CASE REPORT

A silent acute abdomen in a patient with spinal cord injury

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SUMMARY

A 52-year-old man with cervical spondylosis sustained a hyperextension injury to the neck and subsequently developed central cord syndrome after 2 weeks. The diagnosis was confirmed clinically and on MRI. During the admission he was febrile from *Streptococcus anginosus* bacteraemia from a gum infection and was started on penicillin. This resulted in pseudomembranous colitis with abdominal distension and bloody diarrhoea but a lack of expected abdominal complaints.

Unfortunately his neurology deteriorated and a repeat MRI showed a discitis at C5–C7 which required a 2-level discectomy, debridement and instrumented fusion. Owing to his spinal cord injury, an abdominal perforation was initially missed owing to the lack of clinical features of an acute abdomen. He underwent a right hemi-colectomy for ascending colon perforation and eventually made a good recovery and was discharged to a spinal rehabilitation unit. By one year follow-up he had returned to full neurological function.

BACKGROUND

Overall this is a very interesting case where several different pathologies have tied in together to create a diagnostic challenge. An initial accident resulted in neck and dental injury. The dental infection resulted in bacteraemia, and treatment with penicillin caused *Clostridium difficile* colitis. This was not picked up easily owing to altered sensory function from spinal cord injury (SCI). The deterioration in neurology resulting from discitis was possibly caused by the bacteraemia. Finally, the colonic perforation also presented as a 'silent acute abdomen'.

We particularly highlight the difficulties encountered in patients with spinal cord injuries who develop an acute abdomen owing to the lack of clinical signs and symptoms. Apart from distension, our patient had no abdominal complaints at the time of suspected perforation.

It is well documented that the acute abdomen is difficult to diagnose in this group of patients as visceral and somatic abdominal sensation can be affected. We hope to remind doctors who may come across such patients that a high index of suspicion should be maintained in order not to miss a potentially serious intra-abdominal condition.

CASE PRESENTATION

The patient in this case report is a 52-year-old man with a background of alcoholism, hypertension, hyperlipidaemia and cervical spondylosis. He presented at the emergency department 2 weeks after a fight. He had been punched on the face causing a

hyperextension injury of his neck and lost his upper right incisor. His symptoms included progressive neck pain, with development of radiculopathy, weakness of upper and lower limbs, resulting in a fall leading to hospital admission.

Initial examination revealed Glasgow coma scale 15, upper limbs were weaker than lower limbs, and patchy sensory loss in his arms and legs with no distinct sensory level. He was found to have up-going plantar reflexes, intact anal tone, intact perianal sensation and residual urine of 1.5 litres requiring catheterisation. He had neck tenderness and was also noted to be febrile. He was placed in a hard collar and sent for cervical radiographs followed by a CT of the cervical spine. A lamina fracture of C6 among degenerative changes was noted. An MRI (figure 1) was obtained later which confirmed spondylotic disease and C6 lamina fracture with cord contusion at C4–C5 levels. Treatment with intravenous dexamethasone was given for 3 days and neurology had showed some improvement.

The presence of low-grade fever and raised inflammatory markers since admission had prompted the taking of blood cultures, from which *Streptococcus anginosus* was isolated. The patient was started on intravenous benzylpenicillin day 6 postadmission, and a source was sought. Cardiac ECHO revealed no vegetations and repeat cultures on starting antibiotics were negative. The source was eventually localised to the missing incisor with an underlying local gum infection requiring treatment by the dental team.

On day 8 postadmission, abdominal distension was evident. The main symptom was difficulty in breathing owing to distension. He also started having diarrhoea. A CT abdomen was done initially but did not raise concerns from the general surgeons. However, a few days later he had blood in his stool. A gastroscopy and colonoscopy revealed appearance of *C difficile* colitis. Stools were positive for *C difficile* toxin and the penicillin was stopped and replaced with oral vancomycin and metronidazole.

Incidentally, on day 10 postadmission, neurology had deteriorated further and an urgent MRI showed an increased fluid signal in C5–C7 vertebrae and intervening discs with a left para-vertebral fluid collection and canal stenosis. The diagnosis of spondylodiscitis was made. In addition to oral vancomycin for *C difficile*, intravenous vancomycin was initiated and plans for spinal surgery were made pending the improvement of bowel pathology.

