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CASE REPORT
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Biphasic pericardial effusion following the implantation of a pacemaker

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The case is reported of a patient who developed pericardial effusion, both early, at 24 hours, and late, at 3 weeks, after the implantation of a pacemaker. The late effusion was accompanied by manifestations of pericarditis. Pericarditis occurring a few weeks after the insertion of a pacemaker is a variant of the post-cardiac injury syndrome (PCIS). This form of pericarditis, which is considered to be autoimmune in origin, was originally described following myocardial infarction and cardiac surgery. In these conditions the incidence of the PCIS is increased among patients with an early pericardial reaction, which is considered traumatic in origin and occurs from hours to a few days after the precipitating event. Although both early and late forms of pericarditis have been documented following the insertion of a pacemaker, no report was found of early and late pericardial reaction occurring sequentially in the same patient. The course of this patient supports the hypothesis that, in agreement with the other forms of the PCIS, there may be a connection between the early and late forms of pericardial reaction following the implantation of a pacemaker.

Διφασική περικαρδιακή συλλογή μετά από την τοποθέτηση βηματοδότη

Περίληψη στο τέλος του άρθρου

Key words

Biphasic pericarditis
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Over the last 60 years, a mainly clinical entity has been described, which is known under the general term post-cardiac injury syndrome (PCIS). It was first reported in 1952, as the post-commissurotomy (valvulotomy for mitral stenosis) syndrome, and a few years later as the post-myocardial infarction syndrome, by William Dressler.[1–4] Virtually, identical syndromes have been described following open heart surgery, thoracic trauma, cardiac catheterization and pacemaker insertion.[5–12] The common denominator is thought to be a traumatic event to the myocardium or pericardium, which appears to be the triggering factor. This is usually followed by a latent period from a few weeks to a few months, after which the syndrome presents. The recent history, latent period, constitutional symptoms (fever, chest pain due to serositis, and occasionally arthralgias and pulmonary infiltrates), markers of inflammation (raised ESR, CRP and WBC) and response to glucocorticoids, appear to connect this condition with the various triggering events and to support its inflammatory nature. The increased frequency of late-onset pericarditis among post-cardiac surgery patients who developed an early pericardial effusion also supports the continuity between the triggering event and the inflammatory reaction.[5] This is the report of a patient who, following the implantation of a pacemaker, developed both an early, at 24 hours, and a late, at 3 weeks, pericardial effusion, supporting the existence of a similar relationship in this variant of the syndrome.

CASE PRESENTATION

An 86-year-old man was admitted with a 20-day history of fatigue and sub-sternal pleuritic chest pain, which had worsened over the previous two days. A pacemaker with a single lead had been implanted in the right ventricle 6 weeks earlier (VVI-R, Biotronik®) in a referral cardiac surgery/cardiology center because of symptomatic (episodes of presyncope) atrioventricular block. He had been transferred to the intensive care unit (ICU) of the same hospital 24 hours after the implantation for a 48 hour period of observation, following an episode of hypotension and the appearance of a small pericardial effusion without signs of tamponade, attributed to possible bleeding within the pericardium. He was eventually discharged after 7 days when reduction of the pericardial fluid was documented. His other known medical problems were hypertension, type II diabetes mellitus, left common carotid artery endarterectomy 15 years earlier and factor XII deficiency. On examination, a pericardial friction rub was heard at the left sternal border. Laboratory findings showed elevated markers of inflam-

mation (ESR: 107–135 mm/h and CRP: 26.9–36.6 mg/dL, with an upper limit of normal 0.8 mg/dL). Cardiac ultrasound (US) showed a moderate amount of pericardial fluid. He was treated with aspirin (3 g per day in divided doses, gradually tapered off over the course of 6 weeks). The chest pain subsided and a gradual reduction in the amount of pericardial fluid was observed on repeated US. A rapid fall in CRP was elicited, to 10.8 mg/dL at one week, 2.83 mg/dL at 4 weeks and 0.9 mg/dL at 6 weeks, and a more gradual decline in the ESR to 100 mm/h at one week, 67 mm/h at 4 weeks and 25 mm/h at 6 weeks following the start of salicylate therapy.

