CASE REPORT

Low back pain at presentation in a newly diagnosed diabetic

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Insulin dependent diabetes mellitus predisposes to a range of different and unusual infections, including epidural and psoas abscesses. However, they occur mainly in adults with longstanding diabetes. We report the case of a 12 year old boy who presented with diabetic ketoacidosis and low back pain, and was subsequently diagnosed with both a left psoas abscess and an extensive thoracolumbar spinal epidural abscess measuring 20 cm in length. This case report highlights the need to maintain a high index of suspicion for epidural abscesses in children presenting with fever and localised back pain. Early diagnosis with appropriate imaging and aggressive management can prevent development of permanent neurological damage as was the case in our patient.

Patients with insulin dependent diabetes mellitus (IDDM) are generally more susceptible to infections, which can cause hyperglycaemia and trigger diabetic ketoacidosis.[1] In many cases, the focus of infection, such as the urinary or respiratory tract, may be apparent at presentation. Occasionally, however, patients can present with unusual foci of infection and vague clinical symptoms and signs. Both epidural[2–5] and psoas[6 7] abscesses have been reported in diabetics, but mainly in adults with longstanding disease. Presentation with an epidural abscess and psoas abscess in the same patient is extremely rare in adults, particularly at first presentation of IDDM,[2] and, to our knowledge, has not been reported in children.

CASE REPORT

A 12 year old boy of mixed race (White/Afro-Caribbean) presented to casualty with a three day history of central abdominal pain, vomiting, lethargy, and low back pain, followed by a five hour history of difficulty in breathing. There was no previous history of respiratory problems, polyuria, polydipsia, or weight loss. His past medical history was unremarkable with normal development and no previous acute hospital admissions.

On examination, he was 10% dehydrated with cold peripheries, dry mucous membranes, sunken eyes, and reduced skin turgor. He had Kussmaul breathing, with a respiratory rate of 60 breaths per minute, but good bilateral air entry on auscultation and no wheeze or crackles. There was no lymphadenopathy but he was noted to be anaemic. Axillary temperature was 36.0°C on admission. His heart rate was regular at 120 per minute with a blood pressure of 130/70 mm Hg. He had mild central abdominal pain with no hepatosplenomegaly. Examination of the spine revealed mild tenderness on palpation over the upper lumbar spine, but peripheral neurological examination was entirely normal.

Initial investigations showed a blood glucose of 30.4 mmol/l, haemoglobin 75 g/l with a raised glycated haemoglobin of 14.5%, white cell count 32.9 × 10^9/l with a neutrophilia of 27.3 × 10^9/l, sodium 133 mmol/l, potassium 3.1 mmol/l, urea 9.2 mmol/l, creatinine 141 µmol/l, and erythrocyte sedimentation rate 114 mm in the first hour. Haemoglobin electrophoresis was negative for sickle cell disease or trait. A venous blood gas on admission showed a pH of 7.08, pCO_2 1.3 kPa, standard bicarbonate 2.7 mmol/l, and base excess of −26.1 mmol/l. Urine dipstick showed glucose 3+, blood 3+, ketones 4+, and a trace of protein. The chest radiograph was clear.

Figure 1 Saggital magnetic resonance image of the lower thoracic, lumbar, and upper sacral spine with gadolinium contrast showing extensive loculated epidural abscesses (arrowed) extending from the T10/11 disc space to S1.

A diagnosis of diabetic ketoacidosis was made; he was resuscitated with normal saline and commenced on intravenous fluids with added potassium and an insulin infusion according to the local protocol. His dehydration was corrected slowly over 48 hours. Intravenous cefotaxime was commenced to treat suspected infection based on the high neutrophil count and erythrocyte sedimentation rate (ESR), although no focus was identified at the time.

