Diabetic Mononeuropathy Involving the Sciatic Nerve

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ABSTRACT

Sciatic neuropathy is caused by a wide spectrum of etiologies including trauma, compression, metabolic, infectious, and inflammatory conditions. Diabetic mononeuropathy remains one of the more elusive disease manifestations due to lack of large-scale prevalence studies and varied classification schemes. In this study, we present the case of a 48-year-old female diagnosed with diabetic mononeuropathy involving the sciatic nerve. Electromyography and nerve conduction studies showed left sciatic neuropathy, and magnetic resonance imaging of the pelvis revealed an enlarged left sciatic nerve. The patient was managed by controlling the blood glucose level and by therapeutic exercise. This case aims to achieve a better understanding of mononeuropathies occurring in diabetes.

Key Words: diabetes mellitus, neuropathy, sciatic nerve

Introduction

Neuropathy is a disorder of the peripheral nervous system causing a spectrum of disorders including neuropathic pain, paresthesia, numbness, weakness, and atrophy. Mononeuropathy is a problem affecting a single nerve group such as the sciatic nerve. The damage causes slowing of the conduction of impulses through the nerve. Usual causes of mononeuropathy include direct trauma and entrapment via prolonged external pressure on the nerve or pressure on the nerve from nearby body structures. Of less frequent incidence are systemic causes like infection, inflammatory conditions, and metabolic conditions.

One of the more common causes of neuropathy is diabetes mellitus (DM). According to Sinnreich, "Diabetes mellitus is one of the 3 systemic illnesses most commonly associated with mononeuropathy, the others being rheumatoid arthritis and hypothyroidism."[1] The internationally agreed definition of diabetic neuropathy (DN) is "the presence of symptoms and/or signs of peripheral nerve dysfunction in people with diabetes after the exclusion of other causes."[2] According to the American Diabetes Association (ADA), 60% to 70% of people with diabetes have mild to severe forms of nervous system damage.[3] Approximately 7.5% of diabetics are expected to have neuropathy at disease onset, and 50% after 25 years from onset.[4]

There were various classifications of DN that have been proposed throughout the years. These include presumed etiology, topographical features, or pathological features. Among these, the classification by pathophysiological features has been the most appropriate. However, since the pathogenesis of DN has yet to be firmly established, classifications based on clinical manifestations are still widely used. Thomas classified DN according to generalized symmetric polyneuropathies or focal and multifocal neuropathies. The symmetric polyneuropathies included presentations that are: chronic sensory or sensorimotor; acute or chronic selective small-fiber painful; autonomic; or symmetric, lower limb, motor. The focal and multifocal neuropathies included: those affecting the cranial nerves; those with asymmetric trunk/limb, single/multiple nerve involvement; those with proximal motor (amyotrophy); and those with coexisting chronic inflammatory demyelinating polyneuropathy (CIDP).[5]

Much has been discussed regarding the symmetric polyneuropathies because these comprise the bulk of DN with a prevalence of 54% in type 1 DM and 45% in type 2 DM. The focal forms of neuropathy are seen in approximately 25% of diabetics.[5] Focal limb neuropathy or mononeuropathy, which is the focus of this case report, is noted to arise with or without diabetes. Seinnreich noted that 7% of patients with mononeuropathy had DM, while 5.8% of diabetics had mononeuropathy.[1] Since the relationship between DM and mononeuropathy is still not clear, it is imperative that other causes of neuropathy be ruled out.

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Nerves that are more commonly affected in diabetic mononeuropathy include the median, radial, ulnar, common peroneal, and lateral femoral cutaneous nerves. Less frequently affected are the femoral, sural, and sciatic nerves.

Case Report

The patient was a 48-year-old female presenting with weakness of the left ankle and foot musculature of three months duration for which she was admitted at a tertiary government hospital. She had been diagnosed with diabetes since 2000, treated with glyburide and metformin 500/125mg three times a day. Family history was significant for diabetes in the father. She underwent an appendectomy in 1978 and delivered a baby via Cesarean section in 1981. At the time of consult, she had been living with her family in a two-story house with two flights of stairs with 10 steps per flight. Her bedroom was on the second floor.

On July 2004, the patient was side swiped by a motor vehicle; however, radiograph studies at the time revealed normal findings and she did not note any neurologic deficits. On November 2004, she accidentally slipped and fell, landing on her buttocks. Radiograph studies revealed lumbar instability (Lippman-Cobb's angle of 50^0) and degenerative osseous changes of the lumbosacral spine. She remained asymptomatic with no note of any neurologic deficits.

