Scholars Journal of Medical Case Reports **ISSN 2347-6559 (Online)**
Sch J Med Case Rep 2015; 3(4)323-325 **ISSN 2347-9507 (Print)**

(An International Publisher for Academic and Scientific Resources)
www.saspublishers.com DOI: 10.36347/sjmcr.2015.v03i04.012

Acute Dilated Cardiomyopathy Complicated by Pulmonary Embolus Formation: A Report of Two Cases

Nikunj R Shah
Cardiology Research Fellow, Faculty of Health and Medical Sciences, University of Surrey, Guildford, UK

***Corresponding Author:**
Name: Nikunj R Shah
Email: nikunjravshah@hotmail.com

Abstract: We report two cases of an acute dilated cardiomyopathy (DCM) with each patient developing acute pulmonary emboli (PE) as a complication of severe right heart failure. This is rarely documented as a complication associated with acute DCM. One patient improved with aggressive heart failure treatment, and one patient died despite diuresis, neurohormonal blockade and inotropic support. Our case highlights the association between acute DCM and PE, particularly those with marked right ventricular involvement. This has prognostic significance and despite conflicting data regarding prophylactic anticoagulation for DCM, our report emphasises individualising the risk versus benefit by such therapeutic decisions in such a vulnerable cohort.
Keywords: Cardiomyopathy, Pulmonary embolism, Anticoagulation.

INTRODUCTION

Systemic thrombo-embolic events have been widely reported, however, pulmonary embolus (PE) formation occurring as a complication of severe right heart failure is rare in the context of an acute dilated cardiomyopathy (DCM). We report two cases of an acute non-ischaemic DCM with each patient developing an acute PE.

CASE REPORT

Case 1

A 42 year old Caucasian lady presented with a 3 month history of exertional dyspnoea following a viral illness. She reported a drastic decrease from a previously normal exercise tolerance to 20 metres on flat ground, orthopnoea and bipedal oedema. Past medical history included a 30 pack year smoking history. The patient was not taking any regular medications, had no significant family history of cardiac disease and denied toxin exposure. Physical examination corroborated the chest X-ray findings of pulmonary oedema. Electrocardiogram (ECG) revealed a narrow QRS complex, sinus tachycardia with lateral T-wave inversion. Troponin T titres were negative (<0.04µg/L) and brain natriuretic peptide (BNP) was elevated at 2593 picograms per millilitre (age adjusted normal value <40pg/ml). Echocardiography demonstrated severe global bi-ventricular impairment with dilatation. The calculated left ventricular ejection fraction (LVEF) was 15% (Fig. 1A). Phaeochromocytoma was excluded from 24-hour urinary catecholamines. Cardiac catheterisation demonstrated an unobstructed left coronary circulation, chronic subtotal stenosis of a non-dominant right coronary artery with extensive collateralisation and hence coronary artery disease was not considered as the predominant cause of her biventricular failure. A viral DCM was therefore diagnosed. Following an episode of witnessed haemoptysis a computed tomography pulmonary angiogram (CTPA) was performed revealing multiple pulmonary arterial emboli, the largest being in the right lower lobe artery (Fig. 1B). The patient further developed a subacute right axillary vein thrombosis and was initiated on warfarin following a negative thrombophilia screen. Although initially considered for a left ventricular assistance device (LVAD), she symptomatically improved and was discharged home for out-patient follow-up and optimisation of heart failure medications prior to consideration for an implantable cardio-defibrillator.

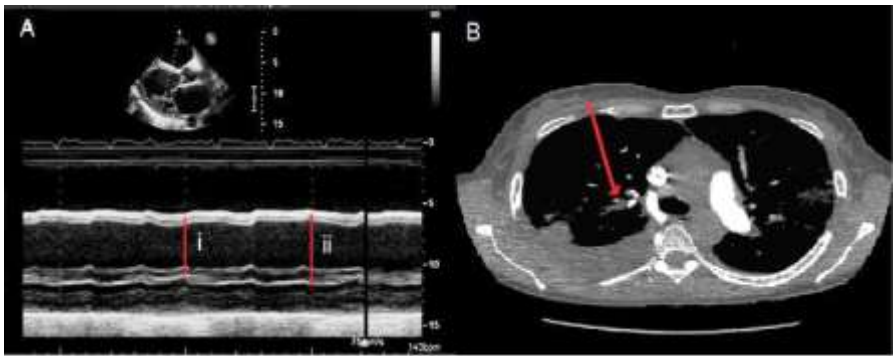

Fig. 1: A) M-mode echocardiographic parasternal long axis view indicating minimal change in dilated LV dimension in systole (i) and diastole (ii), equating to an EF of 15%; B) CT pulmonary angiogram revealing multiple pulmonary arterial emboli, with the arrow demonstrating a prominent right lower lobar arterial embolus.

