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CASE REPORT

Autonomic dysreflexia and myocardial ischemia

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Study design: A case report of silent myocardial ischemia in a man with C5 AIS A (American Spinal Injury Association Impairment Scale) tetraplegia during an episode of autonomic dysreflexia (AD).
Objective: The aim of this study was to show a clinical presentation of myocardial ischemia in individuals with spinal cord injury (SCI).
Case report: A 45-year-old man with chronic C5 complete SCI presented with an episode of uncontrolled AD. He denied any symptoms of typical myocardial ischemia. Despite initiation of the AD management protocol, his blood pressure remained elevated. Additional testing revealed an unexpected horizontal ST depression in the lateral leads with a significant elevation of troponins. A follow-up cardiac angiography and MIBI cardiac perfusion scan revealed normal left ventricular contractility and no evidence of coronary artery occlusion.
Conclusions: In individuals with SCI, the loss of sensory input from the myocardium to supraspinal structures predisposes them to asymptomatic myocardial ischemia. Furthermore, during an episode of AD, a significant increase in visceral sympathetic activity with coronary artery constriction can result in myocardial ischemia, even in the absence of coronary artery disease.
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Keywords: spinal cord injury; myocardial ischemia; autonomic dysreflexia

Introduction

Cardiovascular disease is among the leading causes of morbidity and mortality in individuals with spinal cord injury (SCI).[1] Lack of exercise, decreased muscle mass and the development of metabolic syndrome are among the possible causes for predisposing them to having premature coronary artery disease (CAD).[2] Lee *et al.*[3] showed that up to 64% of clinically asymptomatic individuals with SCI developed myocardial ischemia during pharmacological stress testing. We present a case of silent myocardial ischemia during an episode of autonomic dysreflexia (AD).

Case report

A 45-year-old man with chronic cervical C5 AIS A (American Spinal Injury Association Impairment Scale) complete SCI presented to the Emergency Room with severe headaches, diaphoresis and palpitations. He denied chest pain or any other classic symptoms of myocardial ischemia. No cardiac risk factors unrelated to SCI were identified.

He was afebrile with a blood pressure of 220/105 mm Hg and a heart rate of 50 b.p.m. He had no pedal edema or jugular venous distension. His lungs were clear and heart sounds were normal. There was no abdominal distension or guarding. His chest and abdominal X-rays were unremarkable. His bladder was emptied with an intermittent catheter and had minimal residual volume; however, his urinalysis revealed a significant number of red blood cells and protein, with minimal leukocytes. The established AD management protocol was initiated, including nonpharmacological measures (raising the head of his bed, loosening restrictive devices and checking for bladder and bowel problems) and pharmacological measures (sublingual captopril 25 mg when systolic blood pressure remained over 150 mm Hg). Nevertheless, his blood pressure was persistently elevated and difficult to control.[4] Further investigations were thus sought, including an ECG that showed sinus bradycardia, a significantly shortened PR interval of 104 ms, and an unexpected horizontal ST depression in the V5 and V6 leads. Simultaneous cardiac enzymes showed an elevated troponin T of 0.48 $\mu g\,l^{-1}$ and a normal CK of 111 $U\,l^{-1}$. This was consistent with myocardial ischemia and standard antianginal therapy was initiated. After 48 h, cardiac angiography revealed normal left ventricular contractility and coronary arteries. Urine cultures eventually grew *Pseudomonas aeruginosa*, which likely caused a urinary tract infection and triggered this episode of AD. With normalization of his cardiac enzymes and initiation of antibiotics,

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Table 1 Typical clinical symptoms of myocardial ischemia in ambulatory and SCI population

Typical symptoms of myocardial ischemia	*Present case (2009)*	*Walker and Khokhar (1992)*[6]
Retrosternal chest discomfort	No	No
Radiation of pain to epigastrium, shoulders, arms, neck and throat, lower jaw and teeth or interscapular region	No	No
Shortness of breath	No	No
Nausea/vomiting	No	No
Diaphoresis	Yes	No
Lightheadedness	No	No
Fatigue	No	No

he was discharged home. After 8 months, a pharmacological stress test and cardiac perfusion scan showed no evidence of coronary artery occlusion.

Discussion

We report a case of silent myocardial ischemia in an individual with chronic cervical SCI that was associated with an episode of uncontrolled AD and no evidence of coronary artery occlusion. In the ambulatory population, myocardial ischemia typically presents as angina pectoris, a squeezing chest discomfort that often radiates to the epigastrium, shoulders, arms, neck and throat, lower jaw and teeth or interscapular region. This occurs when myocardial oxygen demand exceeds oxygen supply and is considered the cardinal symptom of myocardial ischemia. The localization of the discomfort is due to a referred pattern of sensation within the corresponding dermatomes that are innervated by the peripheral nerves originating from the same segments of the spinal cord as the segments that receive afferent sensory information from the heart. However, in individuals with SCI, anginal symptoms are frequently absent due to interruptions of the spinal sensory pathways and inability to perceive sensation from the body below the level of injury. Therefore, classic anginal symptoms in individuals with SCI are frequently absent and result in the development of silent cardiac ischemia. Silent myocardial ischemia occurs in ambulatory patients, as well.[5]

To our knowledge, only one case of silent cardiac ischemia has been previously reported in individuals with cervical SCI.[6] Our case, as well as the one described by Walker and Khokhar,[6] showed the absence of typical symptoms of angina pectoris (Table 1). However, our case has numerous distinct and unique aspects. First, in our case, the silent myocardial ischemia developed during an episode of AD. Although both individuals sustained cervical SCI, the individual in our case had a complete injury and was 20 years younger (45 vs 65 years old) than the individual described by Walker and Khokhar.[6] Furthermore, apart from his SCI, the individual in our case had an unremarkable medical history in contrast to the multiple cardiovascular risk factors in Walker's case, which include hypertension, a history of abnormal findings on ECG and a history of smoking for almost 50 years.[6] Finally, the most interesting aspect of our case was the absence of CAD, as established by follow-up angiography and pharmacological stress testing. These aspects show that his myocardial ischemia may have, in fact, been caused by significant cardiac vasculature constriction during his episode of severe AD.

AD is one of the most recognizable secondary complications of SCI and is commonly observed in individuals with high thoracic and cervical SCI. This condition is characterized by hypertension that can be triggered by various stimuli. The hypertension results from the activation of the spinal sympathetic circuits, increase in efferent sympathetic activity within the cutaneous musculature and visceral nerves and constriction of blood vessels in various vasculature beds below the level of SCI.[7] If the episode of AD is difficult to control and there is persistent coronary artery constriction, myocardial ischemia can occur, even in the absence of CAD.

Conclusion

There are no established guidelines for the management of CAD and cardiac risk factors in individuals with SCI. However, it is essential to perform a comprehensive evaluation and provide necessary treatment for patients with SCI and suspected CAD. Documentation and early recognition of AD should be included as part of the standard neurological assessment and management of individuals with SCI.[8] It is crucial that blood pressure be managed appropriately in these individuals and that hospitalization be considered in cases of uncontrolled AD.[4] Owing to the possibility of asymptomatic myocardial ischemia in patients with SCI, routine ECG's and troponin tests should be ordered on patients with SCI presenting with severe and difficult to control AD.

Conflict of interest

The authors declare no conflict of interest.

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