitudinal fracture involving the vertical segment of the right facial canal and displacement of the ossicles (Figure 1(B)). Electroneurography (ENoG) showed greater than 90% degeneration of the right facial nerve compared with the left. His audiogram showed mild conductive hearing loss and acoustic reflex was absent at maximum stimulus intensities. On day 20, facial nerve decompression surgery was performed.
A transmastoid approach was used to provide access to the tympanic and mastoid segment of the facial nerve. Bone fractures and crushing of the nerve were identified in the mastoid segment (Figure 1(C)). The bone fragments placing pressure on the nerve were removed as gently as possible. Gelfoam infiltrated with steroids was placed around the injured nerve. After the surgery, he was followed up for one year. Unfortunately, his facial paralysis had not fully resolved during that period, remaining H–B grade III and Yanagihara grade 24/40. His audiogram was unchanged.

Case 2 is a 10-year-old boy who was admitted to our emergency department after a road traffic accident. His level of consciousness was low at E2V2M4. He had cerebrospinal otorrhea from the right ear. Facial paralysis was not apparent. Brain CT demonstrated an acute subdural hematoma and multiple temporal bone fractures, including displacement of the ossicles. He was moved to the ICU for observation and complete rest. On day 2, his level of consciousness improved. There was no obvious abnormality in motion of limbs. The cerebrospinal otorrhea had stopped, but he complained of right hearing loss. On day 5, his mother noted right facial paralysis. When he was referred to the otolaryngology division, the paralysis was classified as H–B grade III and Yanagihara grade 22/40. Although he was given prednisolone 250 mg intravenously, tapered over 10 days, his facial paralysis worsened to H–B grade V and Yanagihara grade 8/40. ENoG showed greater than 95% degeneration of the right facial nerve. His audiogram showed a normal hearing level, but acoustic reflex was absent at maximum stimulus intensities. No fracture of the facial canal was seen on careful inspection of his temporal bone CT, but enhanced magnetic resonance imaging (MRI) demonstrated high signal intensity of the nerve around the geniculate ganglion (Figure 2). At his mother’s urging, surgery was performed on day 25. Using a transmastoid approach, the facial nerve was decompressed from around the geniculate ganglion to the mastoid section, in a manner similar to the procedure for Bell palsy. Briefly, the incus was temporarily removed, allowing completion of drilling toward the geniculate ganglion. The entire facial canal was thinned to eggshell-thickness with a diamond bur. The thinned bone was fractured with a pick, and the bone fragments were lifted gently with a round knife. The facial nerve was swollen and edematous. On closing, the incus was repositioned and held in place by several small pieces of Gelfoam. The patient’s facial paralysis had completely resolved with H–B grade I and Yanagihara grade 40/40, during the 4-month follow-up period after the surgery.

3. Discussion

In general, complete versus partial paresis is predictive of the degree of recovery. H–B stage II paralysis has a good outcome, while H–B stages III and IV are associated with moderate residual dysfunction. H–B stages V and VI rarely recover [1,2]. The time of onset and progression of facial paralysis are also important for outcome. Immediate-onset paralysis has a relatively poor prognosis, often caused by direct nerve impingement or transection, while delayed paralysis almost

Figure 1. Computed tomography showing longitudinal and squamous fractures of the temporal bone (A) in case 1. The fracture line (B,C) extends to the mastoid segment of the facial canal and involves facial nerve fibers.
always results in at least some improvement. If a patient therefore has incomplete paralysis of delayed onset, the likelihood of full recovery of normal facial function is high. In our cases, both did not show apparent facial paralysis just after the trauma and, several days later, complained of moderate facial paralysis classified as H-B grade III. Therefore, we thought that surgical treatment was not necessary for them at that time. On the other hand, when patients with immediate onset of complete facial paralysis have a relatively poor prognosis, less than 50% patients would recover normal or near-normal facial function unless emergent surgical intervention.