Day 17 postadmission, a repeat abdominal film (figure 2) was performed to assess bowel distension

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Figure 1 These are the two T2-weighted MRIs taken 10 days apart. The left MRI was done early in admission showing cervical spondylosis marrow oedema in C6 and C7 vertebral bodies and patchy signal increase in the cord at C4–C5 levels suggestive of traumatic cord contusion. The second MRI on the right was done after further neurological deterioration and marrow oedema in C5, C6 and C7 vertebral bodies is evident with high fluid signal in C5/6 and C6/7 disc suggestive of discitis.

which was noted to be reduced. The patient complained of no abdominal pain and examination revealed no tenderness/guarding, but obvious distension. The patient was taken to surgery the next day to undergo C5–C6 and C6–C7 debridement, curettage and fusion with bone graft and instrumentation. Seropurulent fluid was collected intraoperatively; however, microbiological examination revealed no organisms.

During the first postoperative day in intensive treatment unit, a routine chest radiograph revealed free air under the right hemi-diaphragm causing alarm (figure 3). It was then noted retrospectively that the preoperative supine abdominal film did show pneumoperitoneum, which was missed by the junior doctors in the ward. General surgeons operated on the patient a few hours later and discovered a perforation in the dilated ascending colon and purulent peritoneal fluid. A right hemicolectomy was performed with double barrel stoma of ileum and transverse colon. A regime of intravenous metronidazole, intravenous vancomycin and intravenous tazocin was used to control sepsis.

OUTCOME AND FOLLOW-UP

The patient's postoperative issues included a sacral sore, as well as intra-abdominal collection and abdominal wound infection,

Figure 2 The two supine abdominal radiographs demonstrate the important change resulting from bowel perforation. The left image shows a radiograph taken during the initial period of abdominal distension and demonstrates dilated colon. The second radiograph on the right was taken the day prior to spinal surgery and the colonic distension appears to be reduced but the air on either side of bowel wall (Riggler's sign) is apparent and should have raised alarms for perforation.

Figure 3 A supine chest radiograph was done in ITU 1 day postcervical surgery in order to confirm the nasogastric tube position, and despite not being an erect film picked up a prominent amount of free air under the right hemi-diaphragm. ITU, intensive treatment unit.

both of which were managed non-operatively with antibiotics. He was eventually discharged from hospital 40 days after operation, with enough improvement in power to ambulate with the help of a walking frame. He went to a community hospital for ongoing rehabilitation for another 3 months. On six months postinitial hemicolectomy, he returned to the care of general surgeons electively and underwent an uneventful reversal of the double-barrel colostomy and ileostomy with reanastomosis. He was followed up in the spinal surgery clinic for a year from initial surgery. He was noted to have full power in his limbs, but owing to unsteady gait, required walking frame for 4 months postoperation and crutches for another 3 months after that. Sensation and function of bladder and bowel were noted to be intact at all follow-ups. Outpatient blood tests revealed no rising inflammatory markers and overall he had a satisfactory surgical outcome from both cervical spine and abdominal points of view.

DISCUSSION

SCI is a well-documented condition with 15–40 cases seen per million people per year.¹ The acute abdomen in SCI patients is thus rare and we have only found a few case series in the current literature with more than 20 patients.^{2–6} The incidence of acute abdomen in SCI varies from 1% to 4%^{2–5} based on the inclusion criteria being gastrointestinal (GI) perforations, obstructions/volvulus and appendicitis. The largest study of appendicitis in SCI by Strauther *et al*⁶ was based on 23 295 patients with only 30 cases of appendicitis, that is, 0.1%.

Acute abdomens may be difficult to ascertain in the face of chronic GI complaints as the majority of patients with SCI suffer from constipation, incontinence, pain, abdominal distension, etc, which are unrelated to acute pathology.⁷ The acute abdomen after SCI is a topic of interest and well documented owing to the challenges faced in diagnosis. Several authors have noted that patients are misdiagnosed or have a delay of 1–4 days in reaching the diagnosis.^{2–5–6} This is often held responsible for increased mortality.²

The death rates in this situation are again difficult to state given the small sizes of case series. Our literature review, however, has indicated the death rates vary from 4% to 16.6%.^{2–6} Diagnostic difficulties exist in SCI owing to the lack of display of normal clinical signs and symptoms associated with

acute abdomen. As in this patient, there have been reports where abdominal pain/tenderness is completely absent.^{6 8 9} Signs of fever and leukocytosis are surprisingly infrequent.^{2 4}

To get a better understanding of why presenting features are unusual in SCI, we need to understand the innervation pathways involved, which have been described well by Miller *et al*⁴ and Neumayer *et al*.⁵ Somatic abdominal sensation (T7–L1) is lost with lesions above T7, while sympathetic innervation to viscera which arises from T6 and below from splanchnic nerves can be affected by lesions above T6. T6 level hereby is the demarcation for differentiating the high cord from the low cord lesions. Visceral sensation may still be preserved in low cord lesions related to the level of cord injury; in high cord injuries, visceral sensation may be present if there is an incomplete cord injury leaving intact fibres through the affected cord level, or if visceral fibres ascend the sympathetic chain to bypass the injured cord level.

