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Dressler's syndrome – a literature review

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Abstract. Dressler's syndrome, first described in 1956, is a form of secondary pericarditis classified within the broader category of post-cardiac injury syndromes. It typically occurs several weeks after acute myocardial infarction and is believed to result from an autoimmune response triggered by myocardial necrosis. Once common, its prevalence has declined significantly due to the widespread adoption of reperfusion therapies, including thrombolysis and percutaneous coronary intervention (PCI). This review synthesizes current understanding of Dressler's syndrome, with particular attention to the recommendations of the European Society of Cardiology (ESC). Clinically, it may present with low-grade fever, chest pain, dyspnoea and elevated inflammatory markers. Diagnosis is based on specific criteria, which require at least two of the following: pleuritic chest pain, a pericardial friction rub, electrocardiographic features suggestive of pericarditis, or new or progressive pericardial effusion. Imaging techniques such as echocardiography and cardiac magnetic resonance enhance diagnostic accuracy and assist in ruling out alternative causes. Treatment involves high-dose aspirin, often used in combination with colchicine to prevent recurrence. For cases that are resistant to standard therapy, escalation to immunosuppressive agents or pericardiotomy may be necessary in carefully selected patients. In modern practice, it should still be considered in patients presenting post-MI chest pain, particularly in those with large infarctions or without prior PCI. Given that most existing literature originates from the pre-reperfusion era, there is a clear need for updated clinical insights.

Keywords: Dressler's syndrome – post-myocardial injury – pericarditis

Dressler's syndrome, or post-myocardial injury syndrome, is a delayed inflammatory pericardial response occurring in the aftermath of acute myocardial infarction (MI) (1). First described in 1956 by William Dressler, this syndrome was initially considered a relatively common condition. Currently, with the routine implementation

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of reperfusion strategies, including thrombolysis and percutaneous coronary intervention (PCI), the incidence of Dressler's syndrome has markedly diminished. However, it still holds clinical importance, as it may result in serious complications (2).

The objective of this review is to evaluate and synthesize the current knowledge on Dressler's syndrome, with particular emphasis on its evolving clinical presentation in the era of contemporary MI management. The article includes its definition, historical context, etiopathogenesis, epidemiology, clinical features, diagnostic approach, treatment strategies, and prognosis. Additionally, the article contains a synthesis of case reports documenting Dressler's syndrome over the past fifteen years.

Methods

A comprehensive literature review was conducted using databases such as PubMed, Medline, and Google Scholar. Articles were included if the term 'Dressler's syndrome' appeared in the title, abstract, or keywords. The primary search period spanned from 2010 to 2025; however, earlier sources were considered in cases where more recent data were unavailable. Additionally, guidelines issued by the European Society of Cardiology (ESC) were reviewed to integrate current clinical recommendations.

Results and Discussion

Definition

As outlined in the 2015 ESC Guidelines on pericardial disease, Dressler's syndrome is defined as a form of pericarditis that arises following MI, typically manifesting between 1 and 2 weeks after the ischemic event (1). Some authors, however, propose a broader window of onset extending up to 10 weeks post-infarction (3).

It is classified within the broader category of post-cardiac injury syndromes (PCIS), which encompasses various forms of inflammatory pericarditis. This group includes conditions such as post-pericardiotomy syndrome (PPS) and post-traumatic pericarditis, the latter of which may result from either iatrogenic causes, including invasive cardiac interventions, or from non-iatrogenic thoracic trauma (1).

Some researchers use the term 'Dressler's syndrome' in the context of other forms of PCIS. However, for the purposes of this article the authors, in accordance with the nomenclature outlined in the ESC guidelines, define Dressler's syndrome exclusively as late pericarditis following MI.

It is important to distinguish Dressler's syndrome from early post-infarction pericarditis, which typically presents within the initial four days after an acute MI and is usually self-limiting (4).

Historical context

The syndrome was initially characterized in 1956 by William Dressler, who reported a constellation of post-infarction symptoms resembling idiopathic, recurrent pericarditis (5). A subsequent publication in 1959 expanded upon these observations through a case series involving forty-four patients (6). These seminal contributions laid the groundwork for subsequent recognition and understanding of Dressler's syndrome as a distinct delayed inflammatory response following MI.

Etiopathogenesis

The underlying pathophysiological mechanisms of Dressler's syndrome are not yet fully elucidated. Initially, in 1956, Dressler hypothesized that the condition resulted from a hypersensitivity reaction to cardiac autoantigens exposed during myocardial necrosis (5). Current evidence suggests that autoimmune and inflammatory responses play a crucial role in its development (7).

The pathogenesis is thought to involve several interconnected mechanisms. Myocardial necrosis leads to the exposure of intracellular antigens, which may trigger an aberrant immune response in genetically or immunologically predisposed individuals (7). The resulting inflammatory cascade can extend beyond the pericardium, with pleural and synovial involvement likely mediated by molecular mimicry and immune cross-reactivity (8).

Dressler's syndrome appears to occur more frequently after larger MI (9), which may be associated with a greater release of myocardial antigens.