The prognosis of pediatric post-traumatic paralysis can be harder to estimate, as the possibility of complete functional recovery is greater in children than in adults. Moreover, very young children may not be able to cooperate in performing the forehead, eye or mouth movements for evaluation of facial nerve function [3]. In such cases, facial asymmetry might be obvious only when the child is crying. In addition, the time of onset is sometimes unclear because thorough assessment after the head trauma cannot always be performed at the time of initial examination. Delayed-onset paralysis may appear 24 to 36 hours following trauma. However, patients at that point may have severe facial swelling and be comatose. Therefore, facial paralysis may not be discovered until several days after the patients awaken and the facial swelling decreases. Especially, in pediatric cases, it can be extremely difficult to estimate the correct time of onset and severity before the child has regained consciousness. In our both cases, the child was comatose until day 2 or 3, delaying assessment of facial asymmetry. Their facial paralysis might have developed immediately after the trauma.

ENoG has been the most valuable electrophysiological test to predict which patients are unlikely to have full recovery [4]. It is usually possible to perform ENoG in children of all ages, regardless of their ability to cooperate [3]. ENoG findings are correlated with the H-B grade to establish a prognosis. In a case series, the majority of patients with ENoG values between 67% and 100% had facial weakness grade I–II, corresponding to neuropraxia, most of whom made a complete recovery within 3 months. Prognosis is generally good for ENoG values above 10%. Below that level, the prognosis is poor and surgical decompression may be recommended. In addition, ENoG works for even the patients with impaired consciousness because it is objective test. If ENoG was undergone to the case 1 at an earlier date, we may have had performed earlier surgical intervention, leading to better outcome.

Post-traumatic facial paralysis is usually associated with temporal bone fractures, but it can sometimes occur in the absence of radiologically demonstrable bone disruptions [5]. In those cases, microtrauma causing nerve edema can be assumed. Microtrauma would be due to severe traction and stretching of the greater superficial petrosal nerve, potentially leading to the formation of an intraneural hematoma and secondary edema that extends in a retrograde direction along the proximal nerve [6,7]. Neural edema in such a case has the same deleterious effect as that in inflammatory palsy and, therefore, enhanced MRI is sensitive to such nerve edema and can clearly display the nerve itself [8,9]. Moreover, MRI can also show a thickened geniculate ganglion. Even in cases of facial paralysis without any bone fractures seen on CT, MRI can reveal abnormal findings in the geniculate ganglion [9]. Actually, case 2 showed abnormal signal intensity around geniculate ganglion on MRI without

Figure 2. High-resolution computed tomography of the right ear showing a normal geniculate fossa without bony fracture (A) in case 2. Contrast-enhanced, T1-weighted magnetic resonance imaging shows an enlarged, enhancing geniculate ganglion extending to the internal auditory canal on the right (B).
apparent bone fractures on CT, which would be helpful in surgical procedure.

Finally, surgical indications in cases of traumatic facial paralysis remain controversial and poorly defined. Most patients with temporal bone fractures causing facial paralysis recover fully without intervention. Some authors advise against surgical decompression of the facial nerve in its labyrinthine segment [10,11] due to the lack of systematic clinical studies demonstrating its effectiveness and because of the risk of sensorineural hearing loss. But, other investigators recommend serial electrophysiologic testing with ENoG between days 3 and 14 and consider facial nerve decompression if there is greater than 95% loss of axonal function compared with the unaffected side [12]. We removed bony fragments from around the facial nerve in case 1 because there was bony impingement. In case 2, no fractures were seen on CT, nor was there visible abnormality during the operation but abnormal enhanced signal was seen around geniculate ganglion on MRI. Therefore, a procedure similar to that for Bell palsy was performed to decompress the edema around the geniculate ganglion.

4. Conclusions

In our cases, the onsets of facial paralysis seemed delayed, but they worsened to H–B grade V. ENoGs showed more than 90% nerve degeneration, suggesting an unfavorable prognosis for both patients. After operative treatments, one child had moderate permanent facial paralysis and the other had complete recovery. The difficulty of estimating prognosis for posttraumatic pediatric facial paralysis is reconfirmed by these cases, suggesting that the fracture line extending to fallopian canal seen on CT would be poor prognostic factor, and ENoG and enhanced MRI should be performed as early as possible in the comatose cases with temporal bone fracture.

Disclosure statement

There is no conflict of interest or financial disclosure to be made.

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