

Acute paraspinal compartment syndrome as a rare cause of loin pain

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ABSTRACT

A significant proportion of emergency urological admissions are comprised of ureteric colic presenting as loin pain. A variety of alternative pathologies present in this manner and should be considered during systematic assessment. We report the case of a patient admitted with severe unilateral back and flank pain after strenuous deadlift exercise. Clinical examination and subsequent investigation following a significant delay demonstrated acute paraspinal compartment syndrome (PCS) after an initial misdiagnosis of ureteric colic. The patient was managed conservatively. We review the current literature surrounding the rare diagnosis of PCS and discuss the management options.

KEYWORDS

Paraspinal compartment syndrome – Back pain – Rhabdomyolysis – Renal colic differential diagnosis

Accepted 28 September 2014; published online XXX

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A 24-year-old man was referred to the urology team by his general practitioner. He presented with a 24-hour history of severe right loin and back pain, which radiated to the front of his abdomen and followed strenuous weight lifting exercises. He denied any trauma or urinary tract and bowel symptoms. He also denied neurological symptoms in his extremities. His previous medical history included renal colic. He admitted smoking cigarettes, drinking alcohol and had previously taken recreational drugs. He was in a long-term relationship, and denied any previous sexually transmitted infections, other sexual partners and foreign travel.

On examination, his observations were within normal limitations and his abdominal examination was unremarkable. Marked tenderness was noted over the right paraspinal muscles and within the right loin. Lower limb neurological examination was normal. The pain improved but did not resolve with opiates.

Urinalysis demonstrated microscopic haematuria. Routine blood tests (including a full blood count, C-reactive protein [$<1.0\text{mg/l}$], electrolytes, renal function and liver function tests) were normal. An initial diagnosis of ureteric colic was ruled out following urgent non-contrast computed tomography of the kidneys, ureters and bladder. A musculoskeletal differential diagnosis was questioned after four days of high opiate and diazepam usage with no improvement. Repeat biochemical analysis remained normal.

An orthopaedic review on day 5 recommended spinal magnetic resonance imaging (MRI). This revealed necrosis of the posterior paraspinal muscles from the midthoracic

level to the pelvis and was consistent with a diagnosis of acute paraspinal compartment syndrome (PCS). The patient was commenced on aggressive intravenous fluid resuscitation and daily renal function monitoring. The first recorded creatine kinase (CK) level was reported at $4,949\text{iu/l}$ on day 9 of the admission. His renal function remained normal. Urine and venous myoglobin levels were not performed given that rhabdomyolysis was confirmed with CK and MRI. The CK normalised over the next ten days, avoiding the need for compartment pressure monitoring. The patient's discomfort improved with conservative measures and physiotherapy.

Discussion

Around 25–35% of all emergency urological admissions present with loin pain, of which 65–94% are found to be ureteric colic.¹ PCS is a rare 'non-stone' differential diagnosis for loin pain, reiterating the need for a systematic approach when assessing patients.

Compartment syndrome was first described by Richard von Volkmann in 1881.² This can occur in any closed compartment throughout the body. PCS was first described by Peck in 1981.³ In 1985 Carr *et al* found that the erector spinae muscles were encased within a fibro-osseous space that could accommodate compartment syndrome.⁴ In 1987 Styf documented pressures in the paraspinal compartments during rest and exercise,⁵ which have been replicated and used in the development of paraspinal compartment pressure monitoring. MRI has a proven diagnostic role in PCS but more usefully differentiates between other pathologies

causing pain such as fractures, abscesses, tumours, aneurysms, discitis and acute intervertebral disc prolapses.

Since 1991 only ten reported cases of acute PCS have been documented following activities such as skiing, weightlifting and surfing.^{4,6} In 1987 the first case of chronic PCS was recorded. Clinically, PCS is easily overlooked as knowledge of the disease is uncommon given its rarity. This appears to be the first documented case of acute PCS case in the UK and the first to mimic a presentation of ureteric colic.

No consensus exists for the management of PCS and case reports provide the only standard for comparison. According to these reports, surgical decompression reduces paraspinal compartment pressures rapidly and facilitates a quicker return to physical activity when no diagnostic or surgical delays occur. Decompression is achieved using either a mid-line or bilateral paramedian incision. Second-look surgery occurs at 48 hours with delayed closure within 1–2 weeks.

The importance of compartment pressure monitoring and the need for urgent surgical intervention for peripheral compartment syndrome has been well documented by McQueen and Court-Brown.⁷ As PCS is rare with non-specific symptoms and signs (often requiring time consuming investigation and imaging), diagnosis within this time-frame is infrequently reported.

Conservative management has been suggested in cases of peripheral compartment syndrome with a delayed diagnosis (>48 hours) as a risk of infection can outweigh the benefits of surgery.⁸ Case reports have demonstrated successful conservative management of PCS with normalisation of compartment pressures and eventual return to exercise.^{4,6} The roll of compartment pressure monitoring in the paraspinal musculature requires further investigation. Conservative measures include aggressive fluid administration and urine monitoring to optimise tissue perfusion, thereby reducing the effects of rhabdomyolysis. Patients undergo early physiotherapy while optimising analgesia. Successful surgical decompression has been reported if conservative measures fail. Conservative monitoring involves serial venous CK estimations, renal function, pain score and compartment pressure (if measured) monitoring, and, occasionally, serial MRI.^{4,6}

The patient described in this case report did not undergo surgical decompression as conservative measures produced biochemical and clinical improvements following the significant diagnostic delay. At orthopaedic follow-up review six weeks later, he was found to have a reduced range of spinal movement and some paraspinal muscle

wasting. He only required simple analgesia, was mobilising and his biochemical markers had all returned to normal with no evidence of infection or renal failure. He remains under follow-up.

Conclusions

Loin pain assessment requires a systematic history and examination to establish the correct diagnosis. PCS is a rare cause of back pain but knowledge of its existence is nevertheless required to manage the potential sequelae of rhabdomyolysis with a view to early surgical decompression. PCS should be considered if back pain is out of proportion to an alternative diagnosis or analgesic requirements are inexplicably high. Timely MRI should be carried out if uncertainty remains. Compartment pressures have a limited diagnostic role but appear more useful in monitoring response to treatment.

Once established as PCS, the patient should be commenced on aggressive intravenous fluids and have hourly urine monitoring, with regular serum creatinine kinase, renal function and pain monitoring. Surgical decompression normalises compartment pressures rapidly and facilitates a quicker return to exercise if no diagnostic delay occurs (ie <24 hours) but is also effective if conservative measures fail. This case and others demonstrate the satisfactory role of conservative management should diagnostic delays occur.

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