Acute Anterior Compartment Syndrome Secondary To Group A β-Hemolytic Streptococcal Myositis

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Compartment syndrome, the acute elevation of pressure within the fascial compartment of a muscle above that needed for capillary perfusion, is almost always associated with trauma. Infection, however, has been reported to cause this pressure phenomenon as well, as can excessive exercise, which is simply repeated microtrauma. Infectious causes of compartment syndrome have been previously reported by Dannemann, et al. in 1984,[1] Bohn and Coleman in 1985,[2] and by Knezevich and Torch in 1990.[3] As the muscle swells in response to these insults, the muscle volume can increase by as much as 20 percent.[4] The fascial compartments are nondistensible, so this increased volume is accompanied by increased pressure. Venous stasis is followed by frank ischemia. Inflammatory mediators are then released, causing further swelling. If left unchecked, muscle necrosis eventually develops. Compartment syndrome usually responds favorably to fasciotomy if performed promptly.[4,5]

We report a case of compartment syndrome caused by acute group A β-hemolytic streptococcal myositis.

Case Report

A 47-year-old unemployed professional male pilot, retired from the US Air Force, came to the Dover Air Force Base Emergency Department complaining of severe pain in the left lower leg that had begun several hours before admission. He reported no history of trauma. On review of systems he had had upper respiratory tract infection signs and symptoms (i.e., nasal congestion, mild nonproductive cough, fullness in the ears, and postnasal drip) during the 2 weeks before admission. Additionally, he had been febrile for 3 to 4 days before admission, and his fever had been accompanied by moderate to severe frontal headache. The patient was a nonsmoker and drank two to three alcoholic drinks daily; he denied risk factors for human immunodeficiency virus (HIV) infection. He did not participate in regular vigorous exercise.

His medical history was otherwise unremarkable.

At the initial physical examination the patient had a temperature of 101.5°F (38.6°C), a pulse of 100 beats per minute, respirations of 22/min, and a blood pressure of 136/85 mmHg. He appeared moderately ill and was in acute distress secondary to left anterior tibial pain. Head, eyes, ears, nose, and throat examination was remarkable for mild nasal mucosal bogginess with marked frontal sinus tenderness; his tympanic membranes had a splayed light reflex but were translucent. His throat was unremarkable aside from cloudy postnasal drainage. He had no anterior or posterior cervical adenopathy. His chest was clear to auscultation and percussion; his heart showed a regular rate and rhythm without murmur or gallop. His abdomen was nontender and not distended, and there was no hepatosplenomegaly or masses. Extremities were remarkable for a very tense, tender anterior compartment in the left lower leg. Stretching of the anterior compartment was accompanied by severe pain. Manometry showed an intracompartmental pressure of 55 cm H_2O on the left, compared with 23 cm H_2O on the right.

The patient was taken to the operating room, where an anterior compartment fasciotomy was carried out. Intraoperatively no purulent material was noted. The anterior tibial muscle was quite pale and ischemic; it promptly returned to its normal red color following fasciotomy. Muscle biopsy was obtained.

Postoperatively the cause of the patient's fever, which had reached 103°F (39.4°C) was investi-

Submitted, revised, 30 August 1994.

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gated. Findings on chest radiograph were normal. Radiographs of the involved extremity were completely normal. His white cell count was 9600/mm^3, and his urinalysis was unremarkable. Sinus radiographs showed marked left frontal mucosal thickening. HIV titer, rapid plasma reagin, antinuclear antibody, and rheumatoid factor were all negative. Urine and throat cultures were negative; the patient was not producing any sputum for culture. Neither an otolaryngologist nor computed tomography (CT) was available in the facility where the patient was treated, and moving the patient for these further studies was considered unnecessary for his immediate care.

Intravenous ampicillin was prescribed to combat the most likely pathogens in the frontal sinus (our laboratory had not reported any resistant strains of *Haemophilus influenzae*). This therapy would also be effective against the other two most likely pathogens in the sinuses, *Streptococcus pneumoniae* and *Moraxella catarrhalis*.[6] A blood culture subsequently grew group A β-hemolytic streptococci. The laboratory examining the muscle biopsy (which was at another facility in another state) was asked to perform a Gram stain of the tissue, which revealed many gram-positive cocci in pairs and short chains.

The patient had a good initial response to intravenous ampicillin, but a wound infection 7 days later required an opening of the operative site at the bedside and changing antibiotics to a nafcillin-gentamicin combination to contend with the most likely pathogen for a wound infection, *Staphylococcus aureus*. Twenty-four hours later the patient's fever again abated. Cultures from the wound site grew no organisms. Our internal medicine consultant believed the most likely cause of the recurrent infection was now anaerobes, so 3 days later, still afebrile and with a clean, dry wound, the patient was discharged home with a prescription for penicillin. He had an unremarkable recovery.

