CASE REPORT

Catastrophic chest pain: blinded by cardiopulmonary disease

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Accepted 21 May 2015

SUMMARY

A 53-year-old man with a history of diabetic foot ulcer, osteomyelitis, coronary artery disease, hypertension and hyperlipidaemia, presented with chest pain of 3 weeks duration. Eleven days earlier, the patient had had a drug-eluting stent (DES) placed in a branch of the right coronary artery (RCA) after similar chest pain, leading to the findings of a positive nuclear stress test. Since discharge, he was not compliant with taking clopidogrel (Plavix), a concern for in-stent thrombosis with recurrent myocardial ischaemia; but work up was negative and medications were restarted. Within 24 h of admission, he developed bilateral flaccid leg weakness, urine retention and loss of sensation from the umbilicus level down. MRI revealed a T4–T6 epidural abscess. Emergent decompression laminectomy and abscess drainage was completed. Neurological symptoms improved hours after surgery with complete resolution of sensory deficits. Cultures grew *Streptococcus* sp., treated with intravenous nafcillin for 8 weeks. He regained leg strength with continued improvement seen in rehabilitation.

BACKGROUND

When patients present with chest pain and shortness of breath, clinicians reflexively think of five differential diagnoses including: ischaemic heart disease, aortic dissection, pulmonary embolism, tension pneumothorax and pericarditis with tamponade. Once these are ruled out, chest pain is usually attributed to either gastrointestinal or musculoskeletal conditions, which reduce suspicion for other chest pain aetiologies that may be fatal. The presence of an acute cardiopulmonary process may blind physicians from recognising other potentially fatal processes even in presence of overt evidence. This case represents a potentially life-threatening process with delayed diagnosis and treatment in the absence of acute cardiopulmonary process on initial work up.

CASE PRESENTATION

A 53-year-old man presented to the emergency room with dull aching right-sided chest pain for 3 weeks. The pain was located on the right anterior chest area. Two weeks prior, he had presented with similar symptoms, and a stress test revealed reversible inferior cardiac wall ischaemia. Cardiac catheterisation was performed, with drug-eluting stent (DES) placement in the right posterolateral branch of the right coronary artery (RCA), which had 80% proximal stenosis. The patient was discharged on metoprolol (Lopressor), atorvastatin (Lipitor),

aspirin and clopidogrel (Plavix). He was unable to afford the clopidogrel (Plavix), hence he did not take it.

His medical history included diabetes with history of left first metatarsal osteomyelitis, hypertension, coronary artery disease and hyperlipidaemia. Vitals revealed a blood pressure of 194/90 mm Hg; pulse 110 bpm, with pulses equal in both arms; temperature 99.8°F; respiratory rate of 14 breaths/min; and SpO₂ 99% on room air. Head and neck finding were unremarkable with the cardiac examination showing no murmurs or gallops. The lungs were clear without adventitious sounds. A left first metatarsal scar consistent with recently healed ulcer was present. The neurological examination was of normal findings.

INVESTIGATIONS

ECG showed no changes from previous with multifocal PVCs. The white cell count (WCC), haemoglobin and platelet counts were normal. Serum chemistries revealed sodium of 125 mmol/L and glucose of 389 mg/dL; all other parameters were normal. Further testing revealed isotonic, euvoelaemic hyponatraemia with inappropriately high urine sodium attributed to diuretics. Sedimentation rate was 5 mm/h. Ischaemic profile was negative. Chest radiograph was normal, but chest CT angiogram revealed a right paravertebral soft tissue mass measuring 5.4×1.8 cm (figure 1). Similar findings were noted on imaging tests carried out on prior admission.

DIFFERENTIAL DIAGNOSIS

1. Musculoskeletal chest pain
2. Gastro-oesophageal reflux-related chest pain
3. Pneumonia

Figure 1 CT of the chest showing a right paravertebral mass 5.4×1.8 cm in size pressing on lung parenchyma.

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To cite: Barreiro TJ, Asiimwe DD, Gemmel D, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2015-209928

4. Lung mass
5. Acute coronary syndrome
6. Aortic dissection

TREATMENT

In the emergency room, the patient's blood pressure was controlled with intravenous β -blocker, nitroglycerin and his home medications. He was given clopidogrel (Plavix) and aspirin. His chest pain was relieved with intravenous morphine. He was admitted to the chest pain unit with concerns for possible in-stent thrombosis, and cardiology was consulted. A pulmonary consultation was requested because the radiologist reported 'mass' seen on images.

OUTCOME AND FOLLOW-UP

Within hours of admission, the patient reported urinary retention, which required straight urethral catheterisation. Hours later the patient was still unable to void and developed numbness in the left leg. At this time, neurological evaluation was unrevealing. A foley catheter was inserted. Symptoms worsened to bilateral leg weakness with the inability to then move his legs. Examination revealed reduced power in bilateral lower extremities with right at 1/5 and left at 2/5. The patient also exhibited no sensation to light touch and pain up to the umbilicus level, and reduced anal sphincter tone was noted. Neurosurgery consultation was made and thoracic spine MRI revealed an epidural abscess at levels T4 through T6 with features of osteomyelitis (figures 2–4). The patient was treated with steroids and emergency decompression laminectomy with drainage of the epidural

Figure 2 Axial view MRI showing normal appearing midline spinal cord, with increased contrast enhancement at the right paravertebral area at T4 level consistent with inflammatory changes (arrows).

