A case of painless inflammatory brachial plexopathy responding to immunotherapy

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BACKGROUND

Due to its location and its anatomical relationships to the adjacent tissues, the brachial plexus can be easily damaged by trauma or other processes. In some cases it can be affected by immune-mediated diseases such as neuralgic amyotrophy, presenting with acute onset of severe pain followed by progressive weakness, an asymmetric form of chronic inflammatory demyelinating neuropathy, with focal slowing localized to the brachial plexus, or multifocal acquired motor and sensory demyelinating neuropathy characterized by the presence of conduction blocks.

CASE REPORT

We report a 39 year old man observed for the insidious onset, seven years before, of progressive weakness of the left upper limb, particularly in the flexion of the elbow, associated to biceps myokimia. Neurological examination showed the absence of reflexes in the left upper limb. We performed a brachial plexus and cervical cord magnetic resonance imaging (MRI) which revealed thickening and increased T2 signal of the left roots from C5 to C7 and of the upper trunk of the left brachial plexus (Fig. 1). Electromyography (EMG) revealed chronic neurogenic changes in the left C5-C6 myotome and an asymmetry in the median sensory nerve action potential (SNAP) through stimulation of the thumb (with right amplitude being 75% bigger than the left amplitude) and of the SNAP of the lateral cutaneus antebrachial nerve (right 40% bigger of the left) suggesting a chronic axonal damage of the upper trunk of the left brachial plexus (Tab. 1). In order to investigate the possible causes we performed an ecography of the left shoulder which confirmed the thickening of the left C5-C6 roots and excluded possible origins of organic compression. Examination of the cerebrospinal fluid (CSF) was decisive revealing slightly increased protein, albumin and immunoglobulins G with the alteration of the "barrier index". Viruses, toxoplasma and oligoclonal bands were absent as well as serological anti-GM1, -GM2 and -GQ1b antibodies. On the basis of an inflammatory hypothesis the patient underwent immunotherapy consisting of intravenous immunoglobulins, with the regression of the symptoms after a few days.

	ENG parameters	
<u>Sensory Nerves</u>	CV (m/s)	Amp uV
Median I [R/L]	53.1/53.1	60.3/24.1
Median II [R/L]	64/57	29.1/14.5
Lat. Cut. Antebrachial [R/L]	58.5/57.3	8.1/5.1

Table 1: Electroneurography revealing an asymmetry in the median SNAP through stimulation of the thumb and of the SNAP of the lateral cutaneous antebrachial nerve.

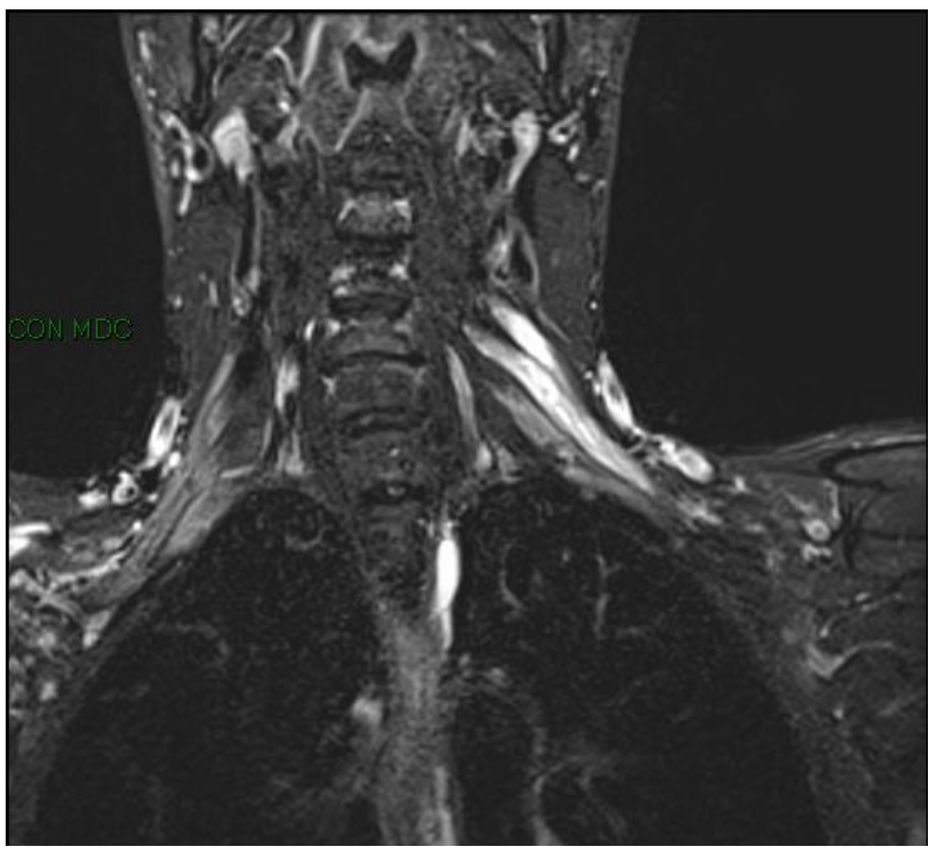

Figure 1. MRI showing thickening and increased T2 signal of the upper trunk of the left brachial plexus

CONCLUSIONS

Even though the brachial plexus damage of our patient did not appear to have the clinical or electrophysiological characteristics of the known types of immune-mediated forms of brachial plexopathy, on the basis of the inflammatory CSF and the absence of compression, we suspected an immune-mediated pathogenesis. Our diagnostic hypothesis was confirmed by the good response to immunotherapy.

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