Surgical Strategies for Brachial Plexus Polio-Like Paralysis

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Background: Brachial plexus polio-like paralysis is an acute flaccid paralysis of the upper limb following viral infection. Surgical approaches to the paralytic limb have not previously been discussed in the literature. This study is the first to describe the clinical course and propose a surgical strategy for disabilities related to poliomyelitis-like paralysis.
Methods: Between 1982 and 2004, 17 patients (11 boys and six girls) presented with acute flaccid paralysis of the upper limb. Their clinical course was reviewed retrospectively. Average age at onset of disease was 2 years (range, 4 months to 13 years). All patients had disability in shoulder abduction and/or elbow flexion. Ten patients underwent reconstructive surgery.
Results: Stage V shoulder abduction (>160 degrees) according to Gilbert's classification was regained in five patients who underwent nerve transfer within 1 year of paralysis. One patient treated by nerve transfer after 3 years of paralysis obtained only stage I abduction (<45 degrees). In two patients, multiple local muscle transfers were performed for the shoulder abduction disability, resulting in mild improvement from stage I to stage II abduction (<90 degrees). In four patients, functioning free muscle transplantation for elbow flexion was carried out, and all regained functional M4 muscle strength. Of seven patients in the nonsurgical group, two had complete spontaneous recovery within 1 year, but five had permanent residual limb paralysis at a mean follow-up of 10 years.
Conclusions: Surgical strategies, including nerve transfer for shoulder abduction deficit within 1 year after attack and functioning free muscle transplantation for the elbow flexion deficit in the late period, should be considered for this disease. Late reconstruction, either by nerve transfer or by using local multiple muscle transfer for shoulder abduction, is ineffective. (*Plast. Reconstr. Surg.* 120: 482, 2007.)

In the past, poliomyelitis infection caused by poliovirus was commonly diagnosed in patients with the clinical symptoms of fever, meningeal irritation, and acute flaccid paralysis of the limbs. Because poliomyelitis has almost been eradicated, a polio-like syndrome caused by other viruses has become a subject for investigation in the patient presenting with acute flaccid limb paralysis.[1–11] Many pathogens such as enterovirus-71,[3–7] Epstein-Barr virus,[8] Japanese encephalitis virus,[9] West Nile virus,[10] and *Mycoplasma pneumoniae* [11] have been reported to cause polio-like paralysis. Characteristics of this polio-like syndrome include sudden onset of flaccid paralysis of the limb without sensory deficit, no history of trauma or other causes, nonspecific symptoms such as fever within 1 week before the paralysis, and asymmetrical limb weakness generally in the proximal muscles of the upper extremities. In endemic areas, enterovirus-71 infection may also manifest foot-mouth-hand disease (vesicular lesions on the hands, feet, and oral mucosa) and herpangina. Virus isolation and identification in viral laboratories may be proven by virus culture or viral serologic tests. The lower motor neuron lesions in the anterior horn of the spinal cord, as with poliomyelitis, are sometimes found using magnetic resonance imaging.[5,6]

Information regarding the surgical management of the brachial plexus polio-like paralysis has not been documented in the literature. This study describes the clinical course of the disease and proposes a surgical strategy with which to treat it.

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PATIENTS AND METHODS

Between 1982 and 2004, 17 patients (Tables 1 and 2) who fulfilled the criteria for polio-like brachial plexus paralysis were treated at the Chang Gung Memorial Hospital by the senior surgeon (D.C.-C.C.). Their charts were reviewed retrospectively. There were 11 male patients (65 percent) and six female patients (35 percent). The mean age at disease onset was 2 years (range, 4 months to 13 years). All patients suffered from a sudden onset of acute upper extremity paralysis without a prior history of trauma. The left upper limb and the right upper limb were affected in nine cases and seven cases, respectively. In one case (case 6), bilateral upper limbs were affected. Most patients experienced prodromal symptoms, such as high fever and rash. Some had foot-mouth-hand disease or herpangina, with bullae on the hand, foot, and in the mouth. Some developed fetal symptoms such as convulsion, meningoencephalitis, or pneumonia. The mean latent period between fever and paralysis was 3.8 days (range, 1 to 7 days). Sensory deficit was not detected in any of the cases. The causative pathogen was identified as enterovirus-71 in nine of the 17 cases. The others were unknown, or not confirmed.

For objective evaluation of shoulder abduction, Gilbert's classification was used (stage 0, no abduction; stage I, 0 to 45 degrees; stage II, 45 to 90 degrees; stage III, 90 to 120 degrees; stage IV, 120 to 160 degrees; and stage V, normal).[12,13] Elbow flexion strength was assessed according to the modified Medical Research Council scale[14]: M1, a flicker of muscle contraction without joint movement; M2, joint movement through the range of motion without gravity; M3, movement through the range of motion against gravity; M3+, able to oppose the examiner's single finger force less than 30 seconds; M4, able to oppose the examiner's single finger force longer than 30 seconds; and M5, the strength against the examiner's four-finger resistance.

