Acute Hypotension and Chest Pain as The Presentation of a Post-Myocardial Infarction Acute Pericarditis (Dressler's Syndrome)

Alfredo De Giorgi, Matteo Guarino, Federico Moro, Dario Gozzi, Christian Molino, Ruana Tiseo, Elisa Misurati, Benedetta Boari, Claudia Parisi, Roberto Manfredini, Fabio Fabbian.

Department of Medical Sciences, Clinica Medica Unit, School of Medicine, University-Hospital of Ferrara, Italy .

Corresponding author:
Alfredo De Giorgi,
Department of Medical Sciences, Clinica Medica Unit,
School of Medicine, University of Ferrara, University Hospital of Ferrara,
Via Aldo Moro 8, I-44124, Italy
Phone: +39–0532–237071,
Fax: +39–0532–236816,
E-mail: degiorgialfredo@libero.it

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We report a case of a 53-year old man admitted because of fever (38.5°C) and atypical chest pain. He also complained of epigastric pain spread to left hypochondrium and exacerbated by breathing. In his past medical history, Hodgkin's lymphoma, gastric MALToma (oncologic follow-up only), hypothyroidism, and hypertension are recorded. Fifteen days prior actual hospital admission, the patient underwent angioplasty with stenting due to a ST elevation myocardial infarction. Moreover, he was enrolled in the experimental clinical double-blind trial GEMINI-ACS [1], designed to compare the safety of rivaroxaban vs aspirin in addition to either clopidogrel or ticagrelor. Thus, his complete therapy included also ticagrelor, bisoprolol, perindopril, levothyroxine, pantoprazole, and atorvastatin. At the time of admission chest X-ray showed bilateral pleural effusion. Blood chemistry panel showed moderate anemia, increase of inflammatory indexes, in particular fibrinogen 1057 mg/dl (normal range 150-400 mg/dl), C-reactive-protein 17.6 mg/dl (normal range <0.5 mg/dl), serum ferritin 650 ng/ml (normal range 11-306 ng/ml), while serum pro-calcitonin was normal. Electrocardiography and cardiac troponin I were not suggestive of further heart ischemic damage. Two days later, the patient showed hypotension, exacerbation of chest pain, as well as a rapid drop hemoglobin values. A thoracic-abdominal CT (figure 1) was performed, showing peri-hepatic and pericardial effusions associated with hyper-reflectivity of pericardial leaflets. After a precautionary discontinuation of the experimental drugs, acetylsalicylic acid and clopidogrel were only given, together with antibiotics, diuretics and steroids. Clinical conditions slowly improved, blood pressure levels raised, together with hemoglobin values, and inflammatory parameters decreased. The patient was discharged in good clinical conditions, with the conclusive discharge diagnosis of Dressler syndrome (DS) related to angioplasty and stenting procedure for acute myocardial infarction.

Discussion

DS, also known as post-myocardial infarction syndrome and first characterized by William Dressler in 1956, is a delayed complication of acute myocardial infarction occurring within 1 to 8 weeks after cardiac acute events. In the pre-thrombolysis era,

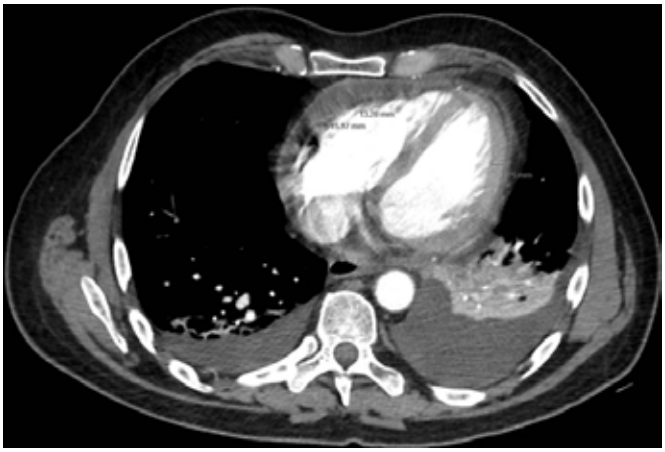

Figure 1. Computer tomography (CT) showing evident pleuritic and pericarditic effusions.

* Corresponding author. E-mail: degiorgialfredo@libero.it
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its prevalence was 7–23%, then decreasing to <5% after the introduction of reperfusion therapies [2]. Typical symptoms of DS include pleuritic chest pain, pericardial and pleural effusion, low-grade fever, increase of inflammatory laboratory tests, enlargement of the cardiac silhouette, ST-elevation and T-wave changes. Diagnosis is typically made by clinical conditions, chest x-ray, EKG, echocardiography, or by chest computed tomography (CT), although cardiac nuclear magnetic resonance (NMR) would represent the gold standard [3].