Discussion

This patient had acute anterior tibial compartment syndrome and acute frontal sinusitis in the absence of trauma. The characteristic findings of this syndrome were confirmed by an elevated intracompartmental pressure. The pressure was measured using a lumbar puncture needle and a saline manometer, connected by a three-way stopcock, all of which can be found on a standard lumbar puncture tray.

Sinusitis has been associated with many other conditions, almost all of them local, including osteomyelitis of the surrounding bones, cavernous sinus thrombosis, and brain abscess. Sinusitis has also been associated with allergic rhinitis and exacerbations of asthma or chronic obstructive pulmonary disease. Sinusitis usually follows a prolonged viral upper respiratory tract infection or, more commonly, an episode of prolonged nasal allergy. Both of these conditions interrupt air flow into the sinus, allowing initial resorption of the air within, then subsequent transudation of fluid, in which the normal flora of the sinus grow quite well. The resulting bacterial infection is usually accompanied by headache, nonproductive cough, fever, and nasal congestion with postnasal drip.[6]

A MEDLINE search from 1973 to the present revealed no previous report of acute compartment syndrome accompanying acute sinusitis. Lower respiratory tract infections are known to cause osteomyelitis by hematogenous spread; in children the osteomyelitis usually occurs in the long bones, which have a rich red bone marrow supply. In adults the osteomyelitis is usually limited to the vertebrae, one of the few adult reservoirs of red bone marrow.[7] Previously reported complications of sinusitis have been limited to those resulting from local spread: orbital complications (from preorbital cellulitis to cavernous sinus thrombosis), intracranial complications (from meningitis to cerebral abscess), pulmonary complications (bronchitis, asthma, and cystic fibrosis exacerbations), and other local complications (mucocele and localized osteomyelitis, known as the Pott puffy tumor).[8-11]

In 1985 Bohn and Coleman[2] reported a case similar to ours of a 44-year-old man who had streptococcal gangrene mimicking compartment syndrome. This patient also had upper respiratory tract complaints several days before admission, as well as a sore throat with a subsequently positive throat culture. In addition, Knezevich and Torch[3] reported a case of streptococcal toxic shock leading to bilateral lower extremity compartment syndrome, rhabdomyolysis, and renal failure. The site of the patient's index infection was the vagina, as is typical of toxic shock. Both of these patients suffered life-threatening crush syndrome in which the sudden release of myoglobin

causes acute renal failure, and the first patient actually lost one leg. Streptococcal fasciitis has also been reported to cause compartment syndrome with rhabdomyolysis.[1]

In our patient, although cultures of the fascial compartment regrettably were not obtained at the time of surgery, the clinical presentation of compartment syndrome in the absence of any trauma, a clear radiographic picture of sinusitis in the proper setting (upper respiratory tract infection symptoms for the preceding 2 weeks), and a blood culture positive for group A β-hemolytic streptococcus with consistent Gram stain results in the muscle biopsy suggest that hematogenous spread is a possibility. It is unfortunate that we were unable to obtain a CT scan, but treatment was able to proceed without it. Group A streptococcus is admittedly an unusual organism in sinus infections (about 5 percent). Other possibilities would include hematogenous spread from another site, such as the oropharynx (throat cultures have been known to miss up to 10 percent of streptococcal infections[6]), the bronchi, or the skin. Another possibility would be direct penetrating trauma to the leg (even an apparently trivial injury), but the patient denied injury, and there was no evidence of injury. The patient did not participate in regular, vigorous exercise, eliminating overuse as a cause; even so, overuse would not lead to a positive Gram stain in the muscle biopsy.

Conclusion

This case suggests that acute sinusitis could be associated with distant infectious complications. Although this patient's apparently infectious compartment syndrome could have come from other sources, it is intriguing to consider the possibility of hematogenous spread from the sinuses. In any patient with infection, especially streptococcal infection, who also has a tense fascial compartment with severe pain made worse by stretching the compartment, the possibility that the two conditions are caused by the same organism must be entertained, and antibiotic therapy should be chosen with this in mind. As the other previously cited cases demonstrate, the consequences of not performing emergency fasciotomy can be devastating; therefore, the patient must have the pressure within this compartment measured immediately. If the pressure is elevated it must be relieved by emergency fasciotomy to avoid the disastrous complications of delayed surgical intervention.

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