The proximal muscles for shoulder abduction and elbow flexion were predominantly involved. At the first clinical assessment, which averaged 3.8 years after onset (range, 2 months to 14 years), all patients had stage 0 shoulder abduction. Eleven of the 17 patients (65 percent) also had weakness of elbow flexion: of these, eight patients were graded M0, and three were graded M2. Three patients (cases 11, 13, and 17) were seen with total palsy initially. Only one (case 13) recovered completely after 6 months' follow-up from distal to proximal. The other two remained with shoulder disability.

Table 1. Patient Demographics and Procedures in Surgical Group

Case	Sex	Age at Onset of Disease	Prodromal Symptoms	Pathogen	Latent Period of Paralysis	First Clinic Visit after Onset	Motor Deficit	Time of Operation after Onset	Operation Method	Outcome
1	M	2 yr	High fever, FMHD	EV-71	7 days	6 mo	Left SA, 0	10 mo	NT for shoulder	Left SA, V
2	M	5 yr	High fever, IICP, skin rash	Unknown	2 days	9 mo	Left SA, 0; EF, M2	10 mo	NT for shoulder, FFMT for elbow	Left SA, V; EF, M4
3	M	6 mo	High fever	EV-71	4 days	2 mo	Right SA, 0; EF, M0	3 yr	NT for shoulder, FFMT for elbow	Right SA, 0; EF, M4
4	M	1 yr 6 mo	High fever	Unknown	5 days	1 mo	Left SA, 0; EF, M0	2 yr 6 mo	FFMT for elbow	Left SA, 0; EF, M4
5	M	13 yr	High fever	Unknown	2 days	2 yr 2 mo	Right SA, 0; EF, M0	2 yr 5 mo	FFMT for elbow	Right SA, 0; EF, M4
6	F	4 mo	High fever	Unknown	2 days	11y	Bil SA, 0	11 yr 2 mo	MLMT for left shoulder	Left SA, II; right SA, 0
7	F	1 yr	High fever	Unknown	Unknown	11 yr 7 mo	Left SA, 0	11 yr 10 mo	MLMT for shoulder	Left SA, II
8	F	4 mo	FHMD	EV-71	Unknown	9 mo	Right SA, 0; EF, M2	1 yr	NT for shoulder	Right SA, V; EF, M4+
9	M	9 mo	High fever	EV-71	Unknown	10 mo	Left SA, 0	1 yr	NT for shoulder	Left SA, V
10	M	6 mo	High fever	EV-71	Unknown	8 mo	Right SA, 0; EF, M2	1 yr	NT for shoulder	Right SA, V; EF, M2

M, male; F, female; EV-71, enterovirus-71; FMHD, foot-mouth-hand disease; IICP, increased intracranial pressure; SA, shoulder abduction (Gilbert's classification); EF, elbow flexion (Medical Research Council scale); NT, nerve transfer; FFMT, functioning free muscle transplantation; MLMT, multiple local muscle transfers.

Table 2. Patient Demographics in Nonsurgical Group

Case	Sex	Age at Onset of Disease	Prodromal Symptoms	Pathogen	Latent Period of Paralysis	First Clinic Visit after Onset	Initial Motor Deficit	Clinical Follow-Up Period (yr)	Final Motor Deficit
11	F	1 yr 7 mo	High fever, herpangina, convulsion	EV-71	7 days	6 yr	Right total palsy	7	Right SA, 0
12	M	6 mo	FMHD, meningoencephalitis	EV-71	2 days	2 mo	Left SA, 0	4	Left SA, 0
13	M	1 yr 3 mo	High fever	EV-71	3 days	6 mo	Left total palsy	<1	Total recovery from distal to proximal
14	F	8 mo	High fever	Unknown	Unknown	5 yr	Right SA, 0	12	Right SA, 0
15	M	1 yr	High fever	Unknown	1 day	6 mo	Left SA, 0; EF: M0	<1	Left, total recovery
16	M	6 mo	Aspiration pneumonia, FMHD	EV-71	7 days	1 yr 4 mo	Left SA: 0; EF, M0	5	Left SA, 0; EF, M0
17	F	11 mo	High fever	Unknown	Unknown	14 yr	Right total palsy	24	Right SA, 0

M, male; F, female; EV-71, enterovirus-71; FMHD, foot-mouth-hand disease; SA, shoulder abduction (Gilbert's classification); EF, elbow flexion (Medical Research Council scale).

In this study, patients were categorized into two clinical courses: surgical (10 patients) and nonsurgical (seven patients) (Fig. 1), depending on the surgical agreement of the patient or parents after full explanation.

The patients were observed closely every month during the first year of paralysis. Up to the end of the first year, the decision of whether or not to perform surgery is discussed and made. The determination of recovery was based mainly based on the clinical examination and adjuvant help by means of electromyography and motor nerve conduction velocities. In the surgical group (Table 1), six patients underwent nerve transfer for shoulder abduction: five had the operation within 1 year after onset of paralysis, and one was operated on 3 years after onset. The nerve used for neurotization was the phrenic nerve and/or spinal accessory nerve (Table 3). Three (cases 1, 2, and 10) had double neurotization with two neurotization transfers, whereas three (cases 3, 8, and 9) had single neurotization. Two patients (cases 6 and 7) underwent multiple local muscle transfers (trapezius muscle transfer to the humerus neck, levator scapulae muscle transfer to the supraspinatus, and latissimus dorsi muscle transfer to the infraspinatus) in an attempt to reestablish shoulder elevation after an average of 11 years of shoulder palsy. Four patients underwent gracilis functioning free muscle transplantation for elbow flexion deficits. Thoracic intercostal nerves (T3 to T5) were used as the donor nerve in all of them. Two of these four patients (cases 2 and 3) had previously undergone nerve transfer for shoulder elevation. One patient (case 8) had spontaneous recovery of elbow flexion, and one patient (case 10) remained with an M2 elbow flexion disability without further reconstruction. Patients in the nonsurgical group (Table 2) have been followed in the clinic for an average of 8 years (range, 1 to 24 years). Some have had continuous rehabilitation therapy.

