Iatrogenic Femoral Nerve Palsy Masquerading as Knee Extensor Mechanism Rupture

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Patellar ligament and quadriceps tendon ruptures, often the result of a mechanical load to failure on the tendon, lead to an inability to actively extend the knee. Optimal treatment is immediate repair using any of a number of methods.[1-3]

Diagnosis of extensor mechanism rupture of the knee can often be made with a complete history of the event and a physical examination and may be supplemented with radiographic studies. Often there is a history of a fall or hyperflexion injury, a subjective sensation of the involved knee "giving way," and difficulty bearing weight after the injury. Examination reveals an inability to actively extend the knee (though an intact extensor retinaculum may allow minimal knee extension), swelling in the region of the patellar ligament or quadriceps tendon, a palpable defect where the tendon would normally reside, and often a superiorly (patellar supera) or inferiorly (patella infera) translated patella. Radiographs often show patella supera or infera, and magnetic resonance imaging (MRI) can confirm the diagnosis.[1-3]

We present the case of a femoral nerve palsy masquerading as a knee extensor mechanism rupture that became symptomatic 1 month after emergent exploratory laparotomy, sigmoid resection, and Hartmann pouch closure for a colon perforation after colonoscopy. This case highlights the need for a thorough history and physical examination, as well as potential complications of abdominal and pelvic surgery that the orthopedist should be aware of. Furthermore, awareness of this potential confusing clinical situation may avoid an unnecessary surgical intervention on an intact extensor mechanism despite a clinical examination consistent with knee extensor mechanism rupture.

Case Report

Our patient was a man in his early 70s who was indicated for routine screening colonoscopy by his internist. During the colonoscopy, iatrogenic perforation of the sigmoid colon was noted. The patient underwent emergent laparotomy and sigmoid colon resection with Hartmann pouch closure of the rectal stump and end colostomy. The abdomen was not closed primarily; instead, a "Bogotá bag" technique was used secondary to wound contamination with fecal material. Two weeks later, the patient returned to the operating room for lysis of adhesions and abdominal wound closure. He was nonambulatory between these surgical procedures.

Four weeks after the second laparotomy, the patient was being ambulated by his physical therapist when the left knee buckled, immediately producing pain and an inability to bear weight or ambulate. An orthopedic consultation was requested for suspected extensor mechanism disruption.

Physical examination by the orthopedic attending and chief resident revealed slight atrophy of the entire left lower extremity with decreased muscle tone. There was a mild knee effusion with only minimal medial joint line tenderness. The knee was stable to anterior/posterior draw and to varus and valgus stress. The patella was intact and nonpainful to palpation. Left hip flexor strength was 4/5, and left knee extension strength was 0/5. There was no active contraction of the quadriceps muscle, but hip flexor contraction, pain about the knee, and a language barrier made confirmation difficult. Other left-side muscle groups and the contralateral right side exhibited normal strength. Reflex examination demonstrated 0 patellar ligament reflex on the left, 2+ on the right, and 2+ Achilles tendon reflexes bilaterally. Sensory examination was somewhat inconsistent, but there appeared to be slightly decreased sensation along the left medial calf. The patient could not actively extend or hold out the left knee. The quadriceps tendon was palpable and intact. The patellar ligament was not palpable. The patient said that he had been able to extend the knee before his recent hospitalization and that he was a normal community ambulator. All laboratory tests were within normal limits. Anteroposterior and lateral plain radiographs of the left knee were normal. There was no evidence of a fracture, patella supera, or patella infera.

A working differential diagnosis of extensor mechanism disruption was made, and surgery was planned, but first, as there was only mild knee swelling, MRI was performed to assess the chronicity of the injury (Figure). The MRI scan was interpreted as showing redundant quadriceps tendon and patellar ligament, consistent with possible intrasubstance tearing. In addition, because there was no active contraction of the proximal quadriceps muscle, MRI of the lumbar spine was performed; it revealed mild spinal stenosis

