



# Delayed onset Post-cardiac Injury Syndrome presenting as bilateral pleural effusion following defibrillator insertion

Rouba Rahal MD - Pulmonary and Critical Care Fellow Under the supervision of Pr Moussa Riachy  Dressler's Syndrome or Post-cardiac Injury Syndrome (PCIS) is a well-defined entity characterised by a secondary pleuro-pericarditis in a setting of myocardial or pericardial insult.

• Dressler's syndrome was originally described after acute myocardial infarction and its incidence seems to be declining, owing to modern reperfusion modalities.

• However, other causes for this syndrome are emerging.

Different causes for PCIS have been identified.

- 1- Post pericardiotomy +++
- 2- Post infarction syndrome +++
- 3- Post-traumatic pericarditis +

4- latrogenic causes including intracardiac interventions and pacemaker lead insertion ???

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- 1- Post pericardiotomy +++
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4- latrogenic causes including intracardiac interventions and pacemaker lead insertion ???

- A 45-year-old middle eastern female
- Dyslipidemia
- History of smoking (1 pack per day for 20 years)
- Congenital hypertrophic cardiomyopathy
- Familial history of sudden cardiac death at an early age

- Single chamber ICD implanted due to her high-risk cardiomyopathy in March 2022
- Follow up
- Chest X-RAY



- ED December 2022 for chest pain dyspnea NYHA III
- Subacute presentation
- Antibiotic course (Levofloxacin)
- She then presented to our hospital for evaluation.

### Physical exam

- Afebrile but tachycardic and tachypneic.
- No fever, chills, cough, or sputum production.



• Cardiac ultrasound

### Laboratory workup

- WBC 11.8 x 10^9 /L
- Hg of 10.9 g/dl
- ESR of 31
- CRP 65  $\rightarrow$  230
- LDH 69
- Protein 192

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	Left Side
рН	7.64
LDH (U/L)	290
Protein (g/L)	44
Glucose (mmol/L)	5
GR	1 400 000
GB	2400
PNN	48%
Lymph	15%
Mononuclear	20%
Macrophage	17%

	Left Side	
рН	7.64	
LDH (U/L)	290	→ Ratio 1.5
Protein (g/L)	44	→ Ratio 0.63
Glucose (mmol/L)	5	
GR	1 400 000	
GB	2400	
PNN	48%	
Lymph	15%	
Mononuclear	20%	
Macrophage	17%	

### Day 2

- Clinical worsening
- Dyspnea
- Oxygen supplementation

## Day 2



	Left Side	Right Side
рН	7.64	7.67
LDH (U/L)	290	228
Protein (g/L)	44	42
Glucose (mmol/L)	5	6
GR	1 400 000	2000
GB	2400	5000
PNN	48%	46%
Lymphocyte	15%	13%
Mononuclear	20%	10%
Macrophage	17%	31%

### Laboratory workup

- Pleural fluid microbiology, including bacterial, fungal, and acid-fast bacilli cultures: Negative.
- Auto-immune panel : Negative
- PPD: Negative
- Pathology : Hematic and inflammatory liquid with immunohistochemistry positive for calretinin and negative for BerEP4 confirming the mesothelial origin of the cells.
- No malignant cells were observed.

#### Delayed onset Post-cardiac Injury Syndrome

- She was treated with NSAIDs (Naproxen) and discharged on colchicine.
- Gradual improvement.
- On follow up patient was asymptomatic with full disappearance of any pleural fluid.

Delayed onset Post-cardiac Injury Syndrome presenting as bilateral pleural effusion following defibrillator insertion • Four published cases of post pacemaker PCIS.



#### Pacemaker-Associated Post-cardiac Injury Syndrome Presenting with Tamponade and Recurrent Pleural Effusion

Young Ju Lee<sup>1</sup>, Mahmood Mubasher<sup>1</sup>, Abir Zainal<sup>1</sup>, Tausif Syed<sup>1</sup>, Mouhand F.H. Mohamed<sup>2</sup>, Matthew Ferrantino<sup>1</sup> and Ryan Hoefen<sup>1</sup>

<sup>1</sup>Unity Hospital, Rochester Regional Health, Rochester, New York, USA. <sup>2</sup>Internal Medicine Department, Hamad Medical Corporation, Doha, Qatar.

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Unusual association of diseases/symptoms

CASE REPORT

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

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



### Post-cardiac injury syndrome: an atypical case following percutaneous coronary intervention

Silvia Paiardi <sup>a,1</sup>, Francesco Cannata <sup>b,\*,1</sup>, Michele Ciccarelli <sup>c</sup>, Antonio Voza <sup>d</sup>

<sup>a</sup> Emergency Department, Humanitas Research Hospital, Via Manzoni 56, 20089 Rozzano, Italy

<sup>b</sup> Cardiovascular Department, Humanitas Research Hospital, Via Manzoni 56, 20089 Rozzano, Italy

<sup>c</sup> Pneumology Department, Humanitas Research Hospital, Via Manzoni 56, 20089 Rozzano, Italy

<sup>d</sup> Emergency Department, Humanitas Research Hospital, Via Manzoni 56, 20089 Rozzano, Italy

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#### Large Unilateral Pleural Effusion with Pacemaker-associated Post-cardiac Injury Syndrome

Sundeep Kumar<sup>1</sup>, Abed Madanieh<sup>1</sup>, Hiren Patel<sup>1</sup>, Ruthvik Srinivasa Murthy<sup>1</sup>, Jose M. Goyos<sup>2</sup>, Mark R. Milunski<sup>3</sup>

1. Internal Medicine, University of Central Florida College of Medicine, Orlando, USA 2. Pulmonary and Critical Care, Orlando Veterans Affairs Medical Center, Orlando, USA 3. Cardiology, Orlando Veterans Affairs Medical Center, Orlando, USA

- Four published cases of post pacemaker PCIS.
- One case of exclusive pleural involvement.
- One case of delayed presentation.

• The first ever reported case of post pacemaker PCIS presenting as a strict pulmonary presentation with bilateral involvement and delayed onset.

### Why this case ?

Similarities vs Differences

### Why this case ?

Similarities vs Differences

- 1- Delayed onset
- 2- Strict pleural presentation
- 3- Bilateral pleural involvement

1- This case challenges the current understanding of PCIS and highlights the need to consider it as a possible etiology for pleural effusion.

2- It also supports an autoimmune etiology for PCIS and calls for further investigation into its pathophysiology.

#### THANK YOU