

compression and do not regain tone for several days. It is the pooling of desaturated stagnant blood in the atonic vessels that gives the cyanotic facial appearance. It has been recorded that the cyanosis does not occur over points of pressure such as hat bands or collars. This has been ascribed to counter pressure on the skin, preventing venous and capillary distension. In two of the fatal cases described by Williams the patients had no warning before the crushing injury; these patients did not develop the classical craniofacial cyanosis. Lee et al reported a fear response in 12 out of 14 patients in their series.<sup>8</sup>

Large variations in duration and weight of compression have been described. Animal studies<sup>6,12</sup> on guinea pigs and dogs show that the incidence of death due to traumatic asphyxia is a function of absolute weight and duration of compression. Weights of up to 7 tons have been tolerated for short periods.<sup>13</sup> Conversely, substantially lower weights have caused death when the compression was prolonged. Seven of the eight fatal cases presented by Williams<sup>6</sup> involved prolonged crushing force. In this situation death results from complete restriction of respiratory movement. In the absence of prolonged compression death is due to associated injuries.<sup>14</sup> The victims of the Hillsborough disaster showed few of the typical signs of traumatic asphyxia, having been exposed to a gradual and prolonged crush rather than a sudden compressive force, thus emphasising the importance of the fear response in generating the traumatic asphyxia syndrome. The term "crush asphyxia" was coined to describe the variation on traumatic asphyxia experienced at Hillsborough.<sup>3</sup>

The two cases we describe illustrate the classical features of traumatic asphyxia uncomplicated by skeletal or thoracoabdominal visceral injury. The child who survived experienced a large crushing force over a very brief period. In comparison the compressing force in the fatal case was fairly modest, but presumably sufficient to prevent all respiratory excursion, leading to hypoxic cardiac arrest. There are several reports of survival following a large crushing weight being applied for a short period of time.<sup>9,13,14</sup> Other reports describe survival after periods of entrapment of between two and ten minutes.<sup>6</sup> Gorenstein et al recorded

only two fatalities in their series of 16 cases of traumatic asphyxia in paediatric patients.<sup>5</sup> Both cases were found to have hypoxic encephalopathy at necropsy; one had severe abdominal injuries in addition. It is not possible to compare outcome in adults and children due to the low number of reported cases; however, fatalities in both groups are related to the weight and duration of compression and associated injuries. The fatal case in our report is notable in that the death occurred in the absence of any other injury. It is all the more tragic in that death is rare in such cases.<sup>5</sup> This case illustrates the hazards of children being left unsupervised in rooms where inadequately secured large pieces of furniture are present. It is obviously desirable that parents should be informed of such risks.

The treatment of traumatic asphyxia is supportive. Assisted ventilation may be required in cases of chest wall disruption, underlying lung parenchymal damage, or respiratory depression secondary to cerebral hypoxia. Subsequent treatment is directed towards other specific associated injuries.

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- 1 D'Angers O. Relation medicale des evenements survenus au Champs de Mars Le 14 Juillet 1837. *Ann d'Hyg* 1837;18:485-9.
- 2 Haller JA, Donahoo JS. Traumatic asphyxia in children: pathophysiology and management. *J Trauma* 1971;2:453-7.
- 3 Wardrope J, Ryan F, Clark G, Venables G, Courtney Crosby A, Redgrave P. The Hillsborough tragedy. *BMJ* 1991;303:1381-5.
- 4 Channer KS, Edbrooke DL, Moores M, McHugh P, Michael S. Acute right heart strain after crushing injury at Hillsborough Football ground. *BMJ* 1989;299:1379-80.
- 5 Gorenstein L, Blair GK, Shandling B. The prognosis of traumatic asphyxia in childhood. *J Pediatr Surg* 1986;21:753-6.
- 6 Williams JS, Minken SL, Adams JT. Traumatic asphyxia reappraised. *Ann Surg* 1968;167:384-92.
- 7 Jongewaard WR, Coghill TH, Landercasper J. Neurological consequences of traumatic asphyxia. *J Trauma* 1992;32:28-31.
- 8 Lee MC, Wong SS, Chu JJ, Chang JP, Lin PJ, Shien MJ, et al. Traumatic asphyxia. *Ann Thorac Surg* 1991;51:86-8.
- 9 Baldwin GA, McNab AJ, McCormack AQ. Visual loss following traumatic asphyxia in children. *J Trauma* 1988;28:557-8.
- 10 Laird WR, Borman MC. Traumatic asphyxia with a report of 5 additional cases. *Surg Gynecol Obstet* 1930;00:578-85.
- 11 Bolt RA. Traumatic asphyxia, report of a case. *Cleveland Med J* 1908;7:647.
- 12 Furuya Y. Experimental traumatic asphyxia, grades of thoracic compression and mortality. *Igaku Kenkyu* 1981;51:117-9.
- 13 Parker FJ. Optic atrophy from traumatic asphyxia with report of a case. *Ann Ophthalmol* 1911;40:159-62.
- 14 Fred HL, Chandler FW. Traumatic asphyxia. *Am J Med* 1960;29:508-17.

