# Unlikely recovery in a delayed diagnosis of compartment syndrome

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Compartment syndrome (CS) is defined as a pathologic elevation of hydrostatic pressure within a closed compartment of muscle leading to the development of ischemia and myonecrosis.[1] Causes of CS include trauma, infection, and neurogenic or musculoskeletal conditions; among these, trauma is most common. Tibial fractures account for up to 12% of all CS cases.[2] Diagnosis is typically based on clinical presentation: pain, pressure, paresthesia, paresis, and diminished pulses. The criterion standard treatment for CS is fasciotomy; outcomes are best when there is no delay in treatment. The use of hyperbaric oxygen therapy (HBOT) in CS has been reported in a handful of case reports[3-5]; however, to the best of our knowledge, there is no report of using HBOT in a delayed presentation of CS. We report a case of a young man with a delayed presentation of CS being followed up by his family physician and treated with a combination of HBOT and physical therapy (PT).

## Case

A 22-year-old South Asian man with a history of type 1 diabetes with poor glycemic control and recreational use of anabolic steroids for at least 6 weeks presented to his family physician with pain and swelling bilaterally around his shins and ankles. The patient's symptoms began 5 days earlier and he had been given a diagnosis of medial tibial stress syndrome (shin splints) on day 4 after presenting to the emergency department. On day 5, the patient was assessed by his primary care physician and bloodwork was ordered based on the clinical presentation to rule out CS. He was found to have elevated levels of creatine kinase (13272 U/L), white blood cells ($22.3 \times 10^9$/L), and alanine aminotransferase (208 U/L). On the evening of day 5, the patient returned to the emergency department for further evaluation.

The patient was assessed for CS by the orthopedic surgeon on call. During physical examination of the patient's legs, the surgeon found tense anterior compartments and bilateral lower limb swelling with erythema on the anterior tibias. Sensory grading using the American Spinal Injury Association Impairment Scale was assessed to be 1 of 2 in the deep peroneal nerve bilaterally and 2 of 2 in the superficial peroneal, saphenous, sural, and tibial nerves. There was decreased motor function bilaterally (American Spinal Injury Association Impairment Scale muscle strength scores: extensor hallucis longus muscle, 0 of 5; extensor digitorum longus muscle, 0 of 5; tibialis anterior muscle, 0 of 5). The anterior compartments of the patient's left and right legs demonstrated compartment pressures greater than 100 mm Hg (normal <10 mm Hg). The lateral compartment pressures of his left and right legs were 46 mm Hg and 49 mm Hg, respectively. The pressures within his superficial posterior compartments were 29 mm Hg and 20 mm Hg for his left and right legs, respectively. His deep posterior compartment pressures were less than 10 mm Hg bilaterally. The patient's creatine kinase level was above 10000 U/L and his blood glucose level was 17 mmol/L.

Given the potential risks versus benefits associated with fasciotomy in a patient with poor glycemic control and delayed presentation of CS, a decision was made to manage conservatively. This approach was taken with the

## Editor's key points

- While surgical decompression is the mainstay treatment for acute compartment syndrome (CS), the management of delayed CS is controversial; delayed fasciotomy has been associated with increased rates of late amputation and infection. The risk is increased in patients with immunocompromising comorbidities.
- This case describes the successful outcome of a more conservative treatment modality for CS, hyperbaric oxygen therapy (HBOT), in combination with physical therapy (PT) for a patient whose delayed presentation of CS and history of poor glycemic control and anabolic steroid use made him a poor candidate for fasciotomy.
- When surgical decompression is not indicated, a trial of HBOT with PT might be warranted if there are no contraindications. The synergistic effects of HBOT and PT need to be further researched to determine which patients will benefit most and which injuries will have a better prognosis.

