A Case of Double Crush Syndrome in the Lower Extremity?

An interesting case of a young athlete is presented that, in order to provide appropriate treatment, demands differential diagnosis and perhaps a bit of out-of-the-box thinking. As foot and ankle specialists, we may encounter patients with concomitant, confounding, or sometimes confusing signs and symptoms that are further influenced by proximal structures or dysfunction. This case illustrates an infrequent but existing phenomenon of symptom generation, more frequently identified in the upper extremities.

Upton and McComas[1] first described a "double crush" hypothesis, stating that axons compressed at one site become especially susceptible to damage at another site. They used the double crush hypothesis to explain why patients with carpal tunnel syndrome (CTS) sometimes feel pain in the forearm, elbow, upper arm, shoulder, chest, and upper back, and further, to explain failed attempts at surgical repairs when neither surgery nor CTS diagnosis appeared faulty. Upton and McComas[1] further suggested that a high proportion (75%) of patients with one peripheral nerve lesion did in fact have a second lesion elsewhere and they implied that both lesions were contributing to the symptoms. They claim that most patients with CTS not only have compressive lesions at the wrist, but also show evidence of damage to cervical nerve roots.

Nakase and colleagues[2] used the term "peripheral entrapment" to explain how neural function could be impaired when single axons that have been compressed in one region, become especially susceptible to damage in another region. They suggested that a discrepancy between neurological manifestation and neuro-imaging sometimes occurs in cervical lesions, and double crush should be considered as a possible pathogenetic mechanism.

Some other studies have addressed this "coexisting nerve entrapment" in association with cervical spine pathologies,[3-6] while others propose the entrapment through structures associated with the thoracic outlet.[7-10]

In 1998, Golovchinsky[11] analyzed results of electromyography and nerve conduction velocity testing in 169 patients with lower back pain, mostly caused by trauma. A total of 289 peroneal, 280 posterior tibial, and 301 sural nerves were included in statistical analysis. Peripheral entrapment of nerves (tarsal tunnel syndrome and anterior tarsal tunnel syndrome) were found in 5.3% of patients, signs of acute or chronic partial muscle denervation of corresponding muscles of lower extremities in 21.8% of patients, and abnormally prolonged F-wave latency in 12.5% of patients. A higher than random coincidence of low back pain and distal EMG compromise allowed the author to conclude a cause-and-effect relationship of damage of the proximal motor nerve fibers and development of peripheral entrapment syndromes in the same nerves rather than a random coincidence of two independent pathologies, and that clinicians should consider simultaneous treatment of the lower back problem as well.

CASE STUDY

The patient was a 13-year-old female athlete who presented with primary complaints of persistent (>2 months) left posterior calf and ankle pain. She reported no specific mechanism of injury but that her pain seemed to begin when she started to attempt to run hurdles for the first time. She could not remember any specific event in which she struck her left ankle on a hurdle but rather that her pain just continued to increase *after* running hurdles. Eventually she experienced increased pain to the point that she could no longer run and was also limping because of pain while ambulating. Approximately 2 weeks after onset of posterior calf and medial foot pain, she was diagnosed with Achilles tendinitis and the foot/ankle was placed in a walking boot that she wore for 5 weeks. She reported the walking boot decreased her discomfort by 25%. The boot was discontinued in exchange for a lace-up ankle brace that she wore for another 4 weeks. During that time, she reported slowly increasing pain to previous levels (8/10).

No other medical problems or significant medical history was discovered. Imaging of the foot and ankle included both radiograph and MRI that were negative for pathology of the foot and ankle. She was provided a lift in her shoe by a doctor of podiatric medicine (DPM) to decrease strain on the Achilles and was prescribed Voltaren cream. She reported no change in pain with the lift or the cream.

She presented to physical therapy 11 weeks after initial injury for evaluation. Evaluation revealed a 13-year-old female ambulating with a significantly antalgic gait. Active range of motion and passive range of motion of the ankle and foot were significantly limited in all directions secondary to pain. Strength testing was not valid secondary to pain levels, although she found force production difficult with plantar flexion. She presented with hypersensitivity and allodynia throughout the left Achilles tendon and calcaneus. Myofascial trigger points were noted in the flexor digitorum brevis and medial head of the gastrocnemius. She had a positive straight leg raise (SLR) and slump test on the left. She had positive signs of adverse neural tension in the left leg. Lumbar examination revealed improved SLR following repeated prone extension with overpressure. She was referred back to orthopaedics by the treating physical therapist for assessment of the lumbar spine. Radiographs were negative for pathology of the lumbar spine. An MRI revealed a moderate bulging disc at L5-S1. She was placed on Medrol dose pack and referred back to physical therapy services. Administration of a Medrol dose pack reduced her discomfort; however, following conclusion of the dose pack, her pain level increased again and no sustained relief of discomfort could be achieved with physical therapy. She was again referred back to the DPM at which point she was placed in a cast. The foot/ankle cast decreased her pain from 8/10 to 2/10. She returned to therapy showing decreased signs of neural tension through the lower extremity.

SUMMARY

The reliance on immobilization of the distal entrapment site, in light of a completely normal MRI examination, suggests that this patient had a form of double crush. The MRI of the lumbar spine established a proximal site of axonal compression, while the foot symptoms (mimicking tarsal tunnel syndrome) suggested a local entrapment of the tibial or plantar nerves. Nerve conduction studies, not performed in this case, might confirm axonal disruption. Yet, the clinician who appreciates the co-existing sites of compression may appropriately adapt the plan of care.

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