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Case Report

Neurogenic muscle hypertrophy in a 12-year-old girl

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Abstract

Muscular hypertrophy secondary to denervation is very rare, but well-documented phenomena in adults. This is the first report of a child with neurogenic unilateral hypertrophy due to S1 radiculopathy. A 12-year-old girl presented with left calf hypertrophy and negative history of low back pain or trauma. The serum creatinine kinase level and inflammatory markers were normal. Magnetic resonance imaging showed muscle hypertrophy of the left gastrocnemius and revealed a protruded lumbar disc at the L5-S1 level. The protruded disc abuts the S1 root on the left side. Electromyography showed mild left S1 radiculopathy. Passive stretching and work load might clarify the origin of neurogenic hypertrophy but there is still a need for further evidence. Clinical, laboratory, magnetic resonance imaging and electromyography findings showed that S1 radiculopathy could be a cause of unilateral calf swelling in youth even in the absence of a history of back or leg pain.

Keywords: Muscle hypertrophy; Disc herniation; Calf enlargement; Radiculopathy; Denervation hypertrophy

1. Introduction

Unilateral calf swelling can be induced by different conditions such as deep vein thrombosis, ruptured popliteal cyst, tumour or trauma. This is the first report of a child with neurogenic unilateral hypertrophy due to S1 radiculopathy.

There is only one report of a child with congenital calf muscle hypertrophy but the underlying etiology is not quite clear. Spinal cord showed no apparent abnormality on MRI and authors related to spina bifida occulta at the S1 level [1].

2. Case report

A 12-year-old girl was referred to our hospital in June 2012 with a 6-month medical history of a swollen left calf. On examination, left calf was 3.5 cm bigger in circumference than the right, but was painless, not tender or weak and ankle reflex was symmetrical. The gait was normal and she was able to walk on tiptoes without difficulty. She has done a deep squat easily but with more stance on the right side. Manual muscle test for plantar flexors of the left foot was 4 +. She had no other complaints and no previous medical history of low back

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pain. Also, she was involved in sport activity regularly in school and participated badminton classes two times per week. Laboratory tests included complete blood count, inflammatory markers, renal and liver function, serum creatinine kinase level; 134 IU/L (55–249 IU/L) were all normal. Doppler ultrasonography of the lower limbs found no evidence of deep vein thrombosis. Electromyography (EMG) and nerve conduction study were performed on both peroneal and tibial nerve, with needle EMG in following muscles: left vastus medialis and tibialis anterior, both extensor digitorum brevis and gastrocnemius. Left medial gastrocnemius showed reduced recruitment during contraction with rare high amplitude (4 mV) motor unit potentials. There was no abnormal spontaneous activity or complex repetitive discharges. Motor conduction velocities (MCV) and amplitudes revealed normal results for peroneal nerve (MCV left 49.0 m/s, distal amplitude 6.65 mV; MCV right 47.2 m/s, distal amplitude 5.25 mV) and tibial nerve (MCV left 49.5 m/s, distal amplitude 7.43 mV; MCV right 54.1 m/s, distal amplitude 5.8 mV). F wave of the peroneal and tibial nerve showed normal mean latency value. H reflex testing was not performed. Magnetic resonance imaging (MRI) of both calves showed a slight enlargement of left gastrocnemius muscle, precisely medial portion. The enlarged left gastrocnemius muscle was isointense on MRI to the remainder of muscles on T1 weighted images (WI), T1 WI with fat suppression and proton density with fat suppression (PD FS) sequences (Fig. 1). No signs of muscle oedema or fatty replacement of the left gastrocnemius muscle were found. Lumbosacral MRI showed left-sided protrusion of the L5-S1 intervertebral disc with narrowing of the neural foramen, causing slight compression of the S1 nerve root (Fig. 2). In addition, our patient also had multiple changes of the lumbar spine including multilevel loss of height of the disc spaces of the lumbar spine, a corner erosion of the lower end-plate of the L3 and bulging of the L4-5 disc (Fig. 3). In consultation with orthopaedic surgeon this protrusion had been managed conservatively.

