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Development of Psoas Hematoma After Lumbar Sympathetic Block in a Patient on Anticoagulants

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Background: Lumbar sympathetic blocks (LSB) play an important role in the therapeutic management of patients who suffer from neuropathic pain conditions such as complex regional pain syndrome (CRPS). LSB is a generally safe and well-tolerated procedure, however rare complications may occur. While case reports of iliopsoas hematomas related to bleeding disorders, surgical interventions, and trauma are well reported in the literature, similar complications after LSB have not been reported.

Case Report: Development of an 18 x 6.1-cm hematoma in the left iliopsoas muscle after a fluoroscopically-guided LSB in an anticoagulated patient despite following recommended periprocedural anticoagulation management guidelines resulted in pain, impaired mobility, and likely femoral neuropathy.

Conclusion: Pain management physicians should be aware that hematomas are a possible complication of LSB and should weigh the risks and benefits of holding anticoagulation therapy in patients undergoing such procedures.

Key words: Anticoagulation, complex regional pain syndrome, hematoma, lumbar sympathetic block, lumbar sympathetic block complications

BACKGROUND

Lumbar sympathetic blocks (LSBs) are performed for painful neuropathic conditions and sympathetically mediated pain states such as complex regional pain syndrome (CRPS) (1). Per the 2018 American Society of Regional Anesthesia (ASRA) Anticoagulation Guidelines for Interventional Spine and Pain Procedures, during the performance of LSB, warfarin should be held for 5 days with an international normalized ratio (INR) < 1.2, and warfarin can be resumed after a minimum 6 hours post procedure but ideally on the next day; enoxaparin bridge is recommended for patients on maintenance warfarin held during the periprocedural period and should be held for 24 hours prior to the procedure and resumed 24 hours post procedure.

Psoas hematomas may form within the context of a variety of settings. An iliopsoas hematoma (IPH) is defined as a spontaneous or traumatic retroperitoneal collection of blood involving the iliopsoas muscle. The precise rate of IPH occurrence remains uncertain, and very few studies are available. The reported incidence of spontaneous retroperitoneal bleeding in patients undergoing anticoagulation ranges from 0.1% to 0.6%. Many case reports in the literature are related to spinal instrumented surgeries, hemophilia, trauma, aortic interventions, and surgeries. No previous case reports of IPH following LSB or other interventional pain procedures could be found (2). Here, we present a case of iliopsoas hematoma following LSB.

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CASE

A 73-year-old man on warfarin 5 mg daily for atrial fibrillation with pertinent past medical history of stroke following coronary artery bypass grafting as well as left total knee replacement complicated by persistent postoperative neuropathic pain presented for evaluation and management of CRPS type I symptoms on referral from the surgeon. The surgery was uncomplicated, and there was no evidence of postoperative infection. The patient developed significant allodynia in the postoperative period along the medial knee and down the medial leg. He had been initiated on gabapentin and titrated to 300 twice a day. He had also been taking acetaminophen 1000 mg every 6 hours, tramadol 50 mg every 8 hours as needed, and oxycodone 5-10 mg one to 2 times daily. Pain persisted despite this regimen. The patient decided to undergo LSB, and permission was obtained to hold warfarin with enoxaparin bridge prior to the procedure per ASRA guidelines and warfarin clinic recommendations. The patient underwent uneventful LSB and achieved almost complete pain relief for 5 days. For the procedure, the patient was positioned prone. A fluoroscopic view of the lumbar spine was obtained, and the L2 vertebra was identified. The skin and subcutaneous tissues were anesthetized with 1% lidocaine using a 1.5-inch, 25-gauge needle. A 22-gauge, 5-inch spinal needle was advanced under fluoroscopic multiplane imaging guidance onto the anterolateral surface of the L2 vertebra on the left side. Contrast dye was injected under live imaging without evidence of vascular uptake. A test dose of 5 mL of 1% lidocaine was injected with no adverse effects elicited. Lastly, 15 mL of 0.25% bupivacaine mixed with 50 mcg of clonidine and 5 mg of dexamethasone was injected incrementally. All needles were removed, and the patient tolerated the procedure well. Warfarin was resumed the evening after the procedure. Five days later, he developed severe left flank, abdomen, and groin pain. Computed tomography (CT) scan in the emergency department (ED) revealed a large left psoas hematoma (7.5 x 6.4 x 18.9 cm). The patient was admitted to the hospital for evaluation and management. Prothrombin time (PT)/INR was 16.7/1.3. Warfarin was held for 3 days before being restarted with gradual improvement of the hematoma on CT (6.6 x 6.2 x 18.1 cm). In outpatient follow-up, the patient had persistent left flank, abdomen, and groin pain, as well as left psoas and quadriceps muscle weakness; he also experienced nocturnal muscle spasms in this thigh. Physical exam revealed a flexed attitude of the left hip, impaired left hip limb advancement during gait, left quad atrophy, reproducible psoas pain with hip flexion, extension, and internal and external rotation, 3 of 5 muscle strength in left hip flexion, 4 of 5 strength in knee extension, and improving hyperesthesia in the left medial thigh and calf. His gabapentin was increased to 300 mg 3 times a day, and he was started on tizanidine 2 mg as needed for spasms. These, in addition to acetaminophen 1000 mg every 8 hours and tramadol 50 mg every 6 hours as needed, helped to manage his symptoms. He was reenrolled in a formal physical therapy program with a focus on ambulatory gait and balance training, left lower extremity desensitization therapies, gentle hip range-of-motion and mobility exercises, and left quad strengthening. Over the course of 3 follow-up visits, his pain and mobility improved, as did his strength with left hip flexion from 3 of 5 to 4 of 5 strength.

