Paradoxical Patellar Reflex As A Presenting Sign Of Acute Inflammatory Demyelinating Polyradiculoneuropathy

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Abstract

We report a case of paradoxical patellar reflex as a presenting sign of acute inflammatory demyelinating polyradiculoneuropathy.

Case report

A 46-year-old man presented with distal limb paresthesias and dysequilibrium. Four weeks prior he had a sore throat and dry cough that resolved over two weeks. One week prior he began to experience brief episodes of dyspnea while recumbent. Three days prior he developed progressive low back and lateral hip pain, symmetric paresthesias of the hands and feet, and dysequilibrium. The day of presentation he developed paresthesias of the lips and nose and it became difficult for him to drink liquids because they would run from the sides of his mouth.

His vital signs, general physical examination, and mental status were normal. There was mild symmetric upper and lower bifacial weakness. Strength, tone, and coordination were normal. Somatosensation was normal except for symmetric dysesthesias of the hands and feet. Gait was normal except for mild unsteadiness with tandem walking. Muscle stretch reflexes, other than the patellar tendons, were moderately hyperactive throughout, and severely so at both Achilles tendons with three beats of clonus. Percussion of either patellar tendon elicited no extension at the knee or contraction of the quadriceps, but instead knee flexion occurred with visible and palpable contraction of the hamstrings. Percussion of the medial knee did not elicit bilateral adductor contraction.

The cerebrospinal fluid had an elevated protein of 151 mg/dl and one white blood cell. Nerve conduction studies showed diffuse slowing and conduction block suggestive of demyelination, as well as absent F waves bilaterally.

Over the subsequent days, the bifacial weakness progressed to absence of volitional contraction, and he developed moderate weakness of bilateral hip and knee flexors and extensors. He lost the paradoxical patellar reflex, and all the muscle stretch reflexes became hypoactive with continued absence at both knees. He stabilized after several days of intravenous immunoglobulin treatment, and after two weeks he began to regain strength in the proximal legs, although the bifacial weakness gradually resolved over months.

Discussion

This patient presented with diffuse hyperreflexia, initially casting doubt on the diagnosis which subsequently declared itself over the following days. Cases of acute inflammatory demyelinating polyradiculoneuropathy (AIDP) presenting with initial hyperreflexia has been reported1-3. The mechanism of this phenomenon is unclear, as the usual characteristic of this disorder is hyporeflexia secondary to demyelination of peripheral nerves. The unusual finding of hyperreflexia in case appeared to provide the substrate for another confounding sign: the presence of an inverted or paradoxical patellar reflex. We are aware of only a single report of two patients with an “inverted knee jerk,” occurring with a low spinal cord lesion4. In the setting of spread of muscle stretch reflexes to neighboring muscle groups if opposing muscles are differentially affected such that the reflex of one is lost while the other is increased, striking the tendon of the muscle with the lost reflex could be enough stimulation to the opposing muscle group to trigger its reflex loop, giving rise to paradoxical movement of the tested joint. This type of muscle stretch reflex spread is typically seen with cervical spinal cord lesions where striking the brachioradialis tendon may cause no response in that muscle but finger flexion instead5.

The clinical, cerebrospinal fluid, and electrophysiologic findings of this case are compatible with the diagnosis of AIDP, but early in the patient’s course there were two clinical findings rarely encountered with this condition, both of which could be potential sources of diagnostic confusion.

References

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