Bilateral brachial plexus palsies due to malpositioning after burn injury

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We reported a case of a 62-year-old man who sustained bilateral brachial plexus palsies resulting from malpositioning while being restrained due to agitation after burn injury. According to the clinical and EMG findings, we selected conservative treatment with rehabilitative intervention. Approximately 1 year after the injury, the patient became able to eat meals, dress himself, and use the toilet independently. To prevent brachial plexus injury in the supine position, the arms should be abducted and flexed to less than 90°. Clinicians should be vigilant regarding positioning when patients must be restrained.

Key words: Brachial Plexus Palsy, Burn, Malpositioning, Rehabilitation, Traction

Although many injurious agents can damage the brachial plexus, they act through a limited number of mechanisms. Traction is the most common, particularly in severe lesions. In adults, the principal causes of traction injuries are traumatic (e.g., vehicular accidents). Characteristically, traction injury in adult causes unilateral lesion [1]. Bilateral lesions are much less common and have been reported only in situation such as classic postoperative paralysis, post-medial sternotomy, pack palsy, and neuralgic amyotrophy [2].

We reported a patient who sustained bilateral brachial plexus palsies due to malpositioning after a burn injury. It is rare for it to occur in this manner.

We presented the patient's clinical course and discussed the onset mechanism.

CASE PRESENTATION

The patient was a 62-year-old, right-handed man. Past medical history was unremarkable and lipid status was normal. He was intoxicated with alcohol at the time of the injury, which was sustained when he mistakenly entered the boiler room of a bathhouse. On arrival at the emergency hospital, he had 3rd degree burns extending from the back to the gluteal region and the dorsal aspects of the upper and lower limbs. A total of 30% of the body surface area was involved. Because the patient was delirious and agitated, he was restrained by the 4 extremities in the supine position. The following day, he demonstrated severe weakness of both upper limbs. He had been prescribed with rehabilitative intervention like as range of motion (ROM) exercise and low frequency electrical stimulation from the onset. Five months after the injury, he was transferred to our hospital for furthermore rehabilitation. He had already undergone the surgical treatment of debridement and skin grafting for the burned skin.

Physical findings on admission

The patient was alert and well oriented regarding time, place, and person. Examination of cranial nerve function revealed no abnormality, and Horner's sign was absent. Mild edema was present on the dorsum of the both hands. Meshed skin grafts from both thighs were evident on the back, gluteal region, dorsal aspects of both arms, and the dorsal and plantar aspects of the right foot (Fig. 1). The grafted skin did not exhibit redness, swelling, or hypertrophy. Weakness of both upper extremities was evident. Results of manual mus-

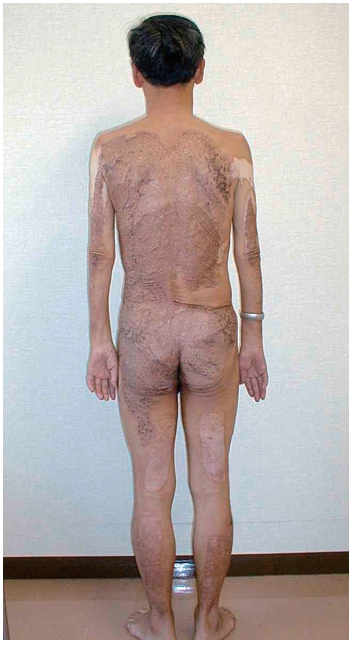

Fig. 1 Appearance of burn injuries.
Skin on the back, gluteal region, dorsal aspect of both arms, and the dorsal and plantar aspects of the right foot are covered by meshed skin grafted from his both thighs.

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Table 1 Nerve Conduction Studies Findings.

