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Peri-Myocardial Infarction Pericarditis: Current Concepts

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Abstract

Peri-Myocardial Infarction Pericarditis (PMIP), or epistenocardiac pericarditis, has been considered a relatively benign condition occurring within a few days after Myocardial Infarction (MI). Although the condition usually does not require specific treatment, the finding of a pericardial friction rub in the post-MI patient does prompt a careful review of post-MI symptoms and test results, including dysrhythmia recordings, Electro-Cardio Grams (EKG), and the Transthoracic Echocardiogram (TTE) to exclude potentially life threatening post-MI mechanical complications such as free wall rupture. The auscultatory findings of a pericardial rub often represent a teaching opportunity for house staff and students, yet given the self-limited course of PMIP, very little is known regarding the mechanism, biology, imaging findings, and management strategy in patients with and without symptoms. We review the current understanding of PMIP for clinicians caring for post-MI patients.

Keywords: Pericardium, Pericarditic injury syndromes, Dressler syndrome, Corticosteroids.

Abbreviations: PMIP- Peri-Myocardial Infarction Pericarditis, MI- Myocardial Infarction, EKG- Electro-Cardio Grams, TTE- Transthoracic Echocardiogram, PCIS- Post-Cardiac Injury Syndromes, PPS- Post-Pericardiotomy Syndrome, NSAIDs- Non-Steroidal Anti-Inflammatory Drugs, ICAP- Investigation on Colchicine for Acute Pericarditis, CMR- Cardiac Magnetic Resonance, AFL- Atrial Fibrillation, AF- Atrial Flutter.

Pericardial Anatomy

The pericardium is functionally a dual layer sac of outer fibrous and inner serosal (visceral) layer that contains the heart and normally less than 50 mL of pericardial fluid [1]. The outer fibrous layer is in continuity with the great vessels above and with the diaphragm below and is in direct contact with many mediastinal structures such as the pleura, bronchi, esophagus, and rib costal cartilage. Measuring less than 1mm in thickness, the pericardium is composed of collagen, elastic fibers, fibroblasts, and mesothelial cell layers along the parietal and serosal pericardium [1]. Regardless of etiology, injury to the pericardium generally results in a non-specific response with generation of fluid and inflammatory cells with formation of fibrinous adhesions during convalescence.

Cardiac-Pericardial Injury Syndromes

The cardiac and pericarditic injury syndromes, often referred to as Post-Cardiac Injury Syndromes (PCIS), are categorized into PMIP, Post-Pericardiotomy Syndrome (PPS), and post-traumatic pericarditis. It is postulated that PMIP and PPS have a similar biologic basis, evoking an antibody response to cardiac cellular components and contractile proteins. However, this response is perhaps magnified in PPS owing to greater tissue injury following cardiac surgery. Immune complexes trigger inflammatory responses in the pericardium [2,3].

Post- traumatic etiologies include iatrogenic and non-iatrogenic factors. The most common iatrogenic culprits are percutaneous cardiac procedures such as pacemaker lead insertion, cardiac ablation procedures, percutaneous coronary interventions, endomyocardial biopsies, and structural heart procedures. Non-iatrogenic factors include trauma to the chest wall or aortic dissection. Post-infarction etiologies are grouped by timing, with early (<7 days post MI) involvement consistent with PMIP while a delayed (>7 days to months) involvement consistent with Dressler syndrome (late or delayed pericarditis). First described in 1956 by William Dressler, the delayed post infarction syndrome may present with symptoms similar to those with acute idiopathic pericarditis including pleuritic chest discomfort or pain across the trapezius ridge, fever, and signs of a pericardial friction rub [4].

The European Society of Cardiology has proposed diagnostic criteria for PCIS when at least 2 of the 5 following criteria are met: 1) fever without alternative explanation, 2) pericarditic or pleuritic chest pain, 3) pericardial or pleural friction rub, 4) pericardial effusion and/or 5) pleural effusion with elevated C-reactive protein [5]. Regardless of etiology, the typical EKG findings of PCIS include widespread ST-segment elevation often sparing aVR and V1 along with PR segment depression. However, these EKG changes are dynamic, evolving over time and ultimately normalize.

