Biphasic pericardial effusion following the implantation of a pacemaker

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.

CASE PRESENTATION

An 86-year-old man was admitted with a 20-day history of fatigue and substernal 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, Biositec®) in a referral cardiac surgery/cardiology center because of symptomatic episodes of presyncope. 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 early pericardial effusion, supporting the existence of a similar relationship in this variant of the syndrome.

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. Virtually, identical syndromes have been described following open heart surgery, thoracic trauma, cardiac catheterization and pacemaker insertion. 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. 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.
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. 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. 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. 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. 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.

**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. Similar syndromes were subsequently described after myocardial infarction, post-pericardiotomy, following non/or penetrating chest wounds, post-radiofrequency ablation and post-intra-cardiac lead insertion. 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. 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. 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. 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.

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καρδίτιδας, που θεωρείται αυτοάνοσης αρχής, περιγράφηκε αρχικά μετά από έμφραγμα μυοκαρδίου και καρδιοχειρουργική επέμβαση. Στις τελευταίες περιπτώσεις, η επίπτωση του συνδρόμου μετά από θωρακοτομία 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’ όλα που υπάρχουν αρκετές αναφορές στη βιβλιογραφία σχετικά με την ύπαρξη και των δύο φάσεων μετά από τοποθέτηση βηματοδότη, δεν βρήκαμε κα- ποια 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 ύπαρξη περικαρ- διακή αντίδραση, η οποία παρατηρείται μετά από την τοποθέτηση βηματοδότη.

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

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