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Bilateral Lower Extremity Anterior Compartment Syndrome in a Severely Hypothyroid Patient

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Compartment syndrome represents a potentially devastating surgical emergency that requires prompt recognition and definitive treatment. Early fasciotomy is essential to maximize the opportunity of a good outcome. The complications of compartment syndrome are often quite severe and include neuropathy, rhabdomyolysis (and its sequelae), and limb loss. The most common causes are typically related to trauma (orthopedic injury, crush injury, severe burns), vascular insult, or external restriction of the compartment, as in the case of an ill-fitted orthopedic cast.¹

Although rare, other causes of compartment syndrome have been reported. One such rare cause, reported only twice previously, is severe hypothyroidism.^{2, 3} The following case describes a third such case encountered at our institution.

The patient was a 60-year-old man who had been admitted the day before undergoing coronary angiography and stenting of a midleft anterior descending coronary artery stenosis diagnosed after an evaluation for new-onset substernal chest pain. This proceeded without incident. The patient was noted to have bilateral foot drop on his initial postprocedure assessment. When asked, the patient admitted that he had progressive anterior leg pain and cramps for several days, which he attributed to his long history of back problems and radiculopathy. When notified, his cardiologist felt that the patient's lower extremities felt tense to palpation and consulted surgery to exclude a possible compartment syndrome.

His medical history was significant for chronic back pain, hypertension, and longstanding hypothyroidism. He admitted to noncompliance with his levothyroxine for the past several months. His review of symptoms was significant for recent weight gain, leg pain, paresthesias, and the aforementioned angina pectoris.

On examination, the patient was noted to have a small multinodular goiter. Other pertinent findings included bilaterally tense lower extremities and bilateral foot drop with complete loss of dorsiflexion and impaired sensation over the dorsum of the foot. Plantarflexion was normal. Pulses were palpable (2+) and symmetrical from the femoral arteries down to, and

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including, both the dorsalis pedis and posterior tibial arteries bilaterally. Pain was present on both palpation of the anterior compartment and on passive movement of the foot.

Laboratory studies revealed the following abnormal values: thyroid-stimulating hormone (TSH) 176 $\mu\text{m}/\text{mL}$, total creatinine kinase (CK) 68,000 IU/L, and serum creatinine 1.6 mg/dL. A complete blood count was within normal limits. His urine was tea-colored and tested positive for the presence of myoglobin. Compartment pressures were obtained at the bedside and revealed pressures of 75 mmHg in both anterior compartments and pressures of 10 to 15 mmHg in both the lateral and posterior compartments bilaterally. Based on these data, the patient was vigorously resuscitated with alkalinized saline and taken to the operating room for four-compartment fasciotomy.

At surgery, the muscles of the both anterior compartments were found to be frankly necrotic, whereas the muscles in the remaining compartments were healthy and viable. After fasciotomy and débridement, dressings were applied and the patient was taken to the intensive care unit. His CK levels began to decrease immediately after surgery and his urine myoglobin quickly became undetectable with no observed renal sequelae. He was started back on oral levothyroxine with steady improvement in his TSH level. Delayed primary closure of the fasciotomy wounds was performed three days later, and the patient was discharged to a rehabilitation center for gait training. He is currently ambulatory with bilateral lower extremity ankle braces.

Compartment syndrome results from an increase in pressure within a confined fascial space. This results in impaired tissue perfusion and subsequent tissue damage. Untreated, it results in severe permanent disability and even limb loss. The symptoms are well established and are listed in Table 1.¹ Although many etiologies exist, they can be divided into two major groups. The first are those that increase the volume of the compartmental contents. These include, for example, swelling from direct tissue trauma or the edema of reperfusion after treatment of a vascular insult. The second group consists of those etiologies that restrict the size of the compartment. Examples include poorly fitted orthopedic casts or constrictive eschar formation after major burns.¹

Hypothyroidism has been implicated as a cause of compartment syndrome in two previously reported cases. In both cases the presentation was subacute, as it was with our patient, and in both cases, the patients had significant permanent disabling sequelae as a result of the delay in diagnosis.^{2, 3}

Although the exact mechanism by which hypothyroidism causes compartment syndrome is not known, a number of theories exist. Skeletal muscle hypertrophy (Hoffman syndrome) may occur as a consequence of hypothyroidism. Additionally, hypothyroidism is associated with deposits of glycosaminoglycans and extravasation of proteinaceous fluid into the interstitial space. Connective tissue proliferation within the muscle compartment from TSH-induced stimulation of fibroblasts may also be a contributing factor.⁴ All of these causes result in a gradual increase in the intra-compartmental contents and, because of its gradual nature, may result in the subacute presentation seen in our case and in the two cases reported previously. Also, the bilateral nature of the compartment syndrome suggests a systemic

(rather than a local) cause. It is unclear why this process appears to have a predilection for involving the anterior compartments of the lower extremity.

In summary, we describe a rare complication of hypothyroidism with potentially devastating consequences if not diagnosed and treated promptly. We stress the importance of a high index of suspicion for the possibility of compartment syndrome in the hypothyroid patient with leg pain and encourage early surgical consultation (and direct compartmental pressure measurement) for cases in which compartment syndrome remains in the differential diagnosis.

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Table 1**Signs and Symptoms of Extremity Compartment Syndrome**

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- Pain in the involved extremity, out of proportion to apparent degree of injury
 - Pain on passive stretching of the extremity
 - Loss of sensation
 - Paresthesias
 - Impaired motor function (ranging from weakness to paralysis)
 - Tenderness to palpation of the affected compartment
 - Tense/edematous extremity to palpation
 - Compartmental pressure >25–30 mmHg
 - Pulselessness (late)
 - Rhabdomyolysis/myoglobinuria (late)
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