OMCR

OMCR 2014 10 (3 pages)
doi:10.1093/omcr/omu050

Case Report

But he has no chest pain. . .

Ioannis Merinopoulos* and David Bloore

Department of Cardiology, The Ipswich Hospital, Ipswich, UK

*Correspondence address. Department of Cardiology, The Ipswich Hospital, Heath Road, Ipswich IP4 5PD, UK. Tel: +44-7800601571; E-mail: ioannis.merinopoulos@doctors.org.uk

Received 3 August 2014; revised 29 September 2014; accepted 2 October 2014

Atypical presentations of acute coronary syndromes (ACS) are not uncommon and have been associated with higher mortality probably, because these patients are misdiagnosed and undertreated. They are most frequently encountered in older patients, women and in patients with diabetes, chronic renal failure or dementia. It is also well described in the literature that many chemotherapy agents are associated with myocardial ischaemic events. In addition to that, patients with cancer frequently receive large doses of opiate analgesics for chronic pain, which can obscure the symptoms of myocardial ischaemia. In this case report, we describe a patient who was receiving chemotherapy and large doses of opiate analgesics and presented with atypical symptoms for ACS. Our aim is to raise awareness of this challenging group of patients and the necessity to pay particular attention to symptoms other than chest pain as potential indicators of myocardial ischaemia.

INTRODUCTION

Atypical presentations of acute coronary syndrome (ACS) are not uncommon [1]. The Global Registry of Acute Coronary Events showed that patients with atypical presentations of ACS have greater morbidity and mortality probably because they are frequently misdiagnosed and undertreated [2]. Over the last few years, it has become well known that the cardiac side effects of chemotherapy are more frequent than what they were considered to be previously [3]. Patients with cancer receiving chemotherapy and potent analgesics represent a challenging group of patients to diagnose and treat. Many of the chemotherapy agents are associated with myocardial infarctions and the large doses of potent analgesics can obscure the symptoms of myocardial ischaemia. The aim of this case report is to raise awareness of the possible atypical presentations of these patients and the necessity to pay particular attention to symptoms other than chest pain as indicators of myocardial ischaemia.

CASE REPORT

We present the case of a 57-year-old gentleman who was admitted from the community with a 5-day history of worsening lower back pain. Nine months previously he had been diagnosed with advanced bladder transitional cell carcinoma (pT2G3N1) and treated with cystoprostatectomy. He was receiving adjuvant chemotherapy (gemcitabine and cisplatin) and his last cycle was 10 days prior to admission. His back pain had started 1 month previously after lifting a heavy object from the floor. He had been investigated with magnetic resonance imaging of his spine, which showed an acute biconcave benign fracture of L1 vertebra with a normal spinal canal. A bone scan at the time did not show any evidence of bone metastases and he was being managed with a spinal brace by the spinal team with good effect. The back pain was improving until 5 days prior to admission. It was worse on movement, without any radiation and there were no symptoms of cord compression. He denied any chest pain or discomfort at any point but complained of diaphoresis and mild breathlessness on exertion, which was initially attributed to the severe lower back pain. Apart from the bladder cancer, he did not have any other significant past medical history and there was no family history of ischaemic heart disease. He had stopped smoking 2 years ago after smoking 4–5 cigars per day for the last 30 years and that was his only cardiovascular risk factor. He was taking paracetamol 1 g QDS (four times per day), ibuprofen 400 mg TDS (three times per day), morphine sulphate 20 mg BD (twice a day) and diazepam 5 mg BD for his back pain. Clinical examination revealed normal