Abbreviations: ESR, erythrocyte sedimentation rate; IDDM, insulin dependent diabetes mellitus; MRI, magnetic resonance imaging; SEA, spinal epidural abscess

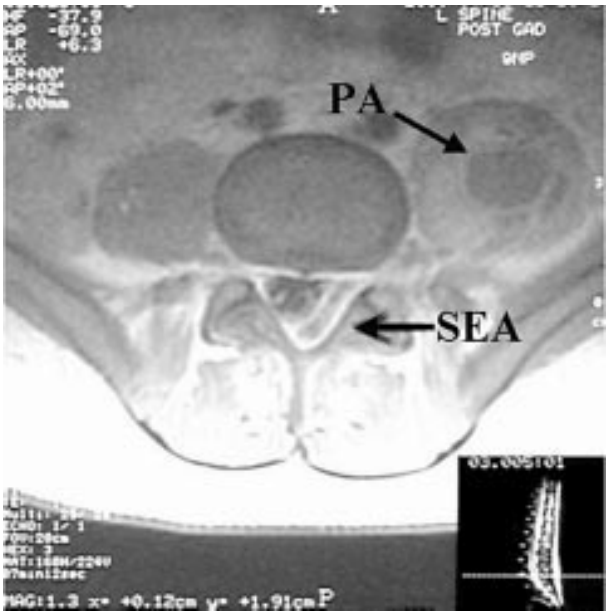

Figure 2 Transverse magnetic resonance image of the pelvis at the level of L4 with gadolinium contrast showing extensive loculated psoas abscess (PA). The epidural abscess (SEA) at this level is also shown.

His condition and metabolic derangements improved over the next 48 hours and he was commenced on subcutaneous insulin. However, he continued to complain of widespread left sided abdominal and flank pain as well as lower back and bilateral buttock pain. A surgical and orthopaedic opinion were sought but no concerns were raised. Abdominal ultrasound scan was also reported as normal. Twenty four hours later, the left sided abdominal pain had progressed to the left anterior thigh. On examination this time, he was unable to extend his left hip, there was reduced power in hip and knee flexion on the left side, associated with diminished knee and ankle reflexes, and loss of sensation in the L2/3 region. Anal and cremasteric reflexes were also absent. The plantar reflexes were downgoing bilaterally and there was no sphincter disturbance.

In view of his focal neurological signs, a magnetic resonance imaging (MRI) scan was performed, which showed extensive, multiple thoracolumbar epidural loculated abscesses from T10/11 disc space to the S1 level, measuring 20 cm in length with a 15–20 mm anteroposterior diameter (fig 1). The largest abscess extended from the T12 disc space at the level of the conus, to L2 and caused significant cord compression and displacement of the cauda equina and the lower third of the conus. There was no evidence of vertebral osteitis or discitis. The MRI scan also revealed a large psoas abscess on the left side (fig 2).

His admission blood cultures grew *Staphylococcus aureus*, and his antibiotics were changed to flucloxacillin and fusidic acid. He was immediately transferred to the local neurosurgical unit and underwent a left L1/2 hemilaminectomy and evacuation under general anaesthetic. At the same time, a catheter was inserted anteriorly to drain the psoas abcess. Histology subsequently showed extensive necrotising inflamed connective tissue and skeletal muscle associated with Gram positive cocci. He was treated with intravenous antibiotics for six weeks and discharged home with no further complications. His neurological abnormality returned to normal and, five months later, he participated successfully at the annual diabetic skiing event.

DISCUSSION

Patients with diabetes mellitus are at increased risk of a range of different infections.[1] Hyperglycaemia can interfere with the activities of phagocytic cells, chemotaxis, adherence, and the respiratory bursts responsible for the intracellular killing of microorganisms.[1] Diabetic patients also have increased carriage rates for many organisms, including *Staphylococcus aureus*.[1 8 9] In adult cases with longstanding diabetes, staphylococcal colonisation and infections of neuropathic foot ulcers and gangrene have been associated with haematogenous spread and development of secondary infections,[8] including epidural[3 4] and psoas abscesses.[6 7 10] In the latter, patients usually present with fever, pain in the hip, flank, or back, and flexion of the ipsilateral hip. *S aureus* is the most frequently isolated organism and both ultrasound and computed tomography are useful investigations for confirming the diagnosis. Treatment involves surgical debridement and drainage of the abscess with long term intravenous antibiotics.

At diagnosis, our patient had a considerably raised ESR and neutrophil count suggestive of bacterial infection, although a C reactive protein, a more specific test for bacterial infection, was not available. Consequently, he was started on cefotaxime. The increased carriage rate of *S aureus* in diabetes might suggest that a more specific antistaphylococcal agent would have been more appropriate; however, these were commenced as soon as the results of the blood culture became available and earlier treatment is unlikely to have made any difference to the outcome given the nature of the pathology and the need for surgical intervention. It is not clear for how long our patient had had the diabetes. He had only had a short duration of symptoms and there were no foot ulcers or any other focus of staphylococcal infection at diagnosis, but the raised glycated haemoglobin of 14.5% suggests that the blood glucose had been raised for several weeks, as is often the case in newly diagnosed diabetes.