Figure 3 Axial view MRI showing mixed density mass with areas of necrosis at the right paravertebral area at level of T5–T6 extending into the epidural space and displacing the cord towards the left (arrows).

abscess. Neurological symptoms improved several hours after surgery with complete resolution of sensory loss. Cultures grew *group B Streptococcus*, treated with intravenous nafcillin for 8 weeks. Osteomyelitis of the left great toe was presumed to be the infection nidus. The patient was discharged to rehabilitation and was able to walk with a walker within weeks.

DISCUSSION

The differential diagnosis of chest pain is wide; however, the immediately life-threatening conditions include: acute coronary syndrome, aortic dissection, pulmonary embolism, tension pneumothorax and cardiac tamponade. Absence of the above processes may result in physicians being less vigilant in urgently searching for and treating other conditions causing chest pain, which are rare, yet could be fatal. Gastrointestinal or musculoskeletal causes of chest pain could be fatal depending on the severity and location of the pathology. For example, musculoskeletal chest pain resulting from trauma, infection, rheumatological diseases or malignancy, especially involving the spinal cord or surrounding structures, could result in permanent disability or death if not identified and treated in time.

Spinal epidural abscess (SEA) is a suppurative infection localised to the extradural space,¹ which commonly presents with back pain. SEA is not a typical diagnosis in patients presenting with chest pain, especially in a patient with known coronary artery disease and recent stent placement. A recent meta-analysis reported incidence of 2–3 cases of SEA per 10 000 hospital admissions, an increase over the incidence reported in retrospective studies from 1990, which spanned 10 years.^{1 2} This increase in the incidence of SEA may be attributed to a number of factors, including better neuroimaging capabilities, improved disease awareness, an increased number of elderly and immunocompromised patients and growing rates of illicit drug abuse.³ Diabetes mellitus, intravenous drug abuse, tattooing, acupuncture, spinal instrumentation procedures, chronic haemodialysis, alcohol abuse, trauma, long-term intravascular devices and immune compromise are other risk factors for development of SEA.^{3 4}

Primary SEAs result from haematogenous spread of pathogens from a distant site into the epidural space; secondary SEAs occur after spinal trauma, injections, surgery or direct inoculation of a pathogen into the epidural space.¹ At least 60% of

Figure 4 Sagittal views, MRI showing increased density at level of T5–T6, consistent with epidural abscess, note slight narrowing of the cord at this level and reactive inflammatory changes (whitish densities) on the vertebral bodies of T5–T6 vertebrae.

SEAs are a result of a primary mechanism.^{1 2} One third of cases are due to a secondary mechanism and the remainder of the cases may have no identifiable source.⁴ The most common isolated organism is *Staphylococcus aureus* (>60%), followed by *Streptococcus* spp.^{1 2} Rarely, other organisms may be isolated. The mechanism by which SEA causes neurological injury is controversial; however, a combination of direct mechanical compression by pus with or without associated vascular damage and local inflammation, leading to thrombophlebitis or venous congestion are thought to play some role.^{2 4} The location of most SEAs appears to occur in spinal regions with relatively larger epidural space and where excessive epidural fat padding is located.^{2 3} Thoracic spine involvement is most commonly reported at 50%, followed by lumbar spine involvement in 35% of cases.¹ Cervical spine involvement is low, at 14%.³ However, some reports show conflicting results as to positional location of the abscess, either anteriorly or posteriorly, to the cord. Osteomyelitis was more common with anteriorly located abscesses. In our case, the patient had suspected T5 to T6 vertebral osteomyelitis associated with the SEA (figures 2 and 4).

SEA presenting features may be non-specific and subacute.^{2 3} Although our patient initially presented with insidious right-sided chest pain, possibly referred from the thoracic spine, his clinical presentation may best fit into Heuser's description of the four stages of SEA evolution. Stage I includes spinal pain; stage II is nerve root pain; stage III involves motor, sensory and sphincter function; and stage IV involves paralysis.⁵ Back pain,

the most common symptom, is usually localised to the midline, with marked tenderness to spinous process percussion.^{2 3} The classic triad of back pain (90%), fever (67%) and neurological deficits (80%), is present only in a small percentage of patients.^{2 3} Therefore, a high index of suspicion is necessary in order to avoid delays in diagnosis and management.³

Despite having sophisticated imaging techniques and laboratory studies, SEA misdiagnosis is still high, with an average rate of approximately 50%.^{2 3} Plain spinal radiographs are generally unrevealing, unless there is significant structural damage.^{2 3} Imaging modality of choice is MRI; CT myelography is an equivalent option.^{2 3} WCC count, C reactive protein and sedimentation rate are usually, but not always, elevated,⁴ as was the case in our patient. Surgical drainage of the abscess confirms the diagnosis.⁴

Symptomatic vertebral osteomyelitis and SEA with neurological deficit is a surgical emergency.¹ The goal of surgery is to provide direct spinal cord and nerve decompression to prevent neurological deterioration and deficits.² Surgical debridement can remove inciting organisms, reduce the risk of vascular-related neurological complications and provide tissue for culture to isolate causative organisms.^{2 3} Treatment of neurologically intact patients with vertebral osteomyelitis alone or minimal epidural abscess is far more controversial. Successful medical treatment for these cases has been reported, as have cases of failed medical treatment resulting in neurological decline.¹ Surgery, in combination with systemic antibiotic therapy, is the treatment of choice.⁴

Learning points

- ▶ Spinal epidural abscess (SEA) may be difficult to diagnose and requires a high degree of suspicion.
- ▶ The classic triad of back pain, fevers and neurological deficits occurs in less than 20% of patients with SEA.
- ▶ Despite better imaging modalities and surgical techniques, misdiagnosis is surprisingly common, and a large percentage of patients suffer significant neurological deficits if diagnosis is delayed.
- ▶ SEA neurological symptoms lasting more than 36–48 h have been associated with a poor outcome.

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Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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