Case Report

Acute sporadic brachial plexus neuropathy

Dirk Deleu, *MD, PhD,* ***André Louon,*** *MD.*

ABSTRACT

A 43-year-old patient presented with acute sporadic brachial plexus. Diagnosis was based on clinical and neurophysiological findings. This case highlights that early diagnosis of this clinical disorder can prevent unnecessary neuroradiological or neurosurgical interventions.

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Reversible brachial plexus neuropathy is not unusual, and is probably underdiagnosed. During the first few days, the clinical picture can have a severe and alarming character, but in general the prognosis is very favorable.[1] Early diagnosis of this clinical disorder can prevent the patient from undergoing unnecessary neuroradiological or neurosurgical interventions.

Case Report. A 43-year-old man presented with a 3-week history of acute severe aching pain in the neck extending to the right shoulder and arm predominantly at night. Over this period, he started complaining of numbness in the right shoulder girdle and became unable to raise his right arm. There was no history of fever, immunization, trauma, systemic disorders or neuralgic amyotrophy. He was initially treated with diclofenac 50-mg 3 times a day. Due to persistence of the pain, he sought medical advice at Sultan Qaboos University Hospital, Oman.

Neurological examination disclosed a mild atrophy and weakness of the right shoulder girdle, mainly of the major pectoral, major rhomboid, deltoid, supraspinate and infraspinate muscles with winging of the scapula (**Figure 1**). No pyramidal signs or fasciculations were found. Generalized areflexia was present in the upper extremities. Sensation for all modalities was normal in both arms and shoulders. Examination of cranial nerves revealed no abnormality. His neck and shoulder muscles were painful at palpation. He was afebrile, there was no dyspnoea and cardiopulmonary examination was normal.

The following laboratory results were within normal range: complete blood count; erythrocyte sedimentation rate; glucose; glycosylated hemoglobin; electrolytes; liver enzymes; renal and thyroid function. Autoantibody screening (including antinuclear antibodies and rheumatoid arthritis) was negative. Complete viral and bacterial screening including serology for *Borrelia Burgerdorferi* and human immunodeficiency virus (HIV) failed to demonstrate any recent infection. An extensive electromyographic examination of the upper limbs showed increased insertional activity with fibrillations and positive sharp waves (signs of denervation) in deltoid, infraspinate and supraspinate muscles of the right shoulder. A poor recruitment pattern and increased number of polyphasic potentials in major rhomboid, infraspinate, triceps, pectoral major muscles at the right side was observed suggestive of a neurogenic lesion. Anatomically, the lesion affected the upper truck of the brachial plexus and part of the ventral root of C5. Conduction velocities and distal latencies were within normal limits. Chest x-ray was normal (inspiration and expiration) excluding phrenic nerve involvement. Magnetic resonance image of the cervical spine was

From the Neurology Clinic (Deleu) and the Department of Anesthesia (Louon), Sultan Qaboos University Hospital, Muscat, *Sultanate of Oman*.

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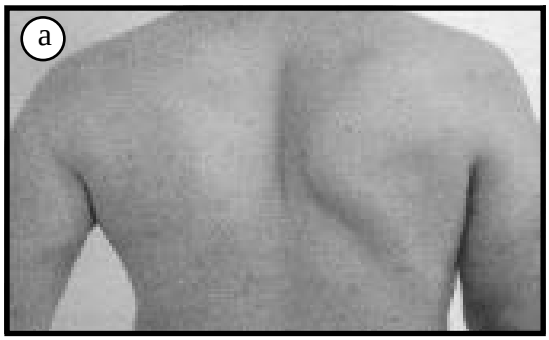
Address correspondence and reprint request to: Dr. Dirk Deleu, College of Medicine, Sultan Qaboos University, PO Box 35, Al-Khod, Muscat-123, *Sultanate of Oman*. Tel. +968 515106. Fax. +968 513419. E-mail: dtodeleu@squ.edu.om



