Brief Report

Foot Dystonia and Lumbar Canal Stenosis

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Summary: We describe a patient who developed involuntary, painless, dystonic contraction of the toes of the right foot on standing or walking. The development of this abnormal movement had been preceded by sensory disturbance on the soles of both feet, triggered by dorsiflexion of the feet. Examination showed that weight bearing on the right foot and walking brought on clawing of the toes of the right foot, which was relieved within seconds of taking pressure off the right foot. There was sensory and reflex evidence of bilateral S1 root disturbance confirmed by electrophysiology. Magnetic resonance imaging of the lumbar spine showed marked stenosis of the lumbar canal with compression of the L5 and S1 nerve roots bilaterally. The patient underwent a lumbar laminectomy with nerve root exit foramina decompression, which abolished the foot dystonia and has considerably improved the sensory disturbance. This case demonstrates that lumbar canal stenosis and/or nerve root compression may be responsible for foot dystonia. Amelioration of the abnormal movement by surgical decompression argues strongly in favour of this hypothesis. Key Words: Foot dystonia—Lumbar canal stenosis—Root compression—F waves.

A number of clinical syndromes exist that involve involuntary movements of a limb or part thereof. Limb dystonia is one way in which idiopathic torsion dystonia may present. Peripheral trauma also has been associated with the onset of a variety of focal or segmental dystonias, including torticollis, writer's cramp, axial, or arm dystonia (1,2), and foot dystonia (3). Whether peripheral nerve or root lesions can cause focal dystonia is less certain. Arm dystonia after peripheral nerve lesion has been described (4), although the possibility that the nerve lesion may be secondary to the dystonia also must be considered (5). The syndrome of painful legs and moving toes is another condition in which lesions of peripheral nerve, nerve roots, cauda equina, and posterior root ganglion (6,7) all have been associated with the onset of involuntary movements of part of a limb.

We describe a patient who developed a dystonia of the right foot that was heralded by sensory disturbance and accompanied by reflex changes suggestive of bilateral S1 root lesions. Magnetic resonance imaging (MRI) of the lumbar spine showed severe canal stenosis at the L4-5 level with bilateral L5 root compression; the thecal sac and S1 roots also were compressed within the spinal canal. Surgical decompression of the lumbar spine with laminectomy and nerve root exit foramina decompression abolished this patient's abnormal movement. This case demonstrates that lumbar canal stenosis and/or nerve root compression can result in foot dystonia in the absence of more typical features of lumbar spinal canal stenosis.

CASE REPORT

A 57-year-old man was well until March 1994, when he noticed paraesthesia and numbness of the soles of both feet while swimming the breast stroke. The abnormal sensation continued intermittently, when he dorsiflexed the feet. This continued as his only symptom for 7 months until October 1994 when he went on a long walk. During this walk he noticed he was beginning to limp, the right foot feeling abnormal. He realised that the toes of the right foot had developed into an involuntary, painless, clawing spasm. Since that time, the toes of the right foot would develop this abnormal posture whenever he put weight on the right foot, or whenever he walked. The time to onset of the abnormal movement after placing weight on the foot, or beginning to walk was not constant, but had gradually deteriorated so that it now appeared by the time he had walked a few paces. The abnormal movement was not painful but prevented him from walking normally. The movements never occurred when he lay with his feet up. He had no back pain or weakness in the legs, and he had had no trauma to the leg or back. There were

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no abnormal movements elsewhere. He had never been
on any neuroleptic or dopaminergic drugs. There was no
family history of neurological disease.

Examination in March 1995 showed a man of high in-
telligence. The cranial nerves and arms were normal. In
particular there was no evidence of extrapyramidal or
pyramidal disturbance. Examination of the legs and feet
showed a normal appearance at rest with no wasting or
fasciculation. Tone was normal, and straight leg raising
was full bilaterally. Flexion of the spine at the hips was
slightly restricted. Power was full in all muscle groups.
There was an abnormal subjective sensation to both light
touch and pin prick over the S1 dermatomes bilaterally.
Reflexes were normal in the arms, but both ankle jerks
were reduced compared with the knee jerks. The right
plantar was unresponsive and the left was flexor.

Examination in the erect position with weight bearing
on the feet (video segment 1) showed spasmodic fanning
of the toes of the right foot, with flexion of the toes at the
interphalangeal joints and dorsiflexion at the metacar-
pophalangeal joints. These spasms were brought about by
simply standing on the foot (although this was not an
invariable trigger), or more consistently, after walking for
three or four paces. Each spasm would last for as long as
he continued walking and would normally be relieved
within 30 s of stopping walking or taking pressure off the
leg. Certain situations and movements were particularly
effective at bringing on the spasms. These included walk-
ing or standing for prolonged periods, walking down-
stairs, and walking indoors with loose slippers. While
walking, although dystonic spasms were not noticed in
any part of the body or limbs other than the right foot, it
is noteworthy that there was evidence of the striatal toe
(dorsiflexion of the hallux) bilaterally. His gait was some-
what overcautious but showed no other abnormal fea-
tures; in particular, there was normal arm swing and no
evidence of spasticity.

Results of routine investigations, including full haema-
tological and biochemical screen, serum caeruloplasmin
and copper levels, were normal. Electrophysiological
studies showed denervation in the right gastrocnemius
consistent with a right S1 root lesion. There was in-
creased persistence and amplitude of the common peroneal
F-responses bilaterally. MRI scan of the lumbar
spine showed marked canal stenosis with narrowing and
dehydration of the L4/5 and L5/S1 discs with posterior
bulges. There was severe hypertrophy of the posterior
articular facet joints and ligamentum flavum at the L4/5
level. The thecal sac and the L5 (Fig. 1) and S1 roots
within the spinal canal were compressed. MRI scan of the
thoracic spine was entirely normal.