Electromyography and nerve conduction velocities were used for electrodiagnostic evaluation in seven patients. Normal conduction velocities over sensory nerves were observed. Low amplitude of the axillary and musculocutaneous nerve motor action potential was revealed in the paralyzed muscles (deltoid and biceps). Electromyography demonstrated denervation of the defective muscles, with increased polyphasic waves and/or fibrillation. Magnetic resonance imaging of the cervical spine was performed in case 11, 2 years after the onset of paralysis. This demonstrated hyperintensities on T2-weighted images over the anterior horn of the spinal cord at the C5 level (Fig. 2).

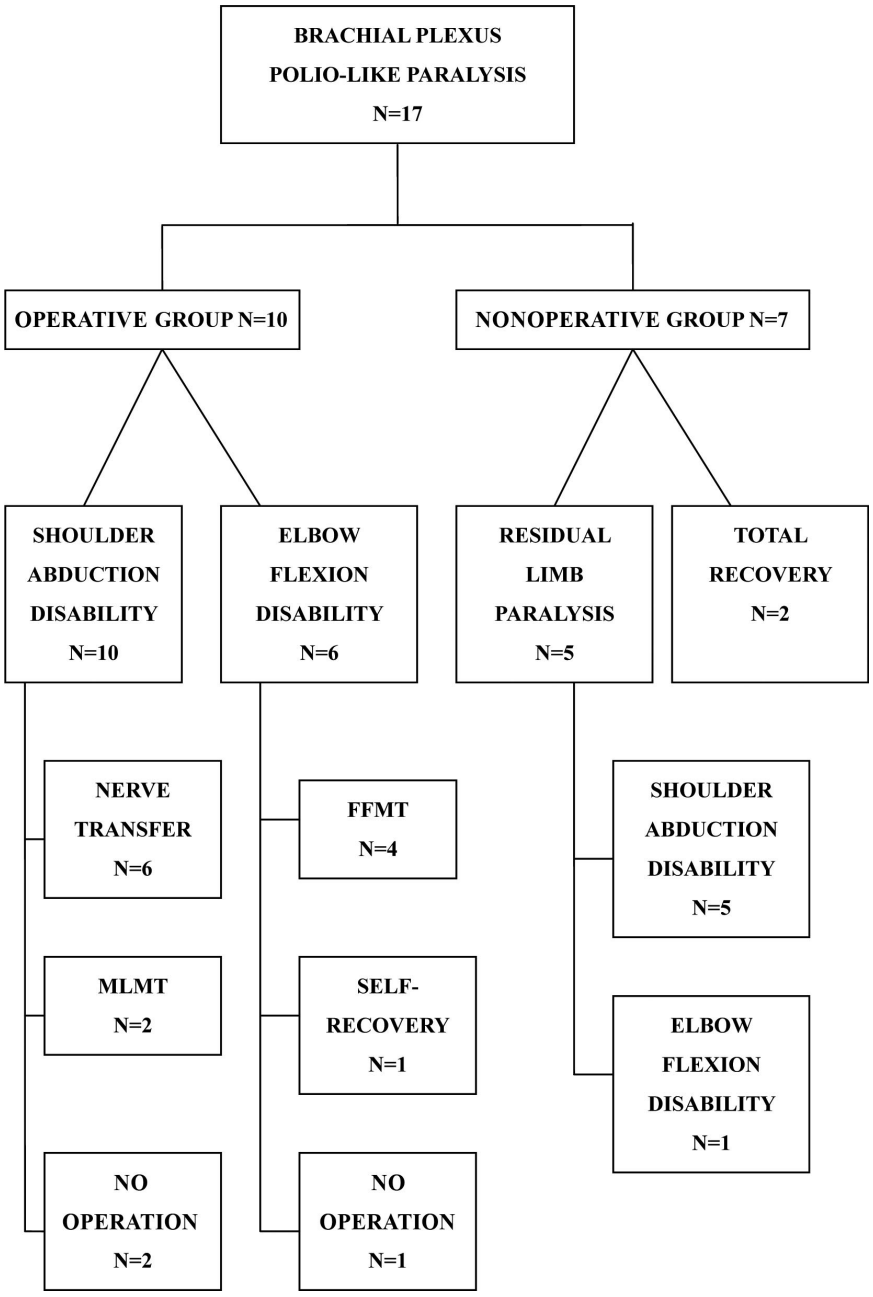

Fig. 1. Schematic review of the patients and their clinical courses (*FFMT*, functioning free muscle transplantation; *MLMT*, multiple local muscle transfers).