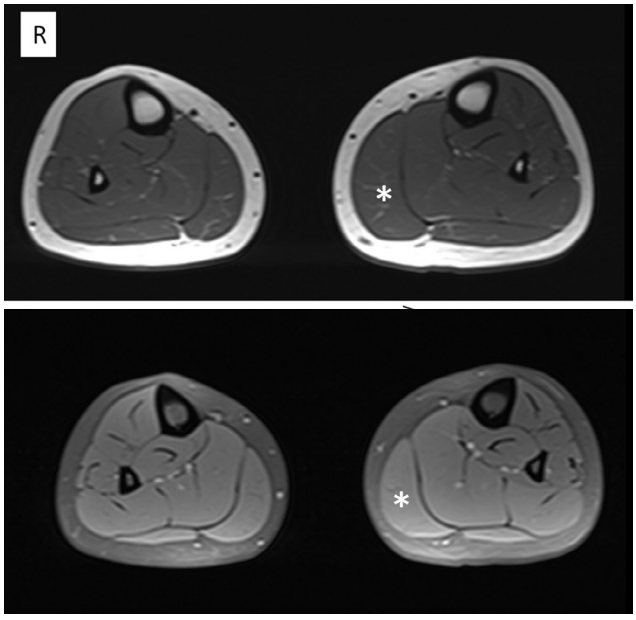

Fig. 1. Lower leg MRI: Axial T1 weighted image shows enlarged left-sided medial head of the gastrocnemius muscle (asterisk).

3. Discussion

Neurogenic muscle hypertrophy (NMH) due to S1 radiculopathy has been reported in adults [2–6]. Enlargement of a muscle may result either from true hypertrophy (increase in the number or the size of muscle fibers) or from pseudohypertrophy (infiltration of the muscle by collagen, fat, parasites, tumour or inflammatory cells) [2]. In our case ultrasound imaging and MRI showed true muscle hypertrophy of posterior compartment, precisely medial portion of the left gastrocnemius. T1 and T1 with fat suppression weighted MRI are considered adequate to show fatty replacement of muscle. Also PD FS is a water sensitive MRI sequence and good enough to show muscle oedema or focal muscle lesion. Contrast was not given due to isointense appearance of the muscle on aforementioned sequences.

The pathophysiology of NMH is unclear. Pareyson et al. outlined possible contributors to NMH: (1) abnormal spontaneous muscular activity, (2) passive stretching by unaffected antagonist muscle and (3) work load [3]. Abnormal spontaneous activity is frequent finding in denervation hypertrophy and may be caused by both denervated muscle fiber hyperexcitability and spontaneous electrogenesis from ectopic foci along injured peripheral nerve fibers, with possible ephaptic transmission to adjacent fibers, inducing a true work hypertrophy [3]. In our case we did not have clinical signs (cramps, pain, fasciculations), electromyography recorded discharges or MRI findings that would suggest

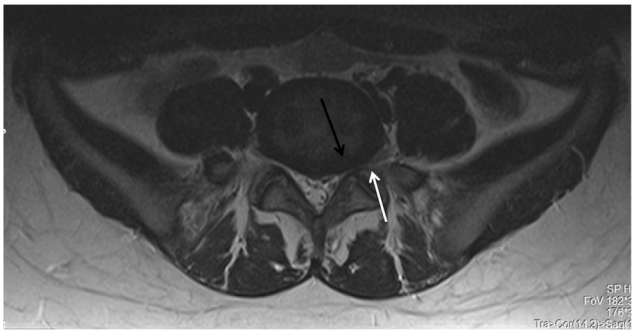

Fig. 2. Axial T2 weighted image shows broad vertebral disc protrusion at L5-S1 with stenosis of the neural foramen on the left side (black arrow). The protruded disc abuts spinal nerve S1 on the left side (white arrow).