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Sciatic Nerve Palsy Secondary to Intensive Care Unit Stay with Prolonged Sitting Position

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Abstract

We report a series of 3 cases who presented to our neurophysiology department with sciatic nerve palsy secondary to prolonged sitting position in the intensive care unit (ICU). Though iatrogenic sciatic nerve palsy is previously described, it is secondary to total hip replacement surgery, neurosurgical procedures and due to injections. This series describes three cases of sciatic nerve palsy related to prolonged sitting position. This will highlight the need to take adequate preventive measures in the ICU setting and also that such sciatic nerve palsy may be much more common than reported.

Keywords: sciatic nerve, pressure palsy, prolonged sitting, intensive care unit

Introduction

Sciatic nerve pressure palsy can occur at many sites. Pressure palsies of the sciatic nerve are often neglected or wrongly diagnosed as peroneal nerve palsies. There has been case reports of sciatic nerve pressure palsies from toilet seat to abnormal postures during surgery. Sciatic nerve injury during total hip replacement is also reported. Most recover completely but some are left with residual sensorimotor dysfunction. Often at the time of ICU admission the sciatic nerve palsy is not at the forefront but instead saving the life is. Sciatic nerve palsy during ICU stay due to prolonged sitting has not been reported previously.

Case reports

We did a retrospective evaluation of 3 cases of sciatic nerve pressure palsies which presented for nerve conduction studies in our neurophysiology lab.

All three patients had clinical and electrophysiological evidence of sciatic nerve palsy. They had involvement of knee flexion, plantar and dorsiflexors of foot and ankle. All had variable sensory loss over the distribution of sciatic nerve. Imaging excluded any focal lesions in spine and also along the sciatic nerve.

Case 1

Adolescent female was admitted with bronchial asthma exacerbation, pneumonia and respiratory failure. She was intubated and mechanically ventilated as all initial medical measures failed to improve her. She was kept

in intensive care unit. Two days later she was extubated and she was kept on oxygen support. Fourth day of admission she was unable to move her right foot. On examination she had weakness of the right knee flexors, dorsiflexors, plantar flexors of foot and toes and also of all small muscles of foot. Sensory loss was noted only on the right peroneal nerve distribution and right ankle reflex was absent. Otherwise all neurologic examination was normal. Her MRI Lumbosacral spine was normal. Nerve conduction studies showed reduced compound muscle action potentials (CMAP) of both peroneal and tibial nerves on right side. Sensory potentials (SNAP) from sural and superficial sensory were reduced on right compared to left. F waves were inelicitable on right side. Electromyography confirmed sciatic nerve axonopathy. Her autoimmune work up including ANA and ANCA was negative. She was started on physiotherapy and at follow up after 1 month showed only mild improvement in knee flexion with persistent distal weakness. At 6 month follow up she had considerable improvement but still had residual numbness of dorsum of foot and mild weakness of dorsiflexion of foot and toes.

Case 2

The second patient was another adolescent girl who was diagnosed with diaphragmatic hernia when she presented with acute onset breathlesness. She was operated and was in ventilator for 5 days and later shifted out from ICU. She was discharged after 3 weeks. Her mother noticed abnormality in her walking. The patient complained of pain over right posterior thigh. On examination, she had high stepping gait bilaterally, weakness of bilateral knee flexors and foot muscles (right more affected). Both ankle reflexes were absent. Nerve conduction studies showed reduced right peroneal CMAP amplitude with mild reduction in conduction velocity and normal distal latency. Left peroneal CMAP amplitude was decreased with normal conduction velocity and distal latency and Tibial CMAP showed normal latency with reduced amplitude and normal conduction velocitiy. Sensory action potentials from sural and superficial nerves on right side were absent while on left side was normal. Follow up NCS done after 6 months revealed recovery of peroneal conductions bilaterally with persistent reduction of tibial motor amplitude on right side. Sensory potentials were still absent on right side. At 1 year follow up she was having good improvement clinically with mild weakness of foot muscles.

Case 3

Third patient was a young pregnant lady found to be covid -19 positive with acute respiratory distress syndrome. She was taken for emergency caesarean and post caesarean section was mechanically ventilated for 9 days. Post extubation she was noted to have left thigh pain and foot drop. She was diagnosed as left peroneal nerve palsy from another hospital. She was seen 1 month later in our clinic. She was improving, but still had burning sensation of sole of left foot and weakness of dorsiflexion. Nerve conduction studies showed reduced amplitudes of left peroneal and tibial motor conductions with normal distal latencies and absent sensory potentials from left superficial peroneal and sural nerves. She was started on physiotherapy and she improved symptomatically with good improvement in motor power.