RESULTS

In the surgical group (10 patients) (Table 1 and Fig. 1), the stage V shoulder abduction (>160 degrees) was regained in five patients who underwent early nerve transfer within 1 year of paralysis (Fig. 3). One patient had a poor result, remaining with stage 0 shoulder abduction. This patient (case 3) had undergone phrenic nerve transfer to the suprascapular nerve after 3 years of shoulder palsy. Single or double neurotization did not seem to make a difference. Two patients achieved mild improvement, from stage I to stage II in shoulder abduction after multiple local muscle transfers (Fig. 4). In four patients, the functioning free muscle transplantation for elbow flexion reached the muscle strength of M4. The surgical reconstruction of both shoulder abduction by nerve transfer and elbow flexion by functioning free muscle transplantation was accomplished in two cases (cases 2 and 3). One achieved satisfactory

Table 3. Methods of Nerve Transfer

Method	Procedure	Cases
I	Phrenic to axillary with nerve graft, plus spinal accessory nerve to suprascapular	1 and 10
II	Phrenic to posterior division of upper trunk, plus spinal accessory nerve to suprascapular	2
III	Spinal accessory nerve to suprascapular	8 and 9
IV	Phrenic to suprascapular	3

results in both shoulder and elbow flexion (case 2) (Fig. 5). The other (case 3) only achieved good result with elbow flexion, but remaining with permanent shoulder disability. Nerve transfer after 3 years of shoulder palsy was ineffective.

In the nonsurgical group (seven patients) (Table 2 and Fig. 3), two had complete spontaneous recovery from distal to proximal musculatures within 1 year (cases 13 and 15). Five patients (71 percent), however, had permanent shoulder disability at an average of 10 years' clinical follow-up (range, 4 to 24 years) (Fig. 6). Four patients (90 percent) had spontaneous recovery of elbow flexion. One (case 16) remained with permanent M0 elbow flexion after 5 years' follow-up.

DISCUSSION

Flaccid limb paralysis is the distinctive feature of damage to the lower motor neurons, either in the anterior horn of the spinal cord or in the peripheral nerves. There are two major differential syndromes of spontaneous acute flaccid limb paralysis: (1) poliomyelitis or polio-like syndrome[1–7], which consists of viral infection and inflammation that directly damages cell bodies of the lower motor neurons in the anterior horn of the spinal cord; and (2) Guillain-Barré syndrome[15], an immunologically mediated parainfectious or postinfectious process causing damage to the lower motor neurons in the peripheral nerve or nerve roots. In general, Guillain-Barré syndrome can be distinguished from poliomyelitis or polio-like paralysis by clinical manifestations and neurophysiologic tests (Table 4). Guillain-Barré syndrome typically occurs 1 to 2 weeks after an acute infection, with an ascending symmetrical weakness. The nerve conduction velocities of both sensory and motor nerves show delayed distal latencies and reduced velocities. Conversely, in poliomyelitis or polio-like syndrome, asymmetrical weakness predominantly involves proximal muscles. These patients always have prodromal symptoms (fever, rash, or diarrhea) before the flaccid limb paralysis. The latent period for the paralysis is usually 3 to 5 days, but not more than 1 week. There are no sensory manifestations in polio or the polio-like syndrome.

The diagnosis of polio-like paralysis is based primarily on clinical manifestations and viral culture or viral serologic tests for confirmation, and assisted by neurophysiologic tests and magnetic resonance imaging. Nerve conduction velocities reveal decreased muscle action potential and electromyography shows positive sharp waves and fibrillation. No poliovirus infection or traumatic injury can be identified in these patients. Although it is difficult to localize the precise neuroanatomical impairment, the electromyographic and nerve conduction velocity data may suggest involvement of the anterior horn cells. Hyperintensities over the anterior horn of the spinal cord on T2-weighted magnetic resonance imaging confirm that the muscle weakness and paralysis are secondary to destruction of nerve cells in the anterior horn (Fig. 2).[5,6] Nevertheless, it is not possible to show these specific magnetic resonance imaging findings in every case.

Many pathogens have been reported in the literature that can cause polio-like paralysis. The polio virus has almost been eradicated. Enterovirus-71 has become the major pathogen of polio-like paralysis in Taiwan.[4–7] Enteroviruses consist of 68 serotypes and usually cause a self-limited infection in children. Enterovius-71 was first isolated in California in 1969.[4] Since then, enterovius-71 has been isolated in many parts of the world. The clinical picture of the infection varies from self-

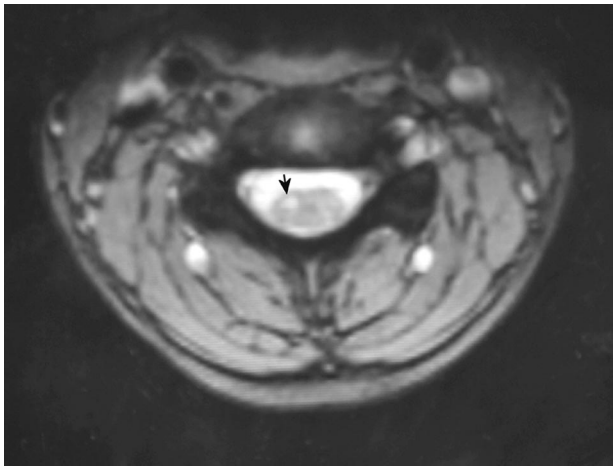

Fig. 2. Magnetic resonance imaging scan of the patient in case 11. The hyperintense area is marked by an *arrow* representing the lesion on the anterior horn at the C5 level.

