

# The magnetic resonance neurography of a parsonage-Turner syndrome case

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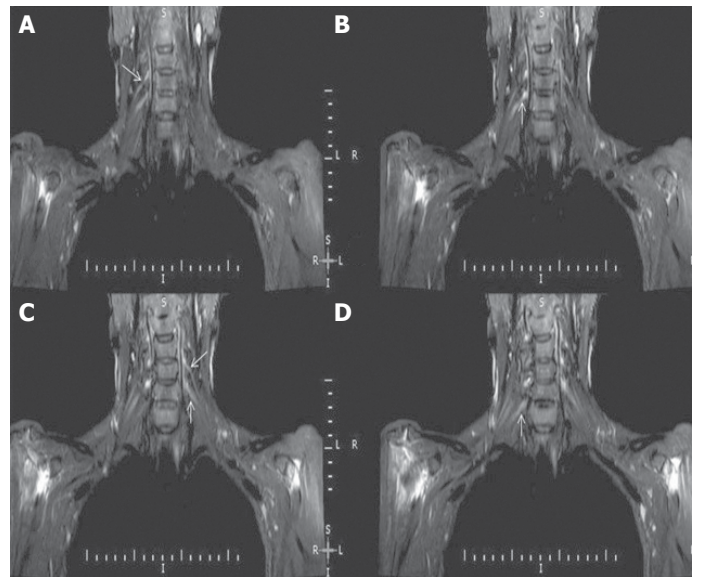
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A 42-year-old woman was seen due to severe pain and weakness in her shoulders bilaterally for ten days. She stated that her symptoms had started suddenly after two consecutive (between few hours) generalized tonic clonic seizures. The pain was present at rest and worse with movement since the second seizure onset. Medical history as regards any fever, diarrhea, infection were noncontributory.

Physical examination revealed restriction in shoulder movements, weakness of the supra/infraspinatus, deltoid, biceps and brachioradialis muscles bilaterally, and normal sensorial findings. Laboratory findings were within normal limits except increased levels of creatine kinase (7300 U/l, normal: 0–170) at the third day of the symptoms onset. Microbial and biochemical investigations of the cerebrospinal fluid and cranial magnetic resonance imaging (MRI) were normal. Electroencephalography was normal. Cervical (C) MRI findings were insignificant. Electroneuromyographic examination showed acute denervation of the axillary and musculocutaneous nerves on the left and axillary nerve on the right upper extremity. Ten days after the first visit (20 days after the symptom onset) shoulder pain and weakness were remained, creatine kinase level decreased to 67 U/l. In respect of the EMG examination outcomes, a brachial plexus magnetic resonance neurography was performed and it showed thickening and high signal intensity from



**FIGURE 1.** Magnetic resonance neurography. Magnetic resonance neurography shows thickening and high signal intensity from the postganglionic C5-C6-C7 at the right (**A, B, D**) and C5-C6 at the left (**C**) in coronal STIR images.

the postganglionic C5-C6-C7 at the right and C5-C6 at the left in coronal STIR images (Fig. 1).

Overall, the patient was diagnosed with neuralgic amyotrophy (NA) and methylprednisolon 120 mg (only one intramuscular dose after 15 days the symptom onset), levetiracetam 1000 mg/day and pregabalin 150

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mg/day were given by neurology department. Additionally, a rehabilitation program including range of motion and isometric strengthening exercises were started. One month after, her pain relieved (visual analog scale decreased 9 to 6) and a mild improvement was detected for weakness. Informed consent was obtained from the patient.

Neuralgic amyotrophy, also known as the Parsonage–Turner syndrome, is characterized with acute and severe pain/paresis particularly in the upper extremities and seen with an incidence of 2–3/100000 per year. By and large, the upper and middle trunks of the brachial plexus

are affected. NA can be sporadic or seen in an autosomal dominant manner. The etiopathology is not clearly understood. However, peripheral nerve biopsies of NA yielded inflammatory and autoimmune responses, perineural thickening, epineural perivascular mononuclear T-cell infiltration, active multifocal axonal degeneration, but not vessel wall inflammation or necrosis. Herein, we are presenting our case with MRI evaluations. Besides, it should be pointed that early rehabilitation program for this syndrome might have benefits for preventing immobilization-related problems as well as improving affected muscle strength and relieving pain.