Etiology of DS is not clearly established, but an immunologic pathogenesis has been suggested, secondary to the release of cardiac antigens during cardiovascular events. This could stimulate antibodies formation, with further formation of immune complexes in pericardium, pleura and lungs, followed by an inflammatory response [4]. A recent French study evaluated 193 patients with a first episode of ST-elevation myocardial infarction, 58.5% of whom complicated by the presence of pericardial effusion [5]. All patients, symptomatic or not, were investigated with cardiac NMR. The authors showed that presence and volume of pericardial effusion were related to infarct size, microvascular obstruction, and systolic wall stress, and early reperfusion and optimal medical therapy resolved the condition. Treatment of DS remains largely empiric due to the absence of evidence based data. In agreement with the European Society of Cardiology guidelines, non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen, represent the first pharmacological choice in early post-infarction pericarditis [6]. On the one hand, such therapy was found to be related with a risk of scar thinning, infarct expansion and myocardial free wall rupture but, on the other, the same complications have also been issued to corticosteroid therapy [7].

High acetyl-salicylic acid dose (500 mg every 8 – 12 hours for 7 days), provides an efficacious anti-inflammatory effect and is generally well tolerated [8]. In particular, however, in patients who received a percutaneous coronary intervention, corticosteroids should be avoided for at least 4 weeks after the procedure, due to their potential impact on cardiac tissues [9]. By means of a PubMed search, we performed a review of the case reports available in literature. We found 19 cases (Table 1). DS occurred more frequently in the middle age (mean age 61±13 years) and in men (13 vs 6 case), on average 26 days after (range 1-120) from the acute event. Even though the clinical manifestations and pharmacological therapies were different, mortality appears to be low (1 fatal case only in our series). Interestingly, despite the above mentioned suggestions by Imazio et al [9], near 50% of DS cases reported received corticosteroid therapy, with positive response.

DS is part of the so-called post cardiac injury syndrome (PCIS), an inflammatory process that occurs in the setting of injury to the pericardium, epicardium or myocardium, following cardiac surgery, myocardial infarction, trauma, intra-cardiac ablation, percutaneous coronary intervention or implantation of a pacemaker or cardioverter-defibrillator. Moreover, DS can represent either a triggering factor of or a consequence of Takotsubo cardiomyopathy [10]. Even if male gender was more represented in our series, the risk of developing pericarditis is similar in men and women, also because women are more likely to present autoimmune diseases [11]. The peculiarity of the present case is given by its acute onset and a slightly anticipated time from infarction (15 vs 26 days), even if quite fitting into the observed range, probably secondary to a high inflammatory trigger. Although in the present case the presentation symptoms resembled that of a recurrence of acute cardiac ischemia, more often DS shows a subclinical evolution, so that some cases may be missed and the low incidence reported may be explained. However, the outcome is generally favorable, and the fatal case reported in this series makes reference to a sudden cardiac death, with autopsy findings of acute myocardial infarction and fibrinous pericarditis. However, in the actual era of aggressive treatment of myocardial infarction and given its the delayed onset, the possible occurrence of a Dressler's syndrome should always kept in mind, and not only by cardiologists.

Table 1. Dressler syndrome after acute myocardial infarction: a literature review of case reports.

Author	Age	Sex	Time from cardiovascular event (days)	Therapy	Outcome
Tramontana & Zamporlini, 1961	60	Male	15	Corticosteroids + antibiotics	Favorable
Maggi & Banno, 1964	43	Male	7	Antibiotics	Not known
Maggi & Banno, 1965	71	Male	11	Corticosteroid + acetylsalicylic acid	Not known
Maggi & Banno, 1966	40	Male	6	Anticoagulant discontinuation	Favorable
Maggi & Banno, 1967	53	Male	34	Corticosteroids + acetylsalicylic acid	Favorable
Maggi & Banno, 1968	52	Female	1	Thiazidics, corticosteroids, anticoagulant discontinuation	Favorable
Maggi & Banno, 1969	67	Male	26	Acetylsalicylic acid, anticoagulant and corticosteroids discontinuation	Not known
Vecchio C. 1965	72	Male	14	Corticosteroids	Favorable
Vecchio C. 1966	42	Male	15	Corticosteroids	Favorable
Lawrence & Wright, 1972	67	Female	10	Corticosteroids	Favorable
Hutchcroft BJ, 1972	60	Male	60	Diuretics, corticosteroids	Favorable
Beaufils et al., 1975	58	Male	120	Digoxin + diuretics	Favorable
Fletcher et al., 2004	78	Female	30	Nonsteroidal anti-inflammatory drugs + corticosteroids	Favorable
Lee et al., 2008	75	Female	42	Nonsteroidal anti-inflammatory drugs	Favorable
Hendry et al., 2012	66	Female	Not given	Pericardial patch	Favorable
Lawley et al., 2013	88	Male	21	Not reported	Not known
Van Kolen et al., 2015	59	Female	14	Not reported	Not known
Feola et al., 2015	57	Male	<30	No therapy (sudden cardiac death)	Fatal
Present case	53	Male	15	Antibiotics, diuretics, corticosteroids	Favorable

Declarations of Interest

The authors declare no conflicts of interest.

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The authors state that they abide by the "Requirements for Ethical Publishing in Biomedical Journals" [12].

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