CASE REPORTS

Abdominal pain as an atypical presentation of meningococcaemia

A P Winrow

Abstract

An atypical presentation of meningococcaemia without purpura poses diagnostic problems. The importance of the identification of shock manifest as delayed capillary refill in two children with meningococcal septicaemia presenting with fever and abdominal pain is discussed. Abdominal pain is an unusual presentation of meningococcal disease.

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Keywords: abdominal pain; meningococcal disease

The meningitic and septicaemic presentations of acute meningococcaemia are well recognised. Usually there is no difficulty in making the diagnosis in a sick child presenting with a purpuric rash. Recent health education initiatives have sought to alert the public to the importance of both the early recognition of purpura by means of the "tumbler test" and the need to subsequently seek urgent medical advice.¹ Studies indicate that there may still be a lack of awareness of the significance of the combination of fever and purpura among health professionals and the public.^{2,3} Furthermore, the correct diagnosis may not be considered, with a consequent delay in diagnosis, if the presentation is atypical with the rash appearing sparse, absent, or maculopapular in nature.⁴ Shock may develop early in the disease and is associated with increased mortality.⁵ Inadequate treatment of shock may be implicated in a poor outcome.⁶ Recent published treatment protocols stress the importance of the early recognition, prompt antibiotic administration, and urgent correction of shock.⁷ The presence of shock is often assessed using the capillary refill time technique.⁸ Delayed capillary refill indicates hypovolaemia and shock. Hypotension may be absent and, when present, it is usually a sign of severe shock.

Two children are reported who had an atypical presentation of acute meningococcaemia with acute right iliac fossa pain and fever. Medical disorders may mimic surgical conditions and the diagnosis of surgical intra-abdominal pathology may be difficult in children as many of the early clinical signs are non-specific. Therefore repeated evaluations may be necessary before the true diagnosis is

evident. In the children reported here, their initial presentation of abdominal pain was potentially misleading. The absence of evolving surgical signs together with fever and shock indicated sepsis and directed appropriate treatment.

Case reports

CASE 1

A 3 year old boy presented to the emergency department with a two hour history of fever and abdominal pain. His abdominal pain was limited to the right iliac fossa. Examination revealed abdominal tenderness without guarding. A fever was recorded at 38.4°C. Although he complained of a headache, there was no photophobia or meningism. There was no evidence of a skin rash. Although normotensive, he had a heart rate of 140 beats/min and his capillary refill was delayed at 4 seconds.

Conservative management was instituted after discussion with the surgical team. It was noted that his tachycardia and delayed capillary refill was excessive for the degree of abdominal pain or fever without signs of perforation or peritonitis. His pain and fever persisted despite analgesia and antipyretics, although his headache resolved. Intravenous fluids were started and intravenous cefotaxime was administered to cover the possibility of sepsis. Shortly after admission he suffered a rigor associated with a temperature of 41°C. Immediately after this episode, his perfusion fell with a capillary refill time of 6 seconds. He remained normotensive. Petechiae developed in the right inguinal area. His heart rate increased to 160 beats/min. A presumptive diagnosis of acute meningococcaemia was made. Over the next two hours he received a total infusion of 40 ml/kg of colloid (human albumin solution) to improve his perfusion. His base deficit was -6. The maximum Glasgow meningococcal septicaemia prognostic score⁹ was calculated as 5.

He improved rapidly over the next five hours despite the appearance of further petechiae. His fever settled and the abdominal pain settled within 12 hours of admission. A full blood count revealed a mild neutrophilia and a mild coagulopathy was noted. Standard biochemistry, urinalysis, urine microscopy/culture, stool culture, and faecal blood testing gave normal results. His initial C reactive

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protein estimation was 13 mg/l which rose to a maximum of 186 mg/l. *Neisseria meningitidis* group B was subsequently grown from blood cultures taken on admission. He was discharged on day 4 to complete his antibiotic treatment uneventfully at home.

CASE 2

A 12 year old girl was admitted after a 10 hour episode of acute right iliac fossa pain, cough, and fever of 38.9°C. Other abdominal signs were absent and chest radiography excluded a right lower lobe pneumonia. A heart rate of 130 beats/min was noted with a delayed capillary refill time of 3–4 seconds. She was normotensive and well hydrated. After discussion with the surgical team, she was managed conservatively as there were no signs of perforation or peritonitis. A presumptive diagnosis of sepsis was made. One hour after admission, scanty petechiae were noted over her lower legs and trunk. Her clinical parameters had not altered and there was no evidence of meningism. Initial clotting studies and blood count were normal but the initial C reactive protein concentration was mildly raised at 16 mg/l. Subsequent faecal culture and faecal blood analysis were normal. She was started on intravenous cefotaxime to cover the possibility of meningococcaemia and she received an infusion of 10 ml/kg of colloid (human albumin solution) to improve perfusion. Her fever and pain resolved over the next six hours and she made an uneventful recovery. After prolonged incubation, blood cultures taken on admission grew *N meningitidis*. Retrospectively, her maximum Glasgow meningococcal septicaemia prognostic score was calculated as 3.

Discussion

The role of shock in the pathophysiology and prognosis of meningococcal septicaemia has been reviewed previously.¹⁰ A combination of capillary leak from the intravascular space and myocardial dysfunction result in hypovolaemia and reduced cardiac output. Both hypoperfusion and acidosis have been implicated in the spectrum of organ dysfunction seen in the disease.

