Severe bilateral atrophy of the spinati muscles in a cadaver

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INTRODUCTION

Bilateral atrophy of the spinati muscles is quite rare. One of the few diagnoses that results in this finding is Parsonage-Turner syndrome. Parsonage-Turner syndrome (Brachial Plexus Neuritis or Neuropathy Amyotrophy) is a rare condition characterised by inflammation of the nerves innervating the muscles of the anterior thorax, shoulder, arm, forearm, and hand. The etiology of this condition is unknown. Patients may first experience a sudden onset of severe pain across the affected areas and have frank weakness to atrophy of the involved musculature within a few hours or days. Although paralysis of the affected regions may persist for months to years, recovery is usually complete. Our current case represents the cadaveric findings in a probable case of this neuromuscular disorder.

CASE REPORT

A 62-year-old male formalin-fixed cadaver was observed to have bilateral severe atrophy of the spinati muscles (Fig. 1). No other musculoskeletal anomalies or pathologies were noted in the upper extremities. The brachial plexus did not appear entrapped within the scalene muscles. There were also no gross nervous anomalies such as entrapment of the suprascapular nerve at the suprascapular notch and the nerve was, in fact, freely mobile underneath a non-ossified suprascapular ligament. The cervical spinal cord (from the foramen magnum to the lower border of the C7 vertebra) was sectioned and observed grossly. Both axial and sagittal sections failed to reveal pathology such as hydrosyringomyelia.

Key words: denervation syndromes, shoulder, muscle atrophy, muscle wasting

During the routine dissection of a 62-year-old male cadaver, bilateral atrophy of the supra and infraspinatus muscles was observed. The suprascapular nerves, cervical spinal cord and surrounding muscles were found to be normal. We propose that, in the face of normal histology and other normal shoulder girdle muscles and normal nerves, this case represents an instance of Parsonage-Turner syndrome. To our knowledge, this is the first report of bilateral spinati atrophy in a cadaver.

Figure 1. The left scapular region from our specimen (the cephalad is to the left). Note the atrophied supra (A) and infraspinatus (B) muscles. The suprascapular nerve is marked as N. Also note the normal muscle bulk of the surrounding shoulder.
The cervical spinal cord was grossly within normal limits and there were no signs of disc herniation within the neck. Microscopic examination of the spinal cord and suprascapular nerves revealed normal histological features with no signs of inflammation. Both the supra and infraspinatus were involved bilaterally. The degree of atrophy appeared symmetric. Histopathology of the spinati simply demonstrated atrophied skeletal muscle fibres with no signs of inflammation or other pathology.

**DISCUSSION**

Bilateral atrophy of the spinati muscles is seemingly quite rare. Motoi et al. [9] report a 49-year-old male with progressive bulbar palsy who on admission had bilateral atrophy of the sternocleidomastoid and spinati muscles. In addition, this patient demonstrated dysarthria and dysphagia. Kasuya et al. [7] report an adult male with hoarseness, dysphagia, and unilateral atrophy of the spinati, deltoid, trapezius, sternocleidomastoid, rhomboid and serratus anterior muscles. This patient was diagnosed with a pharyngeal-cervical-brachial variant of Guillain-Barré syndrome after anti-Gal-C IgM antibodies were found in his serum. Interestingly, Berger et al. [2] describe a dog with atrophy of the spinati, bilateral enophthalmos, superficial inguinal lymphadenopathy, hyperreflexic patellar reflexes, lack of conscious proprioception, and lack of pain in the hind limbs. This animal was found on postmortem to have Trypanosoma cruzi infection. Fink and Haupt [5] have described brachial neuritis affecting bilaterally the spinati, deltoid and rhomboid muscles. This case was found following streptokinase thrombolytic therapy.

Parsonage-Turner syndrome, also known as Feinberg’s, Tinel’s and Kiloh-Nevin III syndrome, is a term used to describe many forms of cryptogenic neurological atrophy of primarily the brachial and cervical plexus [10]. The incidence in the U.S. is approximately 1.64 cases per 100,000 person years [6]. The exact etiology of this syndrome is not known but a viral infection is suspected [5]. In fact, many patients have reported antecedent illness or recent immunisation. This syndrome has been described to be bilateral at times [4, 7]. Indeed, Misamore et al. [8] report that this is the case in up to a third of patients. The clinical differential diagnosis for muscle atrophy of the shoulder girdle can be diverse and includes rotator cuff anomalies, peripheral nerve entrapment, trauma, reflex sympathetic dystrophy, poliomyelitis, brachial neuritis, cervical radiculopathy, spinal cord tumour and traumatic or compressive peripheral neuropathy [8]. Bredella et al. [3] state that the suprascapular nerve is the most commonly affected nerve in Parsonage-Turner syndrome. These authors also described other diagnoses affecting the suprascapular nerve such as arteriovenous malformation in the spinoglenoid notch and cysts of the posterior scapular region. No anomalies such as these were found in our specimen. Other less focal disease processes that may involve atrophy of muscles include multiple sclerosis, Eaton-Lambert syndrome, ageing, mitochondrial myopathies, arthrogryposis multiplex congenita, various glycoprotein storage diseases, acid maltase deficiency, carnitine deficiency, Cushing’s disease, corticosteroid polymyopathy, muscular dystrophy, facioscapulo humeral dystrophy, myasthenia gravis, Charcot-Marie-Tooth disease, diabetes and lead poisoning [1].

We propose that this case represents an instance of Parsonage-Turner syndrome. Although there may be other entities in the differential diagnosis, this pathology is the most likely in the face of other normal gross/neuro anatomy in the region. To our knowledge, this is the first report of bilateral spinati atrophy in a cadaver.

**REFERENCES**