CASE REPORT

The return of a disappearing entity: Dressler's syndrome after transvenous pacemaker implantation

António Tralhão, Diogo Cavaco, Marisa Trabulo, António M Ferreira

Department of Cardiology,
Hospital de Santa Cruz, Centro
Hospitalar de Lisboa Ocidental,
Carnaxide, Portugal

Correspondence to

Dr António Miguel Ferreira,
miguelferreira.md@sapo.pt

Accepted 9 February 2014

SUMMARY

A 76-year-old woman with idiopathic dilated cardiomyopathy and left bundle branch block was admitted for biventricular pacemaker implantation. The procedure was complicated by perforation of a coronary sinus tributary vein and ensuing pericardial effusion without tamponade. Three months later, she presented with fever, pleuritic chest pain and functional class worsening. Pericardial and pleural effusions were noted. Sterile blood and pleural fluid cultures and failure to respond to antibiotic therapy led to the hypothesis of a Dressler-like syndrome. The patient improved clinically with a short course of steroid therapy and was discharged on colchicine. A chest X-ray performed after 2 months showed a complete remission of pleural effusion.

BACKGROUND

Dressler's syndrome was originally described after acute myocardial infarction (MI) and its incidence seems to be declining, owing to modern reperfusion modalities.¹ Alternative pericardial insults can, however, generate a similar clinical picture, making the diagnosis less obvious and delaying a potentially effective treatment. This case report is noteworthy for the greater than expected delay in occurrence and emphasises the possibility of such a complication, in the context of an increasingly growing number of endovascular procedures.

CASE PRESENTATION

A 76-year-old woman with idiopathic dilated cardiomyopathy and a history of hypertension and tuberculosis in youth was admitted for biventricular pacemaker implantation (CRT-P). She had severe left ventricular systolic dysfunction, left bundle branch block and experienced New York Heart Association Class (NYHA) III symptoms despite optimised medical therapy. After placement of right atrial and right ventricular leads, a coronary sinus tributary vein was accidentally punctured and a self-limited mild pericardial effusion followed, precluding placement of the left ventricular lead. The patient was discharged on the following day and the completion of the procedure was deferred.

Three months later, she presented to the local emergency department with fever, malaise, left pleuritic chest pain and worsening functional class in the previous week. The patient was febrile (39.3°C), tachypnoeic and in distress, but vital signs were otherwise unremarkable. The remaining physical examination revealed muffled heart sounds and abolished breath sounds in the left lower thorax,

together with dullness to percussion and increased vocal vibrations, consistent with a pleural effusion (figure 1). There was no jugular vein ingurgitation or ankle oedema. Laboratory workup was significant only for elevated acute phase reactants (mild leucocytosis and C reactive protein of 19 mg/dL). Cardiac troponin I was negative, and the 12-lead ECG showed no signs of acute pericarditis (figure 2).

Transthoracic echocardiogram revealed a moderate, anechoic pericardial effusion (diastolic diameter 17 mm), without cardiac chamber collapse. The patient was transferred to a medical ward and given levofloxacin assuming a diagnosis of respiratory tract infection.

INVESTIGATIONS

A thoracic CT (figure 3) confirmed the presence of a large left-sided pleural effusion and a moderate, circumferential pericardial effusion. Therapeutic and diagnostic thoracentesis was performed, yielding approximately 1500 mL of a yellowish exudate (by Light's criteria). Blood and pleural fluid cultures were negative. One week after admission and despite apyrexia, clinical improvement and marked reduction of the pericardial effusion (video 1), left-sided pleural effusion quickly recurred and C reactive protein remained elevated.

The earlier occurrence of pericardial effusion following pacemaker lead insertion suggested the diagnosis of a Dressler-like syndrome, even though the temporal hiatus seemed longer than expected. Antibiotics were stopped and the patient was

Figure 1 Admission chest X-ray revealing cardiomegaly and a left-sided pleural effusion.

CrossMark

To cite: Tralhão A, Cavaco D, Trabulo M, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-203401

Figure 2 Admission ECG showing sinus rhythm and left bundle branch block with widened QRS.

started on a short course of oral prednisolone 40 mg/day, combined with colchicine 1 mg/day. In the following week, clinical status continued to improve and C reactive protein decreased. Left-sided pleural effusion showed no signs of improvement, but as the patient remained clinically well, she was discharged on colchicine 3 weeks after being admitted and was referred to the outpatient clinic for follow-up.

OUTCOME AND FOLLOW-UP

A chest X-ray performed 2 months after discharge (figure 4) showed a complete remission of pleural effusion. She was rescheduled for left ventricular lead placement 3 months later, which took place uneventfully. Her functional class subsequently improved to NYHA II.

DISCUSSION

Dressler's syndrome was reported in 1956 as a benign triad of fever, pericarditis and pericardial effusion post-MI.² More recently, this and other forms of pericardial lesions have been included in the spectrum of 'postcardiac injury' syndromes, namely postpericardiotomy (after cardiac surgery) and post-traumatic, whether iatrogenic (after coronary angioplasty or postarrhythmia ablation) or purely traumatic due to blunt or penetrating trauma.³

Regardless of the specific type of pericardial insult, these syndromes share a common clinical presentation, ranging from Dressler's pericarditis to more complicated cases with pleuropericarditis and pleural effusion. Albeit rare, cardiac tamponade⁴ or massive pleural effusion can occur.^{3 5} Even more rare is the distant evolution to constrictive pericarditis.⁶

Figure 3 Chest CT showing a large left-sided pleural effusion (arrow), mild right-sided pleural effusion and a moderate pericardial effusion (*).