## Points de repère du rédacteur

- Bien que la décompression chirurgicale soit le traitement habituel pour le syndrome des loges aigu (SL), la prise en charge d'un SL tardif est controversée : un retard dans l'aponévrotomie a été associé à des taux accus d'amputation et d'infection ultérieures. Ce risque augmente chez les patients atteints de comorbidités causant une immunodépression.
- Ce cas décrit l'issue favorable de modalités thérapeutiques plus conservatrices pour le SL, notamment une oxygénothérapie hyperbare (OTHB), combinée à de la physiothérapie (PT), pour un patient dont la présentation tardive du SL , les antécédents de contrôle glycémique médiocre et l'utilisation de stéroïdes anabolisants en faisaient un candidat risqué pour l'aponévrotomie.
- Lorsque la décompression chirurgicale n'est pas indiquée, un essai d'OTHB et de PT pourrait être justifié s'il n'y a pas de contre-indications. Les effets synergiques de l'OTHB et de la PT doivent faire l'objet de recherches plus approfondies pour déterminer les patients qui en bénéficieraient le plus et les blessures qui auraient un meilleur pronostic.

patient's understanding that improvement in his lower limb functionality was improbable. He would likely have permanent foot drop and would require a foot drop splint to engage in physical activity. At discharge, our patient was prescribed naproxen as necessary for analgesia, rapid-acting insulin (dose given according to carbohydrate count), 36 units of insulin glargine, and deep vein thrombosis prophylaxis. Physical therapy and HBOT were started within 1 week of discharge.

Magnetic resonance imaging 1 month after discharge showed signs of myonecrosis on the right and left tibialis anterior muscles and findings of vascular compromise; there was no change in the management plan. Drastic improvements were seen within 6 months; the patient no longer needed foot drop splints and had a strength score of 4 of 5 in both legs. Our patient has since improved his glycemic control (glycated hemoglobin $A_{1c}$ level of 5.9%) and has returned to regular day-to-day activities with minimal limitations.

## Discussion and conclusion

Compartment syndrome is a surgical emergency that requires surgical decompression as soon as possible to avoid the onset of tissue necrosis. The increased compartmental pressure leads to decreased oxygen availability. When fasciotomy is done after 12 hours, only two-thirds of patients recover normal limb function.[6] While most cases of CS are caused by a fracture, other causes include burns, blunt-force trauma, and thromboembolisms. Pre-existing conditions such as anabolic steroid use, as in our patient, and diabetes mellitus increase the risk of developing CS; anabolic steroids cause rapid expansion against muscle fascia secondary to muscle hypertrophy.[7] Anabolic steroids have also been shown to promote insulin resistance. With our patient's physical activity and medical history, the compounding effects of exercise, poor glycemic control, and anabolic steroid use likely led to the development of CS.

While early diagnosis of CS is optimal in managing a patient, delayed diagnoses can occur owing to unclear presentation, slow-evolving presentation, or delays in seeking medical care. Our patient did not disclose his anabolic steroid use, which likely contributed to the delay in his diagnosis.[8] Delayed management of CS can lead to complications such as severe intractable pain, delayed fracture union, loss of function, infection, neurologic deficit, and amputation.[9,10] While surgical decompression is the mainstay treatment for acute CS, the management of delayed CS is controversial; delayed fasciotomy has been associated with increased rates of late amputation and infection.[1,11] The risk is increased in patients with poor glycemic control or other immunocompromising comorbidities.

An alternative treatment modality for CS that has been discussed in the literature is HBOT.[4] Hyperbaric oxygen therapy increases oxygen availability to hypoxic tissues during the early postinjury period, while perfusion is inadequate. It increases tissue and blood oxygen levels by allowing for greater diffusion across edematous tissue and increases plasma oxygen levels. Hyperbaric oxygen therapy also induces vasoconstriction, which aids in reduction of edema.[12-14] Currently, there are no guidelines on how to use HBOT in delayed diagnosis of CS. In an acute case of CS, HBOT aids in diminishing reperfusion injury by destroying oxygen radicals. For acute CS, daily or twice-daily 90-minute sessions are used for 7 to 10 days; our patient completed weekly 90-minute sessions for 6 months for his delayed CS recovery.[14]

In conjunction with HBOT, our patient also underwent intensive PT consisting of soft-tissue massage, progressive resistance training, and neuromuscular stimulation. In total, he completed 64 sessions of PT (twice weekly) over 1 year. It can be postulated that the PT rehabilitation techniques used in our patient helped maintain blood supply and muscle stimulation.[15,16] Another factor to consider in managing delayed CS is the amount of muscle necrosis. Our patient might have had a better prognosis compared with patients discussed in other case reports owing to his creatine kinase level being much lower (10 000 to 15 000 U/L); other cases have reported levels of more than 30 000 U/L.[5]

While we do not suggest any change to the current standard of care in CS—surgical decompression—in cases where surgical decompression is not indicated, a trial of HBOT with PT might be warranted if there are no contraindications. The synergistic effects of HBOT and PT need to be further researched to determine which patients will benefit most and which injuries will have a better prognosis.

