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CASE REPORT
Coronary intervention

Unusual presentation of acute spinal cord injury with ischaemic electrocardiographic changes: a case report

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Background Spinal cord injury (SCI) is a significant cause of morbidity and mortality with an incidence of 40–83/million/year. Sympathetic denervation in SCI leads to cardiovascular abnormalities including orthostatic hypotension, rhythm disturbance, and repolarization changes. Electrocardiographic (ECG) findings include bradyarrhythmias, ectopic beats, long QT interval, and ST-T changes that may be mistaken for myocardial ischaemia.

Case summary A patient in their 40 s with free past medical history was referred to our centre with the diagnosis of non-ST elevation acute coronary syndrome. On presentation, chest pain was diffuse and radiating to the back. Twelve-lead ECG showed deep symmetrical T-wave inversion. Echocardiography and cardiac troponin were normal. The patient was scheduled for multi-slice computed tomography coronary angiography which was normal; however, a few hours after admission, the patient developed rapidly progressive motor weakness in both lower limbs with urine retention. Examination revealed motor power Grade 1 in both lower limbs. All sensations were diminished with a sensory level at T6. Urgent magnetic resonance imaging spine revealed neoplastic infiltration of the whole vertebrae with D5/D6 fracture exerting spinal cord compression. The patient was referred for urgent decompression surgery.

Conclusion Electrocardiographic changes could be the earliest sign for ongoing SCI. ST-elevation is reported in higher levels of complete injury, while ST depression and inverted T waves can occur independent of lesion level or severity. Misinterpretation of these changes may cause a delay in reaching the correct diagnosis. We highlight the importance of considering neurological causes for ischaemic-like ECG changes, as early recognition could prevent irreversible functional loss.

Keywords Spinal cord injury • Paraplegia • Spine metastases • T-wave inversion • ECG • Case report

ESC curriculum 3.1 Coronary artery disease • 3.2 Acute coronary syndrome

Learning points

- Chest pain can be the first presenting symptom of spinal cord injury.
- Dynamic Electrocardiographic changes can happen in the context of spinal cord injury.
- Careful analysis of the complaint and thorough physical examination are crucial to diagnose and provide early intervention for emergency spinal cord decompression.

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Introduction

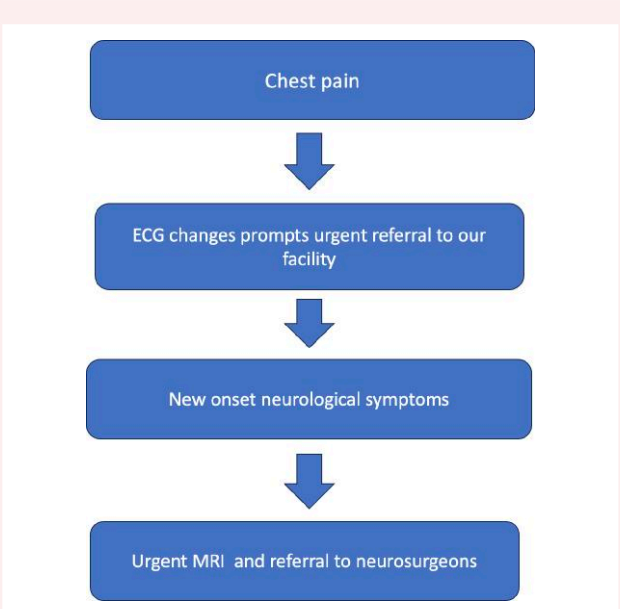
Spinal cord injury (SCI) is a significant cause of morbidity and mortality with a global incidence of 40–83/million/year.[1] Acute SCI is an emergency condition requiring rapid management to prevent irreversible neuronal damage and permanent disability.

Cardiovascular abnormalities have been reported in the acute and late stages of SCI. While autonomic dysfunction is the main mechanism of cardiovascular findings, abnormal myocardial repolarization was frequently described in literature.[2,3] Electrocardiogram (ECG) findings include bradyarrhythmias, ectopic beats, abnormal QT interval, and ST-T changes that may be mistaken for myocardial ischaemia.[4,5]

It is important for physicians to be aware of different ECG patterns associated with acute neurological insults, as some cases may present early before developing obvious neurological manifestations. Careful analysis of the chief complaint in view of clinical risk factors is necessary to avoid ECG misinterpretation and useless workup that may delay the correct diagnosis and management.

