Causes of the acute abdomen: add thrombophilia to your list

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DISCUSSION

This case report presents a patient with a mesenteric venous thrombosis who presented with an acute abdomen. The patient had a thrombophilic condition, namely factor V leiden mutation.

Mesenteric venous thrombosis is an uncommon disorder. The thrombotic process may be idiopathic or secondary to inherited or acquired thrombophilic conditions. It is estimated that between 30 and 40% of cases of mesenteric thrombosis are caused by an underlying inherited thrombophilic state, e.g. protein C and protein S deficiencies, antithrombin III, dysfibrinogenaemia or, as in this case, factor V leiden deficiency (Maloisel, 1996).

The diagnosis can often be difficult because the patient is commonly young with a history of vague abdominal symptoms (Chen et al, 1996). In a review of 16 cases of mesenteric venous thrombosis, the most common presentation was one of gradual onset of abdominal pain. The patient may also present with gastrointestinal haemorrhage or with acute onset abdominal pain. In this series the diagnosis was most commonly established or confirmed with the use of computed tomography and less frequently with selective arteriography or at surgery. Diagnosis should be suspected when a patient presents with acute abdominal symptoms and a history of a previous thrombotic episode with a thrombophilic state (Harward et al, 1989).

Early diagnosis can avoid the need for surgery and reduce the mortality from this condition with the prompt use of thrombolytic, anticoagulant, antiplatelet and antispasmodic therapy (Moriau and Azerad, 1996).

A novel approach to the acute management was recently published where transjugular intrahepatic portosystemic stent placement with thrombolysis was used in a patient with Budd–Chiari syndrome, portal vein and mesenteric vein thrombosis and homozygous for factor V leiden mutation (Leebeek et al, 1998).

The relatively recent discovery of factor V leiden followed the clinical observation that some people with familial or recurrent thromboembolic events did not prolong their partial thromboplastin time (PTT) when activated protein C (APC) was added to their plasma (APC resistance) (Bertina et al, 1994).

APC resistance is caused by a single mutation in the factor V gene resulting in the replacement of the arginine at position 506 with glutamate. A single origin mutation was first suggested by the observation that prevalence was varied among different races. In caucasian individuals,

CASE REPORT

A 47-year-old male was admitted with a 2-week history of periumbilical discomfort. For 48 hours before admission, the patient suffered from associated vomiting and anorexia. Upon admission the abdominal pain had become more severe and began to radiate to both flanks.

His past medical history included a deep vein thrombosis 18 months previously. He was taking Tegretol for epilepsy (seizure free for many years) and regular inhalers for asthma. He had no recent episode of prolonged immobility. He was a non-smoker and did not drink alcohol.

On examination, he was obese (96 kg). He had a low grade pyrexia with a sinus tachycardia (120 beats per minute). He was normotensive. Abdominal examination revealed tenderness over the umbilical and epigastric areas. At this stage, there was no evidence of guarding or rigidity and bowel sounds were present.

Investigations including serum bioprofile, amylase, coagulation screen, chest X-ray, plain film of abdomen and ultrasound of abdomen were all reported as normal. Full blood count revealed a moderate leucocytosis (13.2 x 10^9/litre).

At this stage he was managed conservatively with intravenous fluids and antibiotics. However, over the next 12 hours, the patient's condition deteriorated and he developed diffuse peritonism with guarding and rebound tenderness. He was prepared for theatre. Laparoscopy demonstrated haemosanguinous fluid and gangrenous loops of small bowel. Laparotomy confirmed the presence of a 24" gangrenous segment of distal jejenum and proximal ileum. Perioperatively, this segment was noted to have good arterial pulsation with obvious clot in the venous arcades. Formal resection with end to end anastomosis was completed. Postoperative recovery was uneventful. After surgery he was anticoagulated with unfractionated heparin.

Histology confirmed mesenteric venous infarction. In view of the past history of deep vein thrombosis, a thrombophilia screen was performed. Our patient was heterozygous positive for the factor V leiden mutation. He was anticoagulated and discharged on warfarin indefinitely (under ongoing review).

