Journal of Heart

Short Communication | Vol 2 Sp. Iss 2

Case of Dresslers Syndrome in Non-ST Elevation Myocardial Infarction - A Rare Clinical Presentation

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Received Date: January 22, 2021; **Accepted Date:** February 12, 2021; **Published Date:** February 19, 2021

ABSTRACT

A 58-year-old smoker male with 2 weeks old NSTEMI, presented for cardiology opinion. He was hemodynamically stable and asymptomatic. He was taken up for coronary angiogram showing single vessel disease and advised for optimal medical therapy. 2 weeks later he presented with fever and pleuritic chest pain and pericardial rub pointing towards pericarditis. After a non STEMI that too 1 month old such a presentation is very unusual to have!

KEYWORDS

Coronary angiography; Electrocardiogram; Echocardiography; Pericardial effusion; Chest pain

ABBREVIATION

ECG: Electrocardiography; LVEF: Left Ventricular Ejection Fraction; NSTEMI: Non ST Segment Elevation Myocardial Infarction; QID: Four Times a Day

CASE REPORT

A 58-years old smoker presented to out-patient clinic with a history of typical chest pain 2 weeks back. He was diagnosed as Non-ST elevation MI and managed medically and after 2 weeks came to us for coronary angiography. At presentation he was hemodynamically stable, cardiovascular examination was unremarkable. Electrocardiogram showed normal sinus rhythm with T wave inversion in leads II, III, aVF, V5, V6 (Figure 1). Transthoracic echocardiography showed regional wall motion abnormality in inferior lateral wall with LVEF 50%. Coronary angiogram which showed single vessel disease, right dominant coronaries with total occlusion of major OM2. In view of no ongoing angina and occlusion of major OM2 he was shifted back to cardiac-care unit and was planned for stress exercise test by modified Bruce protocol. He did Treadmill exercise test with baseline pulse rate of 82/minute and blood pressure 127/80 mmHg, on stage 2 of modified Bruce protocol after a duration of 6 minutes he reached 8.4 Mets and achieved target heart rate (172/min) with no chest pain. Patient was advised to continue optimal medical therapy and to follow in cardiology out-patient clinic. After 2 weeks the patient presented to the cardiac out-patient clinic again with complaint of fever since 5 days with left sided pleuritic chest pain. On examination his pulse rate was 110/minute regular, blood pressure was 122/70 mmHg and cardiovascular examination showed high pitched pericardial rub at the base of heart. Electrocardiography done which showed generalized ST elevation in inferior and precordial leads with reciprocal depression in aVR

Citation: Gaurav Chauhan, Case of Dresslers Syndrome in Non-ST Elevation Myocardial Infarction - A Rare Clinical Presentation. J Heart 2(S2): 1-5.

(Figure 2). He was admitted and evaluated for fever. Transthoracic echocardiography showed circumferential pericardial effusion with maximum thickness of 24 mm anterior to RV (Figure 3a and Figure 3b).

Figure 1: 12 lead electrocardiogram showing normal sinus rhythm with T wave inversion in II, III, aVF and poor R wave progression in V5-V6.