In contrast to psoas abscesses, spinal epidural abscesses (SEA) are rare and potentially fatal, requiring immediate neurosurgical intervention to prevent permanent damage. It is described mainly in adults and a recent comprehensive systematic review identified only 915 cases over a 33 year period.[2] Its incidence is estimated to be less than one case per million population but is thought to be increasing,[3] with children accounting for fewer than 10% of cases.[2] The condition is almost twice as common in males, and carries an overall mortality of 16%. Patients with SEA usually present with fever and localised back pain initially (stage 1). If untreated, the condition may progress to radicular irritation with root pain radiating from the tender area (stage 2), early neurological deficit including muscle weakness, sphincter weakness and sensory deficit (stage 3), and finally paralysis (stage 4).[2 3] Our patient had features of stage 1 at presentation, which rapidly progressed to stage 3 by the time SEA was diagnosed. Fortunately, his symptoms regressed completely with prompt and aggressive treatment.

Diabetes mellitus is the single most common condition associated with SEA, accounting for 15% of all cases.[2] Other important risk factors include other infections (44%), particularly staphylococcal skin infections (15%), spinal and extraspinal trauma (10%), intravenous drug use (9%), and alcohol abuse (4.8%).[2] The most common organism isolated from SEA lesions is *S aureus* (73%), but a range of different bacteria, fungi, and parasites may be responsible.[2] A high ESR of greater than 20 mm/h was found in 94% of 110 patients and was the most consistent finding. Leucocytosis was also common, with 78% of 218 patients having a white cell count greater than 10.0 $\times$ 10^9/l.[2] The most useful investigation remains an MRI scan which is considered to be superior to computed tomography because of lower radiation exposure to patients and greater sensitivity (>90% for definitive diagnosis of SEA).[3 5] The use of gadolinium as the contrast medium for MRI scans allows better delineation of SEA from contiguous structures.[2 3] Treatment of SEA primarily involves laminectomy to drain the abscess with intraoperative ultrasound, if necessary, to detect and surgically decompress any anteriorly located abscesses. Long term intravenous antibiotic treatment for 4–6 weeks is also recommended and should cover *S aureus*.

The occurrence of both a psoas abscess and SEA in the same patient is extremely uncommon. The recent systematic review identified only four cases in adults[2] and an extensive literature search revealed no reported cases in children. In particular, such an unusual and severe staphylococcal infection at presentation of IDDM has never been reported. This case report highlights the need for clinicians to maintain a high index of suspicion for early symptoms and signs of SEA.[4] Children presenting with fever and localised back pain should be thoroughly investigated with appropriate imaging because early diagnosis and effective treatment can prevent the development of permanent neurological damage in those with epidural abscesses, as was the case in our patient.

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IMAGES IN PAEDIATRICS

Coughing up a lung

A 10 year old girl presented with a harsh "barky" cough which resolved with sleep. Physical examination revealed lungs clear to auscultation, no cervical crepitus or other neck deformities. An upper gastrointestinal series was performed to investigate whether gastro-oesophageal reflux was causative. During a Valsalva manoeuvre the radiographs revealed a lucency in the neck on the right, representing herniation of a portion of the right lung (see figure). There was mass effect on the trachea with shift to the left.

Lung hernias, uncommon occurrences in children, can be classified using the system devised by Movall-Lovallee. Congenital versus acquired hernias describes the defect based on aetiology. Cervical thoracic versus diaphragmatic lung hernias describes the defect based on location.[1]

Congenital hernias are more common in children than acquired hernias and are primarily the result of weakness in Sibson's fascia.[1] Sibson's fascia is a continuation of the endothoracic fascia over the apex of the lung inserting posteriorly onto the transverse process of the first thoracic vertebra and ramifying anterolaterally around the margins of the first rib.[1 2] The herniation may present throughout infancy and childhood, usually as a painless mass in the neck exacerbated by events increasing intra-abdominal pressure.[1–3] Apical lung hernias are more common on the right.[3]

Treatment of apical lung hernias is seldom necessary because they spontaneously reduce with decreasing intra-abdominal pressure.[4] This patient was diagnosed with a psychogenic cough that responded well to behavioural modification therapy.

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