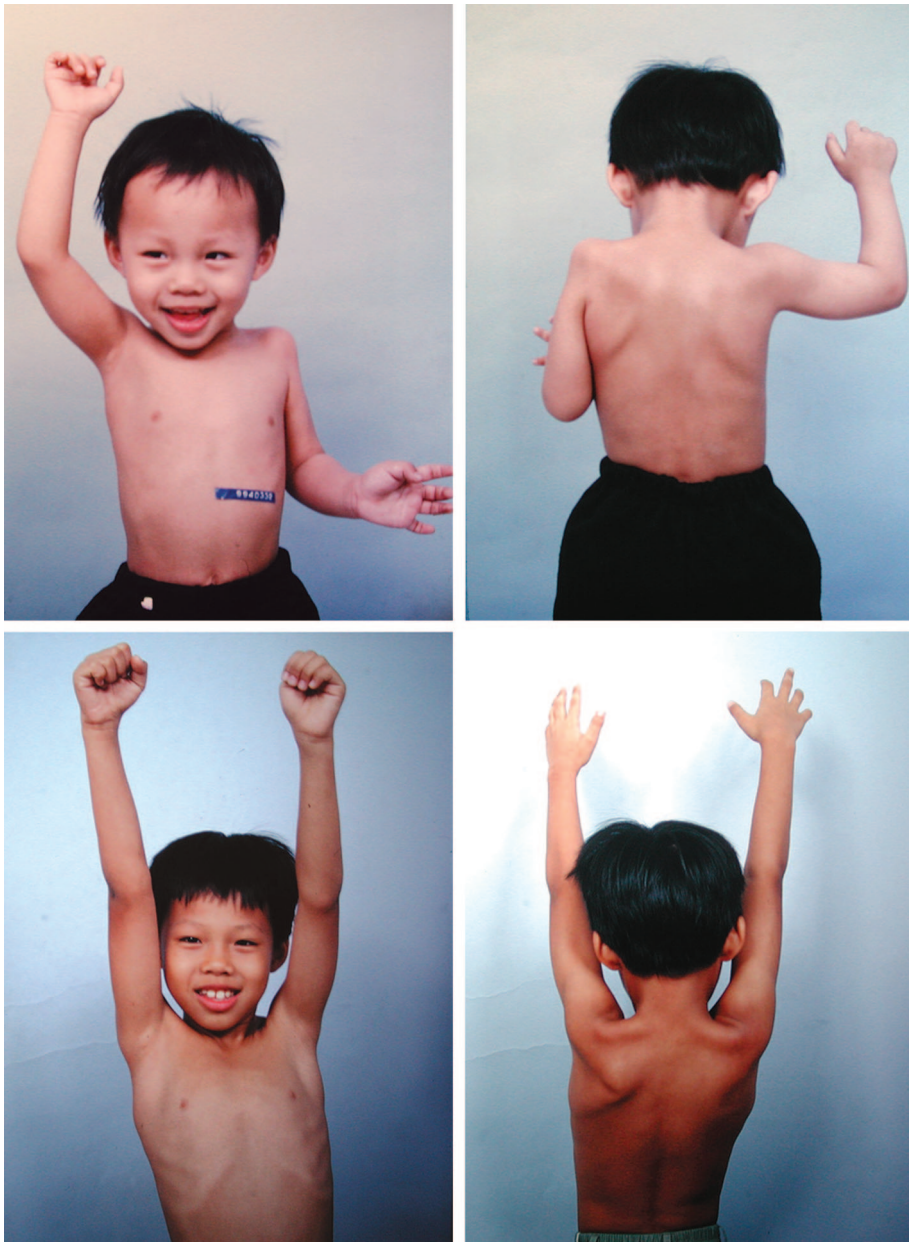

Fig. 3. Case 1. The patient presented with left shoulder paralysis and subsequently underwent nerve transfer for shoulder abduction. Preoperative views (*above*) and 5-year postoperative views (*below*).

limited foot-mouth-hand disease and herpangina to fatal meningitis, pulmonary edema, and limb paralysis. An outbreak of enterovius-71 infection occurred in Taiwan in 1998 and infected over 129,000 patients. Seventy-eight fatalities occurred as a result of neurogenic pulmonary edema. Twenty-five deaths in 2000 and 26 deaths in 2001 were subsequently reported.[7] Acute flaccid limb paralysis mimicking poliomyelitis was also noted during this outbreak, but the total number of affected patients has not yet been reported.

