Combined injuries of the brachial plexus and spinal cord

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A total of 11 patients with combined traumatic injuries of the brachial plexus and spinal cord were reviewed retrospectively. Brachial plexus paralysis in such dual injuries tends to be diagnosed and treated late and the prognosis is usually poor. The associated injuries, which were all on the same side as the plexus lesion, were to the head (nine cases), shoulder girdle (five), thorax (nine) and upper limb (seven). These other injuries were responsible for the delayed diagnosis of brachial plexus paralysis and the poor prognosis was probably because of the delay in starting treatment and the severity of the associated injuries. When such injuries are detected in patients with spinal cord trauma, it is important to consider the possibility of involvement of the brachial plexus.

Combined trauma involving the brachial plexus and spinal cord is very rare^{1,2} and has mostly been reported as single cases.³⁻⁹ The mechanism of the dual injury is unknown and treatment is difficult. The brachial plexus component tends to be diagnosed and treated late and the result of its surgical treatment in such circumstances has not been reported.

Our aim was to determine the clinical features of patients with combined injury to the brachial plexus and spinal cord, discuss the mechanisms involved and assess the outcome of surgical treatment for the brachial plexus component.

Patients and Methods

Between 1990 and 2004, we treated 1523 patients with paralysis as a result of injury to the spinal cord. Of these, 11 had combined injuries of the brachial plexus and spinal cord.

We examined these patients and reviewed their records and radiological investigations, including myelograms, CT and MRI scans.

The grading of brachial plexus paralysis before and after surgery was by the Medical Research Council (MRC) system. Useful arm function was regarded as grade 3 or better. We also evaluated function according to Sedel,¹⁰ as follows: grade 1, manual work performed; grade 2, use of the limb in everyday activities but not strong enough for manual work; grade 3, usefulness very limited, for example as a paper-weight; grade 4, virtually useless but with some movement of elbow or fingers; grade 5, no recovery at all.

Results

The clinical details are given in Table I. All the patients were men, with a mean age of 27.6

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Table I. Details of the 11 patients

Case	Age at injury (yrs)	Cause of injury	Spinal cord lesion	Brachial plexus lesion	Time from injury to diagnosis of brachial plexus lesion (days)
1	32	Motorcycle accident	C4 central cervical spinal cord injury	Complete C5-T1	143
2	65	Fall from a height	C4 central cervical spinal cord injury	Incomplete C5/6	99
3	19	Motorcycle accident	C6 complete paralysis	Complete C5-T1	124
4	21	Fall from a height	C8 complete paralysis	Complete C5-T1	84
5	20	Motorcycle accident	T4 complete paralysis	Incomplete C5/6	149
6	20	Motorcycle accident	T3 complete paralysis	Incomplete C7-T1	108
7	23	Motorcycle accident	T4 incomplete paralysis	Complete C5-T1	186
8	20	Motorcycle accident	T4 complete paralysis	Complete C5-T1	75
9	20	Motorcycle accident	T4 complete paralysis	Complete C5-T1	30
10	25	Motorcycle accident	T3 complete paralysis	Complete C7-T1	106
11	39	Heavy steel object	T10 complete paralysis	Incomplete C5-C7	71

Fig. 1a

Fig. 1b

Case 1 – This patient had central cervical spinal cord injury at C4 and a complete left brachial plexus palsy from C5 to T1. The a) plain cervical radiograph and b) myelogram show separation of the fifth and sixth cervical vertebrae (arrows) on the same side as the brachial plexus lesion.

years (19 to 65) at injury. The causes of injury were: a motorcycle accident in eight patients, a fall from a height in two and being struck by a heavy steel object in one.

Brachial plexus lesion. This was complete C5-T1 in six patients, incomplete C5/6 in two, complete C7-T1 in one, incomplete C5-C7 in one and incomplete C7-T1 in one. The mean time from injury to diagnosis of the plexus lesion was 106.8 days (30 to 186). Ten patients were initially treated at another hospital, five of whom were diagnosed only after transfer to our institution. We evaluated the brachial plexus lesions from electromyograms, myelograms, CT scans and the operative findings.