There may be associated findings on chest X-ray demonstrating a pleural effusion or a pericardial effusion on TTE. Pericardial effusions are usually small without increased intrapericardial pressures and tamponade physiology. However, ongoing inflammation necessitates serial clinical and TTE examinations to determine whether the pericardial effusion is enlarging and requires pericardiocentesis. Treatment of symptomatic patients usually involves anti-inflammatory medications (aspirin 650 mg every 6 to 8 hours or ibuprofen 600mg every 8 hours) for 7 to 10 days. There is general consensus that aspirin is the preferred anti-inflammatory in patients post MI, rather than ibuprofen and other Non-Steroidal Anti-Inflammatory Drugs (NSAIDs). Patients with contraindications to aspirin or NSAIDs (severe renal insufficiency, allergies, GI bleeding, or oral anticoagulants) may be treated with low-dose glucocorticoids (<0.5 mg/kg/day) for 2 to 4 weeks with slow and gradual tapering. In the Investigation on Colchicine for Acute Pericarditis (ICAP) study, which included 20% (n=48 patients) of patients with post-cardiotomy pericarditis, the addition of colchicine to anti-inflammatory therapy significantly reduced the primary outcome of incessant/recurrent pericarditis (16.7% vs 37.5%; RRR 0.55, 95% CI 0.30-0.72) [6]. There were no serious adverse events related to colchicine, which had a similar side effect profile to placebo.

Peri-Myocardial Infarction Pericarditis

PMIP, or epistenocardiac pericarditis, shares some characteristics to Dressler syndrome but also has notable differences. It has been suggested that the incidence of PMIP has declined in the era of reperfusion therapy and may only affect approximately 1% of those with ST-elevation myocardial infarction [7]. Since PMIP is more likely to occur after transmural MI, the EKG findings of pericarditis may be masked by the EKG changes of an evolving MI. Thus, a pericardial friction rub may be the only diagnostic clue to its presence. Indeed, since the typical pyramidal infarction zone of the myocardium has its base at the endocardium and apex at the epicardium, adjacent to the visceral pericardium, it is less likely to produce diffuse ST elevations as seen in Dressler syndromes [8].

Pericardial friction rubs are characteristically dynamic, subtle, change with underlying pericardial insult, and rarely persist beyond a few days. A study of auscultatory and phonocardiographic recordings by an experienced examiner identified that the majority of pericardial friction rubs are composed of 3 components: atrial systole, ventricular systole, and protodiastole (immediately after S2), rather than biphasic "to-and-fro" as had been originally characterized [9]. Rubs have a qualitative characteristic of grating or scratching, and may on rare occasions be palpable [9]. Due to the fleeting nature of rubs and limitations of auscultation to detect pericarditis, PMIP has been increasingly incidentally identified using Cardiac Magnetic Resonance (CMR) early after acute MI. Evidence for pericardial inflammation can be appreciated on late gadolinium enhancement early after acute MI, often adjacent to the region of infarct (**Figure 1**).

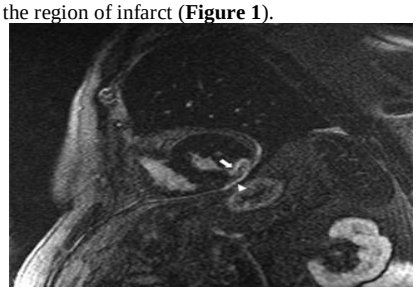

Figure 1: 78-year-old male with evidence of PMIP on cardiac magnetic resonance imaging with late gadolinum enhancement 1 day after acute inferolateral ST-segment elevation myocardial infarction. Note the pericardial enhancement (arrowhead) adjacent to and extending just beyond the inferoateral wall segment with full thickness zone of infarction (arrow).

In a study of 189 patients undergoing CMR 2 to 5 days after primary PCI for acute ST-segment elevation MI, 31% of patients had pericardial inflammation with the majority (60%) of inflammation located in the infarct zone confirmed on late gadolinium enhancement [10]. Interestingly, diffuse involvement was present in 28% of those with pericardial inflammation. Those with pericardial inflammation had larger infarct size, more microvascular obstruction, and higher levels of CRP, as compared to those without inflammation. At 4 month follow up CMR, pericardial abnormalities had resolved in 80% of patients. Indeed, advanced cardiac imaging suggests that PMIP is likely more common than initially appreciated, with identification of subclinical PMIP. Since most of these abnormalities show resolution in follow up, further research is needed to determine the prognostic significance of pericardial inflammation in post-acute MI. **Figure 3** illustrates various degrees of pericardial involvement with late pericaridits and PMIP as compared to normal pericardium.

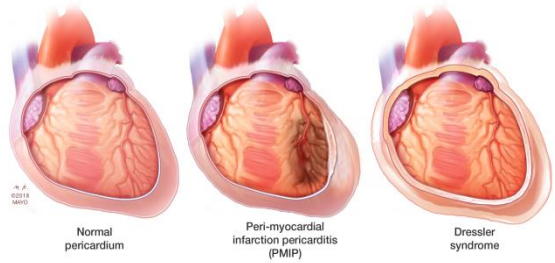

Figure 3: Illustration of normal pericardium as compared to pericardial involvement in peri-myocardial infarction pericarditis (PMIP) and Dressler syndrome. Note the regional involvement of the pericardium adjacent to the infarction zone as compared to a more diffuse and generalized inflammatory involvement in Dressler syndrome.

