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ACUTE LUMBAR PARASPINAL COMPARTMENT SYNDROME: A CASE REPORT AND DETAILED LITERATURE REVIEW

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ABSTRACT: *In this paper, we begin by presenting the 17th case of acute lumbar paraspinal compartment syndrome in the scientific/medical literature. We then review all 17 cases for demographic and clinical characteristics, in particular in terms of how they influence ultimate outcomes. All but one case occurred in a male, and most patients were less than age 40 (71%). Symptoms typically were precipitated by some athletic activity (59%); other causes were surgery (n = 4), drug abuse (n = 2), and direct trauma (n =1). Peak CPK values ranged from 5000 to 350,000 U/L. Ten patients ultimately achieved or were presumed to achieve full recovery, six had persistent low back pain several months later, and one remained in renal failure. The only predictor of final outcome was the treatment given, with six of seven who underwent surgical fasciotomy and both patients administered hyperbaric oxygen experiencing full recovery, versus just two of seven treated conservatively (p = 0.03).*

KEYWORDS: Lumbar Paraspinal Compartment Syndrome, Compartment Syndrome, Fasciotomy, Rhabdomyolysis, Creatine Kinase

INTRODUCTION

The first case of compartment syndrome associated with the ischemic necrosis of muscle was described in Germany in 1881[1]. Compartment syndrome has since been described for almost every major muscle compartment in the body, most commonly in the limbs. In 2012, Nathan et al. published a review of the literature of all cases of acute and chronic paraspinal compartment syndrome they could identify in the literature[2]. They were able to identify just ten cases of the former and eight of the latter. Since that publication, in four papers, an additional five cases of acute paraspinal compartment syndrome have been reported[3-7]; in addition, our review identified an older (2008) case reported that was somehow neglected in the Nathan et al. review[8]. In this paper, we first present our own case, and then re-analyze the essential characteristics of the now seventeen cases[3-17] that have been reported in the scientific literature.

Case Report

On September 23rd, 2012, a 33-year-old male with a history of attention deficit disorder, childhood asthma, chronic sinus infections, episodic depression and possible Raynaud's disease, presented to the emergency room of our hospital with severe lateral hip and back pain and what he described as "Coca-Cola colored urine." The patient states that, earlier that same day, he had participated in a CrossFit competition, in which he performed various weightlifting and aerobic activities. The event began at 10 o'clock in the morning and ended at 2:30 in the afternoon. Afterwards, he felt "sore all over," which he attributed to perhaps inadequate training. At approximately 10 p.m., he went to the bathroom and noticed that his urine was clearly discolored. He was familiar with rhabdomyolysis from other people who had experienced it. He also was having "a ton of pain" in his lower back and right hip, which he rated as 10/10. He tried taking ibuprofen and acetominophen, which did not help, and then drove himself to the emergency room (ER). In the ER, his height and weight were 190

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cm and 90 kg, respectively. His temperature was 97.7^{o}F, pulse 83 bpm, respiratory rate 20, BP 130/87 and oxygen saturation 96% on room air. He appeared well-nourished and in mild distress. The remainder of his physical examination was significant only pain in his low back, both at rest and with movement. His abdomen was soft and non-tender with normal bowel sounds, and he was neurologically intact. His low back was tender. Initial laboratories revealed a serum creatinine of 1.4 mg/dL, potassium 3.2 mEq/L and creatine kinase (CK) of greater than 100,000 U/L, and otherwise were unremarkable. The patient was given 3 L of normal saline in the ER and 1 L of normal saline with 20 mEq of potassium chloride. He was then started on a sodium bicarbonate drip as well as sodium chloride with potassium chloride 20 mEq at 200ml per hour. Both the Nephrology and General Surgery teams were consulted, the latter for a presumed case of compartment syndrome.

When assessed by the Surgery team, he continued to attribute most of his pain to his right low back area, where a subtle soft tissue mass was noted that was not evident on the left. His right paraspinal muscles were tense and very tender, and he had overlying paresthesia of the skin. Muscle swelling extended into the right flank, in the absence of any skin changes. A CT scan of the abdomen was reviewed, which demonstrated no abdominal pathology. Under local anesthesia, a Stryker instrument was inserted into the area of the right lumbar paraspinal muscles, below the fascia, where compartment pressure was measured at 38-40mmHg. Based upon this elevated pressure, a diagnosis of right lumbar paraspinal compartment syndrome was made. The patient and immediate family members were told that surgical decompression was indicated, and consent for urgent surgery was obtained. By this time, with hydration, his renal function had improved and CPK levels had fallen from > 100,000 to roughly 60,000.