The pathogenesis of polio-like paralysis seems to be similar to that of poliomyelitis.[7,16] The virus can attack the anterior horn cells of the spinal cord in two ways. First, it may invade anterior horn cells directly through viremia. From the portal of entry (the mouth), the virus implants and multiplies in the tonsils of the oropharynx and the lymph nodes of the small intestine. Eventually the virus spreads by way of the bloodstream to the anterior horn cells. Enterovius-71 invades only certain types of nerve cells. During the process of

Fig. 4. Case 6. The patient had bilateral shoulder paralysis but intact elbow flexion and underwent multiple local muscle transfers for a deficit in left shoulder abduction. Preoperative views (*above*) and 4-year postoperative views, showing mild improvement of left shoulder abduction (*below*).

intracellular multiplication, the virus may damage or completely destroy the cells, leading to permanent limb weakness. A second type of anterior horn cell dysfunction occurs if the virus temporarily disables the cell. In this type, function may recover completely within several months in mild cases. Why enterovius-71 attacks only certain motor neurons, why recovery is from distal to proximal, and why proximal muscles innervated by C5 and C6 spinal cords are prominently involved are still debated.

Traumatic brachial plexus injury can be classified into four distinct levels as follows: level I, preganglionic root; level II, postganglionic spinal nerve; level III, supraclavicular brachial plexus; and level IV, infraclavicular brachial plexus.[17] The level of injury determines the surgical considerations and prognosis. Poliomyelitis-like paralysis can be considered as a level I brachial plexus neuropathy. Therefore, nerve transfers are the only possible alternative for nerve repair in these cases. In our study, the majority of patients had C5 or C6 motor territory involvement with disability of shoulder abduction and/or elbow flexion. Surgical priority in the polio-like brachial plexus paralysis should be given to restoration of shoulder abduction rather than elbow flexion. Early nerve transfer within the first year of paralysis, with either single or double neurotization, offers a good opportunity to regain adequate stage V shoulder abduction. Late nerve transfer (>1 year) or palliative reconstruction with muscle/tendon transfer or shoulder arthrodesis will not achieve more

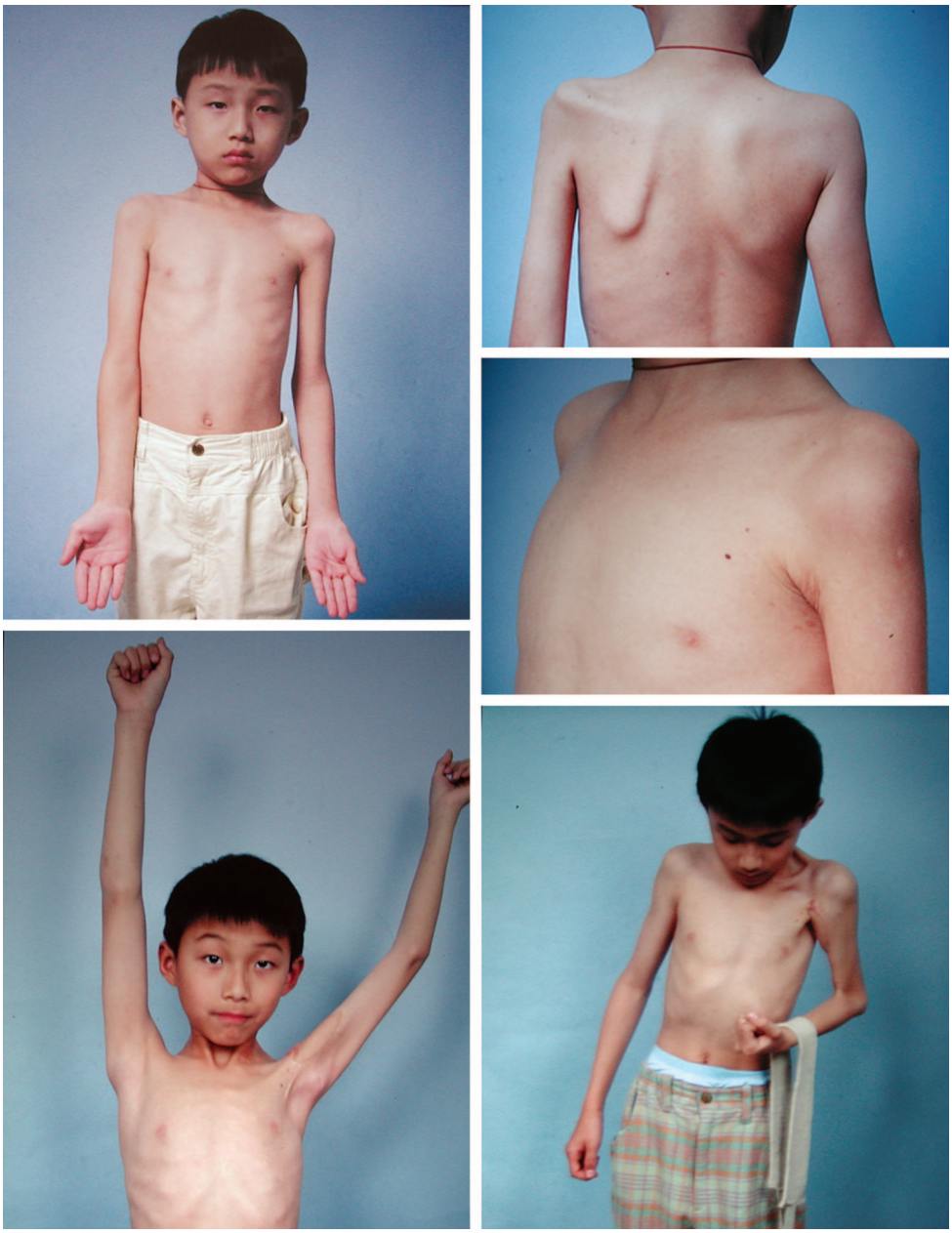

Fig. 5. Case 2. The patient presented with atrophy of the left shoulder and elbow muscles (*above*). He underwent nerve transfer for shoulder abduction and gracilis functioning free muscle transplantation for elbow flexion in different stages. Shoulder elevation (*below*, *left*) and elbow flexion (*below*, *right*) 4 years after elbow reconstruction.