On April 2005, she noted sudden weakness of the left lower extremity involving the distal limb. This progressed to difficulty in ambulation. There was no associated pain or paresthesia. She consulted at UP-PGH Out-Patient Department where fasting glucose was noted to be elevated at 8.3 mmol/L (normal value <5.6 mmol/L), and HbA1c was increased at 7.5% (normal value <7.0%). Complete blood count and thyroid hormone assays were within normal limits. Nerve conduction studies (NCS) of the left peroneal, sural, and tibial nerves revealed delayed distal latency with decreased amplitude of the left peroneal motor action potential and slowed conduction velocity of the left peroneal nerve. The NCS of the right peroneal, bilateral tibial, and bilateral sural nerves were within normal limits. Needle examination or electromyography (EMG) was performed on both paravertebral muscles and on key muscles of the left lower extremity to further determine the extent of involvement. Results showed positive sharp waves, fibrillation potentials, with decreased recruitment over the left semitendinosus, biceps femoris, extensor digitorum longus, and gastrocnemius muscles. Electrodiagnostic findings were consistent with left sciatic neuropathy, incomplete, with acute denervation changes from a level proximal to its innervation to the semitendinosus.

The patient was admitted at the Rehabilitation Medicine Ward of the UP-PGH on August 2005 with an impression of left diabetic lumbosacral plexopathy. She was mesomorphic with stable vital signs. There was tenderness at the dorsum of the left foot and atrophy of the left gastrocsoleus muscles. There was a 2x2 cm lichenified plaque on the dorsum of the left foot. Straight-leg raising test and piriformis stretch test were both negative. Cranial nerve examination was normal. Manual muscle testing revealed weakness of the left knee extensors graded 4/5; the left foot evertors, invertors, ankle plantarflexors and dorsiflexors, and left big toe extensors were graded 2/5. The rest of the muscles were graded 5/5. She reported a 30% deficit to pain and light touch at the left L4-S1 dermatomal levels. Deep tendon reflexes were normal on all extremities.

The patient had good standing balance and tolerance. She was ambulatory without assistive device. On gait analysis, there was an observed foot drop on the left during heel strike, decreased knee flexion on foot flat and hip hike during the swing phase. She was able to ambulate more than 20 meters, but had difficulty in negotiating stairs due to the foot drop. Her Functional Independence Measure (FIM) score on admission was 122/126, with limitations identified in walking and climbing stairs. A FIM score of 126/126 indicates total independence.

The patient was started on a rehabilitation program, primarily physical therapy to improve the motor strength, and sensory and proprioceptive function of the left lower extremity, and to facilitate safe ambulation on level surface and stair climbing. Additional EMG studies done on the left abductor pollicis brevis, left gluteal muscles, right semitendinosus and right extensor digitorum longus were all silent at rest. These findings ruled out the presence of a plexopathy or a polyneuropathy, and confirmed the initial impression of sciatic neuropathy. A lichenified plaque noted on the left foot was diagnosed as a case of lichen simplex chronicus and was managed by the Dermatology Service with topical steroids.

Transvaginal ultrasound done in March 2005 showed adenomyosis with adenomyoma and left peri-ovarian adhesions with normal ovaries. The patient was referred to the Gynecology Service; findings from repeat transvaginal ultrasound did not show peri-ovarian adhesions but revealed a myoma on the left lateral anterior myometrial wall measuring 3.1 x 3.4 x 3.0 cm. Gynecology Service recommended observation of the status of the myoma.

Magnetic resonance imaging of the pelvis showed the left sciatic nerve to be mildly enlarged compared to the right (Figure 1), and there was also dessication of the L5-S1 intervertebral disc.

After ruling out other possible causes of sciatic neuropathy and in the light of poorly controlled diabetes, the patient was treated as a case of diabetic mononeuropathy involving the sciatic nerve. In addition to the rehabilitation program, her blood glucose levels were constantly monitored and her medications were adjusted accordingly. Upon discharge, her left knee extensors were graded 5/5; her left foot evertors and invertors, and left dorsiflexors, 3/5; left

ankle plantarflexors, 2/5; and left big toe extensors, 4/5. There was only 5% deficit to light touch at the left L4-S1 dermatomal levels. She remained ambulatory with no difficulty observed including stair climbing. Her FIM score improved to 124/126. Blood glucose was controlled at 6.07 mmol/L.

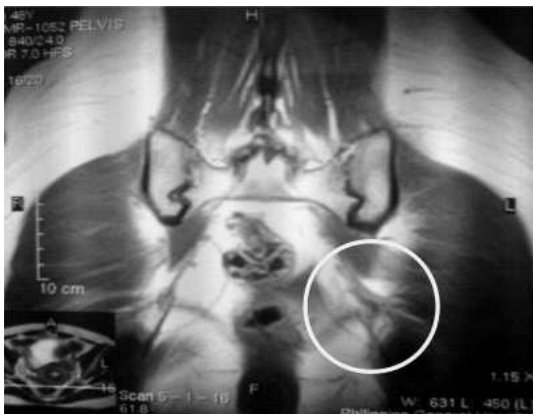

Figure 1. Magnetic resonance imaging study of the pelvis showing enlarged left sciatic nerve