The patient was brought into the operating room, where he was given general anesthesia and intubated while in a supine position. He then was placed in a left lateral decubitus position, right side up for better exposure of his right paraspinal muscles. A 7-8 cm skin incision was made which extended into the deeper layers to expose the fascial layer and deeper compartment. The muscle compartment appeared to be quite under tension; and, as soon as the release incision was made, edematous muscle tissue started to extrude through the incision. In appearance, the muscle was dusky grey and nonreactive to cautery. The skin incision was extended both caudally to the level of the iliac crest and cranially to approximately the T10 level, to make the total incision length roughly 17 cm in the vertical plane. It was extended as high as the thoracic vertebra, until tension was released from the compartment. Other muscles (e.g. latisimus dorsi) were viable, but the paraspinal muscles remained ischemic in appearance. During this entire procedure, the utmost care was taken to achieve hemostasis. The wound cavity was thoroughly irrigated and again secured for hemostasis. The wound was left open for delayed primary closure. A vessel loop was used in a shoelace fashion for partial closure of the wound, and the wound bed was packed with saline-moistened Kerlix covered with 4 x 4s, ABD and a sterile dressing. The patient was transferred back into the supine position, and then extubated with no complications. At the end of the procedure, all counts were correct and the patient was brought into the recovery room without any complications. Estimated intra-operative blood loss was 30 ml.

Post-operatively, the patient did well, with his CPK continuing to trend downwards, and serum creatinine falling to 0.9mg/dL by the second post-operative day, as well as urine volumes and appearance returning to normal. On September 27th (post-op day 4), he was returned to the OR for wound VAC placement of the right paraspinal muscle. On September 29th, the wound VAC was changed at the bedside, at which time the muscle appeared healthy

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and pink, with good granulation tissue and diffuse bleeding. The patient underwent closure of the skin and fascia with removal of the wound VAC on October 3rd. As he was now ambulating without assistance and voiding normally, he was discharged home with PO Percocets as needed for pain, and Colace. He was followed up in the outpatient Surgery clinic, experienced full healing of his wound, and gradually increased his activity level without further complications or setbacks.

LITERATURE REVIEW AND DISCUSSION

Our review of the literature was conducted using the on-line database PubMed and the search terms 'paraspinal compartment syndrome' and 'lumbar paraspinal compartment syndrome', both of which generated 26 abstracts. Of these, twenty were deemed relevant to the topic, and 15 described at least a single case of acute paraspinal compartment syndrome. Of these, thirteen papers described a single case[3-5, 7-11, 13-17], and two described two cases[6, 12], for a total of seventeen cases. However, upon closer inspection, it was discovered that a single case had been presented twice[3, 4]. We therefore have presented here what we believe is only the 17th case of acute lumbar paraspinal compartment syndrome currently published in the scientific literature (Table 1). However, it is the 6th case reported in the last three years (since 2010), and the 5th since the last review by Nathan et al. in 2012. It is also the 10th case since 2008, versus just seven cases in total prior to this, the first of which was reported in 1985[9]. This begs a question that we cannot answer from current data, which is: is the incidence of this disorder increasing, or is the apparent increase in cases merely a reflection of more clinicians reporting their cases? Either way, what is clear is that this condition is now receiving considerably greater attention than it did even five years ago.

Our case was, in many ways, typical of cases already reported. Our patient was male, like all but one other case that involved a 43-year old woman[16]. At age 33, our patient also was relatively young, which also is consistent with the literature, given that 12 of the 17 cases now reported (71%) were under age 40 at the time their paraspinal compartment syndrome was diagnosed (Figure 1). Analyzing further, it is apparent that the mechanism of injury is somehow linked to patient age. Among the twelve cases under age 40, ten (83%) had the onset of their symptoms after intense athletic activity, that activity being weight-lifting in six cases[3-5, 7, 8, 17], downhill skiing in three[9, 10, 13], and surfboarding[14] in a single case. One of the remaining two under-40 cases was related to a gastric bypass procedure for morbid obesity[12], and the other secondary to substance abuse[6], specifically with synthetic cathinone (so-called 'bath salts'). Conversely, none of the five older cases (over age 40) involved athletic endeavors, three cases occurring following aortic bypass procedures[11, 12, 15], one secondary to synthetic cathinone abuse[6], and the single case involving a female secondary to direct trauma to her back[16].