There were two patients with either an incomplete C5/6 or a C7-T1 lesion who were managed conservatively with physiotherapy and analgesia, respectively. In both, useful arm function was achieved. The other nine patients underwent exploration, of whom seven had avulsion of the roots or other severe damage. There were six patients who had neurotisation or nerve grafting and three who underwent a neurolysis. The mean time from injury to exploration was 162.4 days (135 to 206). One patient who underwent exploration was excluded because the follow-up period was less than one year. The results in the remaining eight patients were poor, with four subsequently undergoing a musculotendinous transfer at a mean of 18 months after the brachial plexus surgery. The mean time from the last operation to the final follow-up was 6.1 years (2 to 12). At the final assessment, five of the eight patients had a useless limb (Sedel grade 4 or 5; Table II).

Injury to the spinal cord. In four patients, the spinal cord lesion was associated with a cervical vertebral injury. Two others had partial central-cord syndrome at C4, but without fracture. Of these, one had complete C5-T1 palsy and one a partial C5/6 palsy. The remaining two had complete paralysis at C5 and C8 and both had burst fractures of the cervical spine and a complete C5-T1 plexus palsy. Thus, for patients with injury to the cervical spine, damage to the spinal cord was located at the same level as the brachial plexus lesion (Table I). On plain cervical radiographs, three of the four patients with cervical vertebral injuries showed separation of the vertebrae on the same side as the plexus lesion (Fig. 1), which was a complete C5-T1 palsy in each case.

There were thoracic spinal injuries in seven patients, six of whom showed a complete, and one an incomplete paralysis. These comprised fractures of the mid-thoracic (six patients) and lower thoracic spine (one). In five of these seven patients, plain radiography of the thoracic spine showed lateral flexion of the thoracic vertebrae on the side opposite the brachial plexus lesion (Fig. 2).

Associated injuries (Table III). Nine patients had a head injury, most of which were severe, six of whom were unconscious for more than 24 hours. In the nine patients with thoracic injury, there was an associated head injury. Of the four patients with an injury to the cervical spine, three had a facial injury and the motorcycle helmet visor of the remaining patient was broken. In the five patients who had injury to the shoulder girdle on the same side as the plexus lesion, all had injury to the thoracic spine. Of the eight patients

Fig. 2a

Fig. 2b

Case 9 – This patient had complete spinal cord paralysis at T4 and a complete right brachial plexus palsy from C5 to T1. The a) plain thoracic radiograph and b) myelogram show lateral flexion of the thoracic vertebrae on the side opposite the brachial plexus lesion.

who had a thoracic injury on the same side as the brachial plexus lesion, seven had a fracture of the upper limb on the same side.

Discussion

The incidence of combined traumatic injury to the brachial plexus and spinal cord was reported as 0.6% by Grundy and Silver.² Over a period of 37 years, they encountered 4965 patients with traumatic paralysis of the spinal cord, 31 of whom had brachial plexus injury. Over a period of 15 years we encountered 1523 patients with traumatic spinal cord paralysis, 11 of whom had a brachial plexus injury (0.7%).

We believe that the principle mechanism of injury to the spinal cord is the force at the site of injury. Roaf³ reported four patients who had injury to the cervical spine and complete tetraplegia and brachial plexus paralysis. He speculated that such dual injury was mainly caused by lateral flexion, because radiographs of all four showed separation of the cervical vertebrae on the same side as the plexus lesion. Chechick et al⁴ reported that rotation and hyperextension of the cervical spine were probably the cause of Brown-Séquard syndrome and brachial plexus injury. McGowin et al⁶ reported that traction between the neck and shoulder was the most common cause of brachial plexus lesions, with the upper roots being subject to greatest tension. All the spinal cord injuries in our series were caused by direct spinal injury and no patient had paralysis resulting from secondary damage to the cord from avulsion

of the roots of the brachial plexus.^{7,8} Of our four patients with cervical spinal injuries, three had associated facial injury, while the fourth patient's helmet visor had been broken during the accident. Of these four patients, two had central-cord syndrome, which is the most common hyperextension injury of the cervical spine,¹¹ while three of the four showed separation of the cervical vertebrae on the same side as the brachial plexus lesion. We speculate that their combined injuries were mainly because of excessive hyperextension and lateral flexion.