Discussion

The sciatic nerve involved in mononeuropathy is injured by fractures of the pelvis, gunshot wounds, or other trauma to the buttocks or thigh. Prolonged sitting or lying with pressure on the buttocks may also injure the nerve. The sciatic nerve may also be compressed by pressure from masses such as tumors or abscesses, or by bleeding in the pelvis. Systemic diseases, such as diabetes, can typically damage many different nerves, including the sciatic nerve.[6] In a study by Goh on 29 patients examined at the Neurodiagnostic Laboratory, Tan Tock Seng Hospital from January 1989 to April 1995, external nerve compression was the most common cause of sciatic nerve injury (38%), followed by trauma (21%), while 24% of the cases had uncertain etiology.[7] Other rare causes (17%) reported in this series included intragluteal injections, hip surgery and diabetic mononeuropathy. In a study by Fraser, et al. on 51 patients with diabetic mononeuropathy, it was found that the median, ulnar, and lateral popliteal nerves were the most commonly affected, accounting for 38 out of 51 subjects (75%).[8] In the same study, only three patients (6%) were found to have sciatic nerve mononeuropathy.

There are no studies in the Philippines on the incidence of sciatic neuropathy. The low incidence of sciatic nerve involvement in diabetic mononeuropathies may be due to several factors. As discussed previously, the highly varied classification schemes and the lack of large-scale prevalence studies may significantly affect the true epidemiology of this pathology. Both studies by Goh and Fraser et al. included samples from populations that may not be representative of the target population of individuals with DM, which includes approximately 18.2 million people or 6.3% of the population in the US alone.[3] It is therefore recommended that further studies with large sample sizes be conducted to delve further into the incidence and prevalence of diabetic mononeuropathies.

Another reason for the rarity of the case may be the poorly defined pathogenesis of DN. It has been purported that vascular pathology and an increased propensity to compressive damage make the selective injury of individual nerves more likely in patients with DM. Accelerated atherosclerosis, thrombosis, inflammation, and impaired vasodilatation all contribute to occlusion of arterioles supplying the vaso nervorum, and may, in turn, cause neuropathy. Ischemia classically presents with the triad of sudden pain, weakness, and numbness along the distribution of the nerve. Gooch and Podwall noted that although pain is common, pain is absent in a significant number of patients with DN.[9] Electrodiagnostic studies may show a reduction in both nerve conduction velocity and sensory or motor compound action potential amplitude suggestive of underlying demyelination and axonal degeneration.

Approximately one out of three in the diabetic population is expected to have neuropathy secondary to anatomic compression of the peripheral nerve, supporting the theory that diabetic patients are highly susceptible to nerve compression injuries. It is not currently known how DM increases this susceptibility. Electrodiagnostic studies are used to determine the entrapment sites by identifying the conduction blocks. Mononeuropathies secondary to vasculitis or ischemia are classified as mononeuritis syndromes, while those due to compression are classified as entrapment syndromes. Mononeuritis is a self-limiting condition with sudden onset, involving a single nerve or multiple nerves usually involving cranial nerves III, VI, and VII, and ulnar, median, radial, peroneal, and femoral nerves. Entrapment syndrome, on the other hand, presents with gradual onset, is progressive, and involves single nerves exposed to trauma, such as the median, ulnar, peroneal, plantar, lateral femoral cutaneous, and femoral nerves.[9]

The clinical picture of our patient points to a combination of the two syndromes. Fraser supports such a finding in his statement, "the development of mononeuropathy in diabetic patients may represent a combination of metabolic and vascular factors together with the usual daily trauma to nerves, especially pressure at exposed sites […] such patients were described as having 'autogenous mononeuropathy'."[8]

The enlarged sciatic nerve in the pelvic cavity seen in our patient raises both answers and questions. The enlargement, even in the absence of a space-occupying lesion, is itself a risk factor for compression from adjacent pelvic structures. This location was confirmed by the electrodiagnostic findings of denervation above the thigh

level but below the lumbosacral plexus. Mononeuritis, the entrapment syndromes, and the various metabolic changes in DM have not been reported to cause peripheral nerve enlargement. A nerve biopsy would have been useful in this regard, but was not performed due to financial constraints.

With the limited diagnostic tests done, diabetes was highly considered as the cause of the neuropathy after exclusion of other pathologies and the improvement of the patient's symptoms with glycemic control and rehabilitation intervention.

It must be noted that early onset diabetic symmetric polyneuropathy and early onset chronic inflammatory demyelinating polyneuropathy may initially present as a mononeuropathy. It is only through regular follow-up, serial monitoring of glycemic control with blood glucose levels and electrodiagnostic parameters, and a nerve biopsy can the diagnosis of DM neuropathy be confirmed.

In conclusion, diabetic mononeuropathy remains a diagnosis of exclusion when it involves the peripheral nerves such as the sciatic nerve. More well-designed prospective research is needed to better understand this disease process, prevent or delay its complications, and, ultimately, promote optimal function among patients with diabetes mellitus.

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