Case 2

A previously fit 53 year old Afro-Caribbean man presented with a 10 day history of generalised lethargy, orthopnoea, dyspnoea and haemoptysis. The patient was adopted as a child and with no biological parental contact, a family history of cardiac disease was not available. Admitting ECG revealed poor R wave progression as the sole abnormality. Troponin T titre was 0.09µg/L, BNP was 2191pg/ml and echocardiography demonstrated severe global bi-ventricular impairment with moderate dilatation. Calculated EF was 14%. HIV, viral and an atypical pneumonia screen were negative. Broad spectrum antibiotics were initiated as the patient became more dyspnoeic with a chest X-ray suggestive of a bronchiolitis obliterans organising pneumonia. Lung field images on CTPA in fact demonstrated numerous peripheral rhomboid areas of pulmonary infarction (Fig. 2). Coronary angiography revealed unobstructed coronary arteries. He was anticoagulated with low molecular weight heparin, not warfarin, because of deranged liver function tests secondary to hepatic congestion. Due to cardiogenic decompensation along with worsening chest septicaemia, inotropes were initiated. The patient was deemed unsuitable for LVAD therapy with a concomitant infection and a multidisciplinary consensus was reached that he was too unstable for emergency heart transplant surgery. Despite aggressive diuresis, neurohormonal blockade and inotropic support, the patient eventually died from multi organ failure.

Post mortem revealed widespread fibrosis throughout both ventricles with adherent reorganising right ventricular thrombus. Lack of acute inflammatory changes within the myocardium excluded an infective aetiology but demonstrated the characteristic appearance of an advanced idiopathic DCM. Pathological examination of the lungs revealed extensive areas of pulmonary infarction.

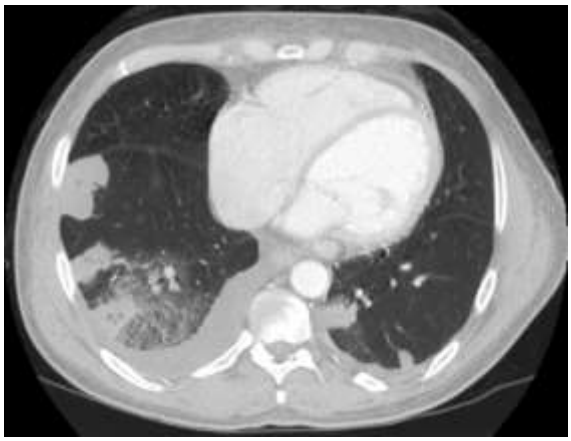

Fig. 2: Multiple areas of consolidation and pulmonary infarctions

DISCUSSION

The acute presentation of severe DCM complicated by large PE has rarely been previously reported [1-3]. Early management of complications such as heart failure improves outcomes [4], however little has been explored regarding the specific risk of PE formation in acute DCM.

The relative risk of PE in systolic heart failure is at least double that of the unaffected population, with risk increasing as LV systolic function decreases. This increased risk in heart failure has been attributed to reduced flow caused by a low cardiac output state [5], abnormalities of haemostasis, platelet dysfunction, and endothelial dysfunction [6]. Acute viral myocarditis causing a systemic inflammatory response compounds a prothrombotic state, as shown with the patient in case 1 developing an axillary vein thrombosis in addition to PE.

CONCLUSION

Our report emphasises that patients with acute DCM and marked right ventricular (RV) involvement may be at significant risk of PE. Post mortem findings in case 2 suggest that a markedly dilated RV acted as substrate for the thrombo-embolic complications that ensued in a hypercoagulable patient. Although there is conflicting data regarding prophylactic anticoagulation for DCM [7, 8], our cases highlight that individual risk versus benefit by such therapeutic decisions must be weighed in such a vulnerable cohort.

Abbreviations

BNP - Brain natriuretic peptide; CTPA - Computed tomography pulmonary angiogram; DCM - Dilated cardiomyopathy
ECG – Electrocardiogram; LVAD - Left ventricular assistance device; LVEF - Left ventricular ejection fraction; PE - Pulmonary embolus; RV - Right ventricular

REFERENCES

1. Aguanne E; Peripartum cardiomyopathy presenting with pulmonary embolism: An unusual case. South Med J., 2008; 101(6): 646-647.
2. Missault L, Koch A, Colardyn F, Clement D; Biventricular thrombi in dilated cardiomyopathy: massive simultaneous pulmonary and systemic embolisation. Eur Heart J., 1994; 15(5): 713-714.
3. Haine SE, De Ridder SM, Van de Vijver KK; Images in cardiology. Ventricular thrombi with pulmonary and systemic embolization. Can J Cardiol., 2008; 24(11): e92.
4. Dennert R, Crijns HJ, Heymans S; Acute viral myocarditis. Eur Heart Journal, 2008; 29(17): 2073-2083.
5. Rho R, DeNofrio D, Loh E; Cardiomyopathy and embolization: risks and benefits of anticoagulation in sinus rhythm. Current Cardiology Reports, 2000; 2(2): 206-211.
6. Weikert U, Kuhl U, Schultheiss HP, Rauch U; Platelet activation is increased in patients with cardiomyopathy: myocardial inflammation and platelet reactivity. Platelets, 2002; 13(8): 487-491.
7. Massie BM, Collins JF, Ammon SE, Armstrong PW, Cleland JG, Ezekowitz M *et al*.; Randomized Trial of Warfarin, Aspirin, and Clopidogrel in Patients With Chronic Heart Failure. The Warfarin and Antiplatelet Therapy in Chronic Heart Failure (WATCH) Trial. Circulation, 2009; 119(12): 1616-1624.
8. Sirajuddin RA, Miller AB, Geraci SA; Anticoagulation in patients with dilated cardiomyopathy and sinus rhythm: a critical literature review. J Card Fail., 2002; 8(1): 48-53.