Support for an immune-mediated mechanism includes the typical latency of several weeks between MI and symptom onset, as well as the favourable response

to anti-inflammatory therapy and the risk of recurrence (1). Elevated titers of anti-myocardial antibodies (such as anti-actin and anti-myosin) have been detected in patients with PCIS, although their exact pathogenic relevance remains uncertain (7, 10).

Naturally, there are also other theories regarding the pathogenesis of this syndrome. An additional hypothesis implicates viral involvement in the pathogenesis, based on observations of seasonal clustering of cases and associations with elevated viral antibody titers in affected individuals (7, 10).

Earlier theories suggested that haemorrhagic pericardial effusion induced by anticoagulant therapy might provoke pericardial inflammation and contribute to the development of Dressler's syndrome. However, subsequent reports of cases occurring in the absence of anticoagulation, combined with the syndrome's declining incidence despite continued use of anticoagulants in the reperfusion era, have largely refuted this as a primary etiological mechanism (8).

Epidemiology

Historically, the incidence of Dressler's syndrome following acute MI was estimated at 1–5% (6, 11, 12, 13). However, its occurrence has markedly decreased over time, and it is now regarded as a rare complication. This decline is largely attributed to the advent and widespread adoption of reperfusion strategies. The use of thrombolysis and coronary angioplasty resulted in the reduction of infarct size and shortened exposure of myocardial antigens to the immune system (8, 14).

In developing countries, where access to previously mentioned therapeutic methods remains restricted, the incidence of Dressler's syndrome may be comparatively higher (15).

It has been suggested that medications commonly used in post-MI management - such as angiotensin-converting enzyme (ACE) inhibitors, beta-adrenolytics, and statins - may exert protective effects against Dressler's syndrome through their immunomodulatory actions. ACE inhibitors and beta-adrenolytics have been shown to lower circulating pro-inflammatory cytokines, while ACE inhibitors may also attenuate myocardial injury (8). Statins, through their pleiotropic properties, have been reported to reduce levels of C-reactive protein, tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) (16).

Dressler's syndrome may be underdiagnosed, particularly following silent or unrecognized MI, as its clinical manifestations are often nonspecific and can closely resemble those of idiopathic pericarditis (17).

Clinical features and additional investigations

Typical clinical presentation of Dressler's syndrome includes fever, dyspnoea, and pleuritic chest pain, often described as sharp and exacerbated by supine positioning, with relief upon sitting forward. On physical examination, a pericardial friction rub may be audible (7, 10, 18, 19).

Inflammatory markers are commonly elevated and may include leucocytosis, increased C-reactive protein (CRP), and a raised erythrocyte sedimentation rate (ESR) (7, 10, 18).

Serum concentrations of troponin I and N-terminal pro B-type natriuretic peptide (NT-proBNP) may be elevated; however, in the setting of a recent MI, these findings lack specificity and provide limited diagnostic utility (18).

Chest radiography may reveal cardiomegaly secondary to pericardial effusion and aid in ruling out alternative causes of symptoms, such as pulmonary congestion or oedema (7, 10, 18).

Electrocardiographic findings frequently mirror those seen in acute pericarditis and may include widespread ST-segment elevation, PR-segment depression, reciprocal ST depression in leads aVR and V1, PR elevation in aVR, and T-wave inversions (10, 18).

Transthoracic echocardiography (TTE) is an essential bedside imaging modality that enables detection, quantification, and characterization of pericardial effusion. It also provides valuable hemodynamic data for the assessment of cardiac tamponade and may demonstrate regional wall motion abnormalities or late complications following MI (7, 10, 18).

In diagnostically challenging or equivocal cases, cardiac magnetic resonance imaging (MRI) may be employed to confirm the diagnosis. MRI can demonstrate pericardial thickening, oedema, and inflammation (18). Late gadolinium enhancement of the pericardium is a characteristic finding, and the modality is known for its high sensitivity and specificity in detecting pericardial pathology (3).

Computed tomography (CT) serves as an alternative imaging modality when MRI is contraindicated or un-

available. It is particularly useful for visualizing pericardial thickening (18).

As part of the immune-mediated process, Dressler's syndrome may involve the pleura, leading to pleuritis and associated pleural effusion (7). These findings can be identified through imaging modalities such as chest radiography, CT, or MRI.NT

Diagnosis

As outlined in the 2023 ESC Guidelines for acute coronary syndromes, the clinical evaluation of Dressler's syndrome follows the same criteria used for acute pericarditis. A diagnosis can be made when at least two of the following features are present: pleuritic chest pain, a pericardial friction rub, electrocardiographic features suggestive of pericarditis, or new or progressive pericardial effusion (4).

The differential diagnosis should encompass a range of conditions that may mimic Dressler's syndrome, particularly in patients presenting with chest pain, dyspnoea, fever, or signs of pericardial or pleural inflammation. Potential alternatives to consider include infectious, autoimmune, neoplastic, or metabolic pericarditis; acute myocarditis; stent thrombosis; reinfarction; pulmonary embolism; and respiratory tract infections such as pneumonia or bronchitis (1, 4, 20).