Our patient had the acute onset of symptoms, having been asymptomatic, in terms of back pain, prior to participating in acute, strenuous athletic activities earlier the same day of his presentation to the emergency room. This pattern too mirrors what has been reported in the literature, most reporting the onset of their severe back pain within 12 to at most 36 hours of a single precipitating event. One potential exception was a case reported in Japan of a 25-year old gentleman who used to go surfboarding every weekend, and admitted to having had chronic back pain for several months, which acutely worsened during the night after a day

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surfboarding[14]. Among these cases, at least one was known to be a high-level athlete, competing as an NCAA football player[5].

Our patient also had the characteristic localized muscle tenderness and swelling, as well as sensory loss over the paraspinal muscles in the absence of any muscle atrophy, as listed previously among diagnostic criteria for this disorder[2].

In our case and in each case in the literature, serum CPK was elevated at presentation, though the literature range was broad, from lows of 5,465 and 7,875 U/L[9, 15] to three peaks surpassing 100,000 U/L, including our own; interesting, the two other cases with CPK peaks above 100,000 involved the abuse of the sympathomimetic drug cathinone[6]. In several cases, serum CPK levels continued to rise despite conservative management that included aggressive fluid replacement; in every case, these dramatic post-treatment-initiation related increases (e.g., from 19,000 to 60,800 U/L[3]) in CPK occurred in a weight-lifter[3, 5, 7, 8]. Categorizing patients into those under versus over age 40, eight of eleven (73%) younger patients ultimately had CPK levels over 50,000 U/L, versus just one of four (25%) over age forty; and the single person > 40 years old who had a CPK peak >50,000 was one whose rhabdomyolysis and consequent compartment syndrome were related to the drug cathinone; this patient's CPK peaked at 162,495[6]. Categorizing patients into athletes, post-operative patients, drug users, and the single case of direct trauma, both drug abusers had CPK peaks > 100,000 (mean = 256,000 U/L). Among athletes, 3 had peak CPK levels < 50,000, six between 50,000 and 99,999, and one (our own case) > 100,000, for an overall mean of 57,000 U/L. The CPKs for the two surgery patients with such values reported, as well as for the single direct trauma case all were 25,000 U/L or less. Renal function was variable, ranging from normal to considerably elevated serum creatinine levels, though presenting oliguria was atypical.

Imaging usually was by T1 or T2-weighted magnetic resonance imaging (MRI) or computed tomography (CT), usually within 24 to 48 hours of presentation; and in no case in which either test was ordered and reported was paraspinal edema not detected[3, 5-10, 13, 14, 17].

Three treatment options were apparent in the literature: conservative management, consisting of aggressive fluid and electrolyte replacement and analgesia[6, 9-12, 16, 17]; hyperbaric oxygen added to conservative management[3, 5]; and, as administered to our patient, surgical fasciotomy[6-8, 13-15]. There was no clear pattern in terms of when each treatment was administered, with one 67-year old male treated surgically despite a peak serum CPK of under 8,000[15], and yet a 43-year old gentleman with a CPK peaking at over 162,000 treated conservatively[6]. In some instances, surgery was considered, but deferred when serum CPK and creatinine levels started to decline with initial conservative management alone[6, 17]. There was a pattern in terms of treatment response however, with only two of seven patients (29%) treated conservatively experiencing complete resolution of symptoms (5 of 7 having continued chronic low back pain) versus complete or presumed complete recovery without pain in eight of nine patients treated either with surgery (6 of 7) or hyperbaric oxygen (2 of 2); this association between treatment administered and complete/incomplete recovery was statistically significant, when tested by Pearson $\chi 2$ analysis ($\chi 2 = 11.17$; $p = 0.03$). There was no association between outcome and any other variable, including patient age ($p = 0.88$), year of presentation ($p = 0.91$), mechanism of injury ($p = 0.34$), or peak CPK ($p = 0.92$).

