Case Report

Acute Paravertebral Compartment Syndrome: Follow-up and Literature Review

Abstract

Objective: To report on a patient with acute paravertebral and posterior thigh compartment syndrome after vigorous exercise. Background: Paravertebral compartment syndrome (PCS) is a rare clinical entity, typically occurring in male athletes after heavy exertion and weightlifting. Case: A 25-year-old man presented with back pain and hematuria hours after back-specific weightlifting. Clinical examination, laboratory markers, MRI, and elevated intracompartmental pressure measurements supported the diagnosis of bilateral paravertebral and posterior thigh compartment syndrome. The patient underwent paravertebral decompression via the Wiltse approach with immediate postoperative relief. He is doing well at 1 year, with recovery of lumbar extension strength, although MRI demonstrates moderate fatty replacement of paravertebral musculature. Conclusions: Although rare, early recognition of PCS and timely decompression can limit myonecrosis. Paravertebral compartment syndrome should be considered in the differential for athletic individuals with acute onset back pain.

Study Design: A case report and review of literature

Acute compartment syndrome (CS) occurs with increased pressure within a myofascial compartment, leading to decreased perfusion and myonecrosis. This typically occurs in the extremities; however, paravertebral compartment syndrome (PCS) has been described in rare circumstances. These cases typically occurred in men aged 20 to 30 years after downhill skiing, weightlifting, cross-training, or aortic aneurysm repair. Herein, we describe a case of simultaneous paravertebral and posterior thigh CS in a former athlete after extreme weightlifting.

Case Report

A 25-year-old African American man, a former professional football player, initially presented with complaints of severe lower back and thigh pain and dark urine 30 minutes after 500-lb deadlifts. The patient had no significant medical history and denied injury during the workout. His presenting examination revealed paravertebral and thigh tenderness, inability to flex his trunk, perform a straight leg raise, or flex the knee. Radiographs demonstrated chronic avulsion fractures.

John C. Roe, MD
Foster Chen, MD
Woojin Cho, MD, PhD

From the Department of Orthopaedic Surgery, Montefiore Medical Center (Dr. Roe, Dr. Chen, and Dr. Cho), and Albert Einstein College of Medicine, Bronx, NY (Dr. Cho).

Correspondence to Dr. Cho: wcho@montefiore.org

None of the following authors or any immediate family member has received anything of value from or has stock or stock options held in a commercial company or institution related directly or indirectly to the subject of this article: Dr. Roe, Dr. Chen, and Dr. Cho.

JAAOS Glob Res Rev 2018;2:e063
DOI: 10.5435/JAAOSGlobal-D-17-00063
Copyright © 2018 The Authors. Published by Wolters Kluwer Health, Inc. on behalf of the American Academy of Orthopaedic Surgeons. This is an open access article distributed under the Creative Commons Attribution-NoDerivatives License 4.0 (CC BY-ND) which allows for redistribution, commercial and non-commercial, as long as it is passed along unchanged and in whole, with credit to the author.
of bilateral anterior-superior iliac spines and possible hemangiomas of fibrous dysplasia on CT. Initial laboratory investigation was remarkable for a creatine phosphokinase (CPK) level of 121,039 mg/L, proteinuria, and hemoglobinuria.

The patient was admitted for rhabdomyolysis and pain control. His pain was unremitting with increasing narcotic demand, and by hospital day 3, he exhibited disorientation and diaphoresis. The lumbar paravertebral and posterior thigh muscles were markedly tender and incompressible, and the patient began to experience paravertebral paresthesias, decreased bowel sounds, and increased CPK levels. MRI revealed bilateral edema and patchy enhancement of the paravertebral (Figure 1), semimembranosus, biceps femoris, and semitendinosus muscles. Compartment pressures of the right and left paravertebral compartments were 103 and 94 mm Hg, respectively, and posterior thigh compartment pressures measured 20 and 40 mm Hg. The patient underwent fasciotomies of paravertebral compartments via the Wiltse approach and posterior thigh compartments via the lateral approach. A significant portion of the musculature appeared dusky and gray, with pathologic examination revealing necrotic muscle in the lumbar region (Figure 2). Drains were placed, and subcutaneous tissue and skin were closed for all compartments.

Immediately postoperatively, the patient described significant symptomatic relief and was able to ambulate by the second postoperative day. The CPK level increased to 58,065 µg/L by the third postoperative day, and he was discharged on day 4. He continued to improve clinically but had wound dehiscence of the lumbar incisions and was treated with repeat débridement at 4 weeks for persistent necro-inflammatory exudate, which slowly improved with negative pressure wound therapy and ultimately closed. His CPK level decreased to 184 µg/L at 6 weeks and 401 µg/L at 6 months. Repeat MRI at 1 year shows residual fatty streaking (Figure 3), but the wound remains

**Figure 1**

Magnetic resonance images taken at L4 as (A) T1, (B) T1-fat suppressed, with contrast, and (C) T2-weighted. Note the posterior paravertebral musculature enlargement and heterogeneous signal abnormality.