We present an unusual scenario of acute SCI which presented with chest pain associated with ECG changes mimicking acute myocardial ischaemia.

Summary figure



Case presentation

A patient in their 40 s with free past medical and drug history was referred to our centre with the diagnosis of acute coronary syndrome, after receiving loading aspirin dose.

On presentation, the patient had persistent chest pain for 2 days, with history of recurrent chest pain attacks in the past 2 months for which she received non-specific analgesics. Pain was diffuse, retrosternal, and radiating to the back.

On physical examination, blood pressure was 110/70 mmHg, pulse rate was 75 bpm, temperature was 37.0°C, respiratory rate 25 per minute, and oxygen saturation was 100% on room air. Generalized tenderness in the upper trunk and shoulders was appreciated.

Twelve-lead ECG showed deep symmetrical T-wave inversion in leads III, avF, and precordial leads V1–V4, and corrected QT interval was 459 ms by Bazett formula (*Figure 1*). Changes were persistent in serial ECGs.

Echocardiography revealed normal left ventricular dimensions and contractility, with no evidence of resting regional wall motion abnormalities.

Initial laboratory workup was unremarkable with normal electrolytes and three negative sets of highly sensitive cardiac troponin; therefore, multi-slice computed tomography coronary angiography was scheduled in the same day.[6]

A few hours after admission, the patient developed rapidly progressive motor weakness in both lower limbs associated with urine retention.

Neurological examination revealed motor power Grade 1 in both lower limbs, hypotonia and hyporeflexia, with absent Babinski's sign bilaterally. All sensations were diminished with a sensory level at T6 and hyperalgesia in the upper trunk. Examination of cranial nerves and both upper limbs was normal.

Multi-slice computed tomography coronary angiography showed no significant lesions. However, computed tomography chest revealed bilateral enlarged axillary lymph nodes and a soft tissue mass measuring 42 × 36 mm in the right breast, suggesting a primary tumour source for spine metastases.

Urgent magnetic resonance imaging spine was done, revealing neoplastic marrow infiltration of the whole vertebrae and pelvic bones with D5/D6 compression fracture associated with para-spinal and intra-spinal soft tissue component exerting compression upon the cord at the same level (*Figure 2*).

After neurosurgical consultation, the patient was started on analgesics, methylprednisolone infusion, and transferred for urgent spinal cord decompression in another facility.

Follow-up of the patient for 3 months after decompression surgery showed partial improvement of motor power to Grade 2 in both lower limbs. The diagnosis of breast malignancy was confirmed with distant metastases that were managed with palliative chemotherapy. Follow-up 12-lead ECG showed persistent repolarization changes (*Figure 3*).

Discussion

Metastatic spinal cord compression is the third commonest cause for SCI after trauma and degenerative spine disease, resulting in complete paraplegia in 15.9% of cases.[1] It affects approximately 2.5–5% of all patients with cancer.[7] Progression of neurological symptoms usually limits the time for pre-operative diagnostic workup. Surgical decompression followed by radiotherapy is mainly palliative with the aim of maintaining or improving neurological function. If not feasible to operate within 24 h, then surgery should be performed within 72 h after the injury.

Preganglionic sympathetic fibres are in the lateral column of the SC from level of T1 to L2. An injury of the SC at one of those levels would cause disruption of connection with the upper autonomic centres in the brain. Depending on the level and severity of injury, sympathetic denervation of the heart and blood vessel may occur.[8]

In the acute stage after SCI, sympathetic denervation of the heart may result in vagal pre-dominance, sinus bradycardia, and conduction disturbances. The incidence of bradyarrhythmia peaks on day 4 after injury and gradually declines thereafter. Abnormalities resolve spontaneously within 6 weeks.[9] In higher or complete lesions, bradyarrhythmia tends to persist and may require permanent pacemaker insertion.[10]

The most described ECG finding during the acute stage after SCI is sinus bradycardia with an incidence of 64–77%.[9]

Sympathetic denervation of blood vessels results in peripheral vasodilatation and hypotension (neurogenic shock). In our case, there were

