Unilateral calf hypertrophy and radiculopathy: an unusual association

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Accepted 20 September 2016

DESCRIPTION

A 46-year-old man presented with a 3-year history of progressive enlargement of the right calf. The patient denied local pain, weakness or paraesthesia of lower limbs. Physical examination confirmed calf enlargement with no neurological deficits (figure 1). Laboratory tests revealed mild elevation of creatine kinase (554 U/L). MRI showed an increased volume of the muscles of the posterior compartment, with signs consistent with local myositis (figure 2). Electromyogram revealed findings of S1 radiculopathy. A muscle biopsy was performed which confirmed a denervation process and revealed a discreet focal myositis process (figure 3). Imaging study of the lumbar-sacral spine, conducted by CT, confirmed a disc herniation, leading to compression of the right L5 and S1 roots (figure 4).

Neurogenic muscle hypertrophy secondary to S1 radiculopathy is a rare but well-documented phenomenon. The pathophysiology is unclear, but it may be secondary to an abnormal electrical activity or occur as a result of increased workload imposed on the non-denervated fibres.¹

Figure 3 Right gastrocnemius muscle biopsy: marked variability in the diameter of muscle fibres with groups of hypertrophied fibres coexisting with groups of atrophied fibres. The number of internal nuclei is increased in hypertrophied fibres. Signs of focal myositis with a mononuclear infiltrate at the level of the perimysium and endomysium of the atrophied muscle fibres.

Figure 1 Enlarged right calf.

Figure 2 Increased volume of right soleus and gastrocnemius muscles with hyperintense signal on T2 compatible with focal myositis.

Figure 4 Disc herniation at L5/S1 level. Calcification of the posterior longitudinal ligament in L5 and S1, forming exuberant bone bridge.

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To cite: Daniel A,
Eugénio G, Silva J, *et al.*
BMJ Case Rep Published
online: [please include Day
Month Year] doi:10.1136/
bcr-2016-217744

Previous case reports provide strong support to the notion that denervated muscles can develop an inflammatory reaction, presenting histopathological features of focal myositis.²

Treatment is not standardised. Oral corticosteroids have been used, particularly when local myositis is identified, with variable results. The benefits of local botulinum toxin A injections or surgical intervention of the disc herniation remain to be evaluated, with only anecdotal reports. In the absence of therapy, the condition tends to stabilise or even regress over several months or years.³

Learning points

- ▶ This case provides further evidence that chronic radiculopathy may be responsible for the development of neurogenic muscle hypertrophy and focal myositis.
- ▶ A careful diagnostic evaluation should be performed to confirm the denervation process, including muscle biopsy.

Contributors AD and GE contributed for the patient study, including planning of the diagnostic examinations and their interpretation. AD was the main doctor responsible for the follow-up of the patient, and JS contributed for the bibliographic research and expedite the performance of muscle biopsy. JdS contributed for the design and conception of this manuscript.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Walcott BP, Nahed BV, Redjal N, *et al.* Pathological mechanism of lumbar disc herniation resulting in neurogenic muscle hypertrophy. *J Clin Neurosci* 2011;18:1682–4.
- 2 Prutki M, Potocki K, Stern-Padovan R, *et al.* Unilateral muscle hypertrophy and focal myositis following S1 radiculopathy. *J Musculoskelet Neuronal Interact* 2013;13:259–61.
- 3 Gross R, Degive C, Darnis E, *et al.* Focal myositis of the calf following S1 radiculopathy. *Semin Arthritis Rheum* 2008;38:20–7.

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