than 90 degrees of shoulder abduction. Deficit in elbow flexion has a high likelihood to spontaneous recovery [four of five (90 percent in the nonsurgical group)] (Table 2). This is why surgical intervention to restore elbow flexion is not as urgent as that to restore shoulder abduction. Functioning free muscle transplantation for elbow flexion can be performed at any time and achieve a good result. Our success in achieving shoulder abduction by nerve transfer within 1 year of paralysis in polio-like paralysis was 100 percent (five of five), which is better than the outcomes following traumatic brachial plexus injury in adults,[18,19] which is more unpredictable.

Local muscle and tendon transfers have been used successfully for obstetrical brachial palsy.[12,17] In our series, however, two patients (cases 6 and 7), each of whom had undergone three local mus-

Fig. 6. Case 11, nonsurgical group. The patient presented with shoulder paralysis. Photographs obtained 5 years (*above*) and 8 years (*below*) after onset of the disease show permanent deficit of shoulder abduction.

Table 4. Comparison of Guillain-Barré Syndrome and Poliomyelitis or Polio-Like Paralysis

	Poliomyelitis or Polio-Like Paralysis	Guillain-Barré Syndrome
Onset time	Rapid onset (several days after acute illness)	Gradual onset (1–2 wk after acute illness)
Motor involvement	Asymmetrical flaccid predominantly involving proximal muscles	Ascending symmetrical weakness
Sensory involvement	No	Yes
Electromyography	Positive sharp waves and fibrillation	Positive sharp waves and fibrillation
Nerve conduction velocities	Low compound muscle action potential, normal sensory nerve action potential	Delayed distal latencies and reduced velocities in sensory and motor nerves

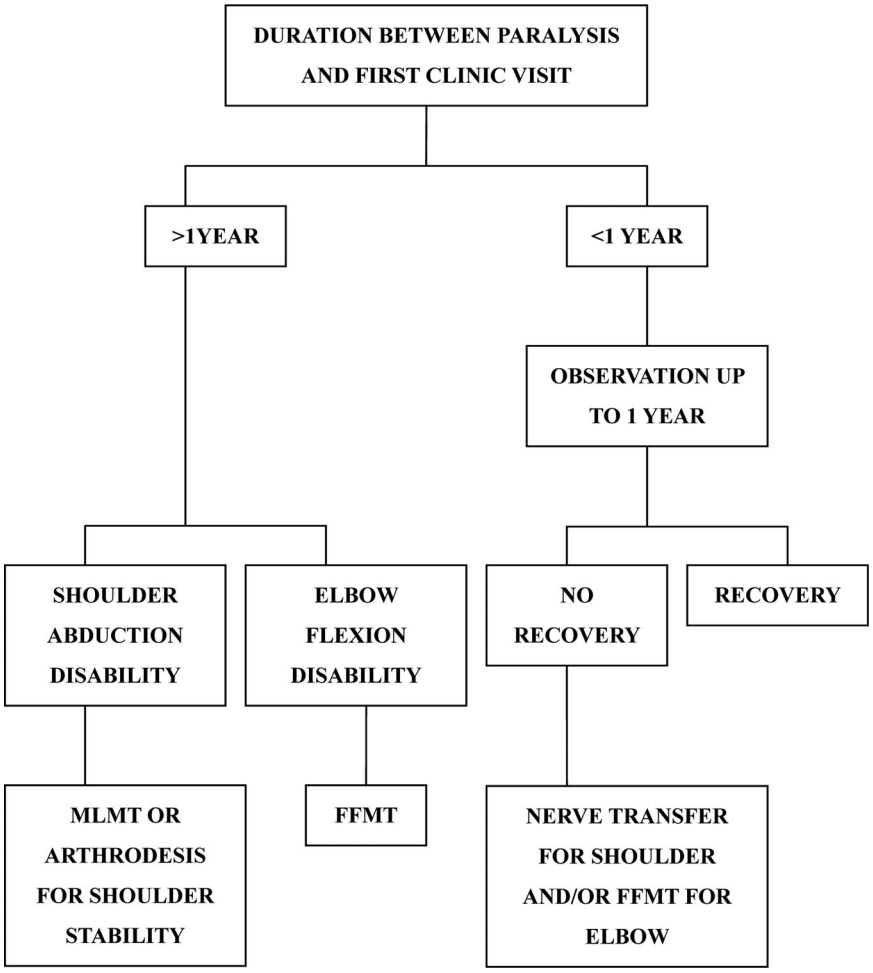

Fig. 7. Summary of the surgical strategies for polio-like brachial plexus palsy (*FFMT*, functioning free muscle transplantation; *MLMT*, multiple local muscle transfers).