Overall outcomes were good, with chronic renal failure occurring in only a single patient who already had lost one kidney secondary to trauma and now developed severe rhabdomyolysis

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(CPK = 350,000) after abuse of cathinone[6]; and cardiac arrest occurring with muscle reperfusion in the 43-year old woman who suffered direct trauma to her back; she was resuscitated successfully and ultimately returned to normal activities, albeit with chronic low back pain[16]. Unfortunately, chronic back pain was not an uncommon result, especially in patients treated conservatively[9, 10, 12, 17]. Though most patients ultimately resumed full activities, this begs a question initially proposed by Peck et al. almost three decades ago[18]: how many people with chronic back pain following an episode of acute severe pain have had an episode of paraspinal compartment syndrome remain undiagnosed? To address this, Konno et al. compared lumbar intramuscular pressure in 102 patients with chronic low back pain against 20 healthy controls, and identified different patterns of increased pressure and decreased muscle perfusion related to different spinal positions (e.g., lordosis versus kyphosis) in those with chronic pain[19]. Unfortunately, given the lack of attention afforded lumbar compartment syndrome until recent years, little further has been done to answer this important question. It seems to be a question that now warrants more dedicated consideration.

CONCLUSIONS

Acute paraspinal compartment syndrome is an uncommonly, but increasingly-recognized disorder, typically brought on by strenuous, acute physical activity, but by other causes as well. Younger males appear to be especially prone, with only one case reported in a female, to date. Typical features are acute back pain following strenuous activity, in the absence of a prior history of pain; focal low back muscle tenderness and rigidity underlying relatively paresthetic skin; and a rapid rise in serum CPK levels. In our patient, "Coco-Cola colored urine" was an accompanying symptom that triggered the patient to immediately and most fortuitously seek emergency medical attention. Imaging studies, whether CT or MRI, almost invariably document paraspinal muscle swelling. Treatment consists of aggressive hydration and electrolyte replacement, with hyperbaric oxygen and surgical fasciotomy as further more aggressive alternatives. Albeit not formally tested within the confines of a clinical trial, which would be almost impossible given the rarity of this syndrome, our review of the literature suggests that aggressive treatment, with either hyperbaric oxygen or surgery, yields better long-term outcomes than conservative management alone, the latter commonly associated with chronic back pain.

Tables and Figures

Table 1: Summary of published cases of acute lumbar paraspinal compartment syndrome

First Author	Year	Gender	Age	Cause	Peak CPK	Treatment	Outcome
Alfaraj	2013	Male	33	Weight-lifting	101,000	Fasciotomy	CPK, Cre normalized; near normal activities by 2 wks
Allerton[3, 4]	2012	Male	25	Weight-lifting	60,800	Hyperbaric O_2	CPK, Cre normalized; near normal activities by 2 wks
Carr[9]	1985	Male	27	Downhill skiing	5,465	Conservative	Resumed full activities; some chronic LBP persisted
DiFazio[10]	1991	Male	27	Downhill skiing	60,000	Conservative	Resumed full activities; some chronic LBP persisted

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Ferreira[11]	2003	Male	55	Aortic bypass	25,000	Conservative	Complete recovery
Haig[12]	2009	Male	57	Aortic bypass	n/a	Conservative	Chronic LBP with strenuous activities persisted
Haig[12]	2009	Male	34	Gastric bypass	n/a	Conservative	Chronic LBP with strenuous activities persisted
Karam[5]	2010	Male	23	Weight-lifting	77,400	Hyperbaric O_2	CPK 8805 w/in 6 sessions over 7 days; pain-free; neuro intact
Khan[13]	2005	Male	35	Downhill skiing	59,863	Fasciotomy	CPK 4600 w/in 5 days post-op; full activities w/in 1 mo.
Kitajima[14]	2002	Male	25	Surfboarding	21,440	Fasciotomy	Much improved w/in 2 days; full recovery w/in 2 mo.
Levine[6]	2013	Male	37	Drug abuse	350,000	Fasciotomy	Pain-free; neurologically intact; but still in renal failure at 5 mo.
Levine[6]	2013	Male	43	Drug abuse	162,495	Conservative	Pain-free; neurologically intact at discharge; CPK & Cre normalized
Minnema[8]	2008	Male	32	Weight-lifting	72,820	Fasciotomy	CPK fell to 5000; pain-free; near normal activities by 2 wks
Osamura[15]	2000	Male	67	Aortic bypass	7,875	Fasciotomy	Patient ultimately resumed normal activities
Paryavi[7]	2010	Male	20	Weight-lifting	72,516	Fasciotomy	Symptoms, CPK and Cre normalized within 13 days
Sava[16]	1999	Female	43	Direct trauma	10,581	Conservative	Suffered cardiac arrest but recovered. Chronic LBP persisted
Wik[17]	2010	Male	30	Weight-lifting	82,000	Conservative	CPK & Cre normalized w/in 1 mo.; pain persisted at 2 mo
Totals		M: 17 F: 1	20-29: 7 30-39: 6 40-49: 2 50-59: 2 60-69: 1 70+: 0	7 weight-lifting 3 downhill skiing 1 snowboarding 4 surgery 2 drug abuse 1 direct trauma	<50,000: 6 50-100,000: 7 >100,000: 3 n/a: 2	Fasciotomy: 7 Conservative Rx: 9 Hyperbaric O_2: 2	Full recovery: 7 Recovering well: 2 Persistent pain: 6 Renal failure: 1 Not available: 1
Means Lower limit Upper limit			36.1 20 67		77,950 5,465 350,000		

