CASE REPORT

A seizure-induced T8 burst fracture re-presenting as an acute abdomen

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SUMMARY

A male aged 44 years presented to the emergency department with a 1-week history of intermittent right-sided abdominal pain radiating to the midline. Examination demonstrated a tender right upper quadrant with voluntary guarding and a low grade fever. One week previously, he had been admitted to hospital after an isolated, self-terminating seizure secondary to an deliberate venlafaxine overdose. His upper abdominal symptoms started immediately postseizure but at the time were attributed to musculoskeletal chest pain by the discharging team. Acute cholecystitis was suspected, but liver function tests, amylase and an erect chest radiograph were unremarkable. The abdominal pain responded well to morphine, permitting a thorough reassessment of the patient, which revealed midthoracic spine tenderness, previously undetected. Prior to this, the patient had not complained of any back pain. A CT scan confirmed a burst fracture of T8 requiring urgent transfer to the local spinal unit for posterior stabilisation.

BACKGROUND

This case demonstrates a rare presentation of a clinically significant thoracic vertebral fracture and the associated diagnostic challenges this entails.

In this scenario, the patient's abdominal signs and symptoms led the clinicians down an incorrect diagnostic pathway with potentially adverse consequences for the patient. The case highlights the importance of clinicians having an awareness of distracting injuries and the value of reassessing after appropriate analgesia which may change the clinical picture, ensuring alternative diagnoses are not missed. From a patient safety perspective, there is a valuable lesson to be learnt as the delay in diagnosis had the potential to cause devastating neurological sequelae as a result of mobilising on an unstable spinal column fracture.

CASE PRESENTATION

A male aged 44 years presented to the emergency department with a 1-week history of intermittent right-sided abdominal pain radiating to the midline. The pain was severe and sharp in nature and had no exacerbating factors. No nausea, vomiting, loss of appetite or fevers were reported, but the patient was complaining of constipation with his bowels last opened 2 days ago. He had been taking regular co-codamol at home with minimal effect. No change in the colour or consistency of his stool or urine was

reported and the patient denied any lower urinary tract symptoms. The patient drank the occasional glass of wine with no history of recent alcohol consumption. He did not have a history of gallstones, pancreatitis or abdominal surgery. His only past medical history was depression with multiple overdoses, the last overdose being taken 1 week before presentation. On that occasion, he had been admitted to hospital after a witnessed isolated tonic-clonic seizure secondary to a deliberate venlafaxine overdose. There was no history of any fall or trauma prior to the seizure.

The patient denied any further overdoses but revealed that his symptoms had started ever since the seizure which at the time had been labelled as musculoskeletal chest pain by the discharging team and therefore no further investigations or follow-up was arranged.

The patient's abdomen was examined in the emergency department by a senior registrar in emergency medicine prior to analgesia, which demonstrated a tender right upper quadrant with voluntary guarding. However, Murphy's sign was negative and no abdominal masses, hepatomegaly or Cullen's sign were elicited. Bowel sounds were active and there were no peripheral stigmata of gastrointestinal disease. After morphine administration, the patient was able to tolerate a thorough reassessment which demonstrated a soft non-tender abdomen, but revealed midthoracic spine tenderness, previously undetected. Prior to this, the patient had not complained of any back pain.

INVESTIGATIONS

Initial investigations showed unremarkable liver function tests and amylase, but demonstrated a raised white cell count of 13.9. An abdominal and erect chest radiograph were performed which were unremarkable. However, the chest radiograph was underpenetrated and therefore the thoracic vertebra could not be adequately visualised. Urinalysis was normal. Subsequent to the new findings on reassessment, a thoracic spine radiograph was requested which revealed a severe compression fracture of the T8 vertebra. In light of the diagnosis, the patient's neurological status was urgently assessed which was normal and a rectal examination was performed which showed preserved anal tone and perianal sensation.

The patient underwent a CT scan which confirmed a complex T8 burst fracture with 75% loss of vertebral body height and an associated kyphotic deformity (see [figure 1](#)). An MRI was also

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Figure 1 A CT sagittal (left) and axial (right) image showing an unstable T8 burst fracture with an associated kyphotic deformity.

performed which showed no spinal cord compression or cord signal changes.

DIFFERENTIAL DIAGNOSIS

The main differential diagnoses to be considered are biliary colic, acute pancreatitis, retrocaecal appendicitis and acute hepatic failure secondary to an overdose. Other less common differential diagnoses include right lower lobe pneumonia and a musculoskeletal injury.

TREATMENT

The patient was admitted for bedrest and analgesia and the local spinal unit was contacted for advice on further management. A decision was made to transfer the patient for urgent spinal surgery. The patient subsequently underwent posterior stabilisation of T8 with a T7-T9 discectomy, cage insertion and left lateral and anterior corpectomy (see figure 2). This operation lasted 9 hours.