Discussion

The common characteristics noted in these 3 patients were the following : all were females, were thin built, and all of them had been ventilated in upright sitting or semirecumbent position for respiratory issues. None had any history of intramuscular injection in the gluteal area. No previous history of any neurologic deficit was reported in any of the patients.The likely etiology of all patients is pressure on sciatic nerve at level of upper thigh due to prolonged sitting position.

Sciatic nerve is the longest and widest nerve in the body derived from multiple lumbosacral nerve roots. It has a lateral and medial divison. Lateral division forms the common peroneal nerve and medial division forms the tibial nerve. Within gluteal region, sciatic nerve lies deep to the gluteus maximus but superficial to other lateral rotators of thigh. Below the gluteal region the nerve passes between the ischial tuberosity and greater trochanter; then it descends between the biceps femoris and adductor magnus muscle to enter the posterior

compartment of thigh. It divides to peroneal and tibial division just above the knee level [1].

Sciatic nerve pressure palsies have been reported due to prolonged sitting in toilet seat and yoga posture [2,3]. Surgeries especially neurosurgical procedures in prolonged sitting position can also cause pressure on sciatic nerves. Lithotomy position for surgical procedures can lead to sciatic nerve palsies due to hip flexion causing stretching of the nerve [4]. Sciatic neuropathy has also been reported due to prolonged immobilisation due to intoxication [7], as a complication of hip and knee surgeries, piriformis syndrome etc.

Seated and semi recumbent positions are adopted usually in critical patients with respiratory illnesses as pulmonary function improves with more erect postures [5]. Prolonged positioning in sitting makes sciatic nerve vulnerable to pressure injuries at the level of ischial tuberosity [6]. Several mechanisms of sciatic nerve damage are described 1) stretching of sciatic nerve and compression against sciatic notch 2) diffuse compression of the nerve in posterior thigh 3) local myonecrosis in gluteal region and direct focal compression.

Severe sciatic neuropathies present with pain and hyperesthesias mainly in sole and dorsum of foot. There will be weakness of hamstrings and all muscles below knee. Usually the peroneal compartment is more severely affected than tibial division. The mechanism postulated for the greater vulnerability of peroneal nerve is that it is it is laterally placed and has fewer and larger fascicles with less supportive tissue. Also it is fixed at sciatic notch and at fibular neck compared to tibial nerve which is more loosely fixed. On physical examination, involvement of non peroneal innervated muscles like ankle inversion (tibial nerve - tibialis posterior), toe flexion (tibial nerve - flexor digitorum longus) and knee flexion (sciatic nerve - hamstrings) suggests involvement beyond peroneal nerve distribution. Isolated sciatic nerve lesions spare sensation over medial calf and foot (saphenous nerve) and posterior thigh (posterior cutaneous nerve of thigh). It also has to be differentiated from lumbosacral plexopathies, radiculopathy and a central lesion.

Weakness when bilateral in a critically ill patient can be mistaken for ICU acquired weakness which is defined as "clinically detected weakness in critically ill patients in whom there is no plausible etiology other than critical illness" [8]. This entity includes critical illness neuropathy, critical illness myopathy and acute necrotising myopathy associated with intensive care. Patients with ICU acquired weakness have more generalised weakness and are difficult to wean from ventilator.

Usual sciatic neuropathies due to external compression are reported in various scenarios like in surgical tables and abnormal sitting postures in hard surfaces. But pressure palsy due to ICU stay has not been reported previously. Though exact mechanism of nerve palsy in our patients could not be confirmed by imaging ad nerve conduction, we speculate that the nerve injury is due to compression on the nerve due to prolonged sitting. The subsequent clinical improvement also supports the diagnosis.

Conclusion

Though measures are taken to prevent the usual complications like pressure sores, infections, deep vein thrombosis, ICU related pressure palsies are not addressed. The need for change of position, avoiding prolonged pressure at small area, passive limb physiotherapy even for critically ill patients and checking the mattress for any missed hard objects underneath the patient is important. Also keeping pads under buttocks to prevent pressure on nerve at level of ischial tuberosity and keeping knee flexed and limiting hip flexion can help to certain level.

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