## Abdominal aortic aneurysm presenting as meralgia paraesthetica

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### Abstract

**A case of abdominal aortic aneurysm is reported in a patient with long standing low back pain, presenting as meralgia paraesthetica and an increase in the**

**severity of back pain. The case highlights the need for objective assessment of new symptoms arising in a chronic condition, and for a systematic approach to the assessment of radiographs performed in**

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#### Case report

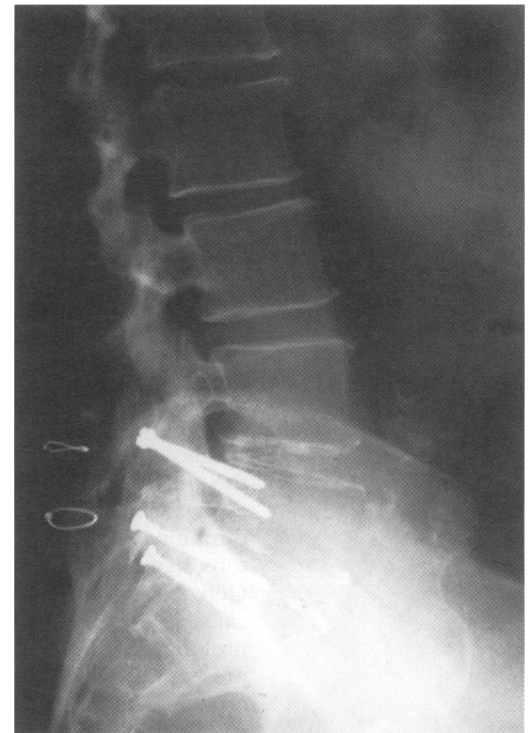
A 62 year old man presented to the accident and emergency (A&E) department complaining of a sudden onset of pain and altered sensation on the lateral aspect of his right thigh. He had a past medical history of two laminectomies in 1963 and 1965, and a lumbar spinal fusion in 1967. He also suffered from chronic obstructive pulmonary disease and had smoked heavily throughout his adult life. Examination revealed a full range of movement in his lumbar spine and normal motor function in his lower limbs. An area of diminished sensation was noted over the lateral aspect of his right thigh. A clinical diagnosis of entrapment of the lateral cutaneous nerve of the thigh was made and he was discharged with analgesia to be followed up by his general practitioner. He represented the next day with the same symptoms and signs and radiographs were performed of his right hip and lumbar spine which were interpreted as showing no new bony injury (figure). A diagnosis of meralgia paraesthetica was made and he was again referred back to his general practitioner. On both occasions his pulse and blood pressure were recorded as normal.

Six days later he was referred directly to the on call medical team with the same symptoms plus increasing back pain. They immediately referred him to the orthopaedic team who admitted him for bed rest and analgesia. His symptoms did not settle with 10 days of bed rest and it was felt that he would benefit from a caudal epidural. At this time it was noted that straight leg raising was reduced to 50% on the right and the femoral stretch test was positive on the same side. After the procedure he was allowed home, to be reviewed in six weeks.

Two weeks later a radiologist's report on the lumbar spine radiograph was received by the A&E department, commenting on a large abdominal aortic aneurysm. The patient was recalled to the department straight away and following surgical referral elective resection was arranged. At this time he reported that the caudal epidural had not affected the intensity of his back pain, nor had it improved the presumed "root" symptoms. The resection was performed without complication and six weeks later he reported that the symptoms affecting his right thigh had vanished almost immediately after the operation, together with the severe back pain, leaving him with the low grade back discomfort which had affected him for many years.

#### Discussion

Meralgia paraesthetica is caused by entrapment of the lateral femoral cutaneous nerve and gives rise to symptoms which are entirely sensory.<sup>1</sup> It is a common condition which is most often seen in middle aged males and in pregnant women. The lateral femoral cutaneous nerve arises from the dorsal branches of



Lateral lumbar spine radiograph, showing the soft tissue outline of a 9 cm diameter aortic aneurysm.

the second and third lumbar ventral rami. After emerging from behind psoas major it passes laterally, crossing iliacus towards the anterior superior iliac spine. On the right the nerve is lateral to the caecum and is separated from it by the fascia iliaca and peritoneum. On the left the nerve passes behind the descending colon, but both nerves pass either behind or through the inguinal ligament. The anterior branch supplies the skin on the anterior and lateral thigh as far as the knee, connecting terminally with branches of the anterior division of the femoral nerve. The posterior branch penetrates the fascia lata to supply the area over the greater trochanter to the mid-thigh.<sup>2</sup>