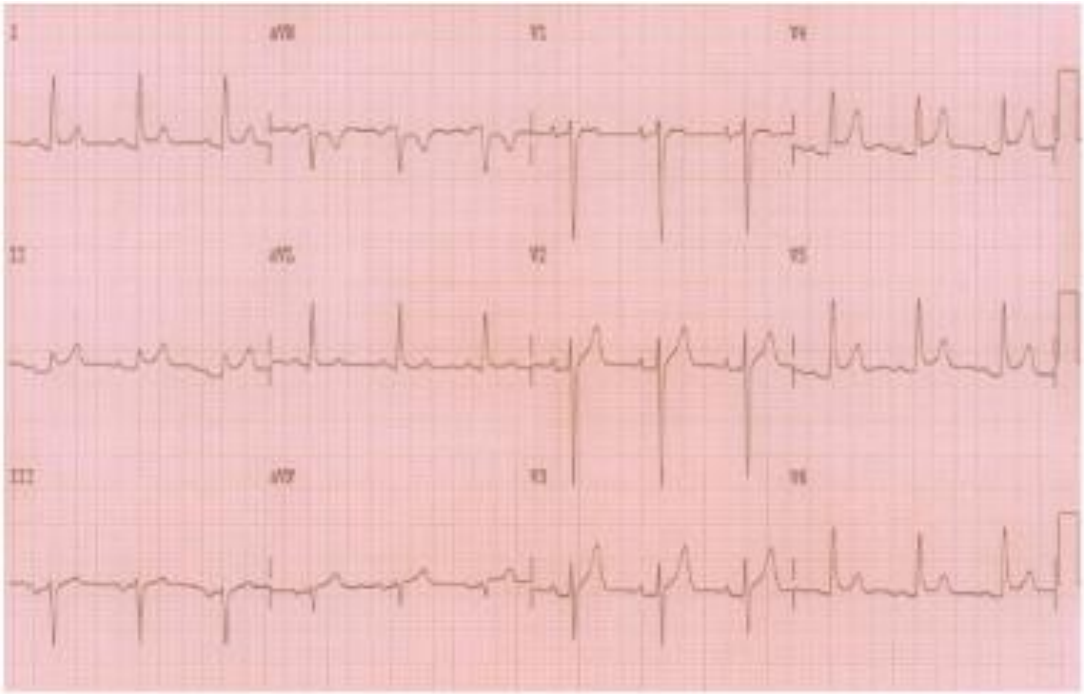

Figure 2: 12 lead electrocardiogram showing widespread concave ST elevation and PR depression is present throughout the precordial (V2-V6) and limb leads (I, II, aVL, aVF). There is reciprocal ST depression and PR elevation in aVR.

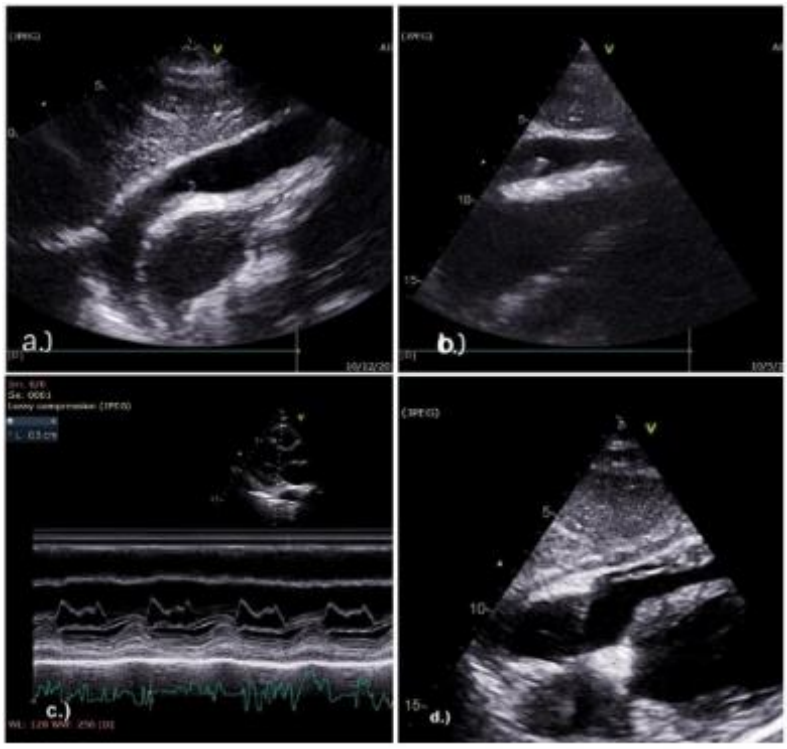

Figure 3: Transthoracic echocardiographic images. A) Showing a subcostal view of a patient with massive 24 mm pericardial effusion anterior to RV. B) Showing transthoracic echocardiography through a subcostal window with pericardial effusion anterior to RV in diastole. C) After 15 days of high dose aspirin pericardial effusion resolved 5 mm anterior to RV seen in M-mode. D) Subcostal view of transthoracic echocardiography of patient showing resolved pericardial effusion 5mm anterior to RV (RV right ventricle).

Past Medical History

No history of prior MI, diabetes, hypertension was there.

Differential Diagnosis

Post viral pericarditis with effusion.

Post myocardial injury syndrome.

Investigation

Inflammatory marker like C- reactive protein was found to be 106.08 mg/dl, total cell counts (7029/mm^3), fever workup (Malaria parasite slide test, widal/typhidot test, dengue IgM antibody and NS1 antigen assay peripheral blood smear, blood cultures, urine cultures, chest skiagram all were negative).

Management

After all clinical, electrocardiographic, echocardiographic findings and investigations a diagnosis of Dressler's syndrome was kept, and he was given a high dose aspirin 650 mg QID. No antibiotics were given throughout the hospital stay. After 15 days of therapy patient responded well and became afebrile and on serial transthoracic echocardiogram showed pericardial effusion resolved to 5 mm anterior to right ventricle (Figure 3c and Figure 3d) and C- reactive protein had fallen to 28 mg/dl and his fever resolved.