Because Dressler's syndrome often presents with nonspecific symptoms, its diagnosis can be challenging, and it is frequently overlooked in favour of more common conditions. Nonetheless, it should remain in the differential diagnosis, particularly in patients with extensive or unrecognized MI, where the clinical context may support an immune-mediated post-injury process.

Treatment

Current ESC Guidelines recommend high-dose aspirin as the first-line therapy, typically administered at 500–1000 mg every 6–8 hours until symptoms resolve, followed by a gradual taper of 250–500 mg every two weeks. Adjunctive therapy with colchicine at a dose of 0.5 mg twice daily for three months is also advised to reduce recurrence rates (4).

Alternative nonsteroidal anti-inflammatory drugs (NSAIDs) may also be considered (1); however, their use has been associated with adverse effects such as infarct expansion and impaired scar formation (9, 21). Corticosteroids may be considered as a secondary option, though their use is limited due to their risk of chronic disease progression and dependency (1).

An isolated case report has described successful symptom control using a combination of low-dose colchicine (0.5 mg/day) and paracetamol (2000 mg/day) in a patient with Dressler's syndrome. Although this approach is not currently included in guideline-recommended management, it may represent a potential alternative in selected individuals who are at increased risk from NSAID use. Nevertheless, further investigation is required before such a strategy can be considered clinically relevant (22).

Management of recurrent, treatment-resistant pericarditis may require escalation to immunosuppressive agents such as azathioprine, intravenous immunoglobulin (IVIG), or interleukin-1 (IL-1) antagonists like anakinra. In select cases, pericardiectomy may be considered as a definitive intervention, although it presents technical challenges, particularly in patients with prior coronary artery bypass grafting (CABG) (18).

Prognosis

Pericarditis tends to occur more frequently in the context of extensive MI. However, available data indicate that patients with Dressler's syndrome do not experience higher rates of hospitalization, one-year mortality, or major cardiogenic complications when compared to those without post-MI pericardial involvement (1).

With appropriate anti-inflammatory therapy, the prognosis is generally favourable, and most patients recover fully. If left untreated, however, Dressler's syndrome may progress to serious complications such as cardiac tamponade or, less commonly, constrictive pericarditis, both of which may necessitate invasive intervention (23).

Persistence of the Syndrome in Contemporary Practice

Although the incidence of Dressler's syndrome has significantly declined, occasional case reports continue to emerge in the literature, indicating that this condition still occurs in contemporary clinical practice. A total of six cases of Dressler's syndrome, defined as delayed-onset pericarditis following MI, published between 2010 and 2025 were identified and reviewed (2, 15, 17, 18, 22, 24).

The time from myocardial infarction to symptom onset ranged from two weeks to over two months. One case occurred following a silent, previously unrecognized and untreated MI (17), while another was diagnosed as late as nine weeks post-infarction (24).

Despite the presence of established ESC criteria for pericarditis, diagnosis was not always straightforward. Some patients presented with incomplete or atypical symptoms, and in one instance the syndrome was initially misdiagnosed as infection, delaying appropriate treatment (17).

Diagnostic workup typically included ECG, inflammatory markers, and transthoracic echocardiography. In selected cases, CT or cardiac MRI was employed to confirm pericardial involvement (15, 17, 18, 24).

Treatment strategies varied depending on severity and tolerance. Ibuprofen was administered in one case (24), while aspirin was used in two (15,18). Colchicine was included in two treatment regimens (18,22); in one case, it was combined with paracetamol due to NSAID intolerance (22). One patient required prednisone and subsequent interleukin-1 receptor inhibitor (18). In one case, pharmacological treatment specific to Dressler's syndrome was not reported (17), while in another therapy was not implemented due to spontaneous improvement and the mild course of the disease (2).

These reports illustrate the heterogeneity of clinical presentation, diagnostic complexity, and therapeutic response in Dressler's syndrome. They underscore the importance of clinical awareness and vigilance in recognizing this rare but potentially serious post-infarction complication.

Conclusions

Dressler's syndrome remains a rare but clinically relevant complication following MI. Although its incidence has declined markedly in the reperfusion era, it should still be considered in the differential diagnosis of post-MI chest pain-particularly in patients presenting with persistent inflammatory markers or pleuropericardial symptoms.

The condition is typically characterized by fever, pleuritic chest pain, pericardial friction rub, and pericardial or pleural effusions, occurring weeks after MI. Its pathogenesis is thought to involve autoimmune and inflammatory mechanisms triggered by myocardial injury.

According to current ESC guidelines, high-dose aspirin is recommended as the first-line treatment for Dressler's syndrome, administered in divided doses and tapered gradually as clinical improvement is observed. Colchicine may be added to reduce the risk of recurrence. While this regimen is generally effective, an isolated report has described the use of paracetamol in combination with colchicine as a potential alternative in selected patients. Although such approaches are not part of standard care, they may be considered in specific clinical contexts where NSAIDs are contraindicated or poorly tolerated. In recurrent refractory pericarditis, immunosuppressive agents, IVIG, IL-1 receptor inhibitors and pericardiotomy remain viable treatment options.

Given that much of the existing literature predates contemporary interventional strategies, further research is needed to update our understanding of Dressler's syndrome in the modern clinical context.

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