DISCUSSION

Reports of pericarditis following the first cardio-surgical procedures of mitral valvulotomy for mitral valve stenosis in the early 1950s represent the first description of a series of kindred syndromes referred to as PCIS.[1,2] Similar syndromes were subsequently described after myocardial infarction, post-pericardiotomy, following non/or penetrating chest wounds, post-radiofrequency ablation and post-intracardiac lead insertion.[3–12] The post-operative appearance of anti-heart antibodies in cardiac surgery patients supported the hypothesis that this is an inflammatory reaction to cardiac muscle products released during the various procedures or injuries.[13–15] Alternative evidence suggests that the appearance of antibodies may be an epiphenomenon and that the syndrome, similar in many aspects to "idiopathic pericarditis", may reflect a viral infection introduced during the various precipitating events.[16] Whatever the evidence for the pathophysiological mechanism, the various reports of the syndrome appear to have in common a history of recent cardiac injury, a similar clinical picture, laboratory markers of inflammation, a mostly benign course and a similar response to treatment.

The evidence that supports a cause and effect relationship, leading to an inflammatory reaction, apart from the recent history, is the increased frequency of PCIS among patients with early evidence of cardiac trauma, such as a pericardial effusion, in the first few days after the precipitating event. This has been documented in a series of patients following cardiac surgery, where 83% of the pericardial effusions were present by the second post-operative day, preceding the appearance of PCIS-related symptoms.[5] Ninety percent of the early effusions were not echo-free, a finding consistent with the presence of serosanguinous fluid or clotted blood, indicating a traumatic etiology.

The first recorded case of appearance of fever and pleuritic chest pain 3 weeks after transvenous pacemaker implantation was described in 1975.[9] There have been subsequent reports of early pericardial effusion appearing a few days after the insertion of a pacemaker, and several reports of PCIS occurring several weeks later.[9–12,17–19] That this may be a biphasic phenomenon with an early traumatic and a late inflammatory component is not always clear, since most reports are retrospective and tend to attribute all effusions to PCIS. In support of the presence of a traumatic component is the fact that the majority of cases PCIS are associated with both an atrial component (related to thinner wall of the atrium) and screw-in active fixation of the lead, presumably more traumatic than other types of fixation.[12,17] In this respect, the patient presented here is unusual, since neither active fixation nor an atrial lead was used (he had a single lead placed in the right ventricle). To the authors' knowledge, there have been no reports of both an early and late component of pericardial reaction in the same patient following pacemaker implantation. This report describes a patient who, following the insertion of a pacemaker, developed both an early (at 24 hours) and a late (at 3 weeks) pericardial effusion, with constitutional signs, supporting the notion of a cause and effect relationship between the two episodes of pericardial reaction in this variant of the syndrome, also.

ΠΕΡΙΛΗΨΗ

Διφασική περικαρδιακή συλλογή μετά από την τοποθέτηση βηματοδότη

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Περιγράφεται ασθενής που εμφάνισε τόσο πρώιμη, δηλαδή εντός 24 ωρών, όσο και όψιμη, δηλαδή μετά από 3 εβδομάδες, περικαρδιακή συλλογή, μετά από την τοποθέτηση βηματοδότη. Η όψιμη περικαρδιακή συλλογή συνοδευόταν από τα τυπικά συμπτώματα της περικαρδίτιδας. Η περικαρδίτιδα, που συνήθως εμφανίζεται εβδομάδες μετά από την εμφύτευση βηματοδότη, θεωρείται ως παραλλαγή του συνδρόμου μετά από θωρακοτομή. Αυτή η μορφή της περι-

καρδίτιδας, που θεωρείται αυτοάνοσης αρχής, περιγράφηκε αρχικά μετά από έμφραγμα μυοκαρδίου και καρδιοχειρουργική επέμβαση. Στις τελευταίες περιπτώσεις, η επίπτωση του συνδρόμου μετά από θωρακοτομή είναι αυξημένη, με την εμφάνιση μιας πρώιμης φάσης περικαρδιακής αντίδρασης, που θεωρείται τραυματικής αρχής και συμβαίνει λίγες ώρες έως λίγες ημέρες μετά από την τοποθέτηση του βηματοδότη. Παρ' όλο που υπάρχουν αρκετές αναφορές στη βιβλιογραφία σχετικά με την ύπαρξη και των δύο φάσεων μετά από τοποθέτηση βηματοδότη, δεν βρήκαμε κάποια αναφορά για την εμφάνιση και των δύο φάσεων της περικαρδιακής συλλογής στον ίδιο ασθενή. Η περίπτωση του ασθενούς μας ενισχύει την υπόθεση ότι πρέπει να υπάρχει σχέση ανάμεσα στην πρώιμη και την όψιμη περικαρδιακή αντίδραση, η οποία παρατηρείται μετά από την τοποθέτηση βηματοδότη.