Grundy and Silver² reported that the cervical and mid-thoracic spine is particularly vulnerable to excessive lateral flexion. Of the 22 thoracic spinal fractures which they examined, 13 involved T5 or T6. Since damage to the upper trunk of the brachial plexus is likely to be due to forcible separation of the shoulder and head by excessive lateral flexion of the cervical spine, traumatic injury to the brachial plexus is usually associated with both head injury and loss of consciousness.¹² In our series, all seven patients with a thoracic spinal fracture had a head injury, and five suffered excessive lateral flexion of the thoracic spine on the same side as the plexus lesion. Of the seven patients, five also had injury to the shoulder girdle on the same side as their plexus paralysis. It is possible therefore, that a strong direct blow to one side of the head may cause excessive lateral flexion of the cervical spine and brachial plexus paralysis. If the same force extends to affect the trunk, it could cause injury to the shoulder girdle and fracture of the thoracic spine, as

Table II. Outcome of surgery for brachial plexus lesions. Of the nine patients evaluated, one was excluded from evaluation of the surgical results because the follow-up period was less than one year

Case	Brachial plexus lesions by operative findings	Nerve surgery for brachial plexus lesion*	Musculotendinous transfer for brachial plexus lesions†	MRC grade‡		Sedel grade ¹⁰	Follow-up after last operation (yrs)
				Pre-operative	Post-operative		
1	Avulsion of C5-T1	ICN(3,4,5) to MCN transfer	-	SA 0 EF 0 EE 0 WE 0 WF 0 FE 0 FF 0	SA0 EF 0 EE 0 WE 0 WF 0 FE 0 FF 0	5	3
3	Rupture of C5 Avulsion of C6-T1	C5 – Posterior cord nerve graft; SAN to MCN transfer; DSN to SSN transfer	-	SA 0 EF 0 EE 0 WE 0 WF 0 FE 0 FF 0	SA 2 EF 1 EE 0 WE 0 WF 0 FE 0 FF 0	5	2
5	C5-T1 in continuity	Neurolysis	Latissimus dorsi – humerus	SA 0 EF 4 EE 4 WF 4 WE 4 FE 5 FF 5	SA 3 EF 5 EE 5 WE 5 WF 5 FE 5 FF 5	1	7
7	Avulsion of C5-T1	ICN(3,4,5) to MCN transfer	Trapezius – humerus	SA 0 EF 0 EE 0 WE 0 WF 0 FE 0 FF 0	SA 2 EF 3 EE 0 WE 0 WF 0 FE 0 FF 0	4	11
8	Rupture of C5/6 Avulsion of C7-T1	C5 – MCN graft; C5 – MN graft; C6 – AN graft; ICN(4,6,7) to RN transfer	Trapezius – humerus Triceps – biceps	SA 0 EF 0 EE 0 WE 0 WF 0 FE 0 FF 0	SA 2 EF 3 EE 0 WE 0 WF 0 FE 0 FF 0	4	12
9	Rupture of C5/6 Avulsion of C7-T1	C5 – Lateral cord nerve graft; C6 – Posterior cord nerve graft	-	SA 0 EF 0 EE 0 WE 0 WF 0 FE 0 FF 0	SA 2 EF 2 EE 0 WE 0 WF 0 FE 0 FF 0	4	9
10	C5/6 in continuity Avulsion of C7-T1	Neurolysis	FCR-EDC	SA 5 EF 5 EE 2 WE 0 WF 0 FE 0 FF 0	SA 5 EF 5 EE 3 WE 5 WF 3 FE 3 FF 3	2	3
11	C5-T1 in continuity Posterior cord tear	Posterior cord – AN graft Partial RN transfer		SA 0 EF 3 EE 0 WE 5 WF 5 FF 5 FE 5	SA 4 EF 5 EE 5 WE 5 WF 5 FF 5 FE 5	1	2