Video 1 Transthoracic echocardiogram (parasternal short-axis view, at midpapillary muscle level) performed one week after admission, showing severe systolic dysfunction of the left ventricle, remission of pericardial effusion and persistence of a left-sided pleural effusion.

Fever and pleuritic chest pain (that can simulate acute MI) and elevated acute-phase reactants are the usual findings. Physical examination can disclose a pericardial friction rub and signs of pleural effusion. Chest X-ray and transthoracic echocardiography are the preferred imaging modalities. A distinctive but often forgotten feature is a latency time between the original insult and disease onset, typically between weeks and months.⁷

The most accepted pathogenic mechanism is an inappropriate immune system reaction after the pericardial aggression, allowing the exposure of myocardial neoantigens and the development of a delayed inflammatory response of varying extension and magnitude.^{6 8} The presence of overt pericardial effusion is not a requirement, but occult microtrauma is presumed to occur.³

The incidence of postcardiac injury syndromes differs according to the type of insult and is reported to be inferior to 1% post-MI and between 10% and 40% after cardiac surgery.^{1 3} Dressler's syndrome is infrequent nowadays, due to the reduction in transmural necrosis associated with reperfusion therapies and infarct size limitation. However, postcardiac injury syndromes are not uncommon, owing to the increasingly growing number of endovascular procedures, including pacemaker implantations.^{1 3 9} The exact incidence, seemingly low and under-reported, is unknown. According to Cevik *et al*, only nine cases have been reported following pacemaker implantation, between 1975 and 2009.

Figure 4 Chest X-ray performed two months after discharge showing complete remission of left-sided pleural effusion.

Symptoms appeared 5–56 days after the procedure and all had pleuropericardial involvement. Six cases improved with medical therapy and three required a pericardial window.¹⁰

Non-steroid anti-inflammatory agents such as aspirin and ibuprofen (or alternatively colchicine) can be used, during 3–6 weeks and gradually tapered after clinical improvement. Corticosteroids (eg, prednisolone) are usually reserved for failure of first-line treatment. Recurrence is possible and colchicine can be given prophylactically.^{3 11} Response to treatment can also be considered indicative of a correct diagnosis.³ A long-term prognosis is favourable, but long-term follow-up is advisable due to the intermediate risk of constrictive pericarditis.³

Learning points

- ▶ Although uncommon, physicians should consider the possibility of postcardiac injury syndrome following transvenous pacemaker insertion.
- ▶ Diagnosis is often unclear and can be delayed due to other confounding entities and late presentation.
- ▶ Prognosis is usually benign, and therapeutic response to anti-inflammatory drugs can aid in the diagnosis.

Contributors AT was responsible for medical data acquisition and drafting of the manuscript. DC, MT and AMF revised the manuscript for important intellectual content. All authors gave final approval of the version to be published.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Bendjelid K, Pugin J. Is Dressler syndrome dead? *Chest* 2004;126:1680–2.
- 2 Dressler W. A post-myocardial infarction syndrome; preliminary report of a complication resembling idiopathic, recurrent, benign pericarditis. *JAMA* 1956;160:1379–83.
- 3 Imazio M, Hoit BD. Post-cardiac injury syndromes. An emerging cause of pericardial diseases. *Int J Cardiol* 2013;168:648–52.
- 4 Hertzeanu H, Almog C, Algom M. Cardiac tamponade in Dressler's syndrome. Case report. *Cardiology* 1983;70:31–6.
- 5 Dombey WR, Whitcomb ME. Pleural effusion as a manifestation of Dressler's syndrome in the distant post-infection period. *Am Heart J* 1978;96:243–5.
- 6 Beaufils P, Bardet J, Temkine J, et al. Dressler's syndrome: constrictive pericarditis following myocardial infarction operated on with success. *Arch Mal Coeur Vaiss* 1975;68:651–6.
- 7 Wessman DE, Stafford CM. The postcardiac injury syndrome: case report and review of the literature. *South Med J* 2006;99:309–14.
- 8 Bartels C, Honig R, Burger G, et al. The significance of anticardiolipin antibodies and anti-heart muscle antibodies for the diagnosis of postpericardiotomy syndrome. *Eur Heart J* 1994;15:1494–9.
- 9 Shahar A, Hod H, Barabash GM, et al. Disappearance of a syndrome: Dressler's syndrome in the era of thrombolysis. *Cardiology* 1994;85:255–8.
- 10 Cevik C, Wilborn T, Corona R, et al. Post-cardiac injury syndrome following transvenous pacemaker insertion: a case report and review of the literature. *Heart Lung Circ* 2009;18:379–83.
- 11 Vinit J, Sagnol P, Buttard P, et al. Recurrent delayed pericarditis after pacemaker implantation: a post-pericardiotomy-like syndrome? *Rev Med Interne* 2007;28:137–40.

Copyright 2014 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit

<http://group.bmj.com/group/rights-licensing/permissions>.

BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow