Acute Compartment Syndrome in Osteochondromatosis

JOAO O. TAVARES, MD

In 1881, Volkmann described muscular ischemia as the cause of “paralytic contractures” that occurred after upper extremity trauma. Eaton and Green depicted a cycle of increasing muscle ischemia in which ischemia leads to anoxia of muscle, which produces histamine-like substances that dilate the capillaries and increase endothelial permeability and edema. This increases the pressure in the compartment, leading to further ischemia until irreversible muscle and nerve damage occurs. Multiple causes include fractures, arterial injury, snake bite, drug overdose, and burns. Exertional states are also believed to produce acute and chronic compartment syndrome in the leg.

In experimental animals, hyperemia caused by exercise was shown to increase the muscle mass within the anterior compartment by approximately 20%. Reactive hyperemia after tourniquet release produced the same effect. After 2 hours of tourniquet use, muscle mass increased 20%-30%, and after 3 hours, 30%-50%.

Conversions of chronic to acute anterior compartment syndrome are known to occur with exercising and surgical repair of muscle herniation of the anterior compartment of the leg. This article presents a patient who developed an acute compartment syndrome after surgery for osteochondromatosis. He had a history of exercise-related pain in the same lower extremity.

CASE REPORT

A 13-year-old boy presented with left knee and calf pain, which occurred after walking for >30 minutes. He had a 3.2-cm leg-length discrepancy, with the right lower extremity longer than the left. He also had bilateral genu valgum and a valgus deformity of the left ankle.

On examination, a large osteochondroma was palpable on the posterolateral aspect of the proximal left calf. The deformities were visible on radiographs (Figure 1). Computed tomography revealed a large osteochondroma arising from the fibula, and compromising the lateral and posterior compartments (Figure 2).

On the right lower extremity, a percutaneous epiphysiodesis of the distal femur was performed and the osteochondroma was removed from the proximal medial tibia. Tourniquet time was 59 minutes. On the left side, staple epiphysiodesis of the medial femur and tibia and screw epiphysiodesis of the medial malleolus were performed. Tourniquet time was 69 minutes.

On postoperative day 1, the patient experienced a sudden increase of pain in the left calf 90 minutes after epidural catheter removal. Maximum tenderness existed over the posterolateral fibular osteochondroma. No pain occurred with active or passive flexion and extension of toes; pain increased with passive dorsiflexion of the ankle.

The patient reported that his left foot felt “funny,” but sensation was intact to pin prick, especially on the first web space. Blood pressure was 130/85 mm Hg. Anterior compartment pressure was 15 mm Hg; posterolateral pressures in the area of maximum tenseness pressure were 25 mm Hg.
were 25, 27, and 25 mm Hg; posterior pressure was 18 mm Hg. Observation was continued. The patient was advised to roll on his right side to avoid pressure on the osteochondroma.

On postoperative day 2, the patient reported feeling better, but still required intravenous morphine sulphate for pain. On postoperative day 3, he had numbness on the lateral aspect of the ankle and foot, weak peroneal muscle function, and extreme pain with passive dorsiflexion of the ankle. Active and passive motion of the toes was painless. Blood pressure was 127/65 mm Hg. Compartment pressures were 16 mm Hg anterior, 42 and 38 mm Hg posterolateral.

A one-incision perifibular fasciotomy was performed with decompression of the anterior, lateral, and posterior superficial compartments. The lateral and posterior superficial compartments were involved. Closure of the incision was achieved 4 days after fasciotomy, with tension-relieving suture. No muscle necrosis was observed.

At 6-month follow-up, the patient had no improvement of the genu valgum of the left knee; however, complete recovery of muscle function on the left leg occurred (Figure 3). The episodes of claudication on the leg resolved.

**DISCUSSION**

Osteochondromas represent the most common benign bone tumor. Disturbances of growth may occur in multiple osteochondromatosis. Widening of the metaphysis of long bones and valgus deformities of the knees and ankles are also often seen.

Tumor resections are not routinely performed, except if the tumor interferes with function or a deformity requires correction. Serious complications can occur from resection. In 1997, Wirganowicz and Watts reported 80 patients with multiple surgeries. The most common complications were peroneal neuropraxies. An arterial laceration and a compartment syndrome were also reported. These bony masses may become symptomatic by causing pressure on tendons, resulting in bursitis and compression of vessels and nerves. Chronic compartment syndrome may potentially cause pain with activity.

Our patient reported leg pain with activity that was relieved by rest. In retrospect, this represents intermittent claudication, similar to chronic compartment syndrome, an accepted entity. Symptoms of claudication were relieved by fasciotomy, without removing the offending large osteochondroma in the fibula.

Some patients with osteochondromatosis, by virtue of their bony masses, have intermittent claudication suggestive of a chronic compartment syndrome. In these patients, surgery and a tourniquet can precipitate an acute compartment syndrome, especially if the offending osteochondromas are not removed. In these cases, postoperative epidural anesthesia should be used cautiously, as it can mask the extreme pain that is the hallmark of impending compartment syndrome. Careful observation and early diagnosis are critical in preventing irreversible damage caused by compartment syndrome.

**REFERENCES**


