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Case Report

Contrast induced spinal myoclonus after percutaneous coronary intervention

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ABSTRACT

We present a case of a 77-year-old man diagnosed with contrast-induced spinal myoclonus following primary percutaneous coronary intervention. After being admitted with a diagnosis of anteroseptal myocardial infarction, he underwent primary percutaneous coronary intervention to the left anterior descending artery and was prescribed aspirin, clopidogrel, and intravenous heparin.

The following day he developed non-intentional irregular jerky movements confined to the truncal area. In view of rhythmic jerking confined to muscles innervated by a restricted segment of the spinal cord, resistance to supra-spinal influences and voluntary action, and no preceding electroencephalography activity in the contralateral sensorimotor cortex, a diagnosis of spinal myoclonus was made.

Spinal myoclonus is a rare entity in which myoclonic movements occur in muscles originating from few (segmental), or many adjacent spinal motor roots (propriospinal). Structural lesions are found in the majority of cases but the actual pathophysiology is still unknown. Contrast-induced spinal myoclonus is an even rarer phenomenon with few published reports. We describe postulated mechanisms and the management of this phenomenon.

<**Learning objective:** Myoclonus is a jerky movement due to abrupt involuntary contractions involving agonist and antagonist muscles. Spinal myoclonus is a rare disorder where myoclonic movements occur in muscles originating from spinal motor roots. The cause is usually a structural lesion, but in rare cases it can be induced by contrast. A video of this rare phenomenon is available with this article and the proposed pathophysiological mechanisms and treatment are discussed.>

Introduction

Myoclonus is defined as the sudden development of involuntary, irregular contractions of a group of muscles triggered by an event originating from different regions within the central nervous system. Spinal origin for myoclonus has been studied since 1881 when it was first reported by Friedreich [1]; however, it remains a rare phenomenon and is usually secondary to a structural lesion. Reports of contrast-induced spinal myoclonus are extremely rare, with the latest report published in 1989, and are more often secondary to contrast used in myelography and aortography. This is a report of a case of contrast-induced spinal myoclonus following primary percutaneous coronary intervention (PCI).

Case report

We present a case of a 77-year-old man diagnosed with contrast-induced spinal myoclonus following primary PCI.

He presented to the accident and emergency department with sudden onset retrosternal chest pain radiating to the left upper limb. He was known to suffer from non-insulin dependent diabetes, with poor glucose control, on metformin. There was otherwise no past medical or surgical history of note. He was an ex-smoker and non-drinker, and was independent in activities of daily living.

On examination, he was alert and hemodynamically stable. He was pale and sweaty but examination of the cardiovascular and respiratory systems was otherwise unremarkable. An electrocardiogram taken on admission to the accident and emergency department was consistent with an anteroseptal myocardial infarction. Initial biochemical investigations revealed a raised cardiac sensitive troponin.

He was admitted under the care of the cardiologists, and underwent primary PCI through a right radial approach. Iohexol

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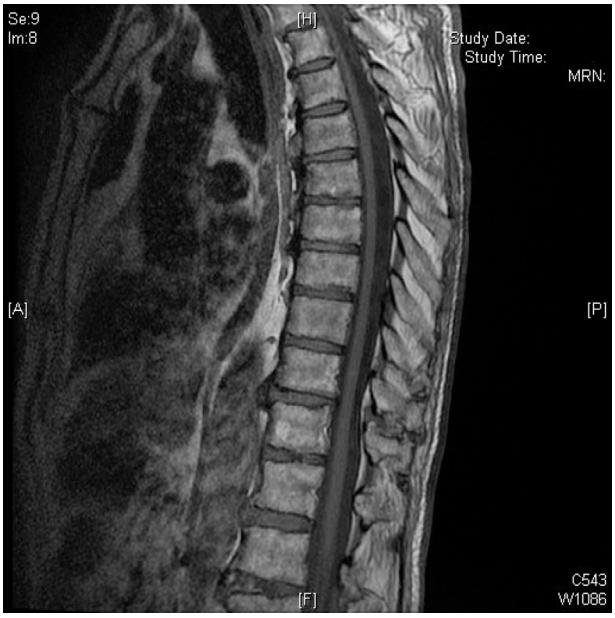

Fig. 1. MRI thoracic spine T1 sagittal view: T2/T3 and T10/T11 disc herniation.

