Post-myocardial infarction (Dressler's) syndrome following early reperfusion

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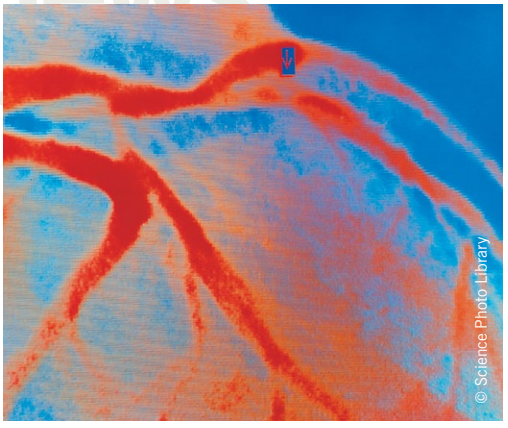
We present a case of a 55-year-old female with a successfully reperfused myocardial infarction in whom Dressler's syndrome was subsequently diagnosed. There have been no reported cases in the literature of Dressler's syndrome following documented early coronary reperfusion, and its continued existence in the era of reperfusion has been questioned. In conclusion, this case demonstrates that this syndrome is still a possibility in the current realm of thrombolysis and cardiac catheterisation.

Introduction

Dressler's, or post-cardiac injury, syndrome is a well-recognised complication of myocardial infarction;[1,2] however, following the introduction of thrombolysis and cardiac catheterisation its continued existence in the era of reperfusion has been questioned.[3] We present a case of Dressler's syndrome following a myocardial infarction that was successfully reperfused via primary catheterisation. This demonstrates that despite the advances in medical care Dressler's syndrome continues to exist and should be considered in the differential of anyone presenting with chest pain in the setting of a recent myocardial infarction.

Case presentation

A 55-year-old black female smoker with diabetes, hypertension and dyslipidaemia presented to the emergency room (ER) with acute chest pain of three hours' duration. Her electrocardiogram (ECG) was consistent with an acute inferior ST-segment elevation myocardial infarction. She was taken urgently to the catheterisation lab where she was found to have an occlusion in her right coronary artery. She was successfully stented and reperfused within one hour of her presentation to the hospital. Following loading doses of aspirin and clopidogrel in the ER she was started on metoprolol, captopril and atorvastatin.



Following the catheterisation, symptoms resolved and she remained chest-pain free. Her cardiac troponins peaked at 36.6 ng/ml, and she was discharged from the hospital within 48 hours. Her echocardiogram on discharge showed inferior hypokinesis with otherwise preserved left ventricular systolic function and no pericardial effusion.

One week following her discharge, she returned to the ER complaining of sharp non-exertional chest pain and dyspnoea persisting for one day. Following assessment she was discharged from the ER with up-titration of her anti-ischaemic medications. Three days later she came back to the hospital complaining of continued respirophasic chest pain with bilateral radiation to her shoulders and neck. This was associated with dyspnoea and cough. She also noted anorexia, weakness and chills. There was no documented fever. Her vital signs were as follows: heart rate 91 beats per minute, blood pressure 128/86 mmHg, respiratory rate 22 breaths per minute, temperature 98.2 degrees Fahrenheit and oxygen saturation of 98% on 2 L oxygen via nasal cannula. On examination, she was uncomfortable and in mild respiratory distress. Her cardiac examination did not reveal any murmurs or rubs, and there was no jugular venous distension or lower extremity oedema. She was noted to have bilateral inspiratory rales and was hypoxic on room air. Her ECG was notable for old Q waves in the

inferior leads, but showed no acute ischaemic changes or persistent ST segment elevation. Lab tests were notable for a leukocytosis of 13 x $10^3/\mu$L with left shift and an elevated B-natriuretic peptide of 3,760 pg/ml. Her chest X-ray demonstrated cardiomegaly with pulmonary vascular congestion and bibasal pleural effusions. A computed tomography of her chest revealed cardiomegaly, small bilateral pleural effusions, bibasal atelectasis and a small pericardial effusion. She was admitted to the cardiac service and diuresed. Her symptoms of chest pain and dyspnoea remained unchanged the next day without any ECG changes or cardiac enzyme elevations. Repeat echocardiography showed mild left ventricular hypertrophy with preserved systolic function, the previously noted inferior hypokinesis and a new moderate pericardial effusion. An erythrocyte sedimentation rate (ESR) was checked and found to be elevated at 106 mm/h. She was given the presumptive diagnosis of Dressler's syndrome and started on naproxen.

Within the next 24 hours there was total resolution of her symptoms and she was discharged from the hospital the next day. She was seen in clinic five weeks later, where she noted no recurrence of her pericardial symptoms and a repeat echocardiogram showed resolution of the pericardial effusion.

Discussion

Post-myocardial infarction (Dressler's) syndrome was initially described by Dressler in 1956.[1] Essentially a clinical diagnosis, it is characterised by the presence of pleuro-pericardial chest pain, low grade fever, pericardial friction rub, elevated ESR, and pericardial/pleural effusions following a myocardial infarction (or some other form of cardiac insult). It is differentiated from the acute pericarditis following a myocardial infarction (peri-infarction pericarditis) by the presence of a latency period that is characteristically weeks to months, although it has been documented to present as early as 24 hours following the infarct.[4] While the exact mechanism has yet to be elucidated, it has been postulated to be an immune mechanism given the presence of the latency period, its correlation to actin and myosin antibody titres[5] and its excellent response to anti-inflammatory agents.[6] Treatment usually consists of high-dose non-steroidal anti-inflammatory drugs (NSAIDs) or steroids. Prognosis is usually benign, and mostly dependent on the severity of the underlying coronary artery disease.

While older studies measuring the incidence of Dressler's syndrome found conflicting results,[7-10] there has been a relative absence of the syndrome in recent decades. In a prospective study of 201 patients with acute myocardial infarctions who received thrombolysis, Shahar *et al.*[3] diagnosed only one patient with Dressler's syndrome in the three-month follow-up period. Of note, this was a patient that did not demonstrate any clinical evidence of early reperfusion. The authors postulated that by reducing infarct size and, hence, exposure of cardiac antigens, reperfusion was primarily responsible for the disappearance of this syndrome. Bendjelid *et al.*[11] noted that, despite the widespread introduction of thrombolysis and cardiac catheterisation, a significant number of patients continue to develop large infarcts secondary to unsuccessful or late reperfusion, and suggested that the recent introduction of novel therapies with immuno-modulatory properties, namely angiotensin-converting enzyme (ACE) inhibitors, statins and beta blockers, were at least partly accountable. While the exact mechanism is not yet clear, there seems to be a general consensus that, in the era of reperfusion, Dressler's syndrome has become virtually absent.

To date, there has been no reported case of Dressler's syndrome following documented early reperfusion of a myocardial infarction. The presented case demonstrates that early reperfusion does not prevent this syndrome from occurring. In addition, since the patient received the standard anti-ischaemic medications (including ACE inhibitors, statins and beta blockers) the immuno-modulatory actions of these drugs are not completely protective. Essentially, this case serves as a reminder that Dressler's syndrome is a potential complication following a myocardial infarction, even in the era of reperfusion and with recent advances in medical care. Whether this syndrome is indeed on the verge of disappearing or it is simply being under-diagnosed, given lack of clinical suspicion, is a question that remains to be answered

Conflict of interest

None declared.

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