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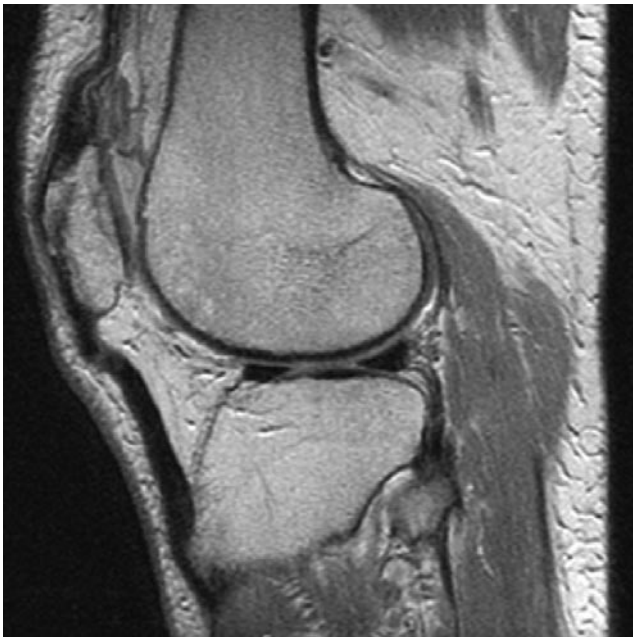

Figure. Sagittal magnetic resonance imaging of affected knee shows redundant patellar ligament and quadriceps tendon.

at L4–L5 and no compressive lesions involving the L2–L4 nerve roots of the femoral nerve. The recent surgery performed in the area of the femoral nerve, as well as decreased sensation along the medial calf, raised concern about a femoral nerve injury, so an electromyographic (EMG) and nerve conduction velocity (NCV) study was performed of the lower extremities. Results were significant for a femoral nerve palsy (Table). On the basis of this imperative information, the patient did not undergo surgical reconstruction of either the quadriceps tendon or the patellar ligament. He was fitted with a locking knee orthosis to promote ambulation with the hope of timely resolution of the neuropraxia.

Over the next year, the patient progressively recovered femoral nerve function. After 3 years, he was fully ambulatory, and there were no inhibitions to his activities of daily living or active lifestyle. Motor strength and sensation were equal to those on the unaffected side, as were reflexes.

Discussion

Femoral nerve palsy is an extremely rare complication of abdominal and pelvic surgery. There are case reports of nerve injury after hysterectomy, renal transplantation, vascular surgery, cystectomy, and colorectal resection (diverticular disease, inflammatory bowel disease, malignancy).[4-8] In the orthopedic literature, femoral nerve palsies have been reported as resulting from iliopsoas muscle hematoma and arising in association with pelvic trauma—specifically, surgical repair of acetabular fractures.[9] Acetabular fractures, primarily associated with anterior hip dislocations, transverse fracture patterns, and ilioinguinal exposure, carry a small risk for femoral nerve injury. Incidence of traumatic femoral nerve injury with acetabular fractures has been low, a reported 0.2% to 0.4%. In a series of 726 patients with acetabular fractures, Gruson and Moed[10] found only 4 femoral nerve injuries, 2 that occurred before surgery (caused by the traumatic event) and 2 diagnosed after surgery (caused by iatrogenic traction or compressive neuropraxias in the setting of an ilioinguinal approach).

The anatomical distribution of the femoral nerve is well documented. The femoral nerve arises from the dorsal divisions of the L2, L3, and L4 nerve roots. Once formed, the nerve passes through the psoas muscle, descends within a groove of the psoas and iliacus muscles, and exits the pelvis beneath the inguinal ligament to enter the lower extremity. The motor portion of the femoral nerve controls the iliacus, pectineus, sartorius, and quadriceps muscles (hip flexion and knee extension, respectively). The sensory fibers innervate the anteromedial thigh, the medial calf, and the medial aspect of the foot.[2,8,11]

Symptoms associated with femoral nerve palsy include varying degrees of motor and sensory innervation loss and loss of the patellar ligament reflex. Most cases exhibit sensory changes over the anteromedial aspect of the thigh and over the medial calf and weakness of the quadriceps muscle. On occasion, presenting symptoms are limited to loss of sensation or paresthesias, though often the patient will have difficulty ambulating because of weakness of the affected limb and may have a history of falls. Chronic nerve palsy can result in muscle wasting and atrophy of the quadriceps, iliacus, sartorius, and pectineus muscles.[7-10,12]

The differential diagnosis for patients who present with these symptoms and history includes injury to the extensor mechanism (patellar tendon rupture, quadriceps tendon rupture, patella fracture), femoral nerve palsy, and psychogenesis. In cases in which a traumatic event is the possible mechanism of injury to the extensor mechanism of the knee, there are seldom any sensory alterations. There usually is a palpable defect associated with pain along the patellar ligament, the patella, or the quadriceps tendon. Other signs and symptoms are swelling, ecchymosis, joint effusion, and inability to actively extend the knee.[1-3]