* ICN, intercostal nerve; MCN, musculocutaneous nerve; SAN, spinal accessory nerve; DSN, dorsal scapular nerve; SSN, suprascapular nerve; MN, median nerve; AN, axillary nerve; RN, radial nerve

† FCR-EDC, transfer of the flexor carpi radialis to extensor digitorum communis tendon

‡ SA, shoulder abduction; EF, elbow flexion; EE, elbow extension; WE, wrist extension; WF, wrist flexion; FE, finger extension; FF, finger flexion

Table III. Associated injuries in the 11 patients. Duration of loss of consciousness is shown in parentheses

Case	Side of brachial plexus injury	Head/facial injury	Thoracic injury*	Shoulder girdle injury	Limb fracture
1	Left	Jaw bone fracture			Lt forearm Bilateral femora
2	Right	Frontal contusion	Bilateral multiple rib fractures		
3	Right	Head injury (a few hours)	Rt lung contusion		Rt forearm
4	Left	Frontal laceration Head injury (a few days)	Lt lung contusion Lt haemothorax		Lt humerus
5	Left	Head injury (a few days)	Lt lung contusion	Lt clavicular fracture	Lt thumb
6	Right	Head injury (a few days)	Rt multiple rib fractures Bilateral haemothorax	Rt clavicular fracture Rt scapular fracture	Rt radius
7	Right	Head injury (a few days)	Rt haemothorax		
8	Left	Head injury (40 days)	Lt multiple rib fractures Bilateral haemothorax		
9	Right	Head injury (6 hours)		Rt subclavian artery injury Rt acromioclavicular dislocation	Rt Monteggia fracture
10	Right	Head injury (a few days)	Rt lung contusion Rt haemothorax Bilateral multiple rib fractures	Rt clavicular fracture Bilateral scapular fractures	Rt radius
11	Right	Head injury (a few hours)	Rt multiple rib fractures	Rt scapular fracture	

* Lt, left; Rt, right

well as rib fracture, lung damage and injuries to the upper limb on the same side as the plexus lesion.

In our series, the results of operative treatment were poor, possibly because of delay in starting treatment, in addition to the severe associated injuries. Bentolila et al¹³ reported that the interval between injury and intervention is one of the most significant prognostic factors for brachial plexus lesions and that the delay should be less than four months if optimum results are to be achieved. In our series, the mean time from injury to diagnosis of the brachial plexus lesion was 106.8 days (30 to 186) and the mean time to exploration was 162.4 days (135 to 206). When surgery is undertaken for brachial plexus lesions, the available nerves may be limited due to spinal cord trauma and severe associated injuries. For example, lung contusion, haemothorax, or multiple rib fractures on the same side may mean that the intercostal nerves or phrenic nerve are unavailable for neurotisation.

Brachial plexus injuries are often missed initially because the neurological deficit is attributed to or masked by associated injuries.^{1,2} With time, the neurological deficit becomes clearer. The rarity of combined brachial plexus and spinal cord injury may be a factor which delays the diagnosis of brachial plexus lesions, because attention is mainly directed towards the spinal cord. In cervical cord injury, all paralysis is usually attributed to damage to the spinal cord and a brachial plexus lesion is often masked by co-existing tetraplegia. In associated thoracic spinal cord and head injuries, the patient may be confused for a variable period, with the diagnosis only being established after recovery from the head injury.

Life-threatening conditions such as lung contusions, haemothorax, and multiple major limb fractures can also

divert attention from neurological deficits of the upper limbs.

Supplementary Material

A further opinion by Mr Alistair Ross is available with the electronic version of this article on our website at www.jbjs.org.uk

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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