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Idiopathic Neuralgic Amyotrophy Presenting as Debilitating Unilateral Lumbosacral Radiculoplexopathy

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Introduction

Neuralgic amyotrophy is characterized by neuropathic pain and subsequent motor weakness. Little is known regarding the etiology of this disorder. We describe a case of neuralgic amyotrophy involving unilateral lumbosacral radiculoplexus which progressed from proximal to distal, and finally more severe affected in the distal muscles.

Case presentation

A previously healthy 63-year-old man visited our outpatient clinic with low back pain and left lower extremity pain which was developed after in-car traffic accident about 4 months before. He showed motor weakness of left lower extremity (grade 3 in hip flexion and 4 in ankle plantarflexion on the Medical Resource Council scale, MRC), diffuse muscle atrophy and severe pain (grade 10 on the Visual Analogue Scale, VAS). These symptoms had been gradually progressed. In initial electrodiagnostic study, nerve conduction study showed normal conduction, however, needle electromyography (EMG) showed abnormal spontaneous activities (positive sharp waves or fibrillation potentials) in the left lumbosacral paraspinal, adductor longus, tibialis anterior, peroneus longus, vastus medialis, rectus femoris and gluteus maximus muscles. Also, interference patterns are decreased in almost muscles. However, in lumbar spine MRI, only mild bulging disc in L4/5 level was seen. Positron Emission Tomography-Computed Tomography (PET-CT) and laboratory test with tumor markers were performed to rule out the cancer related disorder. There were no abnormal findings suggesting malignancy. The pain was temporarily reduced to grade 7 on VAS by taking medicines such as opioid analgesics or prednisolone and receiving interventions. Two months later, pain worsened again to grade 10 on VAS. Weakness and atrophy of left lower extremity were also aggravated. Manual muscle test of his ankle dorsiflexion and plantarflexion became zero. In follow up electrodiagnostic study, nerve conduction study showed abnormal values in compound muscle action potential (CMAP) of left femoral, tibial, peroneal, and sciatic nerves. Also, sensory nerve action potential (SNAP) of left lateral femoral cutaneous, superficial peroneal, saphenous and sural nerves showed abnormal values, such as decreased amplitude or absent response. Needle EMG showed abnormal spontaneous activities in all muscles of left lower extremity with more aggravated interference pattern. Pelvic MRI showed diffuse thickening and enhancement of the left S1 plexus and sciatic nerve, and follow-up PET-CT showed focal increased uptake in the left sciatic and femoral nerves. These findings were compatible with left lumbosacral radiculoplexopathy.

Conclusion

We report a rare case of debilitating left lumbosacral radiculoplexopathy that progressed from proximal to distal peripheral nerves. We could diagnose the idiopathic neuralgic amyotrophy of the left lumbosacral radiculoplexus, clinically more severe involving distal than proximal nerves.

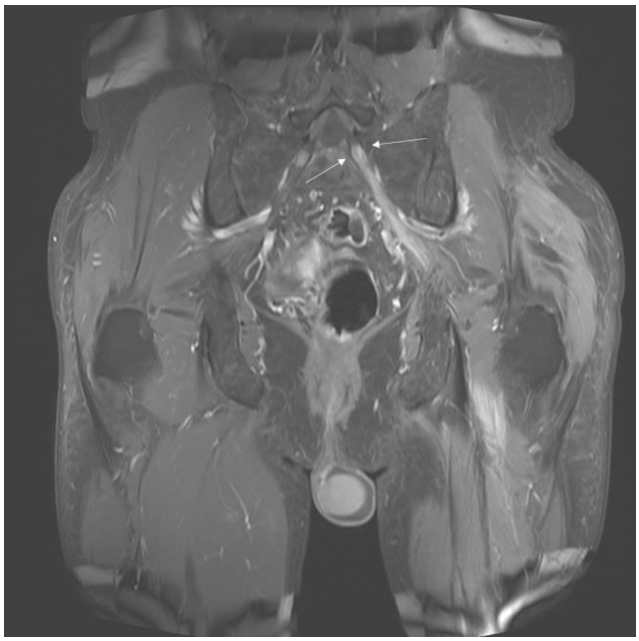

Fig 1. Pelvic MRI contrast enhanced T1WI. Diffuse thickening and enhancement of S1 plexus and sciatic nerve

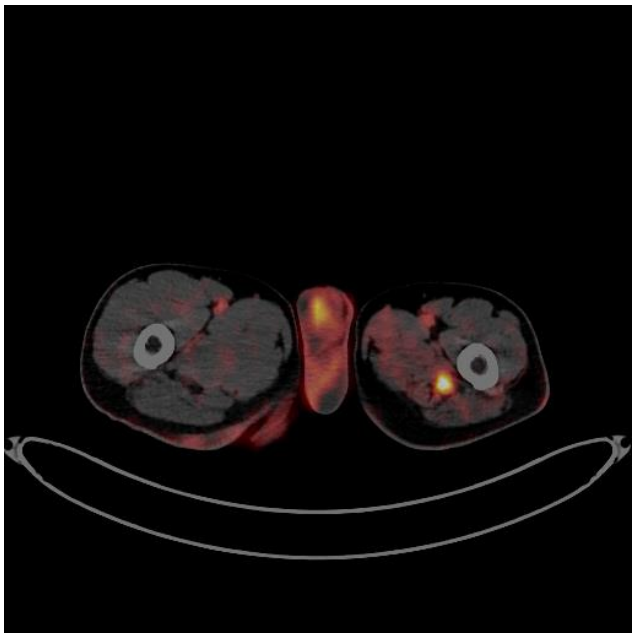

Fig. 2. PET-CT. Diffuse hypermetabolism in the left sciatic nerve (maxSUV 8.32)

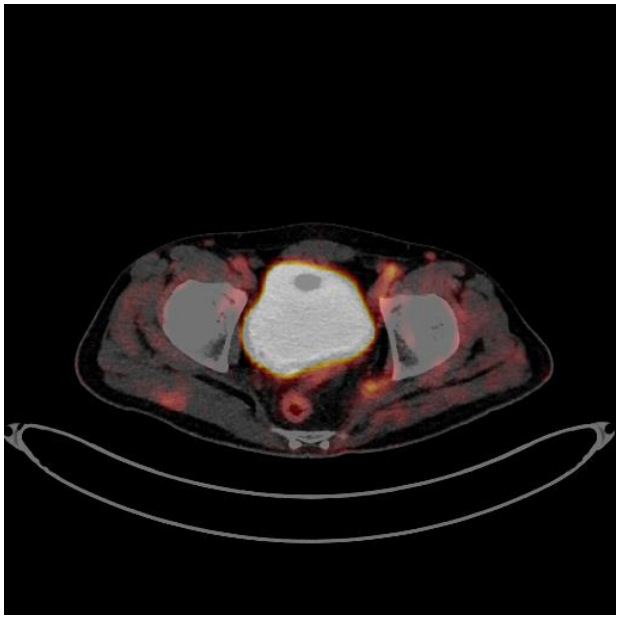

Fig 3. PET-CT. Focal mild hypermetabolism in the left femoral nerve