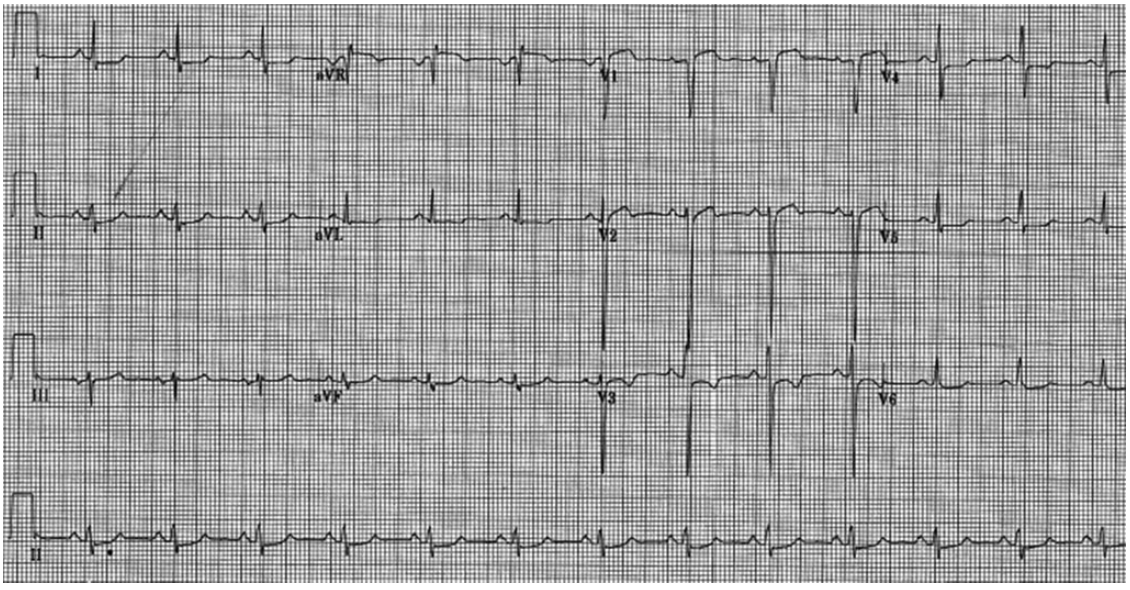

Figure 1: Electrocardiogram showing fixed ST elevation in V1–2 and anterolateral ST depression.

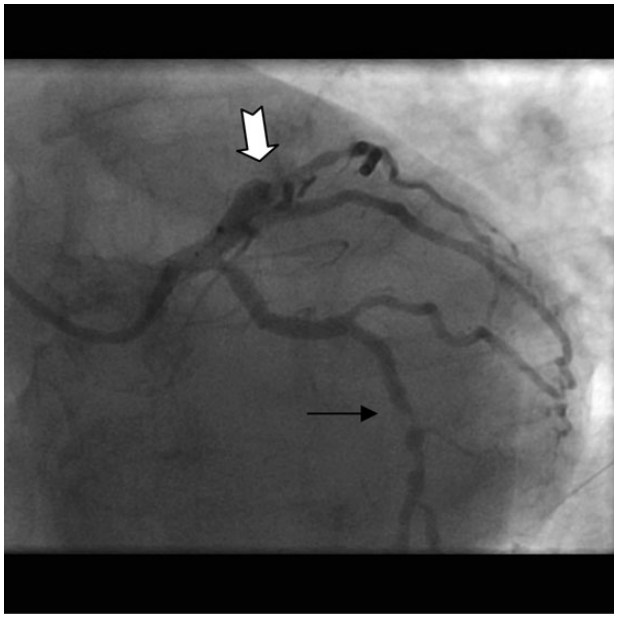

Figure 2: Angiography showing occluded LAD (white arrow) and severe lesion in the circumflex coronary artery (black arrow).

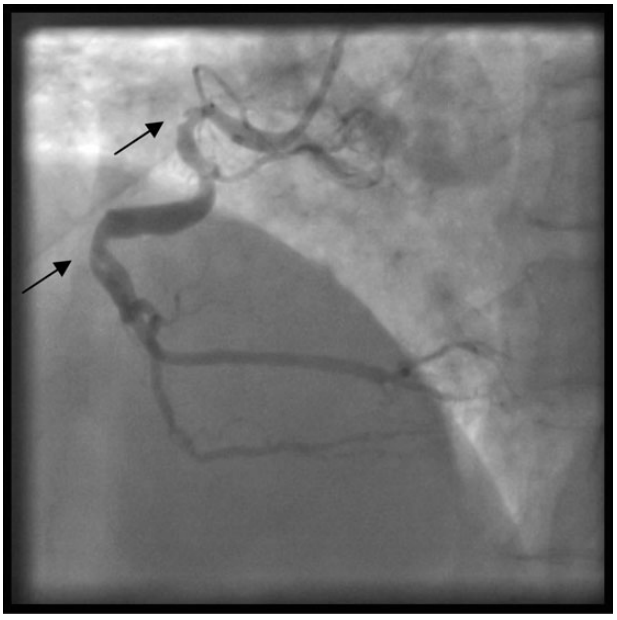

Figure 3: Angiography showing severe lesions in the right coronary artery (black arrows).

