### CASE REPORT

**Restoration of shoulder function and elbow flexion by nerve transfer for poliomyelitis-like paralysis caused by enterovirus 71 infection**

We report the case of an eight-month-old girl who presented with a poliomyelitis-like paralysis in her left upper limb caused by enterovirus 71 infection. She recovered useful function after nerve transfers performed six months after the onset of paralysis. Early neurotisation can be used successfully in the treatment of poliomyelitis-like paralysis in children.

Enterovirus 71 (EV 71) infection causes hand, foot and mouth disease in young children. Although the initial viral illness is self-limiting, it is sometimes followed by a poliomyelitis-like paralysis as a result of damage to the anterior horn cells.\(^1\) In the past, children with this condition were treated conservatively, since spontaneous recovery often occurred. Surgery was reserved for those who failed to recover and were left with residual paralysis.

Nerve transfer has been used to restore shoulder and elbow function after traumatic brachial plexus injury, and obstetric paralysis and useful functional improvements have been achieved.\(^2,3\) We describe the case of an eight-month-old girl who presented with a poliomyelitis-like paralysis caused by EV 71 infection, but with no spontaneous recovery of function in the left shoulder and elbow.

**Case report**

An eight-month-old girl presented with a fever of 38˚, oral ulcers and a vesicular rash on the hands and feet. A diagnosis of hand, foot and mouth disease was made at her local hospital. Her temperature rose to 40˚C for three days, when she developed a complete paralysis of the left upper limb. The function of her wrist and fingers gradually recovered, but her shoulder and elbow remained paralysed. She was referred to our hospital five months later for treatment of her residual paralysis.

On examination, the muscles of the left shoulder-girdle and arm were atrophied. The left deltoid, infraspinatus and biceps were paralysed, whilst the left triceps, forearm and intrinsic muscles were of normal power. Sensation appeared to be intact since she reacted to painful stimuli. The lower limbs and right arm were normal.

There was no radiological evidence of paralysis of the phrenic nerve. MRI of the cervical spinal cord revealed a linear lesion between C3 and C7. It was of high signal intensity on the sagittal T2-weighted images and of low signal intensity on the T1-weighted images (Figs 1a and 1b). It was located in the left anterior horn between C3 and C7 on the axial T2-weighted images (Fig 1c). Although the virus had not been isolated in the acute phase, the EV 71 antibody level was elevated to 128 times its normal value. This strongly supported a diagnosis of a left poliomyelitis-like paralysis caused by EV 71. We could not perform electromyographic studies since she was too young to cooperate. However, clinically there was no recovery of deltoid, infraspinatus and biceps after six months. Nerve transfers were therefore undertaken in an attempt to restore shoulder and elbow function.

The left brachial plexus was explored through a supraclavicular incision of 5 cm. There was no adhesion or scarring of the plexus. Direct stimulation of the upper trunk elicited no response from deltoid, infraspinatus and biceps. Stimulation of the musculocutaneous and axillary nerves also failed to evoke contraction of biceps and deltoid. Deltoid, infraspinatus and biceps were, therefore, completely paralysed. Stimulation of the middle and lower trunks evoked a normal response. The clinical findings indicated complete paralysis of the muscles innervated by the C5 and C6 nerve roots, albeit without sensory disturbance. On the basis of the clinical and MRI findings, the lesion was thought to be located in the anterior horn at the C5/6 level. We therefore carried out neurotisation of the musculo-
cutaneous nerve using the third and fourth intercostal nerves and transferred the middle branch of the spinal accessory nerve to the suprascapular nerve to restore elbow and shoulder function.

Three months later the biceps began contracting and after another two months shoulder abduction and external rotation started to recover. After two years, she had regained a full range of elbow flexion and was able to elevate her arm more than 100˚ with good external rotation (Fig. 2). Her Mallet score (for grading shoulder function) was 25, which is the highest score available and a great improvement on her pre-operative score of 5. The deltoid muscle remained paralysed and severely atrophic since the axillary nerve had not been repaired.

Discussion

A poliomyelitis-like paralysis can be caused not only by the polio virus, but also by an enterovirus, the Epstein-Barr virus, and Japanese encephalitis. Since it was first recognised in California in 1969, EV 71 infection has been responsible for several outbreaks of poliomyelitis-like paralysis worldwide. It is now one of the leading causes of an acute flaccid paralysis, since poliomyelitis has virtually been eradicated.

In our case, the marked elevation of the level of the EV 71 antibody and the typical skin lesions suggested a diagnosis of EV 71 infection. The high-intensity lesion located in the anterior horn on the T2-weighted MRI image is characteristic of the acute flaccid paralysis caused by enteroviruses such as EV 71 and poliomyelitis and suggests the presence of oedema or necrosis of the anterior horn.

While palliative surgery such as tendon transfer or arthrodesis has been performed for this condition, neurotisation has not. This is probably because the technique was developed well after vaccination had dramatically reduced the worldwide incidence of poliomyelitis.

The timing of surgery is controversial. Many cases of poliomyelitis-like paralysis can recover spontaneously, and therefore the decision to operate may be difficult. The prognosis of the paralysis caused by EV 71 is unknown. It may be the same as that of poliomyelitis since the lesions caused by polio virus and the EV 71 virus are similar.

Sharrard reported 142 patients with poliomyelitis and examined their muscle recovery in detail. He concluded that 90% of muscles which were completely paralysed after six months remained so. Elliot concurred, noting that muscles which showed some functional improvement within the first few weeks mostly recovered, while those which remained totally paralysed by the sixth month generally failed to improve. Since our patient did not recover function in biceps or her shoulder muscles by six months, neurotisation remained the only possible treatment. This decision was further justified by the fact that the deltoid remained atrophic two years later.

Nerve transfers are usually performed to restore shoulder and elbow function after traumatic brachial plexus injury.
or obstetric palsy, and good results have been reported.\textsuperscript{2,3} The choices of donor nerves to restore elbow flexion include the intercostal,\textsuperscript{2} the spinal accessory,\textsuperscript{16} the phrenic,\textsuperscript{17} the contralateral C7\textsuperscript{18} and part of the ulnar nerve.\textsuperscript{19} Partial transfer of the ulnar nerve to musculocutaneous nerves is easy to perform and in adults the outcome is reported to be good. In our case, the infant initially had a total paralysis of the left upper limb. On MRI, the high-intensity signal on the T2-weighted image involved the lower aspect of the cervical anterior horn. It could not be determined whether the ulnar nerve was completely normal and we therefore chose to transfer the intercostal nerves.

Orthopaedic surgeons should be aware of poliomyelitis-like paralysis caused by enterovirus infections. Although many patients recover spontaneously, some do not. If there are no signs of recovery after six months, neurotisation should be considered.

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References