Chronic Musculocutaneous Nerve Injury: An Important Differential in Progressive Arm Atrophy

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Introduction

Traumatic injuries to the nerves in the upper limbs most frequently involve the ulnar nerve [1]. In contrast, isolated musculocutaneous nerve (MCN) injuries are rare [2]. The reported causes include direct stab injury, crush injury, strenuous activity, repetitive sports injury, and prolonged awkward positioning of the arm while asleep [2,3]. The MCN receives its nerve supply from the C5, C6, and C7 nerve roots. It arises from the lateral cord of the brachial plexus and supplies the muscles of the anterior compartment of the arm. It also gives off a cutaneous supply to the lateral aspect of the forearm [4].

In addition to being rare, such injuries may remain unnoticed, as elbow flexors such as the brachioradialis muscle and pronator teres muscle, which are innervated by the radial and median nerves, respectively, compensate for the weakness of the biceps brachii and brachialis muscles. We present a rare case of an isolated MCN injury in a young man that remained undetected for a long period.

The aim of reporting this case is to highlight the critical importance of taking a detailed medical history, performing a thorough clinical examination, and then ordering appropriate investigations to reach a correct diagnosis.

Case Report

A 28-year-old man presented to an electrodiagnostic clinic with the complaint of painless and progressive wasting of the...
right arm for last 6 months. The referring physician had a query of cervical radiculopathy or monomelic amyotrophy. On initial history-taking, the patient stated that he had no significant past medical history or neck pain. An examination revealed visible wasting of the right biceps brachii muscle (Fig. 1), with an arm girth 1.5 cm less compared to the left side (10 cm proximal to lateral epicondyle). The active range of motion at the elbow was full, and the right biceps brachii muscle strength was grade 4 on the Medical Research Council (MRC) scale, with only mild elbow flexion and forearm supination weakness compared to the left side. A sensory examination revealed impaired dull and sharp pinprick sensations on the anterolateral aspect of the right forearm. The deep tendon reflexes were intact (grade 2+), except right biceps jerk, which was grade 1+. The Hoffman, Lhermitte, Spurling, and Gower signs were negative, with a flexor plantar response. The power in the other muscles of the upper limbs was graded 5 on the MRC scale. A neurological examination of the cervical spine, cranial nerves, and cerebellum (finger nose test, dysdiadochokinesia test, and heel-to-shin test) was unremarkable. The patient was re-enquired to recall any significant events preceding his current complaints. As a result, the patient recalled that he was in a motorbike accident 14 months previously and sustained a right shoulder contusion due to an impact on the cement track. Immediately after the injury, patient experienced severe pain, weakness, and numbness in the right upper limb. The patient stated that he had no history of an open or penetrating wound or head injury at the time of injury. He was evacuated to a tertiary care hospital. He visited a local medical facility, where he was advised on a home-based physiotherapy plan (hot pack and range of motion exercises) in addition to analgesics. He privately received magnetic resonance imaging of the cervical spine and right shoulder, which were reported as normal. Over the next few months, the patient had significant symptomatic relief and came up with trick movements to compensate for his elbow flexion weakness, relying basically on the brachioradialis muscle. However, mild numbness was present but now localized to the lateral aspect of the right forearm. Initially, he had a slight suspicion of arm asymmetry in front of the mirror, which he ignored as he engaged in routine activities of daily living without any activity limitations or participation restrictions. Later, the right arm wasting gradually progressed without any associated pain, when he finally visited a tertiary care hospital.

Following a correlation between the clinical history and physical examination, the differential diagnosis included isolated MCN injury, upper trunk plexopathy, lateral cord plexopathy, C5/6 radiculopathy, and monomelic amyotrophy involving the C5/6 myotomes on the right side. To confirm a definitive diagnosis, nerve conduction studies (NCS) and electromyography (EMG) were carried out. For motor NCS of the MCN, recording and reference surface electrodes were placed on the belly of the biceps brachii muscle and biceps brachii tendon, respectively [5]. A side-to-side comparison showed a reduced compound muscle action potential amplitude and prolonged distal motor latency of the right MCN nerve (Table 1). For sensory NCS of the lateral antebrachial cutaneous nerve (LACN), a recording surface electrode was placed on the lateral forearm 12 cm distal to the cubital fossa and a reference surface electrode was placed 4 cm distal to the recording electrode.5 A side-to-side comparison showed a reduced sensory nerve action potential amplitude of the right LACN. Needle EMG of the right biceps brachii muscle showed no abnormal involuntary activity, but large polyphasic motor unit action potentials with decreased recruitment resulting in incomplete interference, an EMG finding that is consistent with reinnervation. EMG of the cervical paraspinal muscles (to rule out cervical radiculopathy), deltoid, brachioradialis muscles (to rule out upper trunk plexopathy), pronator teres muscle (to rule out lateral cord plexopathy), triceps brachii, extensor digitorum and first dorsal interosseous muscles showed no abnormal involuntary activities, with normal motor unit action potentials and interference patterns. The clinico-electrophysiological correlation was consistent with chronic, proximal MCN neuropathy.

