

## Delayed Cardiac Tamponade Post Pacemaker Implantation

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### Abstract

Pericardial effusion is defined as the acute or chronic accumulation of fluid in the pericardial space, This fluid collection may exceeds the maximum capacity of pericardial space resulting in serious complication known as cardiac tamponade. We present a case of massive pericardial effusion two weeks after a dual chamber pacemaker implantation for sick sinus syndrome (SSS) with paroxysmal atrial fibrillation (AF). Transthoracic echocardiogram demonstrated large circumferential pericardial effusion with RA/RV diastolic collapses suggestive of cardiac tamponade. He was managed with emergency pericardiocentesis and pericardial fluid send for biochemical analysis. This case report highlights the importance of early diagnosis and treatment of pericardial effusion which are crucial for survival, thus high index suspicion is required even in patients who present late after the procedure.

**Keyword:** Pericardial effusion, Cardiac tamponade, Sick sinus syndrome, Atrial fibrillation, Pericardiocentesis

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### I. Introduction

We describe a case of massive pericardial effusion two weeks after a dual chamber pacemaker implantation for sick sinus syndrome (SSS) with paroxysmal atrial fibrillation (AF). Early diagnosis and treatment are crucial for survival, thus high index suspicion is required even in patients who present late after the procedure, the index patient was also started on oral anticoagulants (dabigatran) for stroke prevention. patient underwent pericardiocentesis and fluid analysis was done. we discuss the differentials and management options for such a case scenario.

### II. Case Report

A 59 year old diabetic and hypertensive male presented with sudden onset of chest pain and shortness of breath. he had undergone dual chamber pacemaker implantation 2 weeks back for sick sinus syndrome. Also he had a history of paroxysmal atrial fibrillation in the past, CHA2DS2VASc SCORE of more than 2 and so needed oral anticoagulants. Anticoagulant therapy with dabigatran was ongoing which was stopped 48 hours prior to procedure and restarted 48 hours prior to this admission to prevent the thromboembolic phenomenon that can occur as a complication of AF. Physical examination revealed he was afebrile, JVP elevated, blood pressure was at 90/60 mmHg, heart rate; 120 beats per minute. RR; 20/minute, spo<sub>2</sub>; 94%, local examination of pacemaker site was normal, cardiovascular examination revealed pulsus paradoxus and muffled heart sounds.

LAB RESULTS	ON ADMISSION
WBC COUNT	20700 cells/mm <sup>3</sup>
HB	11.5 g/dl
PLATLET COUNT	3.39 lakh/mcl
RBC	4.27 million/mm <sup>3</sup>
PCV	32.3
MCV	75.5fl
MCH	26.9 pg
MCHC	35.6 g/dL
BLOOD UREA	75 mg/dL
SERUM CREATININE	3 mg/dL
SERUM SODIUM	124 mmol/L
SERUM POTASium	5.3 mmol/L
ESR	9 mm/hour
CRP	17 mg/L

ECG showed atrial paced rhythm, pacemaker interrogation revealed parameters within normal limit, chest xray showed cardiomegaly with sharp borders, pacemaker lead insitu. Transthoracic echocardiogram demonstrated large circumferential pericardial effusion, RA/RV diastolic collapse SUGGESTIVE OF CARDIAC

TAMPONADE, left and right ventricular systolic function were normal (fig.1). It was decided to do emergency pericardiocentesis, after taking an informed consent. 250ml of hemorrhagic pericardial fluid is aspirated and send for biochemical analysis. pigtail is inserted and left in place till drain became negligible.

PERICARDIAL FLUID ANALYSIS	
Volume	6ml
Color	Reddish
Turbidity	Turbid
Glucose	204 mg/dl
Protein	5.52 g/dl
WBC	5850 cells/mcl (N:70%,L:18%,M:08%,E:0%,OTHERS:4%)
TB-PCR	Not detected
ADA	43.1 U/ml
AFB	-

(WBC: White Blood Cells, ADA: Adenosine Deaminase, AFB: Acid Fast Bacilli, TB-PCR: Tuberculosis-Polymerase Chain Reaction)

He was then treated with I V antibiotics, analgesics and other supportive medications, oral anticoagulants withheld and he is clinically improved after few days of ICU care, repeated echo at discharge showed only thin rim of pericardial effusion all around the heart and mild B/L pleural effusion ( Fig.2), patient is discharged on day 7 with complete resolution of symptoms. Follow up was taken weekly for one month and monthly thereafter, patient was asymptomatic and repeated echocardiogram showed no signs of recurrence of effusion.