Nerves	Distal latency (ms)	Amplitude (mV) (distal/proximal)	Conduction velocity (m/s)
Motor			
Rt Median	4.25	0.2/0.1	33.8
Lt Median		NER	
Rt Ulnar	3.25	0.8/0.7	42.3
Lt Ulnar	3.7	0.1/0.2	36
Rt Musclocutaneous		NER	
Rt Axillary		NER	
Sensory			
Lt Median		NER	
Rt Ulnar		NER	
Rt Radial		NER	

Rt, right; Lt, left; NER, no evoked response

Table 2 Needle Electromyography Findings.

Muscles	Fibs	PSWs	Amplitude (mV)	Duration (ms)	Poly.	Int. Patt.
Rt. Serrat. Ant.	−	−	0.2-3.0	4-10	+	++
Lt. Serrat. Ant.	−	−	0.2-2.0	4-10	+	++++
Rt. Delt.	2	3	0.1-1.5	5-18	+++	++
Lt. Delt.		1	0.1-2.0	8-20	+++	+++
Rt. Biceps Brachii	2	2-3	0.1-0.5	10-20	++++	++
Lt. Biceps Brachii	2	2-3	0.1	12	++++	1MUAPs
Rt. Triceps Brachii	2	2-3	0.1-0.5	5-20	++++	++
Lt. Triceps Brachii	1	1-2	0.1-0.5	5-15	++++	++
Rt. APB	3	2	0.1-1.5	12-14	++++	++
Lt. APB	3	3	0.2	10	+	1MUAPs
Rt. 1st DI	3	3	0.1-2.0	10	++	++
Lt. 1st DI	3	3	E.S			

Fibs and PSWs result from excitation of individual muscle fibers and represent membranous instability.
Poly., appearance of polyphasic MUAPs; +, ++, +++, ++++ indicate that less than 25%, 25 to 50%, 50 to 75%, more than 75% of MUAPs are polyphasic, respectively.
Int. Patt., interference pattern, ++++ means that we cannot recognize base line in oscilloscope by occupation with MUAPs in maximum contraction. 1MUAPs means that we can recognize 1 MUAPs. ++ and +++ indicates the qualitative classification based on the extent of MUAPs appearance.
Fibs, fibrillation potentials; PSWs, positive sharp waves; Poly, polyphasic unit; Int. Patt, interferens pattern; Serrat. Ant, serratus anterior; Delt, deltoid; APB, abductor pollicis brevis; 1st DI, 1st dorsal interosseous

cle testing were as follows: trapezius, serratus anterior, and muscles of the lower extremities, 5/5 bilaterally; shoulder flexors, 2/5; elbow flexors and extensors, and wrist extensors, 0/5. Grip power was bilateral 0 Kg. Sensory examination revealed hypesthesia to touch and pain in the C5-T1 dermatome. Deep tendon reflexes were normal and symmetric, and no pathologic reflexes were elicited. As to passive range of motion, the shoulder flexion was limited to 90°/80° (right/left), and both forearms supination, 0°. He could stand up from bed and walk independently, but he could not to eat, dress, toilet or bathe unaided. His Barthel index score was 66.

EMG (electromyography) findings and electrodiagnosis

Nerve conduction studies showed decreased CMAP (compound muscle action potential) amplitude and reduced conduction velocity for the right median and bilateral ulnar motor nerves. No CMAP of APB (abductor pollicis brevis), Biceps Brachii, deltoid could be obtained following stimulation to the left median, right musculocutaneous, and right axillary nerves, respectively. No sensory nerve action potential was obtained from the nerves examined (Table 1).

Needle electromyography (Table 2) revealed membrane instability in the muscles innervated by C5-T1. Severe polyphasic motor unit action potentials (MUAPs) and decreased interference pattern were observed in the same muscles on voluntary contraction. So called "nascent motor unit potentials" were bilaterally recognized in deltoid. Serratus anterior exhibited normal findings bilaterally.

These findings were consistent with bilateral brachial plexus lesions characterized by axonal degeneration distal to the division of long thoracic nerve.