![Fig. 1. Relative muscle wasting of the biceps brachii (right).](image-url)
(right) with good recovery (right biceps brachii muscle strength of grade 4 on the MRC scale and the presence of recovery potentials on needle EMG).

The patient was advised to take a mecobalamin tablet (500 μg) once daily. He was further advised to engage in a resistive exercise plan to strengthen the right biceps brachii muscle and scheduled for follow-up at 12 weeks. However, he was lost to follow up.

Discussion

NCS/EMG is an extension of the clinical examination and it always starts with a brief history-taking and clinical examination [6]. It is the most important diagnostic test to confirm the diagnosis of peripheral neuropathy and to comment on whether peripheral neuropathy is acute or chronic, hereditary or acquired, and axonal or demyelinating [6]. In cases of peripheral mono-neuropathies, NCS/EMG helps to localize the location of nerve damage [6].

MCN neuropathy commonly occurs due to trauma, iatrogenic causes (shoulder arthroplasty, rotator cuff repair, humeral shaft reconstruction, and shoulder arthroscopy) and idiopathic causes. It may be of mixed type, isolated motor neuropathy of the MCN, or isolated sensory neuropathy of the MCN [7]. Following an acute MCN injury, the patient might not present for medical care at all, as intact pronator teres and brachioradialis muscles also assist in elbow flexion. If it remains undiagnosed, an atypical presentation of chronic MCN injury in later life might become a diagnostic challenge.

Our patient had a motorbike accident during the peak of the second wave of coronavirus disease 2019 and could not access a tertiary care facility due to lockdown conditions. He was able to perform elbow flexion by contraction of the brachioradialis muscle, which was also reported by Tinel [8]. Fortunately, the patient showed spontaneous, clinically significant motor recovery. However, based on an isolated complaint of painless progressive arm wasting, his primary physician referred him for NCS/EMG with differentials of cervical radiculopathy and monomelic amyotrophy. Visible atrophy of the arm warrants a detailed clinical history and examination to search for the possible causes, including (but not limited to) central pathology, anterior horn cell disorder, radiculopathy, plexopathy, peripheral nerve injury, or atypical myopathy. This case report also highlights the need to keep unusually delayed presentations of such peripheral nerve injuries in mind while examining a patient with muscle wasting without any other marked neurological symptoms.

The management of MCN neuropathy includes conservative and surgical treatment. A comprehensive multidisciplinary rehabilitation plan formulated by a physiatrist facilitates recovery and prevents dependence in activities of daily living by avoiding or reducing possible complications secondary to immobility (such as muscle wasting, contracture, joint stiffness), neuropathic or musculoskeletal pain, and adhesive capsulitis. However, some cases, like our patient, recover on their own over time and use self-learned trick movements to assist in activities of daily living. Surgical options include conventional nerve transfer or double fascicular nerve transfer [9].

In conclusions, chronic isolated MCN injury is a rare presentation. Such cases may become a diagnostic challenge and warrant a meticulous clinical history and thorough physical examination, especially when an electrodagnostic facility is not available. In most cases, conservative management is successful and is consid-

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**Table 1. Motor and Sensory Nerve Conduction Studies**

<table>
<thead>
<tr>
<th>Nerve sites</th>
<th>Latency (ms)</th>
<th>Amplitude (mV)</th>
<th>CV (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Right</td>
<td>Left</td>
<td>Right</td>
</tr>
<tr>
<td>Motor NCS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>3.4</td>
<td>3.7</td>
<td>11.9</td>
</tr>
<tr>
<td>Ulnar</td>
<td>2.5</td>
<td>2.7</td>
<td>10.2</td>
</tr>
<tr>
<td>Radial</td>
<td>2.8</td>
<td>2.9</td>
<td>5.8</td>
</tr>
<tr>
<td>Musculocutaneous</td>
<td>5.8</td>
<td>4.6</td>
<td>6.8</td>
</tr>
<tr>
<td>Sensory NCS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>2.9</td>
<td>2.8</td>
<td>40*</td>
</tr>
<tr>
<td>Ulnar</td>
<td>2.3</td>
<td>2.2</td>
<td>38*</td>
</tr>
<tr>
<td>Radial</td>
<td>1.8</td>
<td>2</td>
<td>40*</td>
</tr>
<tr>
<td>Lateral antebrachial cutaneous</td>
<td>1.8</td>
<td>1.5</td>
<td>7*</td>
</tr>
</tbody>
</table>

NCS, nerve conduction studies; CV, conduction velocity; -, not applicable. *Amplitudes are measured in microvolt (μV).
ered the first line of treatment. If unsuccessful, a surgical intervention is needed. An accurate and timely diagnosis with an appropriate intervention ensures the best possible outcome.

**Conflict of Interest**

No potential conflict of interest relevant to this article was reported.

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