Λέξεις ευρετηρίου: Βηματοδότης, Διφασική περικαρδίτιδα, Πρώιμη και όψιμη περικαρδιακή αντίδραση

References

1. JANTON OH, GLOVER RP, O'NEILL TJ, GREGORY JE, FROIO GF. Results of the surgical treatment for mitral stenosis; analysis of one hundred consecutive cases. *Circulation* 1952, 6:321–333
2. SOLOFF LA, ZATUCHNI J, JANTON OH, O'NEIL TJ, GLOVER RP. Reactivation of rheumatic fever following mitral commissurotomy. *Circulation* 1953, 8:481–497
3. DRESSLER W. A post-myocardial infarction syndrome; preliminary report of a complication resembling idiopathic, recurrent, benign pericarditis. *J Am Med Assoc* 1956, 160:1379–1383
4. DRESSLER W. The post-myocardial-infarction syndrome: A report on forty-four cases. *AMA Arch Intern Med* 1959, 103:28–42
5. DRUSIN LM, ENGLE MA, HAGSTROM JW, SCHWARTZ MS. The postpericardiotomy syndrome: A six-year epidemiologic study. *N Engl J Med* 1965, 272:597–602
6. GOODKIND MJ, BLOOMER WE, GOODYER AV. Recurrent pericardial effusion after non-penetrating chest trauma: Report of two cases treated with adrenocortical steroids. *N Engl J Med* 1960, 263:874–881
7. SEGAL F, TABATZINK B. Postpericardiotomy syndrome following penetrating stab wounds of the chest: Comparison with the postcommissurotomy syndrome. *Am Heart J* 1960, 59:175–183
8. WESSMAN DE, STAFFORD CM. The postcardiac injury syndrome: Case report and review of the literature. *South Med J* 2006, 99:309–314
9. KAYE D, FRANKL W, ARDITI LI. Probable postcardiotomy syndrome following implantation of a transvenous pacemaker: Report of the first case. *Am Heart J* 1975, 90:627–630
10. SPINDLER M, BURROWS G, KOWALLIK P, ERTL G, VOELKER W. Postpericardiotomy syndrome and cardiac tamponade as a late complication after pacemaker implantation. *Pacing Clin Electrophysiol* 2001, 24:1433–1434
11. ELLENBOGEN KA, WOOD MA, SHEPARD RK. Delayed complications following pacemaker implantation. *Pacing Clin Electrophysiol* 2002, 25:1155–1158
12. LEVY Y, SHOVMAN O, GRANIT C, LURIA D, GUREVITZ O, BAR-LEV D ET AL. Pericarditis following permanent pacemaker insertion. *Isr Med Assoc J* 2004, 6:599–602
13. KHAN AH. The postcardiac injury syndromes. *Clin Cardiol* 1992, 15:67–72
14. DE SCHEERDER I, DE BUYZERE M, ROBBRECHT M, DE LANGE M, DELANGHE J, BOGAERT AM ET AL. Postoperative immunologic response against contractile proteins after coronary bypass surgery. *Br Heart J* 1986, 56:440–444
15. NOMURA Y, YOSHINAGA M, HARAGUCHI T, OKU S, NODA T, MIYATA K ET AL. Relationship between the degree of injury at operation and the change in antimyosin antibody titer in the postpericardiotomy syndrome. *Pediatr Cardiol* 1994, 15:116–120
16. ENGLE MA, ZABRISKIE JB, SENTERFIT LB, GAY WA Jr, O'LOUGHLIN JE Jr, EHLERS KH. Viral illness and the postpericardiotomy syndrome. A prospective study in children. *Circulation* 1980, 62:1151–1158
17. SIVAKUMARAN S, IRWIN ME, GULAMHUSEIN SS, SENARATNE MP. Postpacemaker implant pericarditis: Incidence and outcomes with active-fixation leads. *Pacing Clin Electrophysiol* 2002, 25:833–837
18. OHLOW MA, LAUER B, BRUNELLI M, GELLER JC. Incidence and predictors of pericardial effusion after permanent heart rhythm device implantation: Prospective evaluation of 968 consecutive patients. *Circ J* 2013, 77:975–981
19. ELMOUCHI DA, ROSEMA S, VANOOSTERHOUT SM, KHAN M, DAVIS AT, GAURI AJ ET AL. Cardiac perforation and lead dislodgement after implantation of a MR-conditional pacing lead: A single-center experience. *Pacing Clin Electrophysiol* 2014, 37:4–10

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