Compressive Neuropathy of the Brachial Plexus and Long Thoracic Nerve*

A Rare Complication of Heparin Anticoagulation

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We present a case of a 69-year-old woman who developed brachial plexopathy and long thoracic nerve palsy secondary to compression from a hematoma while receiving heparin therapy for the treatment of a stroke. The patient was treated conservatively with discontinuation of heparin and had complete resolution of her compressive neuropathy. This is the first report of a patient with long thoracic nerve palsy with a brachial plexopathy complicating anticoagulation. We review the literature on hematoma-induced compressive neuropathies and treatment options. Our review concludes by emphasizing the importance of clinical judgment in determining the best therapeutic modality.

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Key words: anticoagulation; brachial plexus; compression; hematoma; long thoracic nerve

Abbreviation: APTT = activated partial thromboplastin time

Anticoagulant therapy is frequently used in both the inpatient and outpatient settings. As a consequence, clinicians frequently have to deal with the side effects of anticoagulation. In an attempt to increase general awareness, we report an unusual complication of heparin treatment and explore treatment options after a brief review of the literature.

CASE REPORT

A 69-year-old woman with a history of hemicolectomy for Dukes class D colon cancer was admitted to the hospital for evaluation and treatment of the ileus in association with findings of multiple mesenteric and omental soft tissue masses that were consistent with metastases on CT scans of the abdomen and pelvis. A chest radiograph was normal, and the results of a total body bone scan were negative.

Two days later, she developed acute binocular vision loss and was started on IV heparin therapy. A CT scan of the head revealed ischemic changes in both occipital lobes, an ultrasound of the neck showed normal flow in the vertebral arteries, and transesophageal echocardiography showed a small patent foramen ovale with trivial right-to-left shunting. No evidence of deep venous thrombosis was found on duplex ultrasonography of the lower extremities. The patient’s vision returned to normal over the next few days.

On day 5 of heparin therapy, the patient was grasping the bedrail with her left hand in an attempt to roll over and developed a sudden onset of moderate left shoulder pain. Initially, there were no positive findings on physical examination, and she was treated with morphine. The results of laboratory tests at that time showed the following: activated partial thromboplastin time (APTT), 101 s (range, 21 to 33 s); hemoglobin level, 11.5 g/dL (range, 12 to 15.5 g/dL); platelet count, 233,000/μL (range, 150,000 to 450,000/μL). The rate of heparin infusion was decreased to achieve a target APTT of 60 to 90 s.

By the following day, the patient’s left shoulder pain had increased. Inspection revealed a large ecchymosis over the left axilla and flank, as well as mild left scapular winging, but no radiculitis. The patient was not able to abduct her left arm beyond 50°, and the scapular winging was accentuated by having her push against the examiner’s hands. She was able to reasonably adduct, internally and externally rotate, and flex and extend the humerus, but a full assessment of left shoulder motor strength was limited by pain. Motor strength was normal in the remainder of the arm. Sensory examination, deep tendon reflexes, and upper extremity arterial pulses were normal. At that time, her APTT was 78 s, hemoglobin level was 10.4 g/dL, and platelet count was 224,000/μL. Heparin therapy was discontinued, and an MRI of the left brachial plexus was obtained, revealing a 7 × 4-cm mass subjacent to the left scapula in the region of the left axilla, which was consistent with a large hematoma (Fig 1). The hematoma involved the subscapularis muscle on the left and compressed the brachial plexus anteriorly (Fig 2). The hematoma also compressed the posterolateral left thorax in the region of the long thoracic nerve (Fig 3). An underlying metastasis was not seen.

Since the patient had only mild deficits, she was treated conservatively with analgesics and by withholding further administration of anticoagulant drugs. She had complete resolution of pain and a return of normal motor strength over the next 5 days, and winging of the scapula was no longer evident after 1 week.

DISCUSSION

The brachial plexus is formed by the union of the ventral rami of C5–T1. The plexus runs in the posterior triangle of...
the neck then emerges between the scalene muscles, passes posterior to the clavicle, and lies on the first digitation of the serratus anterior and the subscapularis muscles. The long thoracic nerve is a supraclavicular branch of the brachial plexus and is formed by the roots from the fifth to the seventh cervical rami. It descends dorsal to the brachial plexus as well as the first part of the axillary artery and crosses the superior border of the serratus anterior to reach its lateral surface. It then continues downward to the lower border of the serratus anterior, supplying branches to each of its digitations.1

Compressive neuropathies from hematomas were first described in the mid-1960s as treatment with anticoagulant drugs became more widely utilized. Of the reports in the literature, this complication seems to be more common with heparin therapy (45%) than with warfarin therapy (18%) and more frequently involves the lower extremity nerves (87%).2 Hematomas causing compressive neuropathies may occur within the intended therapeutic range of anticoagulation (40%).3 A conservative approach to management seems to yield good results in 85% of cases.4 In one review, early surgical evacuation of the hematoma within 48 h, when necessary for severe symptoms, resulted in improvements in all patients, while late operations after 48 h resulted in improvements in about half of all patients.3 Merrick et al,4 in 1991, were the first to report good results after percutaneous decompression of a retroperitoneal hematoma secondary to heparin treatment in a patient who was not an immediate candidate for surgery.