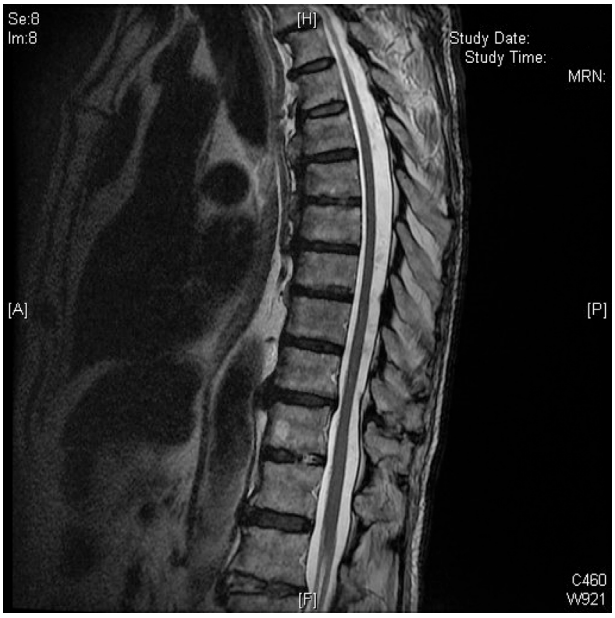

Fig. 3. MRI thoracic spine T2 sagittal view: T2/T3 and T10/T11 disc herniation.

(110 ml) contrast was used. Triple-vessel disease was identified and PCI to the culprit sub-occluded proximal left anterior descending (LAD) lesion was performed. The lesion was crossed with a balanced middle weight guide wire, pre-dilated with a 2.5 × 20 mm balloon and a 2.5 × 30 mm drug-eluting stent was deployed with good end result. The door to balloon time was under 30 min. The patient was transferred to the cardiac ward on aspirin, clopidogrel, and intravenous heparin, with a plan to continue dual antiplatelet therapy for one year.

On day one post-PCI, he was noted to have bilateral non-intentional jerky movements which were uncomfortable for the patient. A neurology consultation was requested. On examination, he was noted to have jerky movements confined to the truncal area. Such movements occurred at irregular intervals, lasting a few seconds and resolving spontaneously. There were approximately 3–5 episodes per minute and were present on movement and at rest, as well as during sleep. Consciousness was not disturbed in any way during episodes. Neurology examination of cranial nerves, upper and lower limbs was otherwise unremarkable. Investigations taken including complete blood count, renal and liver profiles, thyroid function tests, and an infective screen were within normal limits. An electroencephalogram (EEG) was also performed which was unremarkable. MRI of the spine showed degenerative changes but no other intraparenchymal spinal lesion [Figs. 1–3 and Video S1 in Appendix A].

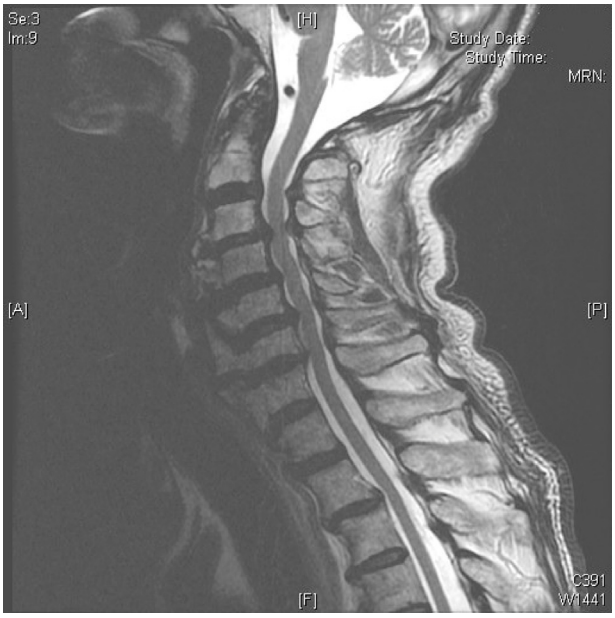

Fig. 2. MRI cervical spine T2 sagittal view: extensive spondylotic change, multilevel disc herniations.

The history and examination findings were consistent with myoclonus, specifically spinal myoclonus. Features consistent with spinal myoclonus are the rhythmic jerking confined to muscles innervated by a restricted segment of the spinal cord, resistance to supra-spinal influences, and voluntary action, and no preceding EEG activity in the contralateral sensorimotor cortex.

Discussion

Myoclonus is defined as a sudden, short-lasting, involuntary contraction involving agonist and antagonist muscles resulting in a jerky movement. Myoclonus is a symptom rather than a diagnosis [2]. It can be divided into positive myoclonus, which is caused by muscle contractions; and negative myoclonus, characterized by brief cessation of ongoing contraction [3]. It usually indicates disease of the central nervous system and is classified by neuroanatomical origin into cortical, subcortical, spinal, and peripheral myoclonus [4]. Cortical myoclonus arises from the sensorimotor cortex and is characterized by aberrant activity relayed down the corticospinal pathway leading to arrhythmic jerks. The origin of subcortical myoclonus is between the cortex and spinal cord, leading to jerky movements of the proximal limbs and axial muscles [5]. Spinal myoclonus is further divided into segmental and propriospinal.

Spinal myoclonus is a rare entity in which myoclonic movements occur in muscles originating from few (segmental), or many adjacent spinal motor roots (propriospinal). It is a reaction to a stimulus on a specific segment of the spinal cord [6]. The cause is

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