**Figure 2**

Intraoperative photographs of the Wiltse approach demonstrating myonecrosis (A) and low-power x40 (B) and high-power x200 (C) slides of hematoxylin-and-eosin–stained tissue from the lumbar spinal musculature in the permanent section, with areas of extensively necrotic skeletal muscle with acute inflammatory infiltrate and abundant necrotic debris.
healed, and he is back to engaging in sports without impairment.

Discussion

Compartment syndrome occurs when an increase in pressure within an enclosed myofascial compartment causes a decrease in tissue perfusion within that compartment. Between 4 and 8 hours, muscle ischemia becomes irreversible, leading to osmotic derangements and further edema. Myocyte necrosis results in rhabdomyolysis, releasing toxic intracellular material into the blood, causing renal toxicity and failure. Classic symptoms include pain, pallor, paresthesias, pulselessness, and paralysis. Among them, pain out of proportion to the injury and exaggeration with passive stretch are the most reliable indicators.1 Diagnosis can be confirmed by intracompartmental pressure (ICP) measurements with a threshold of 30 mm Hg within diastolic blood pressure or an absolute value of 30 mm Hg as diagnostic. The resting ICP of the paravertebral muscles is about 3.11 mm Hg (0 to 11) in the prone position and 7.95 mm Hg (2 to 20) while seated.2

Acute PCS has a more indolent presentation. A constellation of 10 signs and symptoms has been developed for increased reliability. It is characterized by (1) an inciting event causing severe localized back pain worsened by flexion and relieved by extension, (2) no previous back pain, (3) board-like muscular rigidity, (4) hypoactive bowel sounds with diffuse abdominal tenderness, (5) localized sensory loss with possible muscular atrophy, (6) CPK level >2,000 to 40,000 μg/L, (7) elevated serum glutamic oxaloacetic transaminase (SGOT) level, (8) myoglobinuria, (9) ICP of 70 to 80 mm Hg, and (10) homogenous intracompartmental signal increase on T2-weighted MRI.3 Paravertebral compartment syndrome has been an infrequently described phenomenon.1–8 Although exertional PCS has been described <10 times in the literature, deadlifts have become a recognized inciting activity.1,5,7–9 Although an acute event, PCS must be in the differential diagnosis for severe, worsening back pain and muscle rigidity after weightlifting. The above signs, symptoms, and laboratory and imaging tests aid in the diagnosis, and in our opinion, once ICP confirms the diagnosis, fasciotomy is indicated. This patient fulfilled all diagnostic criteria mentioned previously, a prompt fasciotomy was performed, and he returned to baseline laboratory values and athletic activity.

References

1. Mattiassich G, Larcher L, Leitinger M, Trinka E, Wechselberger G, Schubert H: Paravertebral compartment syndrome after training causing severe back pain in an amateur rugby player: Report of a rare case and review of the literature. BMJ Musculoskelet Disord 2013;14:259.

2. Kitajima I, Tachibana S, Hirota Y, Nakamichi K: Acute paraspinal muscle compartment syndrome treated with surgical decompression: A case report. Am J Sports Med 30:283-285.

3. Schreiber VM, Ward WT: Exercise-induced pediatric lumbar paravertebral compartment syndrome: A case report. J Pediatr Orthop 2015;35:e49-e51.

4. DiFazio FA, Barth RA, Frymoyer JW: Acute lumbar paraspinal compartment syndrome: A case report. J Bone Joint Surg Am 1991;73:1101-1103.

5. Rha EY, Kim DH, Yoo G: Acute exertional lumbar paraspinal compartment syndrome treated with fasciotomy and dermatotraction: Case report. J Plast Reconstr Aesthet Surg 2014;67:425-426.

6. Vanbrabant P, Moke L, Meersseman W, Vanderschueren G, Knockaert D: Excruciating low back pain after strenuous exertion: Beware of lumbar paraspinal compartment syndrome. J Emerg Med 2015;49:641-643.

7. Paryavi E, Johin CM, Ludwig SC, Zahiri H, Cushman J: Acute exertional lumbar paraspinal compartment syndrome. Spine (Phila Pa 1976) 2010;35:E1529-E1533.

8. Nathan ST, Roberts CS, Deliberato D: Lumbar paraspinal compartment syndrome. Int Orthop 2012;36:1221–1227.

9. Carr D, Gilbertson L, Frymoyer J, Krag M, Pope M: Lumbar paraspinal compartment syndrome: A case report with physiologic and anatomic studies. Spine (Phila Pa 1976) 1985;10:816-820.

10. Calvert N, Bhalla T, Twerenbold R: Acute exertional paraspinal compartment syndrome. ANZ J Surg 82:564–565.