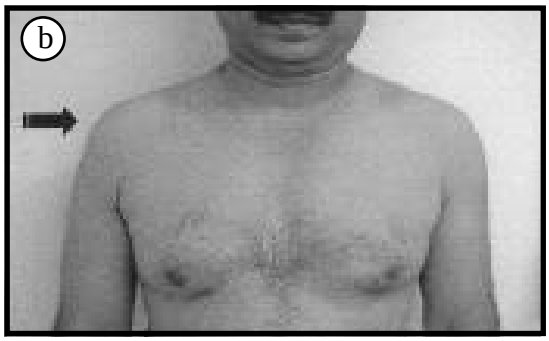


Figure 1 - Photograph of patient showing **a)** winging of the right scapula and atrophy of the right shoulder girdle including deltoid muscle and **b)** deltoid (arrow) and pectoral muscle atrophy (prominent acromioclavicular notch) on the right.

normal. Based on the clinical history, the neurological examination and complementary studies, the diagnosis of acute sporadic brachial plexus neuropathy (Parsonage-Turner syndrome) was made.

In view of the interval between onset of symptoms and his presentation at the clinic, oral corticosteroid therapy was not considered. In addition, most of the pain was thought to be due to muscle spasm. Hence, he was prescribed diazepam followed by physiotherapy. Over a few days, this resulted in substantial relief of the pain. One month later there was gradual improvement of the weakness.

Discussion. Acute sporadic brachial plexus neuropathy, also called acute sporadic brachial plexitis, neuralgic amyotrophy or Parsonage-Turner syndrome, is well-documented in European and American medical literature. However, to the best of our knowledge no reports of this entity originate from this part of the Middle East. Thus, we assume, that this type of brachial plexus neuropathy is underdiagnosed in this area, as the disease is not known to have a geographical predilection.[1]

Acute sporadic brachial plexus neuropathy is a well-defined clinical entity of unknown etiology.[1] The disease can completely or partially affect the upper, lower or the entire brachial plexus. In one-fourth of cases, the presentation may be bilateral. The clinical presentation is very typical with acute onset of severe pain, predominantly or worse during the night, affecting neck, shoulder(s) or the arm(s). Commonly, a non-specific febrile illness or injury precedes the onset of the disorder. The pain frequently extends towards the upper arm, or even below the elbow particularly in lower brachial plexus lesions. Rarely, bilateral phrenic nerve may result in diaphragmatic involvement.[2] After a variable period of time (hours to days) weakness appears confined to the shoulder girdle followed by profound atrophy after a few days to weeks. The prognosis is usually very favorable and most patients recover completely.[3]

Although immune-mediated or inflammatory mechanisms are likely to be involved, the etiology of acute brachial plexus neuropathy remains largely unknown.[1] Reports of acute brachial plexus neuropathy following diphtheria-tetanus-pertussis vaccination, following streptokinase thrombolytic therapy or administration of sera have been reported, suggesting an allergic pathogenic mechanism.[4] Other cases have been related to infectious mononucleosis, cytomegalovirus infection, HIV, systemic lupus, Hodgkin's disease and paraneoplastic syndrome.[5-8] The diagnosis is merely clinical and no investigations are specific. Magnetic resonance imaging findings are also non-specific and include high signal intensity in supraspinate, infraspinate and deltoid muscles.[9] The differential diagnosis of acute brachial plexus neuropathy includes the recurrent hereditary form (an autosomal dominant disorder causing painful, recurrent brachial plexopathies, maps to chromosome 17q25 and is clinically characterized by facial dysmorphism), cervical discal hernia, compression due to cervical spinal hematoma or tumor, vasculitis (Churg-Strauss angiitis, periarteritis nodosa and systemic lupus), Guillain-Barré syndrome, Lyme disease (lymphocytic meningoradiculitis) and plexopathy following surgical interventions.[10-12] The importance of early recognition of the syndrome is prevention of unnecessary neuroradiological or neurosurgical interventions. The conventional treatment of acute brachial plexus neuropathy is oral corticosteroid therapy that usually provides dramatic relief often after the first dosage.

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