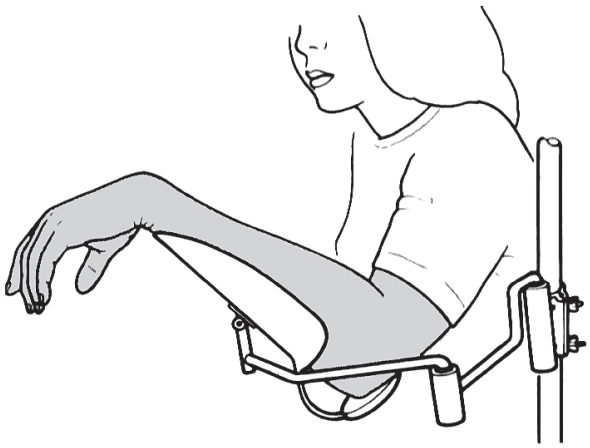

Fig. 2 Mobile Arm Support (MAS) [3]. This orthosis is designed for the patient with shoulder and elbow muscle weakness like as cervical spinal injury, progressive muscular atrophy and so on. The patient can eat dishes independently with this device.

Table 3 Number of cases showing Neuromuscular Problems [5].

Problems	Type of burn		Total
	Nonelectrical	Electrical	
Generalized peripheral neuropathy	31	15	46
Deltoid injection injury	15	4	19
Peroneal nerve palsy	8	2	10
Ulnar nerve palsy	6	1	7
Brachial plexus stretch	5	1	6
Median nerve stretch or compression	5	1	6
Radial nerve compression	0	1	1
Long thoracic nerve compression	1	0	1
Suprascapular nerve stretch	1	0	1
Posterior tibial nerve compression	1	0	1
Cervical radiculopathy	0	3	3
Miscellaneous	0	10	10
Uncertain	6	0	6
Total	79	38	117

Rehabilitative intervention and clinical course

As the burns did not extend to the axillary regions and weakness of both upper limbs was not apparent on admission in the emergency hospital, we excluded the possibility of the direct burn injury of the brachial plexus. According to the clinical and EMG findings, we diagnosed bilateral brachial plexus palsies due to malpositioning after the burn injury. Since the motor and sensory function of the upper limbs had been gradually improving, we selected conservative treatment. ROM exercises and functional electrical stimulation (FES) were performed to avoid joint contracture and muscle atrophy due to disuse.

The patient started exercise for using a mobile arm support [3] (MAS) to eat meals independently (Fig. 2). One month after that, he could eat semi-solid food using the MAS and a wrist-extension cuff with a pocket although his muscle strength did not significantly improve. Seven months after the injury he could eat all meals in his room independently, using the MAS. Approximately 1 year after the injury, manual muscle testing revealed improvement of the muscle strength: shoulder flexors, 4/5 bilaterally; elbow flexors, 3/5 bilaterally; elbow extensors, 4/5 bilaterally; and wrist extensors, 3/5 right, 1/5 left. Passive ROM also gradually improved: shoulder flexion to 160°/150°, forearm supination to 45°/30° (right/left). Eventually the patient became able to eat meals without the MAS, dress himself, and use the toilet independently. Barthel index score improved to 86. The patient was discharged home at this point.

DISCUSSION

1. Neuromuscular complications of burn injuries

Henderson reported that peripheral polyneuropathy was found in 15% of burn patients, with a higher incidence among patients with burns affecting more than 20% of the body [4]. Helm clinically and electrodiagnostically investigated 88 burn patients with weakness or complaints of easy fatigability [5]. Generalized neuropathy was the most frequently diagnosed as the neuromuscular abnormality, while brachial plexus palsy was the fifth most common (Table 3). They attributed this complication to incorrect positioning of the shoulder.