DISCUSSION

Dressler syndrome, also known as post-myocardial infarction syndrome, is a form of secondary pericarditis with or without a pericardial effusion that occurs as a result of injury to the heart or pericardium. In 1956 Dressler's syndrome was first reported as a benign triad of fever, pericarditis and pericardial effusion post-myocardial infarction [1]. More recently, this and other forms of pericardial lesions have been included in the

spectrum of 'post cardiac injury' syndromes, which includes various entities such as:

1) Postpericardiotomy syndrome (after cardiac surgery).
2) Post-traumatic pericarditis from blunting or penetrating trauma.
3) Iatrogenic causes including:
    a. Percutaneous coronary interventions.
    b. Pacemaker lead insertion.
    c. Radiofrequency ablations.

We report a case of post cardiac injury syndrome who presented with non-ST elevation myocardial infarction with the three classical features of Dressler's syndrome i.e.

1) Pleuritic chest pain.
2) Fever not explained by other cause.
3) Raised C- reactive protein.

Classically Dressler's syndrome has been associated with transmural myocardial infarction and presents early after MI within the first 7-days. In our case a patient presented after 2 weeks of ACS NSTEMI with the typical triad of Dressler's syndrome and was correctly diagnosed. He was treated successfully with high dose NSAIDS.

Pericardial effusion arises as a result of immune reaction incited by the pericardium as a result of micro bleed within the pericardium. The timeline of events was atypical for Dressler's syndrome, but all clinical features were in favour. The importance in recognizing this entity lies in the fact that regardless of the cause, Dressler's syndrome can manifest as wide clinical manifestation such as from pericarditis to pleural effusion to cardiac tamponade and very rarely as constrictive pericarditis [2,3]. Fever and pleuritic chest pain (that can simulate acute MI) and elevated acute-phase reactants are the usual findings. A pericardial friction rub on auscultation or reduced air entry in lung fields secondary to pleural effusion can often be elicited on examination. Transthoracic echocardiography and chest ski gram are the preferred imaging modalities. A latency period between the original insult and disease onset, typically between weeks and months is a distinctive but often forgotten feature of this entity [4,5]. The most accepted pathogenic mechanism is an inappropriate immune reaction, allowing the exposure of myocardial neo-antigens and the development of a delayed inflammatory response of varying extension and magnitude [5,6]. The presence of overt pericardial effusion is not a requirement, but occult micro trauma is presumed to occur [7]. Dressler's syndrome is infrequent nowadays, due to the reduction in transmural necrosis associated with reperfusion therapies and infarct size limitation. According to Cevik et al., nine cases have been reported following pacemaker implantation, between 1975 and 2009. Symptoms appeared 5 days - 56 days after the procedure and all had pleuro pericardial involvement. Six cases improved with medical therapy and three required a pericardial window. Non-steroid anti-inflammatory agents such as aspirin and ibuprofen (or alternatively colchicine) can be used, during 3 weeks - 6 weeks and gradually tapered after clinical improvement. Corticosteroids (eg. prednisolone) are usually reserved for failure of first-line treatment. Recurrence is possible after remission so colchicine can be given prophylactically to avoid recurrence [7]. Response to treatment can also be considered indicative of a correct diagnosis [7]. We conclude that although Dressler's syndrome is a rare entity nowadays but with an increasing role of intervention in cardiac diseases the post cardiac injury syndromes are also coming up with unusual presentations, which if diagnosed early are treatable and if ignored then can lead to life-threatening consequences as in our case. In our case the patient developed this rare complication of Dressler's syndrome after 4 weeks of Non-ST elevation MI and responded well to high dose aspirin. Although long-term prognosis is favourable, long-term follow-up is advisable due to the intermediate risk of constrictive pericarditis [7].

The incidence of post cardiac injury syndromes differs according to the type of insult and is reported to less than 1% post-MI and between 10% and 40% after cardiac surgery [8].

Follow Up

He followed up in an out-patient clinic and was asymptomatic, hemodynamically stable, cardiovascular and respiratory examination was unremarkable. ECG was showing a small q wave in I, aVL otherwise normal. Transthoracic echocardiography showed mild regional wall motion abnormality in the lateral wall, all valves are normal and no pericardial effusion visualized.

CONCLUSION

Post MI pericardial effusion to be evaluated in detail and managed aggressively with high dose anti-inflammatory drugs. In case of recurrence it can be managed with colchicine or corticosteroids if used judiciously.

LEARNING OBJECTIVE

Although Dressler's syndrome is very rare nowadays in the post revascularization era, also it was previously more commonly seen with transmural myocardial infarction. But as we have seen from our case that though rare and unusual with NSTEMI this has to be kept in mind especially if the patient develops chest discomfort, dynamic ECG changes or any rub on cardiac auscultation.

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