Lumbosacral radiculopathy secondary to abdominal aortic aneurysms

Report of three cases

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Focal neurological deficits as the initial manifestation of expanding or ruptured abdominal aortic aneurysms are uncommon. When such a situation does occur, the femoral nerve is most often involved due to retroperitoneal or iliopsoas hematoma. Three cases of typical lumbosacral radiculopathy caused by an abdominal aortic aneurysm are reported to emphasize the importance of considering this diagnosis in the older patient with leg pain and radiculopathic findings.

KEY WORDS • abdominal aortic aneurysm • femoral nerve • lumbosacral radiculopathy • pain

Abdominal aortic aneurysms continue to deserve special consideration in the differential diagnosis of back pain. However, infrequently, focal neurological deficits due to peripheral nerve or radicular compression may be the initial manifestation of an expanding or ruptured abdominal aortic aneurysm. Femoral neuropathy with or without obturator nerve or sciatic radicular involvement has been reported in only eight cases as the presenting feature of an abdominal aortic aneurysm. Three cases of typical lumbosacral radiculopathy are presented to emphasize the importance of considering an abdominal aortic aneurysm in the older patient with back pain, radicular pain, or radiculopathic findings.

Case Reports

Case 1

This 62-year-old white man presented with a 3-month history of intermittent back pain and a 3-week history of pain radiating down the right leg to the foot. On neurological examination the right Achilles reflex was absent and pinprick appreciation was diminished over the lateral aspect of the foot. Straight-leg raising was positive at 30°. Plain lumbosacral radiographs showed diffuse degenerative changes. The patient was placed at bedrest, and given muscle relaxants and analgesics. The pain continued unabated for 2 weeks, and the patient was hospitalized. The neurological examination was unchanged except that there was now a trace of right plantar flexion weakness. Lumbar metrizamide myelography was normal. The patient was given a course of physical therapy as well as stronger analgesics and discharged.

Two weeks later he presented to the emergency room still complaining of leg pain but now with the added complication of severe abdominal pain. Abdominal palpation revealed a large aortic aneurysm, confirmed by aortography. An emergency operation was carried out. The aneurysm was found to involve the infrarenal aorta as well as the right internal iliac vessel. A small retroperitoneal hematoma was also present. The aneurysm was replaced with an aorto-iliac interposition graft. Postoperative recovery was uneventful. The right leg pain was completely relieved, and the neurological examination returned to normal with the exception of an absent ankle jerk.

Case 2

This 57-year-old white woman presented with a 6-week history of low-back and left leg pain which had been unrelieved by chiropractic manipulation. On examination, reflexes were intact but mild extensor hallucis muscle weakness was present on the left as well as decreased pinprick sensation over the medial aspect of the foot. When symptoms did not abate after 10 days of bed rest, myelography was performed,
which was negative. The myelogram was followed by a computerized tomography scan of the spine which did not demonstrate any abnormalities of the lumbosacral spine but did demonstrate an abdominal aortic aneurysm approximately 7 cm in diameter. Surgery for the aneurysm was recommended and performed electively. The aneurysm was found to involve the distal aorta as well as both common iliac arteries. A large aneurysmal dilatation of the left internal iliac vessel was present as well. The aneurysm was excised and an aorto-bifurcation graft placed. Postoperatively, the left leg pain resolved slowly over 10 to 14 days, as did the weakness. The sensory changes have persisted for 9 months following surgery.

Case 3

This 72-year-old white man presented with a 1-month history of severe left leg pain radiating from the groin to the instep. Numbness and tingling of the foot were also noticed. Neurological examination showed positive straight-leg raising at 20°, absent Achilles reflex, and a normal sensory and motor examination. Plain lumbosacral spine films showed diffuse degenerative changes as well as a curvilinear calcific density consistent with an abdominal aortic aneurysm. Sonography demonstrated a 6-cm abdominal aortic aneurysm.

At surgery, the aneurysm was found to involve the aorta as well as both common iliac vessels. The involved portion was replaced with interposition grafts between the aorta and both iliac vessels. Postoperative recovery was uneventful. The leg pain along with the numbness and tingling resolved in the first few days after surgery and have not recurred in 16 months.

Discussion

The tendency for abdominal aortic aneurysms to produce back pain similar to that seen in the commonly encountered neurosurgical syndromes of lumbar disc disease or lumbar stenosis is well known and has been emphasized in a recent review by Kramer. Back and referred leg pain in such a setting generally herald an abrupt increase in size or rupture of the aneurysm. Back pain is present in 62% of patients with a ruptured abdominal aortic aneurysm, with 4% having back pain or pain referred to the thighs, hips, or buttocks as the only symptom.