As mentioned earlier, the burn did not extend to the axillary and supraclavicular regions and he did not demonstrate the weakness of both upper limbs on admission in the emergency hospital, we excluded the possibility of the direct burn injury of the brachial plexus. The burn was very severe and intensive treatment had been done on ventilator support for first several weeks, so we could not completely deny the possibility of negative influence of metabolic complica-

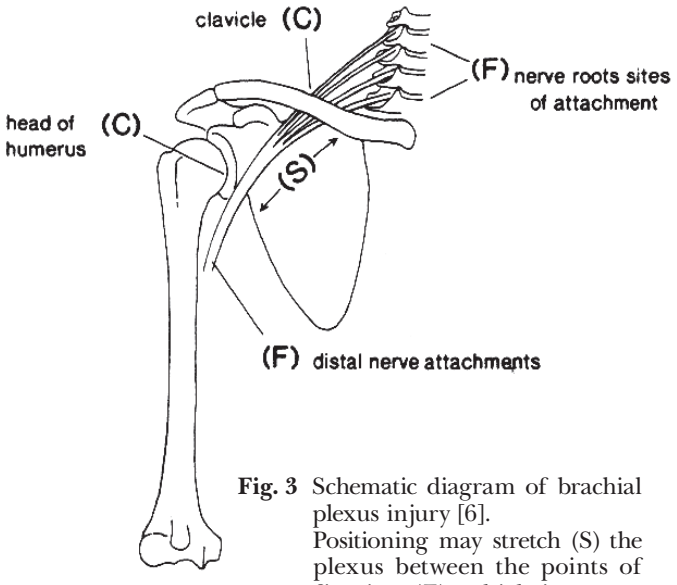

Fig. 3 Schematic diagram of brachial plexus injury [6].
Positioning may stretch (S) the plexus between the points of fixation (F), which increases the likelihood of compression injury at several sites (C).

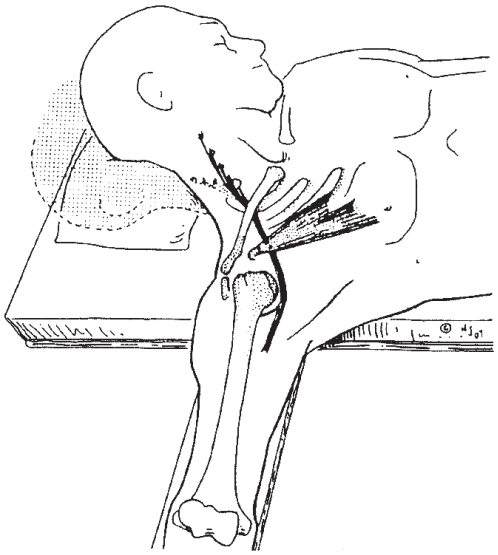

Fig. 4 Incorrect supine position [7].
Extension and lateral flexion of the head to the opposite side stretches the brachial plexus.