Although Imazio et al observed that the incidence of atrial fibrillation/atrial flutter (AF/AFL) in patients with acute pericarditis ranges from 4 -7%, pericardial inflammation may not be independently associated with incident AF/AFL since affected patients were typically older [11]. Nevertheless, some patients with pericarditis develop AF without traditional clinical risk factors for AF (**Figure 2A and 2B**). Furthermore, in an analysis of patients with acute coronary syndrome from several large clinical trials, the incidence of AF was approximately 7% [12]. Unfortunately, the incidence of PMIP or Dresslers was not reported in this study, raising the question whether post infarction pericarditis may have contributed in part to the observed rates of AF [12]. **Table 1** compares and contrasts early PMIP with late pericarditis (Dressler syndrome).

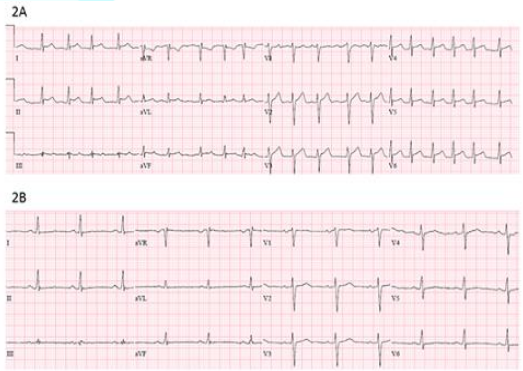

Figure 2: A 46 year old patient developed pericarditis and atrial fibrillation after PCI for non-ST elevation myocardial infarction. Note diffuse ST-segment elevation throughout most leads, sparing lead V1, with ST-segment depression in aVR (2A). A conservative approach was undertaken given no pericarditic symptoms resulting in resolution of EKG abnormalities 5 days later (2B).

	Peri-myocardial infarction pericarditis (PMIP)	Dressler Syndrome (late pericarditis)
Time course	Early (<7 days post MI) (PMIP)	Delayed (>7 days) post MI (Dressler)
Symptoms	Asymptomatic or minimal symptoms, though symptoms often prompt concern of recurrent ischemia post PCI	Symptoms include chest or pleuritic pain, pain along trapezius ridge, pneumonitis, fever
Etiology/Mechanism	Transmural acute MI	Post MI, Post-pericardiotomy syndrome, or post-traumatic* (iatrogenic and non-iatrogenic factors), autoimmune factors, seasonal changes
Laboratories	Often cardiac biomarker elevation given proximity to post MI, inflammatory markers	Elevated inflammatory markers (ESR, CRP), leukocytosis, anti-actin and anti-myosin antibodies
Radiographs	Often unremarkable	Enlarged cardiac silhouette (if pericardial effusion), pleural effusion, possible lung consolidation
Electrocardiogram	Acute MI changes often mask the localized region of PMIP	Diffuse ST-segment elevation with PR segment depression (Classic)
Echocardiogram	Regional wall motion abnormality related to underlying acute MI, pericardial effusion (often small); larger pericardial effusions in this setting may suggest an unrecognized wall rupture	Pericardial effusion (small to large)
Cardiac MRI with gadolinium	Pericardial enhancement adjacent to infarct zone, though may extend beyond zone or have diffuse involvement	Pericardial enhancement often diffuse
Prognosis	Primarily related to the initial acute MI	Related to underlying etiology Recurrence rate 10% Low risk of constrictive pericarditis

Table 1: Comparison between PMIP and Dressler Syndrome.

Treatment

The general approach to patients with PMIP, diagnosed clinically or by advanced cardiac imaging, is usually supportive and no specific therapy is recommended since symptoms and signs usually resolve without intervention. For symptomatic patients, acetaminophen 650 mg scheduled every 6 to 8 hours for up to 10 days is useful. If patients remain symptomatic, aspirin 650 mg every 6 to 8 hours can be initiated with tapering once symptoms improve, usually after 7 to 10 days. Of note, there is a potential bleeding risk with high dose aspirin in the post PCI patient and is not recommended in patients on ticagrelor since it reduces drug efficacy [13].

Colchicine may be considered as an adjunct to reduce recurrence risk. This recommendation is based on practice guideline recommendations for acute idiopathic pericarditis and limited data [5,6]. Non-steroidal medications (NSAIDs) are generally avoided in post MI patients since observational studies have documented increased cardiovascular risk and events (MI and stroke) in patients with known heart disease [14]. Corticosteroids are also generally avoided in the post MI patient due to propensity for fluid retention, hypertension, and their adverse effect on atherosclerotic vascular disease.

Future Directions

Advanced imaging with CMR has identified both localized and generalized pericardial enhancement in PMIP. This observation suggests that pericardial involvement post MI is likely more common than previously thought since the clinical findings of pericarditis alone may be subtle and go unnoticed. Further research is needed to establish whether the finding of pericardial enhancement in PMIP has prognostic significance since clinically diagnosed PMIP generally has a benign and self-limited course.

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