Neuropathy in association with a hematoma likely results from local injury to nerves by compression. In minor cases, this may result in temporary demyelination of the nerve locally, but in more severe cases axonal injury may occur with wallerian degeneration distal to the site of compression.4

Our patient had progressive signs and symptoms of brachial plexopathy. This was evident by her inability to fully abduct her left arm, even with control of her pain by analgesics. Abduction of the arm is a coordinate function of several muscles. The deltoid and supraspinatus muscles, aided by the stabilizing early contraction of the subscapularis, teres minor, and infraspinatus muscles, result in early abduction to 90°; this is followed by the action of the serratus anterior muscle, which is indispensable for full abduction (90° to 180°).

The scapular winging and difficulty with abduction implicates compression of the long thoracic nerve in our patient. The impairment of other nerves or nerve roots supplying the subscapularis, teres minor, supraspinatus, and deltoid muscles also may have contributed to her difficulty with abduction. Her mild scapular winging at rest could have been a manifestation of the subscapular hematoma itself, but it is very unlikely for static winging (ie, that due to an anatomic defect) to be accentuated by pushing against a wall.5

The hematoma in this patient may have resulted from trauma or from bleeding into a metastasis, or it may have occurred spontaneously. It is very rare for colon cancer to metastasize to muscles, although a few cases have been

![Figure 1. T2-weighted axial image shows a 7 × 4-cm hematoma with fluid-fluid hematocrit level (black arrow) subjacent to the left scapula.](image1)

![Figure 2. T1-weighted sagittal view through the hematoma shows it within the subscapularis muscle (black arrow) and compressing the brachial plexus in the region of the axillary artery and vein (white arrow).](image2)

![Figure 3. T1-weighted axial image showing the hematoma (black arrow) compressing the lateral thorax adjacent to the serratus anterior muscle and long thoracic nerve.](image3)
described. Rather, the more likely explanation is a minor muscle or soft tissue injury that occurred while she was pulling herself over in bed and resulted in a capillary bleed.

On review of the literature, no previous report of long thoracic nerve palsy and very few reports of brachial plexopathy as a complication of anticoagulation came to our attention. Salam reported a case of a 68-year-old woman receiving warfarin therapy and using crutches who developed total paralysis and a complete loss of sensation in the left arm. That patient underwent evacuation of a large tense hematoma from her left axilla, with satisfactory return of both motor and sensory function. Another report included two cases of brachial plexus compression by a hematoma following jugular puncture. The two patients, one of whom was receiving warfarin therapy, developed partial deficits of upper limb motor and sensory function and were managed successfully with conservative treatment. In a third report, a 68-year-old woman receiving warfarin therapy experienced a fall and had progressive motor loss below the shoulder. A large hematoma compressing the brachial plexus was drained. The patient had progressive return of function over 2 years. Finally, Hoyt et al reported on a 61-year-old man receiving warfarin therapy who fell and developed progressive right extremity weakness culminating in a right wrist drop after 7 days. This patient underwent surgical evacuation of a large hematoma within the coracobrachialis muscle, which was displacing the brachial plexus. Unfortunately, a follow-up examination did not demonstrate significant improvement in his motor or sensory functions.

While the literature contains only a few reports of brachial plexopathy as a complication in patients receiving anticoagulant therapy, there are frequent case reports of brachial plexopathy secondary to a compressive hematoma as a complication of axillary arteriography or arteriotomy. In these cases, the mechanism of plexopathy is thought to be an expanding hematoma within the axillary sheath with secondary compression of the nerves and cords within this sheath. Satisfactory results in affected patients are usually obtained from early surgical intervention, and delaying surgery may result in permanent neurologic damage.

Whether a hematoma compressing the brachial plexus should be treated conservatively or surgically depends on anatomic and clinical features. Surgical intervention should be considered for the treatment of hematomas in the axillary sheath and in patients with severe motor or sensory impairment. On the other hand, when the hematoma is small to moderate in size, free to expand into the surrounding soft tissues of the axilla, and the neuropathy is not progressive or severe, conservative treatment is likely warranted with discontinuation or reversal of anticoagulation therapy using vitamin K, protamine sulfate, or fresh frozen plasma to help halt the expansion of the hematoma.

REFERENCES
4 Sunderland S. Nerves and nerve injuries. 2nd ed. London, UK: Churchill Livingston, 1978; 147
8 Fuller GN, Dick JPR, Colquhoun IR. Brachial plexus compression by hematoma following jugular puncture. Neurology 1994; 44:775–776

Spontaneous Hemomediastinum Complicating Steroid-Induced Mediastinal Lipomatosis*

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Spontaneous hemomediastinum is a rare event, occurring in association with bleeding disorders, intratumoral bleeding, or following an abrupt increase in intrathoracic pressure. We report the case of a patient with systemic lupus erythematosus, nephrotic syndrome, and renal failure, in whom mediastinal lipomatosis (ML) developed following increased corticosteroid therapy. Anticoagulant therapy likely precipitated a massive spontaneous hemomediastinum secondary to diffuse hemorrhage of mediastinal fat, which required emergency decompressive surgery. Steroid-induced ML is common and usually well tolerated, but clinicians should be aware of its potential risk of bleeding when associated with anticoagulant therapy. This case further emphasizes the bleeding complications of treatment with low-molecular-weight heparin in patients with renal failure.

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Key words: drug therapy; complications; hemomediastinum; low-molecular-weight heparin; renal failure; systemic lupus erythematosus