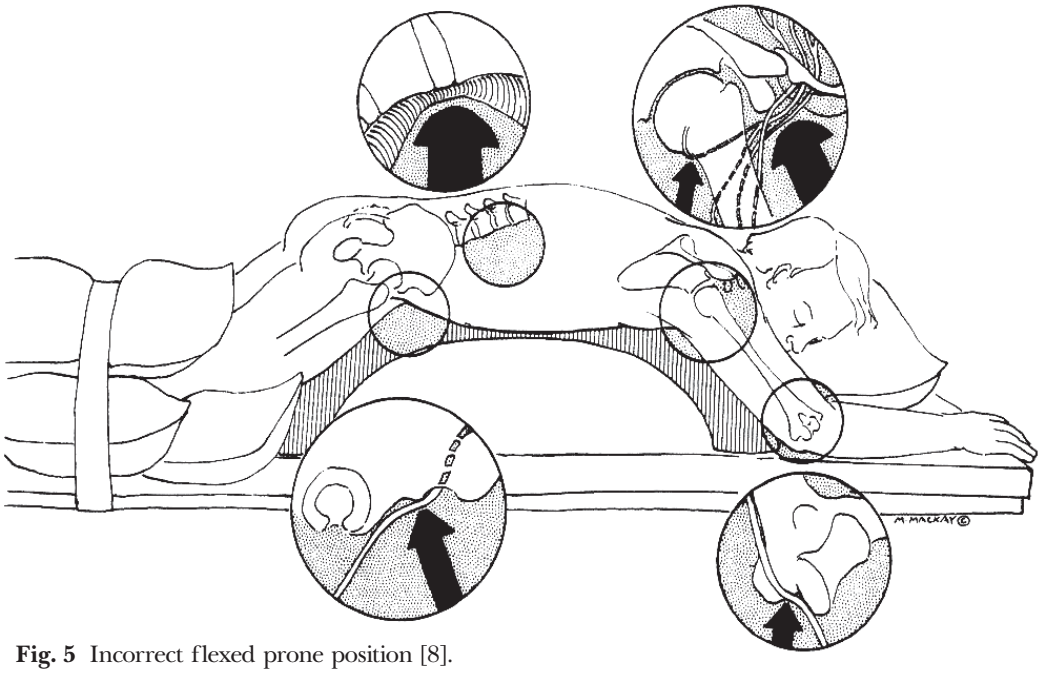

Fig. 5 Incorrect flexed prone position [8].
Abduction and flexion of the arms stretches the brachial plexus.

tions and neurotoxic drugs on the nerve function.

2. The mechanism of brachial plexus injury due to malpositioning

The brachial plexus has a relatively long, mobile, and superficial course in the axilla between two firm points of fixation: the vertebrae and prevertebral fascia above; and the axillary fascia below (Fig. 3). It also lies in proximity to a number of mobile bony structures. Accordingly, the brachial plexus is the nerve structure most susceptible to damage from malpositioning during anesthesia [6].

On admission to the emergency hospital, the present patient was restrained in the supine position by the four extremities for 36 hours, because he was intoxicated and agitated. According to the nursing records and his wife, his upper extremities were constrained with extreme abduction and flexion. In this position, brachial plexus was possibly stretched behind the clavicle and tendon of the pectoralis minor and below the head of the humerus. Moreover, the patient was noted to struggle for several hours. This behavior might exacerbate the brachial plexus injury because extension and lateral flexion of the head increases the angle between the head and the contralateral shoulder tip and thereby stretches the plexus [7] (Fig. 4).

The patient underwent debridement and extensive skin grafts. Therefor, he remained sedated postoperatively in bed in the flexed prone position for 7 days. It is possible that this condition further exacerbated the brachial plexus palsy (Fig. 5) [8].

We assumed these mechanisms caused brachial

plexus injury bilaterally and symmetrically.

To prevent brachial plexus injury in the supine position, the arms should be abducted and flexed to less than 90°. In the prone position, the arms should be kept as close as possible to the patient's side, with the elbow flexed, forearm pronated, and the hands placed on either side of the head [8].

3. Prognosis of the brachial plexus injury due to malpositioning

Since brachial plexus palsy due to malpositioning is relatively mild and transient, few case with the complication has been reported. In this case, the patient transferred to our hospital five months after the onset, he remained the severe weakness of both upper limbs although intensive rehabilitative intervention had been done in an emergency hospital. Fortunately, according to the EMG findings, root avulsion was excluded. We recognized the findings representing axonal degeneration on needle electromyography, so we expected the palsies were long time in recovering. Furthermore the patient showed sign of improvement in the emergency hospital, we anticipated improvement from palsy though starting rehabilitation in our hospital five months after the onset.

CONCLUSION

We report a case of a 62-year-old man with bilateral brachial plexus palsies that developed after his arms were malpositioned during restraint. To prevent brachial plexus injury in the supine position, the arms should be abducted and flexed to less than 90°. When restraint is unavoidable, clinicians must be vigilant regarding patient positioning in order to avoid brachial plexus palsy.

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