■ CASE REPORT

Chronic lumbar paraspinal compartment syndrome

A CASE REPORT AND REVIEW OF THE LITERATURE

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A 25-year-old male weightlifter felt increasing intractable low back pain during training but denied any acute injury. The physical examination, blood parameters, radiographs and MRI were unremarkable. He had been treated non-operatively by various means, with only temporary relief. The pressures in the lumbar paraspinal compartment were abnormally high and he was treated by surgical decompression. This gave rapid relief, he returned to training, and one year later the pain had not recurred.

Compartment syndrome is commonly seen in orthopaedics and sports medicine. It is most frequent in the lower leg, but can also occur in the thigh,^{1,2} foot,^{3,4} forearm^{5,6} and lumbar region.⁷ Theoretically, the condition can affect any compartment in the human body.⁸

A compartment syndrome in the lumbar region was first suggested as a possible cause of low back pain by Peck⁷ in 1981. The condition has subsequently been demonstrated in anatomical and physiological studies and a few case reports.⁹⁻¹¹ There are recent reports of acute paraspinal compartment syndrome^{8,12,13} but little in the literature about a chronic syndrome. We present one such case involving the lumbar paraspinal muscles which responded well to surgical decompression.

Case report

A 25-year-old male weightlifter felt increasing low back pain and a sensation of tightness during training. The pain subsided afterwards, but discomfort remained for most of the day and sometimes prevented sleep. He could also be awakened by it. He had been treated by a number of doctors with massage and physical therapy with only temporary relief. The pain returned whenever he started weightlifting.

On examination there was bilateral paraspinal muscle spasm, swelling and slight tenderness. The range of movement of the lumbar spine was near normal. Straight leg raising and Lasegue's tests were negative. The blood parameters of creatine kinase, aspartate aminotransferase, lactate dehydrogenase and CRP were within normal limits. Radiographs of the lumbar spine and electromyography of

the lower limbs were normal and an MR scan showed only thick lumbar muscle. The possibility of an exercise-related compartment syndrome was considered and pressures were measured in his lumbar compartments with a transducer (Stryker, Kalamazoo, Michigan). The needle was inserted into the paraspinal muscle compartment at the level of maximal tenderness, approximately at L3. The results are shown in Table I.

According to the normal measurements, which range between 3.1 mmHg and 10.8 mmHg at rest and remain below 175 mmHg when exercising,^{7-10,13,14} the diagnosis of chronic compartment syndrome of both paraspinal muscles was confirmed. The patient underwent bilateral minimally invasive fasciotomy, during which small tissue samples were taken from the erector spinae.

Decompression was undertaken under 1% lidocaine local anaesthesia with the patient prone. A 2 cm incision was made at the L3 level, 2.5 cm from the spinous process. After separation of subcutaneous fat, the superficial layer of thoracolumbar fascia was incised over 12 cm. We considered the fascia to be thicker than

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©2009 British Editorial Society of Bone and Joint Surgery
doi:10.1302/0301-620X.91B12.22647 \$2.00

J Bone Joint Surg [Br]
2009;91-B:1628-30.
Received 30 March 2009;
Accepted after revision 1 July 2009

Table I. The measurements of paraspinal intramuscular pressure (mmHg)

	Pre-operative		Post-operative (at four weeks)		
	Left	Right	Left	Right	Normal
Resting	38	31	6.5	7.2	3.1 to 10.8
Exercising	220	190	13.2	156	< 175
Six minutes after exercise	45	39	8.8	9.4	3.1 to 10.8

Fig. 1

Histology showing hyperplasia of striped muscle accompanied with punctiform necrosis (A), hyperplasia of striped muscle with dissolution of muscle fibres (B) and vitreous degeneration (C) (haematoxylin and eosin x 80).

Fig. 2

Electromicroscopy showing scattered disorderly arrangement of skeletal muscle (A) with multiple degeneration (B) (x 2800).

normal. Specimens were taken, the wound closed and the operation was repeated on the opposite side.