DISCUSSION

The L1-L5 lumbar sympathetic ganglia are located posteriorly to the thoracolumbar fascia within the perivertebral space and posteromedially to the psoas muscle. Lumbar vertebral arteries pass backwards off the aorta and course around the vertebral bodies. Lateral branches pass through the psoas and quadratus lumborum muscles. Lumbar veins take a similar course and drain into the inferior vena cava (3). IPH may be caused by spontaneous bleeding secondary to anticoagulation, ruptured abdominal aortic aneurysm (AAA), postoperative AAA repair, hemophilia, status post surgery or biopsy, trauma, or a tumor (4). The clinical presentation of an IPH is often vague and nonspecific. Signs/symptoms may include abdominal, pelvic, back, or groin pain, and/or swelling. Patients may present with signs of hemorrhage including tachycardia, hypotension, or a drop in hemoglobin levels. Pressure effects can cause constipation, urinary frequency, or compression of nearby neurovascular structures, most commonly the femoral nerve. Pressure on the iliac muscle and expansion of fascia contributes to limited hip flexion and extension and compensatory bending deformation of the hip (5). Acute and chronic hematomas may be differentiated using a variety of imaging modalities. On ultrasound, acute hematomas are hypoechoic and may have a similar appearance to a cyst; chronic hematomas are hyperechoic and may show evidence of septa formation, calcification, or appearance as a solid mass. On CT, acute hematomas will present as an area of high

attenuation with or without fluid-fluid level, while chronic hematomas may have a similar appearance to psoas abscesses and present with low attenuation. On magnetic resonance imaging, which is the most sensitive, acute hematomas appear isointense/slightly hypointense to muscle on T1-weighted imaging, and either hypointense or hyperintense on T2. Subacute hematomas present with a high-intensity rim, higher-intensity peripheral zone, and lower-intensity core on T1 and relatively higher signal from core to periphery on T2 when compared to T1. Chronic hematomas show a hypointense rim on both T1 and T2 (4). Treatment includes discontinuation of any anticoagulant or antiplatelet medications as well as reversal of abnormal coagulation parameters and possibly transfusion. The IPH is monitored for expansion or evolution with serial imaging. If the IPH is rapidly expanding or causing significant mass effect with neurologic sequelae, the patient may undergo percutaneous or surgical drainage. Percutaneous drainage should be attempted before surgical decompression despite the difficulty of draining intramuscular hematomas. Conservative treatment is recommended for patients with hemophilia or other coagulation disorders. Allowing for the natural course of spontaneous resolution may be pursued with supportive care including pain control, gentle range-of-motion exercises, physical therapy, etc. If femoral neuropathy is present, recovery may be prolonged, taking up to 3 to 6 months or more depending on the extent of injury (5). Femoral neuropathy most commonly occurs when the femoral nerve is compressed by the hematoma or as it passes under the inguinal ligament anterior to the iliopsoas muscle. The clinical presentation of femoral neuropathy includes groin pain, loss of power in hip flexion and knee extension, loss of/reduced patellar reflex, decreased sensation in the anteromedial thigh and medial aspect of the lower leg, and loss of quadriceps muscle bulk. Common causes are usually iatrogenic in nature and are consequences of pelvic and hip surgeries, vascular procedures requiring femoral access, femoral nerve blocks, prolonged lithotomy positioning, and retroperitoneal hematomas. Non-iatrogenic causes include pelvic masses, trauma, infection, diabetes, alcoholism, and radiation injury (7). Nerve conduction studies of the peroneal and sural nerves will be normal while saphenous nerve conductions may be abnormal or absent. Electromyography (EMG) of the vastus medialis and intermedius will likely show evidence of denervation manifesting as positive sharp waves (PSWs) and fibrillations. The iliopsoas will also likely show significant PSWs and fibrillations. All other muscles (innervated by the sciatic nerve) should be normal. Hip flexion weakness may be secondary to pain limitation or may be pathologic in nature; assessment of recruitment pattern on EMG studies may be helpful in distinguishing between these possibilities. The prognosis of femoral neuropathy depends on the intensity and duration of nerve compression which result in damage to myelin, axons, or both. After Wallerian degeneration takes place, axons regrow at a rate of approximately one mm/day, one cm/week, and 2.5 cm/month (although this is complicated by many factors). Recovery takes several days to several months with most cases resolving within 6 months. Active rest, with gentle progressive mobilization and strengthening, followed by formal physical therapy, are recommended (8).

CONCLUSION

Because of the close proximity to blood vessels in the typical LSB approach, the incidence of inadvertent intravascular injection/trauma can be as high as 12.5% (9). In the case of our patient, despite appropriate INR, he likely experienced a violation of a perivertebral vein resulting in a slow bleed that worsened as anticoagulation was reintroduced. Overall, the natural course of his symptoms improved gradually with supportive care and physical therapy as expected. In summary, the decision to hold anticoagulants is balanced against the risk of adverse cardiovascular or cerebrovascular events. Further studies should be done to evaluate the risk and incidence of hematoma in LSBs, which are frequently performed in our field, to help guide future decision-making regarding procedural safety in anticoagulated patients.

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