It is where the nerve passes under or through the inguinal ligament that entrapment is most likely to occur. It is easy to see how this is possible in obese middle aged men and in pregnancy, but it is not so easy to explain how entrapment or compression might be caused by an abdominal aortic aneurysm. Nevertheless our patient presented with characteristic symptoms of meralgia paraesthetica, which were relieved by laparotomy and resection of a large aneurysm with no evidence of rupture. There are no reports of abdominal aortic aneurysm presenting with symptoms of compression of the lateral femoral cutaneous nerve and although pain in the groin and thigh are well described symptoms,<sup>3</sup> in particular of leaking aneurysm, sciatic or femoral nerve compression is extremely rare.<sup>4</sup>

This case illustrates the danger of a "blinkered" approach to new symptoms in a patient with a chronic condition. It also reinforces the need for the systematic assessment of radiographs in the A&E department and the requirement for a review of the films by a radiologist within a much shorter time period. If

the "Ten Commandments" systematic approach had been followed,<sup>5</sup> and the lumbar spine radiographs had been viewed systematically (Adequacy–Alignment–Bones–Cartilages and joints–Soft tissues), the diagnosis of an aortic aneurysm may have been made within two days of the onset of symptoms. It is recommended that all emergency radiographs are reviewed and a report returned to the A&E department within three working days. The two week delay in this case was clearly unacceptable.

- 1 Maddison PJ, Isenberg DA, Woo P, Glass DN. Oxford textbook of rheumatology. Oxford: Oxford University Press, 1993.
- 2 Williams PL, ed. Grays anatomy, 38th ed. London: Churchill Livingstone, 1995.
- 3 Szilagyi DE, Smith RF, Macksood AJ, Whitcomb JG. Expanding and ruptured aortic aneurysm: problems of diagnosis and treatment. *Arch Surg* 1961;83:395–408.
- 4 Ashleigh RG, Marcuson RW. False aortic aneurysm presenting as sciatic nerve root pain. *Eur J Vasc Surg* 1993; 7:214–6.
- 5 Touquet R, Driscoll P, Nicholson D. Ten commandments of emergency radiology. In: Nicholson D, Driscoll P, eds. The ABC of emergency radiology. London: BMJ Publishing Group, 1995.

## Neck pain as a presenting symptom in malignant hypertension

Joanna Stockwell, Grizelda George

### Abstract

**Neck pain, unrelated to trauma, is relatively common and is usually presumed to be musculoskeletal in origin. A patient presented with an unusual and serious cause of neck pain—malignant hypertension. The mechanism of the neck pain may be incipient tonsillar herniation of the cerebellum caused by raised intracranial pressure.**

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We describe a patient who presented to the accident and emergency (A&E) department complaining of neck pain as a presenting symptom in malignant hypertension.

Malignant hypertension is characterised by very high blood pressure with papilloedema and retinopathy. There is associated organ failure, most commonly of the kidneys and heart. The most frequent presenting symptom is headache, often accompanied by other symptoms of hypertensive encephalopathy such as vomiting, blurred vision, and fits. There may also be symptoms of renal and cardiac insufficiency, for example, lethargy and shortness of breath. Neck pain has not previously been described as a presenting symptom in malignant hypertension.

### Case report

A 33 year old female patient presented to the A&E department complaining of neck pain on waking for the previous 10 days. She had seen her general practitioner (GP) who had diagnosed musculoskeletal neck pain and had prescribed ibuprofen and a soft collar. On further questioning, she complained of headache and blurred vision. She had, in fact, felt unwell for one month, with loss of appetite, nausea and vomiting, lethargy, weakness, intermittent sharp chest pains, and shortness of breath. She had no significant past medical history. She used an oral contraceptive and smoked 15

cigarettes a day. She was a single mother with four children.

On examination, she looked well. However, she was significantly hypertensive with a blood pressure 250/120 mm Hg. The apex beat was not displaced. She had gross papilloedema. She was mildly photophobic. She was tender over the neck but there was no neck stiffness. There were no other abnormal signs. Preliminary investigation showed advanced renal impairment with a plasma creatinine of 543 µmol/litre. Her urine contained blood and protein. ECG and chest x ray were normal. A diagnosis of malignant hypertension was made and she underwent renal biopsy which showed markedly sclerosed glomeruli. She is currently being treated for a rapidly progressive glomerulonephritis.

### Discussion

Malignant hypertension is a potentially life threatening emergency which usually presents with headache. However, in the case described here it presented with neck pain which mimicked non-traumatic, musculoskeletal neck pain.

Although neck pain has not previously been described in malignant hypertension, magnetic resonance imaging (MRI) and single photon emission computerised tomography (SPECT) suggest reasons for it. Hypertensive encephalopathy produces cerebral oedema with regional hyperperfusion of the occipital cortex and cerebellum.<sup>1</sup> The cerebral oedema causes raised intracranial pressure—hence the papilloedema, headache (worse on waking), and the vomiting which are so common in malignant hypertension. Hyperperfusion of the occipital cortex may contribute to the visual symptoms, as may the papilloedema. Cerebellar hyperperfusion, together with raised intracranial pressure, would be expected to cause a degree of tonsillar herniation through the foramen magnum. Neck pain has been described as a presenting symptom in tonsillar herniation.<sup>2</sup> It therefore seems reasonable to attribute the

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