One week later the patient was walking and the vague pain had disappeared. Four weeks after operation he was back to normal activity. The pressures were again measured (Table I). Three months later he returned to weightlifting training for the 2008 Olympics and the pain did not recur during follow-up of one year.

The main microscopic changes were hyperplasia of the striped muscle accompanied by dissolution and vitreous degeneration of muscle fibres with punctiform necrosis of fibrous tissue in the erector spinae (Fig. 1). Electron microscopy showed scattered derangement of skeletal muscle with multiple areas of degeneration (Fig. 2).

Discussion

Following the suggestion by Peck in 1981⁷ that compartment syndrome in the lumbar region was a possible cause of low back pain, Carr et al,⁹ Styf¹⁰ and Styf and Lysell¹¹ assessed the anatomy and physiology. There were no further reports until Konno, Kikuchi and Nagaosa,¹⁵ Stock and Helwig¹⁶ and Mueller et al¹⁴ revisited the subject, concentrating on compartment pressures. From 1991 to 2005 there were only four reports of acute lumbar paraspinal compartment syndrome.^{8,12-14}

In anatomical dissections of the lumbar region, Carr et al⁹ confirmed a clearly defined, well-developed compartment consisting of the erector spinae muscles encased by the posterior and middle lamellae of the lumbodorsal fascia. The posterior layer of the lumbar fascia is composed of superficial and deep layers which cover the iliocostalis, longissimus and multifidus muscles.^{17,18}

In 1987 Styf¹⁰ measured the pressure in the erector spinal muscle during exercise. The paraspinal intra-compartment pressure was 6.1 mmHg (SD 1.4) at rest. The maximum pressure while carrying a load was 175 mmHg, which generally returned to pre-testing levels within six minutes.

Carr et al⁹ arrived at a similar conclusion, and DiFazio et al¹² observed that the intra-compartmental pressure of the erector spinae was 8 mmHg, 175 mmHg and 8 mmHg at rest, during exercise, and six minutes afterwards, respectively. Intra-compartmental pressure is also related to posture and loading.¹⁴ In our case the lumbar paraspinal intracompartmental pressure far exceeded the normal.

Chronic compartment syndrome is commonly induced by hypertrophy of the skeletal muscle and disturbance of liquid exchange after excessive exercise. In this case, microscopy showed changes of chronic injury to the skeletal muscle which had not yet reached the level of an acute compartment syndrome. Compared with the acute condition,^{8,12,13,18} chronic compartment syndrome has a long history; the blood enzyme levels and urine myoglobin do not exceed normal values, and degeneration and necrosis of skeletal muscle is not seen on T2-weighted MR scans.

There is no reasonable explanation for the night pain in this case. A possible explanation might be that lower blood pressure at night leads to slower blood flow, thereby increasing oedema.

Apart from compartment pressure measurements, other methods of investigation can be used.¹⁹ These include radioisotope imaging, methoxy isobutyl isonitrile perfusion imaging, electromyography, MRI, ultrasound imaging and muscle blood flow. It is also suggested that the sensitivity of non-invasive near-infrared spectroscopy is clinically equivalent to invasive compartmental pressure measurements.²⁰ MRI is not ideal for the diagnosis of chronic compartment syndrome and we used MRI and electromyography to exclude other diseases. It is accepted that near-infrared spectroscopy and muscle blood flow studies, which were not done in our case, would probably have shown positive findings.

Surgical decompression is the only satisfactory treatment for compartment syndrome in the limbs, but the authors of previous reports of paraspinal muscle compartment

syndrome all suggested non-operative treatment, including the Graston technique.^{12,18} Because pain in our patient did not improve, we performed a fasciotomy and biopsy. His symptoms rapidly disappeared, and he was walking on the first day post-operatively.

Chronic compartment syndrome should be suspected and intracompartmental pressure measured in patients with low back pain induced by exercise or heavy labour. Once other lumbar diseases are excluded, surgical decompression is appropriate.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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