The patient underwent lumbar laminectomy at L4/5
with root exit foramen (L5 and S1) decompression bilat-
erally, without complication. Postoperatively the patient
was able to walk the length of the ward without evidence
of involuntary movements of the foot. Review several
weeks later demonstrated a dramatic improvement. The
patient can now weight bear and walk for prolonged pe-
riods without development of abnormal movements
(video segment 2). Interestingly, the striatal toe sign per-
sists. Sensory symptoms have abated. Follow-up 9
months after surgery shows a sustained improvement in
gait and sensory symptoms. Interestingly, he notices that
if going downstairs without shoes, his right foot tends to
invert.

DISCUSSION

This patient had a unilateral foot dystonia, the main
component of which involved plantar flexion of the toes
of the right foot and dorsiflexion at the metacarpophal-
angeal joints, occurring in the context of lumbar canal
stenosis and bilateral L5 and S1 root compression as dem-
onstrated with MRI scanning; neurophysiological studies
confirmed the S1 root lesion. Although the most obvious
abnormal movement, i.e. the dystonic foot, was unilat-
eral, the presence of bilateral dorsiflexion of the hallux
(striatal toe) while walking may indicate that this patient's
involuntary motor disorder was in fact bilateral. The foot
dystonia (but not the striatal toes) was relieved by lumbar
laminectomy and nerve root exit foramina decompre-
sion, reinforcing the assumption that it was this pathology
that was responsible for triggering that dystonic abnor-
mality.

The muscles responsible for the dystonic movements
seen in the right foot of this patient are the flexor digito-
rum longus and flexor hallucis longus (innervated by the
L5, S1, and S2 nerve roots), and extensor digitorum lon-
gus (L5 and S1 nerve roots), respectively. Some of the
dystonic movements included fanning of the toes (abduc-
tor digiti minimi, S1 and S2 nerve roots) of the right foot
when he stood or walked. There was no corresponding
motor weakness. Additionally, there were sensory and
reflex changes that suggested bilateral S1 root lesions.

The MRI findings of severe lumbar canal stenosis and
compression of L5 and S1 nerve roots, together with elec-
trophysiological evidence for S1 root involvement, sup-

FIG. 1. Axial T1-weighted MRI scan of the lumbar spine show-
ing compression of the thecal sac and the L5 nerve roots within
the spinal canal.
ported the possibility, derived from clinical findings, that the lumbar canal might be the source of his foot dystonia. The electrophysiological findings were particularly interesting because not only was there evidence of S1 root involvement as shown by denervation changes, but a persistence and increased amplitude of the common peroneal F waves were seen. Furthermore, these changes were bilateral. We have observed this phenomenon in other focal dystonias (unpublished observations) and believe it may indicate an increased excitability of the lower motor neuron pool at that level. This raises the possibility that in focal dystonia, an abnormal physiological response of the lower motor neuron pool may be induced by a peripheral nerve or root lesion.

With regard to aetiology of this patient’s foot dystonia, whether it is the canal stenosis, the root compression, or a combination of the two that triggered the abnormality remains unresolved. Similarly, whether the local pathology is mechanical or ischaemic in origin is unclear. The possibility that the lumbar canal stenosis (rather than for example the L5 and S1 root compression alone) might have contributed to the focal dystonic movement is suggested by the fact that the movement only came on when he stood or walked; this association is consistent with other more typical features of lumbar canal stenosis (such as pain, weakness or numbness), which are classically brought on by standing or walking. Action leg dystonia has been described in a patient with severe lumbar canal stenosis (8). In that patient, however, although the sensory symptoms resolved after operation, there was only moderate reduction in frequency of the abnormal movements. That patient had also undergone surgery to the lumbar area before the development of the abnormal movements. It is difficult to be certain that the patient’s dystonia was due to the canal stenosis because not only was there incomplete resolution of the movements after surgery, but the patient had also undergone previous surgery to the lumbar spine, raising the possibility of trauma-related dystonia.

To our knowledge the present case is the first to demonstrate convincingly that lumbar canal stenosis together with root compression can trigger focal limb dystonia. This case raises the possibility that it is the canal stenosis with root claudication per se that may be the causative factor in this man’s abnormal movement due to the fact that the dystonic spasm came on only when he stood on the foot or when he walked, features that are classical triggers for other more typical symptoms of lumbar canal stenosis such as pain and weakness. Furthermore, the abnormal dystonic spasm has been relieved by surgical amelioration of the canal stenosis and root compression. We suggest that the possibility of spinal canal stenosis with or without root entrapment should be considered in patients presenting with focal dystonia of a limb.

**LEGENDS TO VIDEOTAPE**

**Segment 1.** Presurgery. This section demonstrates the patient walking before lumbar laminectomy. The dystonic contractions in the right foot begin after walking for a few paces. The main movements can be seen to include flexion of the toes at the interphalangeal joints and dorsiflexion at the metacarpophalangeal joint. The dystonic movement is abolished by removing pressure from the foot or by sitting down. The patient has some evidence of what has come to be known as the striatal toe—dorsiflexion of the big toe as he walks. This was evident in both feet, and when asked about it the patient claimed that this was a deliberate manoeuvre aimed at reducing the likelihood of developing the dystonic spasms.

**Segment 2.** Postsurgery. This segment was recorded 7 weeks after surgery. The patient is now free of abnormal movements of the right foot. He can walk several lengths of the room without discomfort or dystonic spasm. It is interesting that the striatal toe sign is still present.

**REFERENCES**