Rarely has it been reported that focal neurological deficits from peripheral nerve or lumbosacral root compression were the primary initial manifestations of an abdominal aortic aneurysm. Five cases of femoral neuropathy secondary to abdominal aneurysm enlargement or rupture have been recorded. Razuk, et al., were the first to describe this phenomenon. Their two patients presented with back and leg pain and quadriceps weakness. While large pulsatile abdominal aneurysms were appreciated in each patient, both underwent extensive neurological work-ups including myelography. Over the next 3 to 4 weeks there was progressive worsening of the pain and weakness. By the time surgery for the abdominal aneurysms was performed both had quadriceps atrophy, absent knee jerks, and sensory deficits over the medial thigh and leg. Only partial restoration of femoral nerve function was observed postoperatively.

Owens described the onset of a painful femoral neuropathy in two patients 24 to 48 hours preceding the signs of frank abdominal aortic aneurysm rupture. Guite and Trembly presented a patient with a 3-to-4-month history of back and leg pain who subsequently presented with concomitant signs of aneurysm rupture as well as a femoral nerve palsy. One year following repair of the aneurysm, femoral nerve function was normal.

Three cases of abdominal aortic aneurysm rupture with associated obturator and sciatic nerve involvement have been reported. However, the case reported by Kubacek appears more likely to represent ischemic paralysis secondary to embolization from a large fusiform abdominal aneurysm.

Lumbosacral radicular involvement associated with abdominal aortic aneurysm has been reported in two cases. Bolton and Blumgart reported the onset of painful foot drop and L-5 root sensory loss 24 hours prior to frank abdominal aortic aneurysm rupture. However, because of distal extremity ischemic changes present at the time of diagnosis, ischemic paralysis is more likely to be the cause than L-5 root compression. Chapman, et al., reported one patient with left L-5 and S-1 root deficits who had a normal myelogram and evidence of a large calcified abdominal aneurysm. Operation on the aneurysm was deferred because of the patient’s advanced age. Over the subsequent 10 months, the patient developed a progressive left leg monoplegia until death resulting from acute aneurysm rupture.

In all reported cases of abdominal aortic aneurysm producing focal neurological deficits, the aneurysm was found at operation or autopsy to be ruptured with either an associated retroperitoneal and iliopsoas hematoma or a false aneurysm on the side of the neurological deficit.

Involvement of the femoral nerve with abdominal aortic aneurysm rupture is not surprising when one considers the anatomy of this region. The femoral nerve is formed in the substance of the psoas muscle from the L2-4 nerve roots which join at the upper border of the L-5 vertebra, slightly below the level of the aortic bifurcation. The nerve then descends through the muscle to exit at its lateral aspect slightly above the inguinal ligament. It then lies in the groove between the psoas and iliacus muscles deep to the iliacus fascia, until it passes under the inguinal ligament. The femoral nerve provides motor innervation to the iliacus, pectineus, sartorius, and quadriceps femoris muscles. Its sensory distribution involves the
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distal two-thirds of the anteromedial portion of the thigh and the medial aspect of the leg.

It is extremely unlikely that an enlarging aortic aneurysm would cause femoral compression unless rupture had also occurred, which was the situation in all reported cases. The femoral nerve together with the psoas and iliacus muscles are bound within a very tight fascial envelope and are therefore readily compressed by retroperitoneal or intramuscular hematomas or by expanding pseudoaneurysms.

The obturator nerve arises from the same divisions as the femoral nerve within the fibers of the psoas, and descends to exit from this muscle on its medial surface. It supplies innervation to the adductors of the thigh and sensation to the proximal medial aspect of the thigh. The obturator nerve can therefore likewise be compromised by expanding retroperitoneal or psoas hematomas or enlarging false aneurysms.

The roots of the lumbosacral plexus (L5–S2) lie directly posterior to the common and internal iliac arteries. Thus, involvement of these roots would require the aneurysm to include these vessels or have the dissection of a false aneurysmal sac over the pelvic brim. The former situation was present in the case reported by Chapman, et al., as well as in the three cases reported here.

It must be borne in mind that in the older patient a femoral or obturator neuropathy or lumbosacral radiculopathy may rarely signal the presence of a leaking or ruptured abdominal aortic aneurysm. If the iliac vessels are involved early in the process, a lumbosacral root compression syndrome may be the presenting feature. In the majority of patients, an abdominal aortic aneurysm can be diagnosed by simple abdominal palpation, and routine lumbosacral spine films will often show a curvilinear density if the aneurysm wall is calcified. Abdominal sonography can be utilized to rule out the diagnosis of abdominal aortic aneurysm.

The importance of recognizing an abdominal aortic aneurysm as a potential cause for neurological deficits lies in the urgent need for operative intervention on the aneurysm without subjecting the patient to prolonged neurological investigation. The mortality rate for operation on unruptured aneurysms is 5% to 7%, while that for symptomatic ruptured aneurysms is 35% to 40%. For these reasons, the neurosurgeon should always consider the diagnosis of abdominal aortic aneurysm in any patient presenting with back pain, leg pain, or radiculopathic findings.

References


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