Figure 2 An intraoperative lateral radiograph showing the cage and posterior stabilisation of T8.

OUTCOME AND FOLLOW-UP

The operation was successful and the patient made a good recovery with no neurological sequelae. Two days after the operation, the patient was mobilising with assistance from the physiotherapists and his good progress meant that he could be discharged directly back home from the spinal unit.

DISCUSSION

Vertebral fractures are an uncommon complication of seizures with a reported incidence of 1%¹ according to some studies. They tend to occur in younger patients and are believed to result from the powerful contractions of the paraspinal musculature causing hyperflexion of the spine during seizure activity.² Our case describes a midthoracic spine burst fracture in a male aged 44 years resulting from an isolated drug-induced seizure, a phenomenon rarely described in the literature. Burst fractures are characterised by disruption to the anterior and middle columns of the spine during substantial axial compressive loads and are considered unstable when associated with >50% loss of vertebral body height,³ as reported in our case. Burst fractures carry a higher risk of spinal cord injury than simple compression fractures due to the potential for bony fragments to be ejected into the vertebral canal. In this instance, no neurological complications occurred as a result of the fracture or subsequent treatment.

To date, only five case reports⁴⁻⁸ exist in the published literature since 1994 that describe thoracic spine burst fractures resulting from seizures, only two of which relate to drug overdoses. McCullen and Brown⁴ described a female aged 41 years who had an isolated seizure after a deliberate tricyclic antidepressant overdose, sustaining bilateral humeral neck fractures and a T7 burst fracture, without neurological complications. Interestingly, the patient presented with delayed onset back pain 1 week after the seizure. The patient underwent spinal decompression and fusion of T7 and was able to mobilise on day 5 postoperatively with a Jewett brace.

More recently, Rahimizadeh *et al*⁵ reported a male aged 20 years who sustained a T12 burst fracture after a seizure secondary to a deliberate tramadol overdose. Consistent with the case by McCullen and Brown,⁴ the patient also presented with delayed onset back pain and lower extremity paraesthesia

1 week following the seizure. The MRI showed compression of the spinal cord by a bony fragment for which the patient underwent decompression. The patient made a full neurological recovery after 2 weeks.

In contrast to the literature, we wish to draw our reader's attention to the atypical way our patient presented, from which important clinical lessons can be learnt. Our patient was not initially asymptomatic postseizure unlike previous reports but rather complained of vague musculoskeletal symptoms from the outset. The significance of these symptoms were not recognised by the discharging medical team which led to the patient being mobilised and discharged with an unstable spinal column fracture with a risk for significant neurological complications. Therefore, to reduce the likelihood of this diagnosis being overlooked in future, clinicians should consider performing a secondary survey postseizure to exclude clinically significant musculoskeletal injuries prior to mobilisation and discharge.

Furthermore, where the majority of patients described in the literature presented with back pain, to our knowledge, we present the first example of a midthoracic spine burst fracture representing as an acute abdomen. We hypothesise that our patient's right upper quadrant pain may have been referred pain in the right T8 dermatome due to impingement of the right T8 spinal nerve root. Although the MRI did not show evidence of this, it is possible that nerve impingement still occurred but had resolved by the time the scan was performed, 2 days after presentation.

Consistent with this hypothesis, Lyon *et al*⁹ described a woman aged 55 years who complained of intermittent right upper quadrant pain and was considered for a cholecystectomy until an intradural mass at the T8 level was discovered. The authors suggested that the pain may have resulted from intrinsic compression of the thoracic nerve root at that level. Other plausible hypotheses for the patient's pain include an inflammatory nerve root irritation or traumatic muscular injury caused by hyperflexion during the seizure.

Similar to the report by Lyon *et al*,⁹ our patient's presentation mimicked acute cholecystitis, leading the patient down an incorrect diagnostic pathway with a further delay in diagnosis. This stresses the importance of clinicians being aware of the potential for spinal pathology associated with a seizure to cause acute abdominal pain.

Learning points

- ▶ Seizure-induced thoracic spine burst fractures are a rare but clinically significant injury postseizure.
- ▶ The most common presentation of this injury is back pain but may also rarely present as acute abdominal pain.
- ▶ Other musculoskeletal injuries to be aware of postseizure include anterior or posterior shoulder dislocations, proximal humerus and femoral neck fractures. Therefore to ensure these distracting injuries are not overlooked, consider performing a secondary survey on any patient postseizure.

Contributors RAJS was responsible for the conception, design and writing of the article and acquisition of images. NP helped to critically revise the article. SC helped to critically revise and approve the final version of the article.

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