Although health promotion programmes have targeted the need for early diagnosis, it is now apparent that recognition and correction of shock is also vital and is addressed in treatment protocols.⁷ Delayed capillary refill time has been considered to be an indicator of hypovolaemia in several clinical situations. However this clinical measurement does have some limitations¹¹ and authors have claimed that it is an insensitive marker of shock due to acute blood loss in adults.¹² Its usefulness lies with its simplicity and repeatability, although variations in site of measurement and measuring technique must be avoided. A recent study did not detect a clinically important effect of fever on capillary refill.¹³

In the two cases reported in this paper, the initial presenting symptom was abdominal pain with fever. The differential diagnosis was extensive. The presence of shock was unusual for a surgical pathology unless visceral perfora-

tion or peritonitis had occurred. There were no features of these complications in either child. Therefore this constellation of features was indicative of sepsis. Both patients responded to reversal of their hypovolaemia with colloid infusions. This suggests that mesenteric hypoperfusion may have been implicated in the aetiology of the abdominal pain. Intestinal haemorrhage or ischaemia due to disordered coagulation appears less likely in the absence of positive faecal blood analysis and normal coagulation profiles. Abdominal pain is a recognised feature of other paediatric disorders characterised by mesenteric hypoperfusion such as nephrotic syndrome.

Other gastrointestinal complications of acute meningococcaemia have been reported. Britto *et al* described gut perforation in critically ill children with meningococcal sepsis complicated by multiorgan failure.¹⁴ However these children developed gastrointestinal complications during intensive care requiring dialysis rather than on presentation. Meningococcal peritonitis occurring shortly after presentation was reported by Buckmaster and Boyce.¹⁵ However fever and abdominal pain had been present for at least 24 hours before admission, unlike the short time noted in the cases above. Splenic rupture as a complication after fine needle aspiration of the lesser sac has been noted as a complication but again the patient was critically ill in intensive care, unlike our patients.¹⁶ There have been anecdotal reports of abdominal pain and fever on presentation, although these are infrequent and the diagnosis is usually suggested by the presence of purpura.¹⁷ Our patients differed from all these either by the degree of severity of the illness, length of time of abdominal symptoms before admission, or the absence of a purpuric rash on presentation.

The key to the recognition of the underlying diagnosis in these two cases was the assessment and evaluation of the delayed capillary refill time. This should be an integral assessment of perfusion in any sick child. Reliance on the presence of a spreading characteristic purpuric rash for diagnosis is hazardous and may delay resuscitation and may jeopardise the prognosis.

In conclusion, the diagnosis of acute meningococcaemia should be considered in any febrile child with a delayed capillary refill time. The absence of a rash should not exclude the diagnosis. Hypoperfusion may result in abdominal pain and the presence of such pain should not be considered at variance with the diagnosis of sepsis. Abdominal pain is an atypical presentation of meningococcal disease.

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Hyponatraemic convulsion secondary to desmopressin treatment for primary enuresis

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Abstract

The case of a 6 year old child who presented with convulsions and coma after unsupervised self administration of intranasal desmopressin (DDAVP) for nocturnal enuresis is presented. Children with enuresis can be embarrassed by their condition and may believe that multiple doses of their nasal spray may bring about a rapid resolution.

Water intoxication is an uncommon but serious adverse effect of treatment with intranasal DDAVP. These patients may present with seizure, mental state changes, or both.

Basic management consists of stopping the drug, fluid restriction, and suppressive treatment for seizures. Recovery is usually rapid and complete.

Administration of the nasal spray in children should be supervised by parents to prevent highly motivated children from accidental overdose. The risks of high fluid intake need to be carefully explained to both parents and children.

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Keywords: desmopressin; enuresis; water intoxication; hyponatraemia

Desmopressin (DDAVP) is a synthetic analogue of the neurohypophyseal nonapeptide arginine vasopressin. It has greater antidiuretic potency, a reduced pressor activity, and a greater half life and duration of action than the natural hormone vasopressin.¹ DDAVP is administered intravenously to treat central diabetes insipidus, haemophilia A, and von Willebrand's disease.

Intranasal administration has become popular in the treatment of primary nocturnal enuresis.² The intranasal dose is approximately 10 times greater than the intravenous dose. DDAVP acts on the distal and collecting tubules of the nephron, via the V2 receptors by

a cyclic AMP mediated mechanism, to increase their permeability to water and solutes.

A literature search has identified 20 reports of patients in whom intranasal DDAVP was associated with hyponatraemia. All of these patients experienced seizures, altered mental status, or both.²⁻⁴

Case report

A 6 year old boy was brought to the accident and emergency department by ambulance after collapsing at school. He had suddenly become drowsy and was witnessed to have a generalised seizure. His parents denied any significant previous medical conditions. The child was reported to have had some behavioural problems. He had been prescribed intranasal DDAVP a week before attendance for control of nocturnal enuresis (10 µg daily).

On arrival, he was irritable and had a Glasgow coma score of 9 with dilated and sluggishly reactive pupils. He had a respiratory rate of 16 breaths/min, an axillary temperature of 35°C, a pulse of 68 beats/min, and a blood pressure of 104/65 mm Hg. Blood glucose was 8.1 mmol/l. There were no meningeal or focal neurological signs.

While in the department, he had two episodes of jerky uncoordinated movements of the upper limbs which mimicked seizures. Both episodes responded to intravenous diazepam. He was vomiting continuously and had a fluctuating level of consciousness. Blood samples were taken at cannulation and sent for biochemistry and haematology. A non-contrast computed tomogram of the brain was normal.

His blood results were received after the computed tomography. His plasma sodium concentration was 119 mmol/l, potassium 3.5 mmol/l, urea 1.2 mmol/l, and creatinine 52 mmol/l.

On further questioning his parents revealed that the child was highly motivated to resolve his enuresis and had self administered the DDAVP without their supervision. His parents considered it likely that the child might have

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