cle transfers (trapezius, levator scapular and latissimus dorsi muscles) to correct shoulder abduction after 11 years of shoulder palsy, obtained only mild improvement. In the polio-like paralysis, it was difficult to achieve satisfactory shoulder abduction by one or multiple local muscle transfers because of damage to the motor neurons of the spinal cord. This high level of injury may influence the entire shoulder muscle group, including the transferred muscles, to varying degrees. Clinical evaluation of muscle strength around the shoulder and neck may not be adequate. Although these transferred muscles are seemingly powerful on physical examination, they may not actually be strong enough for transfer. Shoulder arthrodesis is another method for providing shoulder stability but yields a poor range of motion.[20,21] Because multiple local muscle transfers or shoulder arthrodesis were not better than nerve transfers for functional restoration of the shoulder in patients with polio-like paralysis, nerve transfer is now strongly recommended in the early stage (within 1 year) of shoulder paralysis.

Nerve transfers for shoulder abduction have been attempted using a variety of donor nerves, including the phrenic nerve, the spinal accessory nerve, and motor branches of the cervical plexus.[18,19,22–24] In our series, we preferred to use spinal accessory nerve and phrenic nerve transfer for restoration of shoulder abduction. There are several advantages to using the spinal accessory nerve. First, transfer of only the distal portion of the spinal accessory nerve offers powerful reinnervation and preserving the proximal branches to the upper trapezius preserves shoulder function. Second, this transfer is synergistic because the trapezius normally provides scapular rotation along with shoulder abduction, making postoperative rehabilitation relatively straightforward. The phrenic nerve is also a good choice for the donor nerve. Terzis and Papakonstantinou demon-

strated that the number of myelinated axons in the spinal accessory nerve ($n = 2145$) and the phrenic nerve ($n = 1756$) is greater than the number of motor axons in the intercostal nerve ($n = 1093$) and the cervical plexus donors ($n = 893$).[23] Postoperative respiratory distress after the use of the phrenic nerve is not a problem in children older than 2 years. However, one should be aware of the possibility of respiratory distress in infants. Chuang et al. previously reported one infant suffering from severe respiratory distress after harvest of the phrenic nerve.[18]

In a meta-analysis of nerve transfer for the restoration of shoulder function, Merrell et al. reported that the suprascapular nerve was a significantly better recipient nerve than the axillary nerve.[24] In five patients who underwent nerve transfer using either single or double neurotization within 1 year of paralysis, excellent shoulder abduction (Gilbert stage V) was regained. The only poor result in the nerve transfer group was in a patient who was operated on 3 years after the onset of paralysis. Even though nerve regeneration in children occurs over a relatively shorter distance with a stronger potential for recovery and a greater capacity for brain adaptation than in adults,[25] nerve transfers in delayed cases may not offer satisfactory results because of target muscle atrophy and motor endplate degeneration.

Functioning free muscle transplantation is a good option for restoration of elbow flexion.[26] The gracilis is an ideal muscle for arm or forearm muscle reconstruction because of its size, shape, long single dominant vascular pedicle, long single innervated motor nerve, good excursion, and muscle strength.[26,27] The gracilis functioning free muscle flap innervated by T3 to T5 intercostal nerves was preferred for restoration of elbow flexion in our series (Fig. 7). Although latissimus dorsi transfer is also an option for the restoration of elbow flexion,[28] the latissimus dorsi muscle may not be reliable in this series because of its innervations by C5, C6, and C7. Gutowski and Orenstein[29] reported no pulmonary complications after phrenic nerve transfer, even when the intercostal nerves were concurrently transferred. Similarly, in one of our cases (case 3), nerve transfers of both the spinal accessory nerve and the phrenic nerve for shoulder abduction and elbow flexion was reconstructed by using gracilis functioning free muscle transplantation innervated by three intercostal nerves. Even though both the phrenic nerve and T3 to T5 intercostal nerves were used, respiratory problems were not observed.

CONCLUSIONS

Brachial plexus polio-like paralysis syndrome is a term that describes an acute flaccid paralysis of the upper limb caused by a virus other than poliovirus. The lesion is located on motor neurons of the anterior horn of the spinal cord; C5 and C6 motor neuron involvement compromises shoulder abduction and elbow flexion. On the basis of this study, a surgical strategy for polio-like brachial plexus palsy was developed (Fig. 7). The major contributing factor in the decision regarding surgical approach is the duration of paralysis. If the interval is less than 1 year, we advise continued observation for up to 1 year after the onset of paralysis. If improvement is not detected during the observation period, nerve transfer to restore shoulder abduction is urgently recommended. Functioning free muscle transplantation for elbow flexion can be performed simultaneously with shoulder nerve transfer in a one-stage or, separately, as a two-stage procedure. However, if the shoulder paralysis persists more than 1 year after the onset of paralysis, nerve transfer is ineffective. Multiple local muscle transfers or arthrodesis may be helpful for the instability of the shoulder. In the event of permanent weakness in elbow flexion, gracilis functioning free muscle transplantation can be performed for elbow flexion, usually after the age of 4 years.

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DISCLOSURE

None of the authors has a financial interest in any of the products, devices, or drugs mentioned in this article.

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