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**Competing interests**
None declared

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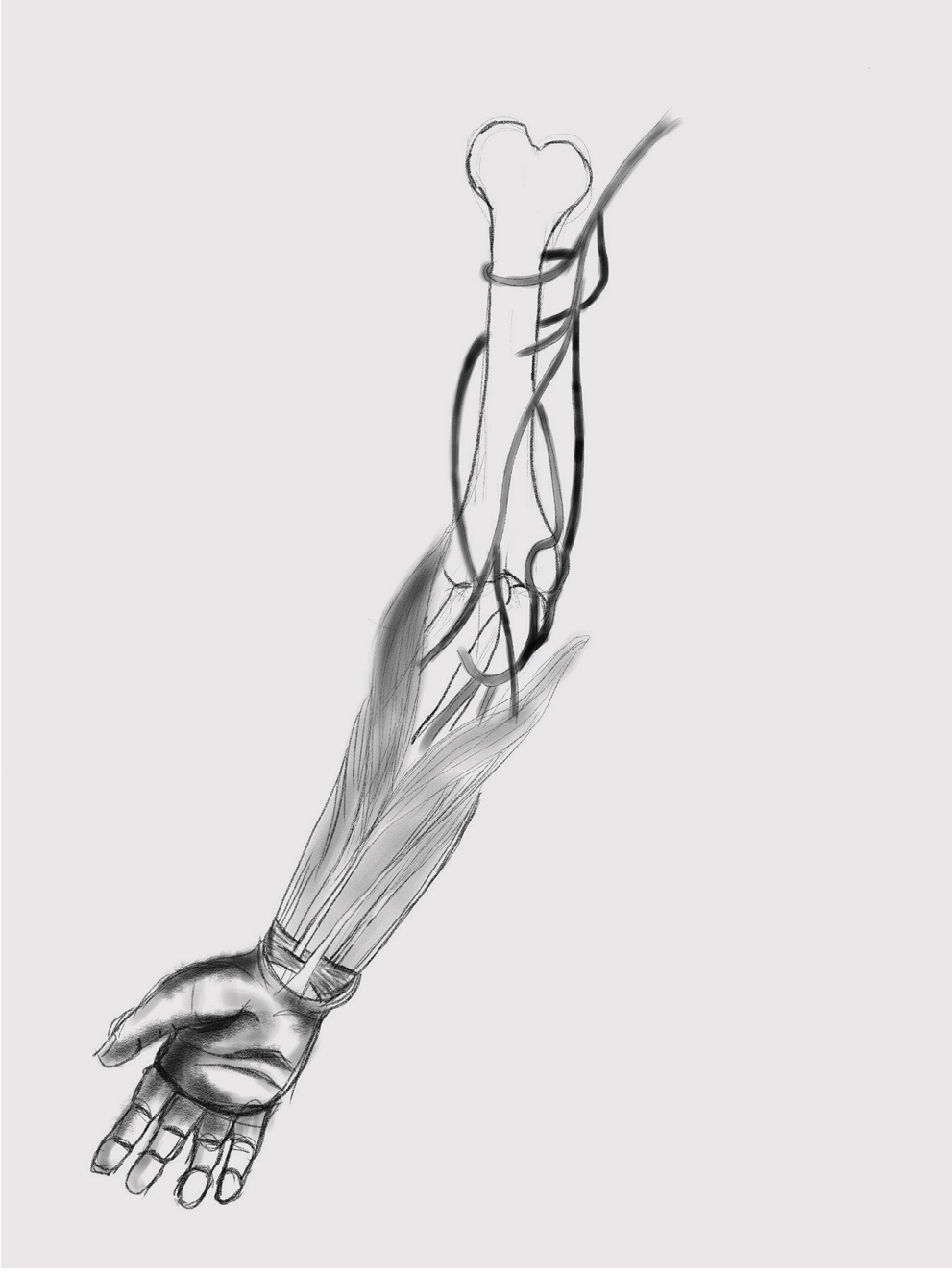

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