Mechanisms of injury to the femoral nerve can be traumatic, toxic, or related to inflammatory, immunologic, metabolic, or systemic disease. Causes of traumatic nerve injury may be accidental, iatrogenic, or compressive. Iatrogenic injuries have been attributed to use of deep-bladed retractors during abdominopelvic surgery compressing the nerve against the pelvic brim before the nerve exits beneath the inguinal ligament.[4,5,10] In the orthopedic literature, iatrogenic femoral nerve injury is most often associated with the ilioinguinal approach for surgical stabilization of acetabular fractures.[10]

Conservative therapy is the mainstay of treatment for femoral nerve palsy, with most patients recovering completely within 12 months after the insult.[5,9,10,12] In cases of nontraumatic causes, they should be corrected, if possible; correction may include medical management of inflammatory, immunologic, metabolic, or systemic disease. Several studies of hemophilia patients with femoral nerve palsies secondary to spontaneous iliopsoas hematomas have found that resolution of motor function occurs within 1 year in 90% of patients but can vary according to control of the coagulopathy and factor inhibitor levels. In cases of iatrogenic femoral nerve palsies

Table. Sensorimotor Evaluation of Bilateral Lower Extremities Revealed Isolated Left Femoral Nerve Dysfunction With Secondary Motor Axon Degeneration

Motor Nerve Conduction Velocity Study

Nerve	Sites	Latency (ms)	Amp (mV)	Area (mV ms)	Duration (ms)	Resp	Distance (cm)	Velocity (m/s)	Temp (°C)
Right femoral —vastus medialis	1. A. Ing. Canal	6.55	2.6	21.5	12.40	—	—	—	—
Left femoral —vastus medialis	1. A. Ing. Canal	NR	—	—	—	—	—	—	—

Needle Electromyography

	Spontaneous				Motor Unit Action Potential				
Nerve	IA	Fib	PSW	Fasc	HF	Amp	Duration	PPP	Recruitment Pattern
Left rectus femoris	N	4+	4+	None	None	N	N	N	No activity
Left add magnus	N	None	None	None	None	1+	1+	1+	Reduced activity
Left iliopsoas	N	None	None	None	None	N	N	N	N
Left vastus medialis	N	4+	4+	None	None	N	N	N	No activity

Abbreviations: A. Ing Canal, anterior inguinal canal; Amp, amplitude; Fasc, fasciculation; Fib, fibrillation; HF, high frequency; IA, insertional activity; N, normal; NR, no response; PPP, polyphasic potentials; PSW, positive sharp wave; Resp, response; Temp, temperature.

after abdominal and pelvic surgery, most patients recover nerve function within 4 to 6 months. The orthopedic literature is relatively deficient in reports on femoral nerve injury and its possible long-term results. Excluding traumatic nerve lacerations or disruptions, in which the possibility of recovery of nerve function is poor, most of these patients recover complete nerve function within 6 months.[10]

There is controversy as to whether surgical intervention is needed for femoral nerve palsies. The literature based on the hemophilia population supports avoiding surgical exploration, as recovery of nerve function is expected without surgery, and the primary factor involved in recovery is control of the coagulopathy.[12] Cases of iatrogenic compressive or traction nerve injuries include only 1 postoperative neuropathy after abdominal rectoplexy and Burch colposuspension in which the patient did not recover any function. The treating physician decided to surgically explore the nerve after EMG and an NCV study. Severe scarring encasing the nerve within the iliopsoas groove was discovered, and external neurolysis was performed. The patient had complete resolution of symptoms within 2 months after the neurolysis.[4]

Conclusions

This case report highlights the potential for misdiagnosis of an apparently simple problem—the inability to extend the knee after a fall. In the absence of a thorough history and careful physical examination, this patient may have been subjected to open repair of the patellar ligament and the accompanying risks of anesthesia and surgery. In addition, the surgical team and the hospital may have faced the legal ramifications of performing unindicated surgery. Fortunately, the patient recovered full nerve function without operative intervention. The importance of a thorough history and careful physical examination, along with judicious use of advanced imaging and functional modalities (MRI, EMG, NCV) in the clinical scenario described in this article, cannot be overemphasized.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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This paper will be judged for the Resident Writer's Award.