CASE REPORT

Hypothermia and the acute abdomen

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SUMMARY

A 64-year old man presented to A&E with a 24 h history of persistent hypothermia and increasing confusion. His medical history included congestive heart failure, hypertension and intermittent claudication secondary to distal abdominal aortic occlusion. Examination revealed profound hypothermia—31°C, bilateral reduced power in the lower limbs and no palpable peripheral pulses. Initial blood tests revealed a metabolic acidosis with hyperlactataemia, neutrophilia and deranged electrolytes. CT scan of the abdomen revealed extensive thromboembolic disease of the abdominal arterial tree and diagnosed acute mesenteric ischaemia. The patient was taken to theatre where an emergency laparotomy demonstrated a perforated duodenum, which was subsequently patched, followed immediately by an axillofemoral bypass with subsequent femoral–femoral crossover. Sadly, the patient passed away shortly after completion of surgery.

BACKGROUND

The classical presentation of acute mesenteric ischaemia (AMI) is that of abdominal pain disproportionate to findings on examination. This patient, however, presented in a unique fashion with non-specific symptoms of hypothermia, confusion and reduced consciousness. This presenting symptomatology provides an insight into the pathophysiology underlying AMI. Not only does this case provide a unique presentation of AMI but also serves as a reminder that patients with non-specific symptoms require thorough investigation.

CASE PRESENTATION

A 64-year-old man presented to A&E by ambulance, accompanied by his wife, who reported a 24 hour history of initially feeling persistently cold to touch, followed by increasing confusion and a gradual reduction in consciousness. His medical history included congestive heart failure, hypertension and long-standing intermittent claudication secondary to distal abdominal aortic occlusion.

On arrival his observations showed profound hypothermia—31°C, automated blood pressure 55/51 mm Hg, heart rate 68 bpm and saturating 97% of 50% FiO₂. His Glasgow Coma Scale (GCS) was 12 of 15 (E₃, V₃, M₆).

INVESTIGATIONS

Full systems examinations revealed very few significant findings. Peripheral neurological examination demonstrated bilateral reduced power (1/5). Cardiovascular examination revealed no palpable pulses except for apex and carotid pulses.

Initial venous blood results were significant for: a white cell count of $27.4 \times 10^9/L$, neutrophilia $23.4 \times 10^9/L$, sodium 128 mmol/L and potassium 7.2 mmol/L. The simultaneous arterial blood gas showed severe metabolic acidosis: pH 6.98, pCO₂ 2.2 kPa, lactate >20, BE -27.6, HCO₃ 4.2.

DIFFERENTIAL DIAGNOSIS

The initial investigations offer various medical and surgical differential diagnoses. Severe inflammatory response syndrome (SIRS), congestive heart failure (CHF) and cerebrovascular disease (CVD) are all medical differentials; while surgical pathologies associated with SIRS, such as: acute mesenteric ischaemia, gastrointestinal perforation and an abdominal aortic aneurysm pathology formulated the surgical differentials.

Hypothermia and neutrophilia found on initial investigation provide two criteria necessary for diagnosis of SIRS. There were various findings in the history and examination that suggested a possible origin for this reaction. CHF is a feature of this patient's medical history and is associated with metabolic acidosis when pump failure leads to severe organ hypoperfusion.¹ Cerebrovascular disease is supported by both the history of confusion and reduced consciousness.^{1 2} Furthermore, bilateral lower limb weakness supports the differential of a CNS insult, particularly a spinal stroke.³

Severe metabolic acidosis arising from organ hypoperfusion can too arise from surgical pathologies. In particular, poor perfusion of the bowel and kidneys can produce highly acidotic environments (pH<7).⁴ The deranged electrolytes further support an underlying pathology of organ and tissue hypoperfusion. Gastrointestinal perforation is another common cause of metabolic acidosis.

AMI, however, ties in all of the above disease pathologies. AMI can result from thromboembolism, pump failure or due to proximal expansion of a pre-existing aortic occlusion. Thus, a preliminary diagnosis of acute mesenteric ischaemia was made and urgent imaging requested.

A CT scan demonstrated extensive thromboembolic disease of: the abdominal aorta, coeliac axis, superior mesenteric artery, renal arteries, inferior mesenteric artery and the common iliacs. The diagnosis was confirmed as AMI.

