CASE REPORT

Constrictive pericarditis from an endocardial pacemaker lead

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SUMMARY

Constrictive pericarditis secondary to endocardial pacemaker inflammation, without associated pericardial effusion or infection, has yet to be described in the literature. We present a case of a 42-year-old man who developed recurrent ascites from regional constrictive pericarditis following pacemaker implant. Symptoms resolved after pericardiectomy.

BACKGROUND

Endocardial pacemakers and defibrillators (EPD) are routinely used in the management of arrhythmias. Although generally considered safe, a number of complications have been described.¹ Constrictive pericarditis (CP) has been reported with the use of EPD four times twice in the setting of a significant pericardial effusion post-procedure^{2–3} and twice associated with EPD sepsis.^{4–5} CP due to EPD-related inflammation in the absence of a reported effusion or infection has never been reported.

CASE PRESENTATION

A 42-year-old man with a history of bradycardia status post dual-chamber endocardial pacemaker implant developed worsening dyspnoea and ascites 2 months post-procedure. Symptoms progressed and he was admitted to an outside hospital. After diuresis, a paracentesis was performed, revealing a protein-rich fluid with some atypical cells. Liver function tests, complete blood count and a metabolic panel were normal; viral hepatitis panel, autoimmune hepatitis panel including rheumatoid factor (RF), antinuclear antibodies (ANA) and antinuclear cytoplasmic antibody (ANCA) were negative and immunoglobulin levels were normal (table 1). Abdominal imaging did not show evidence of cirrhosis or hepatitis. Transthoracic echo at the outside institution was reported normal. Eventually, a diagnostic laparoscopy without a biopsy was performed which was also unrevealing. After adequate diuresis, he was referred to hepatology clinic at our institution where a liver biopsy was performed to evaluate ascites of unknown aetiology. A biopsy revealed mild chronic hepatitis with no cirrhosis; however, during the biopsy, the right atrial pressure was noted to be 25 mm Hg and hence, he was referred to the cardiology clinic. Physical examination in the clinic was remarkable for tense ascites, mild bilateral pedal oedema and a jugular venous pulse elevated 10 cm above the sternal angle with associated rapid y-descent.

Figure 1 Mitral inflow Doppler with a restrictive filling pattern and mitral annular tissue Doppler with normal velocities.

Pacemaker interrogation revealed <1% ventricular pacing with normal lead parameters.

INVESTIGATIONS

An echocardiogram was repeated showing normal biventricular function, a septal bounce, restrictive mitral filling pattern and normal mitral annular velocities without annulus reversus (figure 1). Given these findings, CP was suspected; a subsequent haemodynamic catheterisation revealed elevated filling pressures with diastolic equalisation, 'dip and plateau' waveform in the right ventricle (RV) tracing, and discordant left ventricle (LV) and RV pressures (figures 2 and 3). He was referred

Figure 2 Simultaneous right atrial and left ventricular pressure waveform. Note the prominent 'x' (red arrow) and 'y' (blue arrow) on right atrial pressure tracing.

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Table 1 Autoimmune workup

Test	Value	Interpretation
Antinuclear antibody	1:40	Normal
Rheumatoid factor	1:24	Normal
Cytoplasmic antineutrophil cytoplasmic antibody	1:40	Normal
IgA	159 mg/dL	Normal
IgG	1239 mg/dL	Normal
IgM	100 mg/dL	Normal

to cardiothoracic surgery for pericardiectomy. Interestingly, no pericardial calcifications were seen on CT chest.

TREATMENT

Pericardiectomy was performed and intraoperatively, the pericardium was excessively adhering to the right side, with an associated organised haematoma around the pacer lead in the right atrium (RA). A pericardial biopsy revealed chronic inflammation. Bacterial and viral cultures, acid fast bacilli staining and viral nucleic acid PCR on the pericardial biopsy were negative.

OUTCOME AND FOLLOW-UP

On 1-month clinic follow-up, the patient noticed a significant improvement in ascites and dyspnoea. He returned to his baseline level of activity.

Figure 3 Discordance between RV and LV tracing seen. RV pressures increase and the LV pressures decrease and vice versa. LV, left ventricle; RV, right ventricle.

DISCUSSION

Negative infectious workup on a pericardial biopsy and negative ANA, RF, ANCA serology along with normal immunoglobulin levels done as part of hepatitis workup ruled out infection and connective tissue diseases as the cause for CP. The patient was not on any long-term medications, ruling out medication-induced pericarditis. A temporal association of symptoms with a pacemaker implant and intraoperative findings of localised inflammation and haematoma around the RA lead suggest that the aetiology of CP was from pericardial inflammation associated with a possible microperforation from the RA lead. This regional constriction favouring the right side results in less constriction of the lateral LV wall, explaining the absence of annulus reversus.

Learning points

- Constrictive pericarditis (CP) can be a rare complication of endocardial pacemakers and defibrillator placement.
- In the setting of unexplained ascites, there should be a high clinical suspicion for CP.
- Restrictive mitral inflow pattern with normal to high mitral annular tissue Doppler velocities should raise suspicion for CP.
- On noticing a 'dip and plateau' pattern on right ventricle tracing, a simultaneous left ventricle recording should be obtained to evaluate for ventricular interdependence.

Contributors NA: evaluated patient in clinic and hospital, did the literature search on this topic and played an important role in writing the article. MKM: contributed in patient evaluation and preparation of the manuscript, helped edit the manuscript and obtain the images. MA: the attending supervisor who helped in working up the patient, played a significant role in establishing the diagnosis and helped edit the manuscript contents.

Competing interests None declared.

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