### III. Discussion

Pericardial effusion is defined as the acute or chronic accumulation of fluid in the pericardial space which is the space between parietal and visceral pericardium, the fluid can be either bloody or serous. This fluid collection may exceed the maximum capacity of pericardial space resulting in serious complication known as cardiac tamponade where cardiac output is significantly reduced due to the compression of heart. Most common cause of pericardial effusion after permanent pacemaker implantation is lead related autoimmune reaction known as post pericardiotomy syndrome (Ref.7). It is characterized by fever, malaise, leukocytosis, myalgia, atrial arrhythmia, pleuritic chest pain, shortness of breath and occasionally pericardial tamponade. The syndrome has been reported to occur in up to 17% of the patient after cardiac surgery/procedures (Ref.1&2). Similarly, studies showed that patients who are excessively anticoagulated after any cardiac procedures are more prone to develop large pericardial effusion (Ref.3). Here the patient is presented with cardiac tamponade after 14 days of Permanent Pacemaker Implantation (PPI). Diagnosis is confirmed by an echocardiogram.

Risk factor for pericardial effusion could be hypertension, increased body surface area, immunosuppression, coronary artery disease, pulmonary thromboembolism, renal failure, urgency of operation, type of operation (ventricular assist device insertion, valve surgery, aortic aneurysm and heart transplant) and previous operation with cardiopulmonary bypass or prolonged cardiopulmonary bypass time (Ref.4).

In our case perforation excluded because of no alteration in lead threshold or any other pacing parameters and no change in fluoro position of leads (fig.3). Patient had a history of anticoagulant (dabigatran) use 2 days prior to the onset of symptoms given for prevention of complication of AF, there is an increased risk of incidence and severity of hemorrhagic complication resulting from pacemaker and ICD in patients treated with oral anticoagulants. Direct acting oral anticoagulants (DOAC) and warfarin has consistently been shown to reduce the risk of stroke, but if the patient is not kept at a target INR (2-3) for AF, the risk for hemorrhage outweighs the benefit, so a risk stratification model is used between the risk of hemorrhage and risk of thromboembolic event in such patients (Ref.5,8). The mechanism of action of DOAC is inhibition of either thrombin or activated factor X, and were designed in response to the need for an oral anticoagulant that did not require frequent monitoring and it neither has much dietary and medication interaction. Dabigatran is a potent direct competitive inhibitor of thrombin, its peak plasma concentration is reached at 6 hours, therefore patients are well anticoagulated immediately after first dose which may lead to pericardial effusion and early/late cardiac tamponade (Ref.8). So DOAC interruption is necessary with or without bridging with heparin which is dependable on severity of patient's condition and presence of comorbidities (Ref.9,10). Treatment option for pericardial effusion depends on the patient's clinical condition and echocardiographic findings by looking whether it is mild, moderate or large pericardial effusion. Typically mild effusion will require no specific treatment for the effusion itself instead the underlying cause is treated, for moderate effusion the treatment option depends on individual cases and its effects on heart function (Ref.6) and large pericardial

effusion/cardiac tamponade as in this case pericardiocentesis is the treatment of choice and with holding the anticoagulants which may need to be reintroduced 4-6 weeks after complete resolution of effusion in serial echocardiography (Ref.11,12).

#### **IV. Conclusion**

A high degree of suspicion, clinical correlation and experience are integral in diagnosing and managing a case of cardiac tamponade. Transthoracic echocardiography is the diagnostic procedure of choice and prompt intervention with careful evaluation of cardiac functions are essential for a better outcome.

**Figure 1:** Transthoracic echocardiogram showing large circumferential pericardial effusion with RA/RV diastolic collapse.



**Figure 2:** Post procedure echocardiogram showing only thin rim of pericardial effusion all around the heart



**Figure 3.** pacemaker fluoro showing leads in position



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