TREATMENT

The patient was taken to ITU for urgent fluid resuscitation, intubation and central venous access. An emergency exploratory laparotomy revealed a perforated duodenal ulcer at D1 and poorly perfused but not overtly ischaemic bowel. A patch repair of the perforation was performed immediately

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followed by an axillobifemoral bypass combined with a femoral–femoral crossover to reinstate blood supply to the lower limbs.

OUTCOME AND FOLLOW-UP

Intraoperatively, the patient's cardio output declined and became increasingly resistant to inotropic support. He developed an agonal ventricular rhythm and died after going into cardiac arrest shortly after surgery. The coroner's post mortem examination determined the cause of death was due to duodenal perforation resulting in SIRS/septicaemia.

DISCUSSION

The causes of AMI can be divided into four subtypes according to its underlying cause

1. Embolic
2. Arterial thrombosis
3. Non-occlusive
4. Venous thrombosis

An arterial embolus is the single most common cause of AMI—responsible for 40–50% of all cases—and typically occludes the superior mesenteric artery (90% of cases).^{5–9} Historically, rheumatic valve disease was the most common cause; however, dysrhythmias are now the primary aetiology.¹⁰

Thrombosis of the mesenteric arteries accounts for 25–30% of cases.¹¹ These patients will often have a history of atherosclerotic disease.¹² Collaterals develop allowing the gut to remain viable¹² eventually resulting in chronic mesenteric ischaemia. The patient becomes 'acute' once the origins for the collaterals become occluded.¹³

Non-occlusive ischaemia is seen in those with poor cardiac function. While the exact mechanism is poorly understood, it is hypothesised that low cardiac output leads to peripheral hypoperfusion.⁹ This activates the sympathetic nervous system, thus resulting in further vasoconstriction while attempting to increase the cardiac output. The splanchnic vessels are particularly susceptible to this biofeedback.¹⁴

Mesenteric venous thrombosis accounts for less than 10% of cases.¹² It is thought to be a result of other intra-abdominal pathologies, such as malignancy, sepsis or pancreatitis.¹⁵ It acts synonymously with deep vein thromboses. The venous clot results in vascular stagnation, ultimately resulting in inadequate tissue oxygenation.

There is a variety of clinical presentations of AMI; however, the classical presentation is a dramatic onset of severe abdominal pain disproportionate to physical examination findings.^{7 16 17} The other most common presentations include: nausea, vomiting and diarrhoea, bleeding per rectum and constipation.⁷ Chronic mesenteric ischaemia is most commonly associated with postprandial pain, nausea and weight loss.⁶

This patient differed in that his presenting symptoms were non-specific. The primary concern of the patient and his family was his hypothermia. On examination there was no abdominal pain or other commonly associated gastrointestinal symptoms. Literature review of the presenting features of AMI revealed no prior cases where hypothermia was the primary symptom.

AMI also has various associated biochemistry results. Up to 98% of venous blood tests show a leukocytosis and, when tested for, a raised aspartate aminotransferase and creatine kinase is seen in about 70% and 35%, respectively. ABGs will tend to show a metabolic acidosis with a raised lactate (91% of patients) and a base deficit (52% of patients).^{6 7}

AMI has proven to be a challenging surgical emergency to treat. It comprises 1% of acute surgical admissions,¹⁸ yet over

the past 70 years, the survival rate has not substantially improved and continues to range between 40% and 70%.^{12 19} Even with advances in diagnosis and treatment, such as widened availability of CT scanning, improved resuscitation management and reduced time before theatre, the biggest challenge in treating AMI is diagnosis before bowel infarction occurs.¹⁰ Indeed, just a 24 h delay decreases survival rates by 20%.²⁰

Even once AMI has been diagnosed and management initiated, the mortality rates are still exceptionally high. The most common perioperative complications and potential causes of death are⁷:

Complication	Risk (%)
Respiratory failure	36
Multiorgan failure/SIRS	31
Bowel infarction	26
Coagulopathy	26
Cardiac arrest	22

Learning points

- Hypothermia is a part of the diagnostic criteria of severe inflammatory response syndrome (SIRS) so needs to be investigated urgently and thoroughly.
- SIRS can occur as a result of a variety of pathologies, both medical and surgical, and is not only associated with sepsis.
- The acute abdomen should always be part of the differential diagnosis in a non-specifically unwell patient.
- Urgent diagnosis and management is key to improving survival rates of acute mesenteric ischaemia.

Competing interests None.

Patient consent None.

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