Traffic Jam Neuropathy

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A 45 year old, overweight and short Thai male was trapped in a compact Japaness car during a 4 hour traffic jam. Both femoral neves were compressed long enough to produce a state of neuropraxia. This was confirmed by clinical findings and electrodiagnostic tests. He had a complete recovery within less than one month.

Keywords: Traffic jam, Prolonged hip flexion, Femoral neuropathy, Nerve conduction.

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ขาดไทหย่าๆ 45 ปี รู้ensburg เพื่อท่าน ข้ารองผู้ใหญ่ชนะเล็ก ใจร่วนก่อนอ้นปี 2533 ทอดศีลปิยมา
ประมาณ 4 ข้าวโบโล ปรากฏว่าแล้วประสงค์พิมานรักทั้ง 2 ข้างอุทกภัยกับอ้นนาพรท่านให้เกิดความรุ่งเรืองท่าน
ข้าควีใต้ ซึ่งพุ่มใหญ่ให้การถาวร การตรวจสอบภาพและการตรวจวินิจฉัยตัวกลับขึ้น และภาษามีไม่ถึง
1 เดือน ผู้ป่วยรายนี้พึงศึกษาพฤติกรรมทุกปัจจุบัน
Femoral neuropathy due to compressive lesions has been previously recognized. These included haemorrhage, neoplasms, infection, blunt trauma, operative injuries, anticoagulant therapy with haemorrhage, stretch injury, sudden growth spurt, common iliac artery occlusion, stress fracture of the iliac bone, immobilization and poor position under spinal anaesthesia and traumatic rupture of the iliacus muscle. However, no previously reported included encounters following prolonged hip flexion.

Case report

A 45 year old, overweight and short-Thai male came to the Chulalongkorn Hospital out patient clinic on January 3rd, 1991 with the complaint of weakness of both knees; the right being effected more than the left. Symptoms appeared 4-5 days previously. He had been caught in a Bangkok traffic jam for 4 hours sitting in a compact Japanese car. When he left his car, he found that he could not lock his knees in full extension in the standing position. On physical examination, there was weakness of both quadriceps femoris muscles with a MRC grading of 2 on the right and 3 on the left. No sensory deficits were found. The deep patellar tendon reflexes were absent on the right and diminished on the left. Motor nerve conduction velocity studies were performed on the following day. These revealed a significant reduction of motor Nerve conduction velocity (NCV) of both femoral nerves: 30 and 25 m/sec on the left and right respectively (normal 40 m/sec). Motor NCV of CPN and PTN were preserved (Table 1).

<table>
<thead>
<tr>
<th>Nerve tested</th>
<th>Nerve conduction</th>
<th>Remark</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lt. Femoral nerve</td>
<td>30 meters/second</td>
<td>Slow conduction</td>
</tr>
<tr>
<td>Rt. Femoral nerve</td>
<td>25 meters/second</td>
<td>Slow conduction</td>
</tr>
<tr>
<td>Lt. common peroneal nerve</td>
<td>45.32 meter/second</td>
<td>Normal</td>
</tr>
<tr>
<td>Rt. common peroneal nerve</td>
<td>47.04 meter/second</td>
<td>Normal</td>
</tr>
<tr>
<td>Lt. posterior tibial nerve</td>
<td>42.0 meter/second</td>
<td>Normal</td>
</tr>
<tr>
<td>Rt. posterior tibial nerve</td>
<td>41.0 meter/second</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Electromyography of both quadriceps femoris muscle showed no signs of denervation or myopathy. The duration and amplitudes of the motor unit action potentials were normal. There was some decrement in recruitment. Routine lab investigation did not reveal evidence of diabetes mellitus. There were no family members effected with hereditary neuropathy. He was treated with high dose vitamin B1, B6, B12 and intensive physical therapy.

One week later, the patient gained some strength in his quadriceps; grade 3 on the right, grade 4 on the left. Nerve conduction velocity on the left was 34.09 meters/second and on the right was 30.05 meters/second. The EMG of both quadriceps femoris showed a normal pattern and no signs of denervation.

4 weeks after the injury the patient gained full strength of both quadriceps femoris (grade V). The NCV of the Lt. Femoral N. was 45.9 meters/second and of the Rt. it was 42.6 meters/second.

Based on the clinical course with full recovery and the EMG findings, we concluded that this patient had a neurapraxia of both femoral nerves.

Discussion

Prolonged flexion of the hips in an overweight patient may be the cause of Femoral neuropathy. The iliopsoas muscle arises within the abdomen from the anterolateral aspect of the lumbar vertebrae and from the inner surface of the ilium. The muscle descends, passing somewhat laterally, and leaves the abdomen behind the inguinal ligament to reach the anterior aspect of the thigh. The femoral N. passes behind the inguinal ligament but lies on the anterior surface of the iliopsoas muscle. With hip flexion, the femoral N. will be trapped between the inguinal ligament which retracts between the iliopsoas muscle which is brought forward anteriorly and it is long enough to produce a compression of the femoral N.
Conclusion

To our knowledge, this is the first case report of femoral neuropathy due to being trapped in a traffic jam injury of peripheral nerves.

References


