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Case Report

Isolated musculocutaneous nerve palsy in a spinal cord injury

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Isolated musculocutaneous nerve palsy is rare. We report one case of a bilateral palsy of this nerve following a road accident which led to a complete thoracic level paraplegia.

Keywords: musculocutaneous nerve palsy; spinal cord injury

Introduction

Musculocutaneous nerve injuries usually go together with other nervous injuries of the brachial plexus and thus are rarely isolated. The lesions are frequently due to a direct trauma: surgical wound, bullet or knife wound, direct and violent impact while practising a contact sport or crushing injury during working activities.[1] Several reports also mention indirect trauma involving a violent extension of the forearm,[2] or involving strenuous exercises.[3] A postural mechanism is also evoked in one case of isolated musculocutaneous nerve injury following abdominal surgery.[4] In 1984, Kim and Goodrich[5] described a palsy occurring the day after a football match. More recently, one case of isolated musculocutaneous neuropathy following shoulder arthroplasty has been observed.[6] Another case, associated with repetitive carrying of a heavy rolled object supported by a shoulder and held in place by the arm has been reported.[7]

We present the observation of a paraplegic patient showing a post-traumatic and isolated musculocutaneous nerve lesion, without any osteo-articular and muscular trauma of the upper limbs.

Case report

Mr C, a 34-year old man, was the victim of a road accident in July of 1993 in which he was subjected to a violent thoracic impact against the steering wheel and dashboard of his car. A complete and permanent sensory and motor level paraplegia ensued, linked to a fracture of T4. Some fractures of the first five left ribs and a hemothorax on the left side were also noted. The spinal column was stabilized through the use of Cotrel-Dubousset equipment and the hemothorax was drained. When the patient was admitted to a rehabilitation center, 4 weeks later, a wasting of both arms was noticed. The muscular strength of the biceps brachii was graded 0/5 on the right-hand side and 4/5 on the left-hand side. The biceps tendon reflexes were absent. There was a decreased sensation to touch and pinprick on the radial side of both forearms. The rest of the neurological examination of the upper limbs was normal.

Forty-five days later, needle electromyographic sampling revealed a complete denervation in the right biceps brachii and partial denervation in the left biceps brachii associated with a rich spontaneous activity such as fibrillations. The other muscles of the shoulders and arms, innervated by C5 and C6 were normal, but, unfortunately, the coracobrachialis was not tested by EMG. Stimulodetection of Erb's point provoked, in the right biceps brachii, low amplitude potentials whose latency was within normal limits.

A CT scan and a magnetic resonance imaging (MRI) of the shoulders eliminated a post-traumatic lesion and other lesions liable to lead to a musculocutaneous palsy.

Four months later, the patient had recovered normal strength in his left elbow flexors. The motor deficit remained unchanged in his right arm. Electromyographic examination of the left biceps brachii muscle revealed normal insertional activity and showed an important reduction of voluntary motor unit action potentials which were notably polyphasic. Stimulodetection of Erb's point revealed a low amplitude evoked potential (4.5 mV) with a normal latency (6.7 ms). In the right biceps, there was still evidence of denervation with spontaneous activity. The stimulodetection of Erb's point showed no response.

In spite of these deficits, general wheelchair mobility skills were acquired, but the patient suffered from muscular fatigue after propelling his wheelchair for a long time.

Six months later, the deficits were still noticeable in the right arm and we observed a compensating hypertrophy of the brachioradialis. The EMG showed some 'nascent' potentials of reinnervation in the right

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biceps brachii while a rich spontaneous activity persisted. The evoked response remained of low amplitude (0.6 mV) and a normal latency was obtained in this muscle (6 ms).

Nine months later, the motor deficit of the right biceps persisted, and the functional difficulties as well. EMG in the right biceps brachii still showed 'nascent' potentials of reinnervation with a moderate spontaneous activity. Stimulodetection of Erb's point gave a polyphasic response, with a greater amplitude (2.1 mV) but latency was very long (21 ms). During this examination, the integrity of the right coracobrachialis was confirmed by a normal sample in relation to strain. Finally, the EMG proved to be compatible with a bilateral isolated musculocutaneous nerve injury.

From the functional point of view, the patient became more independent in all daily living activities. Eventually, he returned home and even contemplated working in an office.

Unfortunately, Mr C went abroad which prevented us from reporting the clinical outcome.

Discussion

The musculocutaneous nerve originates from the lateral cord of the brachial plexus and ends in the elbow's bend. Its course is characterized by a number of points of anchorage. In most cases,[8] the principal of these points corresponds to the traverse of the coracobrachialis that it innervates at the level of axilla, before supplying branches to the biceps brachii and the brachialis and before ending in a sensitive lateral cutaneous nerve. This nerve innervates the anterior and external area of the forearm. A musculocutaneous nerve lesion while crossing the coracobrachialis muscle is possible, as suggested by Braddom and Wolfe[3] who evoked, in three cases, a musculocutaneous nerve injury caused by either intermittent vigorous contractions or by a chronic pressure secondary to hypertrophy.

In one of the three cases published by Trojaborg,[2] the musculocutaneous nerve is stretched by a violent extension of the forearm as it comes over the head of humerus. The mechanism has also been attributed to a traction along the nerve while the patient was held in supine position with both arms abducted to 90° and externally rotated.[4]

In our case-report, a direct impact seems unlikely to bring about a bilateral lesion of the musculocutaneous nerve because it should have led to other injuries of the upper limbs, considering the violence of the trauma which caused a spinal cord injury and a severe thoracic lesion. According to the findings of EMG, the lesion is believed to be located below the coracobrachialis muscle, affecting the biceps, brachialis and cutaneous nerves of the forearm. In all likelihood, the mechanism involved is a stretched nerve occurring at the site of passage through the coracobrachialis. This may be caused by the frontal impact which jammed the upper limbs in hyperextension. The second possibility is an isometric and rough contraction of the coracobrachialis.[3] The early clinical recovery on the left-hand side and the later electromyographical recovery on the right-hand side lend equal support to both theories: that of the traction and that of the compression.

In addition to the rarity of isolated musculocutaneous nerve palsies, this case appears to be of great interest because, on the one hand, the lesion was bilateral and, on the other hand, it affected a person with paraplegia. To the best of our knowledge, nothing has ever been written about this condition before.

Despite the persistence of a complete deficit of the right biceps brachii, general wheelchair mobility skills have been acquired. The deficit has been compensated by the brachioradialis. Nevertheless, the resistance to strain remained decreased. Clinical examination of the upper limbs in people with paraplegia is indispensable since it allows detection of any damage to a nerve trunk which might have gone unnoticed in the first weeks and which requires specific management.

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