The literature describes cases where pain is in fact a presenting feature which could be well localised^{3–5} or vaguely localised presenting as a ‘sense’ of discomfort.^{3–6 8} Pain of an acute abdomen with low cord lesions is better localised and similar to normal patients^{3–5} whereas pain is more likely to be absent/vague in high cord lesions.^{4 5} However, incomplete high lesions may allow normal pain sensation.³ Tenderness despite a perforation is often absent⁶ but when present should be taken seriously as is highly likely to be owing to perforation.³ Another important symptom is referred pain to the shoulder. This feature has been commonly noted^{2 3 5 9} and tends to be more common in high cord lesions.^{3 5}

Other features that may be seen in the spectrum of acute abdomen include: loss of bowel sounds,³ abdominal distension,^{2 5 6 8 9} nausea/vomiting^{4 6 8 9} and increased abdominal spasticity.^{2–5} Spasticity is the result of intact spinal reflexes below the level of cord injury. Increased visceral sensory input from abdominal pathology initiates the reflex arc, targeting the abdominal wall muscles and this is more prevalent in high cord lesions.^{3 5} A special mention must be made to the presence of autonomic dysreflexia in SCI. Increased visceral afferent stimulation (by irritation/distension) can result in excessive autonomic stimulation producing vasoconstriction below the level of cord injury. This produces characteristic hypertension (enough to cause headaches), sweating and tachycardia/bradycardia. Dysreflexia has been reported by several authors and is more likely in higher cord lesions above T6.^{3 5 6}

In the context of our case, the patient developed *C difficile* colitis which caused abdominal distension and difficulty in breathing. It took 2–3 days before this diagnosis was confirmed. It is also unclear as to when the perforation occurred. However, the lack of pain and tenderness resulted in a low suspicion for acute abdomen, and an erect chest radiograph pre-operatively was not even considered preoperatively. It probably would have revealed free air that is more obvious than a Riggler’s sign on a supine abdominal film. It is interesting to note that American Spinal Injury Association (ASIA) scoring carried out on our patient revealed no classic pattern. Initial presentation of upper limb weakness greater than lower limb weakness fits with central cord syndrome but with time there was significant deterioration in lower limb power. The areas of diminished sensation were in a patchy distribution that did not clearly fit a sensory level. We feel that although some abdominal somatic sensation was present, this was an incomplete SCI where the visceral afferent pathways may have been more affected than the somatic assessment would suggest. This is an important feature we would like to highlight in this case—that a painless abdomen in the SCI patient with apparent somatic abdominal sensation does not guarantee that ‘all is well’.

The cause of our patient’s cord injury was thought to be a delayed central cord syndrome as per clinical presentation and MRI findings of cord contusion and oedema at C4–C5. There was a period of recovery followed by worsening of neurology which was attributed to the spondylodiscitis and associated epidural collection. As the *S anginosus* bacteraemia had been discovered earlier and the patient started on appropriate antibiotics, this may be the reason why intra-operative cultures were negative. *S anginosus* is within the *Streptococcus milleri* group of bacteria that are commensals of the oropharynx and GI tract. *S anginosus* bacteraemia leading to spondylodiscitis has been previously reported.¹⁰ However, the commonest organism causing spondylodiscitis is *Staphylococcus aureus*.¹¹

We advocate that any suspicion of the acute abdomen in SCI patients should prompt the use of appropriate imaging. In one case series⁵ the correct diagnosis was achieved via radiological investigation in 77% of patients. Although the acute abdomen is difficult to pick up in SCI patients, clinicians should be aware of these potential pitfalls and conduct more thorough assessments, actively looking for concerning features both on history/examination and via appropriate investigations.

Learning points

- ▶ Spinal cord injury can result in altered visceral sensation thus making the diagnosis of an acute abdomen challenging, even in the face of perforation.
- ▶ The clinical presentation correlates to the level and completeness of cord injury. In high cord injuries, symptoms of shoulder tip pain and signs of autonomic dysreflexia should not be ignored.
- ▶ Any abdominal pain or tenderness should be investigated even if the patient is afebrile or lacks leukocytosis. An appropriate imaging is one of the most useful tools in diagnosis. Riggler’s sign is uncommonly seen in practice but is an important radiographic feature not to miss.

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