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the prevalence is estimated at about 5%. However, in other ethnic groups the prevalence is dramatically lower, being extremely low in people of African and Asian ethnic origin (Deschiens et al, 1996; De Stefano et al, 1998).

The increased risk of thrombosis is more pronounced in venous rather than arterial thrombosis. It is not believed to be a major risk factor for arterial thrombosis. It is believed to be responsible for approximately 7% of episodes of venous thromboembolism (Rodeghiero and Tosetto, 1999). Heterozygosity of this allele is associated with about a fourfold increase in risk of first time venous thromboembolism (Siminoni et al, 1999).

Treatment of the venous thromboembolic event is anticoagulation initially with heparin and subsequently with warfarin. However, the duration of treatment is controversial because there are no published randomized controlled trials available wto date which address this question. In our case, we chose to recommend long-term anticoagulation because it was the patient's second venous thromboembolic event and the second episode involved an unusual site, i.e. the mesenteric vein. Long-term anticoagulation with an international normalized ratio target of 2–3 is associated with a risk of major bleeding of about 3%, therefore other less intensive approaches to prevention are being investigated, e.g. the use of low dose warfarin which is currently being studied in the Prevention of Recurrent Venous Thromboembolism (PREVENT) trial (Ridker, 1998).

In medical college we learn the long list of causes of the acute abdomen. This case is an important reminder that this list is continually evolving. **HM**

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In the Public's View...

Even if surgery is possible, is it ethical?

But why were they here at all? Of all the thousands of words written about Jodie and Mary, the Siamese twins from Gozo, near Malta, none answered this question. Columnists put their ethical, legal and religious hats on and opined whether it was right or wrong for a judge to find in favour of an operation, against their parents' wishes, to save Jodie at the cost of Mary's life. On balance, I agreed with Richard Nicholson, editor of the *Bulletin of Medical Ethics* writing in the *Independent on Sunday*. He saw the ethical issue as primarily one of means and ends: the same judgment he reasoned, would support taking the organs from an anencephalic to save the life of another child, even if that child's parents did not want the operation. Dame Mary Warnock, writing in the *Guardian*, thought the judge's decision wise, and one with which most people would be comfortable. She likened Mary to a tumour that otherwise would inevitably kill her sister.

But how did the parents come to the attention of the specialized medical team who would do the operation? So far the parents remain anonymous, and the details mysterious, although we now know that their government had an arrangement with ours, which is why they were here. They will remain so unless the parents sell their story to a tabloid, which is possible: according to the parents' statement in court, they are from a poor, remote community. They worry that they won't be able to cope if Jodie is left with any important disability after the operation. As their objection to the operation to save Jodie's life is religious, one wonders how Jodie will be accepted back into their similarly religious community even if Jodie is not left doubly incontinent. The decision from the High Court has been made to separate them, and the parents have agreed.

In poor communities all around the world there are people suffering from diseases that don't get treatment. If we stay with surgery and incontinence, what about the thousands of women in Africa who are ostracised because of vesico-vaginal fistulae sustained in childbirth? Surgery for most of these women is comparatively easy (if we compare separating Siamese twins). Even if the fistula surgery itself is technically difficult, the anaesthesia and postoperative support is far less intensive. Mothers with fistulae already have live children, whom they often have to abandon. If we think in utilitarian terms — which is the ethical basis for saving Jodie at Mary's expense: one life saved is the maximum 'benefit' — then we do far more with the resources needed to 'save' Jodie by healing a number of women.

A kindly anaesthetist whom I met when I was a trainee helped me anaesthetize a terribly retarded and malformed 17 year-old-boy with spina bifida. He was undergoing his umpteenth insertion of ventricular drain. 'You know what they should have done', he said to me, 'they should have thrown him in the bucket and started again'. An uncomfortable metaphor, but Jodie and Mary's parents would have been happy to have nursed them lovingly until they died, and they should have been allowed to do it. If that was not possible in their own community, they could have been supported outside it. Instead, by a technological 'triumph', yet again Western medicine distorts the perception of what is important in health care. **HM**

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