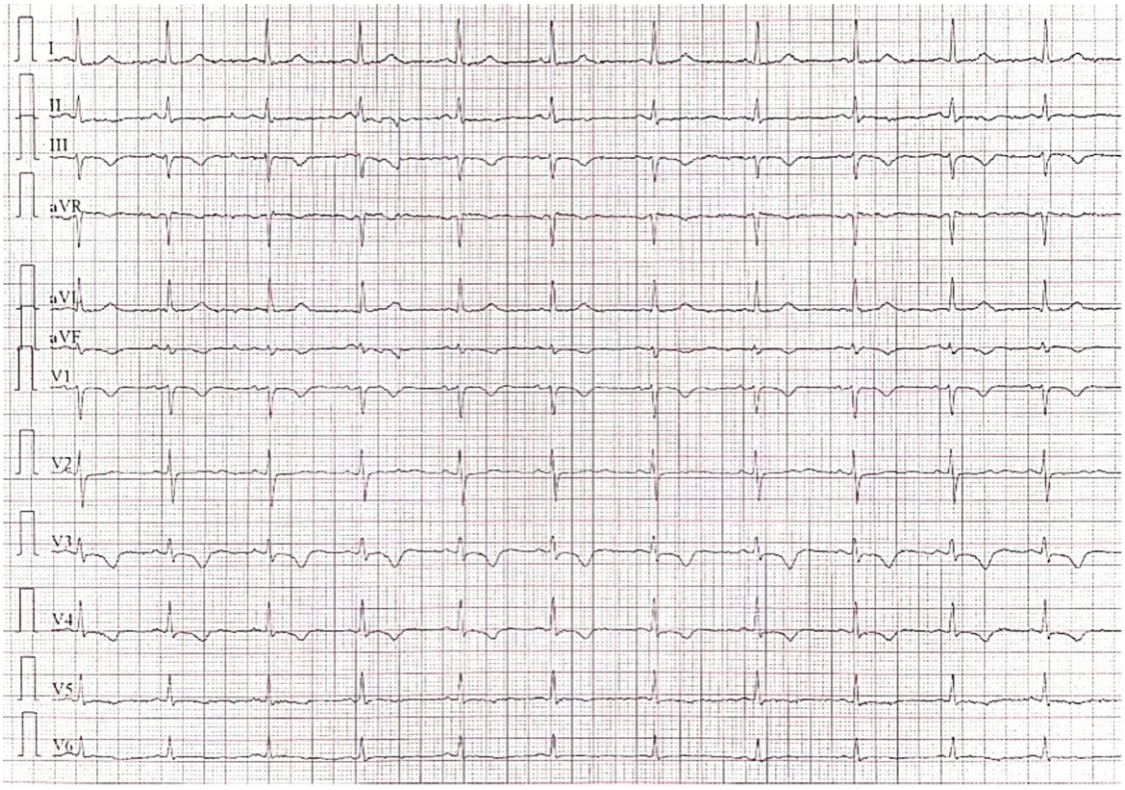


Figure 1 Twelve-lead electrocardiogram showing symmetrical T-wave inversion in leads III, avF, and V1–V4.

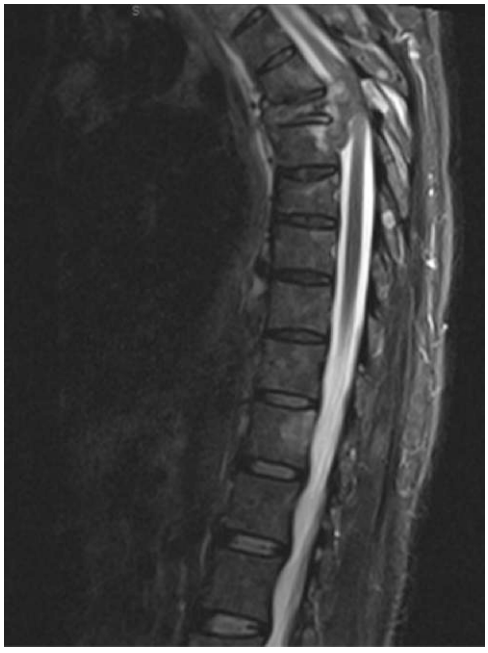


Figure 2 Magnetic resonance imaging spine sagittal view showing D5/D6 fracture with cord compression.

no significant blood pressure changes during the first few hours after diagnosis.