cardiac sounds, clear chest and normal neurology without any evidence of cord compression. His chest X-ray was clear and lumbosacral X-rays did not reveal a new fracture. However, his electrocardiogram (Fig. 1) showed fixed ST elevation in V1–2 and anterolateral ST depression. His troponin T was elevated at 1327 ng/l (normal <14) and he was started on aspirin, clopidogrel, fondaparinaux and secondary prevention including beta-blocker, ACE inhibitor and statin. The rest of his blood tests revealed Hb = 14.7 g/dl (normal 13.5–17.5 g/dl), WBC = 10.5×10^9/l (normal 4–11 $\times 10^9$/l), Plt = 139×10^9/l (normal 135–450 $\times 10^9$/l), Ur = 9.1 mmol/l (normal 2.5–8.5 mmol/l) and Cr = 105 μmol/l (normal 65–120 μmol/l). The next day he had another episode of diaphoresis while at rest with worsening ST depression laterally and so he was started on nitrate and tirofiban infusions, and plans were made for urgent transfer to the regional cardiothoracic centre. Coronary angiography showed an occluded left anterior descending (LAD) artery (Fig. 2, white arrow and Supplementary Video 1), severe lesions in the circumflex artery (Fig. 2, black arrow and Supplementary Video 1) and the right coronary artery (Fig. 3, black arrows) and collaterals from mid-circumflex to distal LAD. Following discussion with

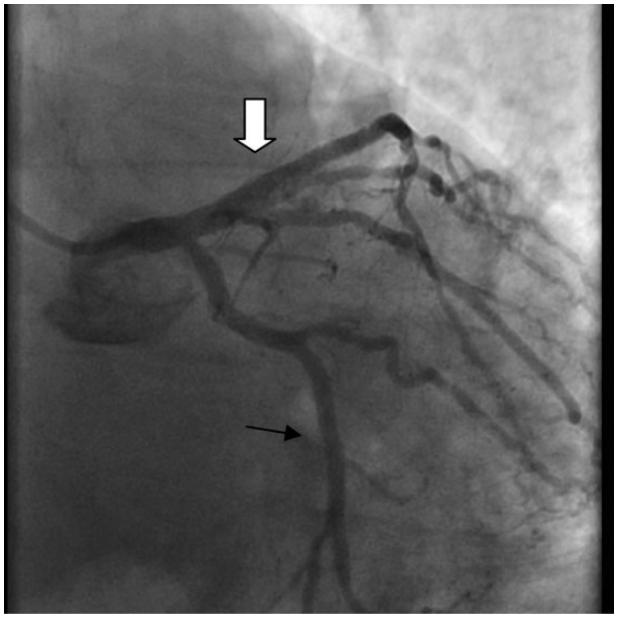

Figure 4: Excellent angiographic result post-angioplasty to the LAD (white arrow) and the circumflex coronary artery (black arrow).

cardiothoracic surgery, his LAD and circumflex arteries were successfully treated with drug-eluting stents. Opening the occluded LAD was challenging due to semi-organised thrombus throughout the mid-LAD. It required serial pre-dilatations, thrombus aspiration with a Pronto LP catheter which produced limited yield, administration of Abciximab (ReoPro) and serial doses of vasodilators before deployment of the 2.75 × 38 mm Xience drug-eluting stent from the mouth of the bifurcation with the diagonal to just above the second diagonal in the mid-vessel. The disease in the circumflex artery was treated with pre-dilatation and a further 2.5 × 33 mm Xience drug-eluting stent. The final angiographic result was excellent as shown in Fig. 4 and Supplementary Videos 2 and 3. Intervention to the right coronary artery lesion is planned as a staged procedure.

DISCUSSION

Atypical presentations of ACS are not uncommon and most frequently encountered in older patients (>75 years old), women and in patients with diabetes, chronic renal failure or dementia [1]. Our patient did not belong into the group of patients where atypical presentations of ACS are most frequently encountered. However, he was being treated with gemcitabine and cisplastin, which are associated with myocardial ischaemic events, and he was receiving opiate analgesics for his back pain, which might have obscured his symptoms. Patients with cancer receiving chemotherapy represent a challenging group of patients to diagnose and treat appropriately. It is well described in the literature that many of the chemotherapy agents can be associated with myocardial ischaemic events. The incidence rate of ischaemic events in relation to cisplatin is estimated at 1–5%, while Numico *et al*. documented two acute myocardial infarctions in a prospective trial of 108 patients with non-small-cell lung cancer treated with gemcitabine–cisplatin chemotherapy [3, 4]. In addition to that, it is not uncommon for these patients to receive large doses of potent analgesics for chronic pain, which can obscure myocardial ischaemic pain. It is therefore important to maintain a high index of suspicion for this group of patients and pay attention to associated symptoms such as dyspnoea, diaphoresis and nausea as potential indicators of myocardial ischaemia.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *Oxford Medical Case Reports* online.

CONFLICT OF INTEREST STATEMENT

None declared.

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