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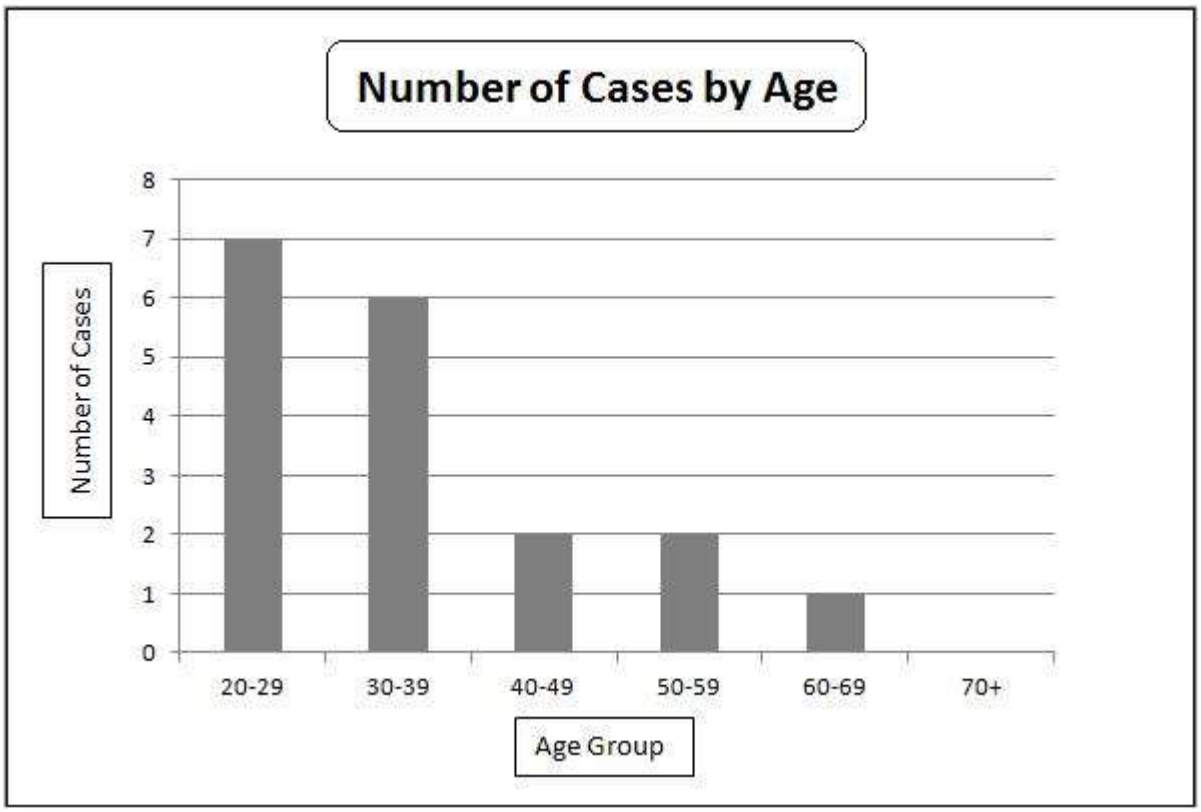

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