Lehmann *et al.*[3] proved that central sympathetic denervation in SCI is associated with myocardial repolarization changes, as patients with higher levels of complete injury showed ST-segment elevation in multiple leads that persisted with arm ergometry exercise but normalized on exogenous isoproterenol infusion. ST depression and inverted T waves were also reported in the acute phase independent of lesion level or severity. Furlan *et al.*[5] documented T-wave inversion in 59 of total 89 patients within 72 h of SCI. Long PR and QT interval were described in acute SC lesions above the level of T6.[4]

Patients also develop loss of sympathetic control of temperature, sweat regulation, cardiac pain sensation (in lesions above T4), and loss of ventricular muscle mass (denervation atrophy) in cervical lesions.

A neurogenic cause for acute ECG changes should be suspected in the presence of any neurological deficit, atypical chest pain, or normal echocardiography and laboratory workup. Careful history taking, chest pain analysis, and meticulous physical examination are important to reach a correct diagnosis. In our case, ECG changes preceded the occurrence of any neurological symptoms. However, chest tenderness was a key sign suggesting possible acute SCI.

Conclusion

ECG changes could be the earliest sign for ongoing neurological injury either in the brain or spinal cord. Misinterpretation of ischaemic-like ECG changes may lead to hazardous interventions or cause delay in

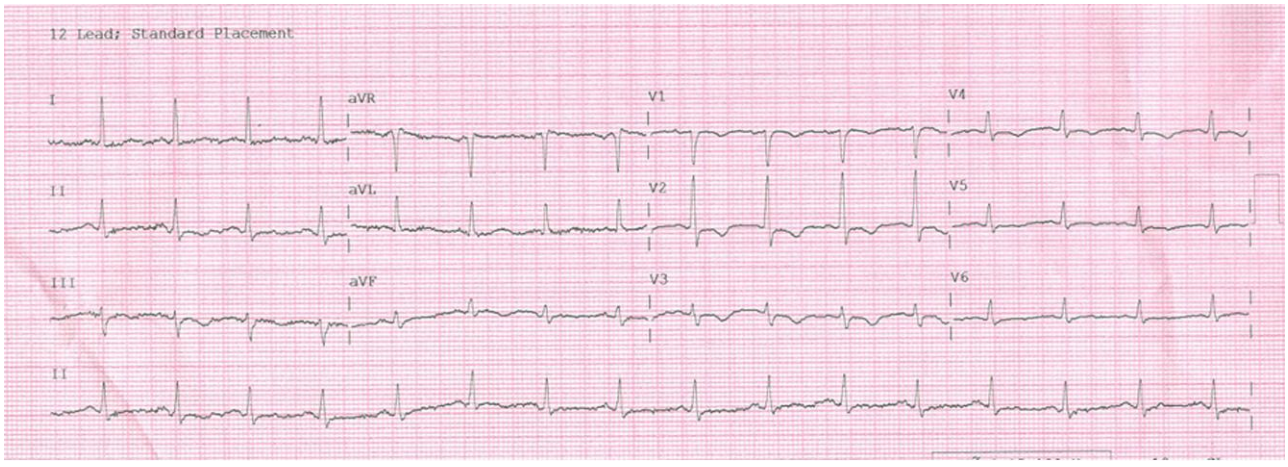


Figure 3 Twelve-lead electrocardiogram at follow-up after 3 months showing persistent symmetrical T-wave inversion in leads III, avF, and V1–V4.

reaching the correct diagnosis, especially, in the presence of atypical presentations. Our case highlights the importance of considering non-cardiac causes for ischaemic-like ECG changes, as early recognition and prompt management of acute neurological insult is crucial to prevent irreversible functional loss.

Lead author biography

Dr Hussein is an assistant lecturer and a PhD student of cardiovascular medicine at Kasr Al-Ainy Medical school, Cairo University. He is a member of the Royal College of Physicians of London. He obtained a Master's degree in cardiovascular medicine in 2019 from Cairo University, and currently holds the position of associate specialist in adult cardiology department at Aswan Heart Center. Dr Hussein has a special interest in the field of coronary and structural interventions.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient.

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Data availability

The data underlying this article are available in the article and in its online supplementary material.

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