CASE REPORT

Reversible cardiac dysfunction in long-standing hypertension may be global variant of stress cardiomyopathy

Anand Chockalingam, Senthil A Kumar

Division of Cardiovascular Medicine, University of Missouri, Columbia, Missouri, USA

Correspondence to

Dr Anand Chockalingam, chockalingama@missouri.edu

Accepted 28 May 2018

SUMMARY

An adult man with long-standing poorly controlled cardiac risk factors presented with acute decompensated heart failure (ADHF). Echocardiogram, cardiac MRI and catheterisation suggested idiopathic dilated cardiomyopathy, severe systolic dysfunction, ejection fraction 25% with global left ventricular (LV) dilation and apical thrombus. He responded well to diuretics and gradual uptitration of lisinopril and carvedilol. Follow-up echocardiogram in 2 months demonstrated complete recovery of systolic function, normalisation of LV size and shape with severe LV hypertrophy. This presentation is potentially a global variant of stress cardiomyopathy with recovery of LV function, highlighting the importance of appropriate imaging, catheterisation and clinical monitoring in patients with ADHF.

BACKGROUND

Available literature is not clear about reversible left ventricular (LV) dysfunction which can be a manifestation of uncontrolled hypertension. Understanding this would improve patient care and reduce procedures.

CASE PRESENTATION

A 48-year-old white male smoker with long-standing and poorly controlled hypertension, hyperlipidaemia presented with 2 weeks of progressively worsening dyspnoea and non-productive cough. He was afebrile and in mild respiratory distress, with a heart rate of 110 beats/minute and elevated blood pressure (BP) at 176/120 mm Hg. Clinical examination did not reveal murmurs or gallops, and crackles were noted in the lung bases. ECG revealed sinus tachycardia at 101 beats/minute, left atrial enlargement and LV hypertrophy with secondary ST-T wave changes. Chest X-ray showed cardiomegaly with alveolar pulmonary oedema, and N terminal-pro Brain Natriuretic peptide (NT-proBNP) was elevated at 2539 pg/mL. Troponin T peaked at 0.02 ng/mL. An echocardiogram revealed moderate-to-severe global hypokinesis of the LV, ejection fraction (EF) estimated at 30%, a 19×7 mm thrombus in the LV apex and a mildly dilated left atrium with tissue Doppler features of diastolic dysfunction (figures 1 and 2, video 1). Acute decompensated heart failure (ADHF) responded appropriately to aggressive diuresis with intravenous furosemide, with over 7

L volume removal in the next few days. Symptoms and vitals stabilised, and the patient tolerated initiation of lisinopril, carvedilol, spironolactone and warfarin during the hospitalisation. Cardiac MRI showed a moderately dilated LV with mild-to-moderate concentric LV hypertrophy with wall thickness of 13 mm. LV systolic function was severely reduced with quantitative EF of 22% and severe global hypokinesis. The right ventricle (RV) was also dilated with moderately reduced systolic function. There was non-specific fibrosis at the RV insertion point and mid-inferolateral wall with a small focal transmural myocardial infarction at the inferoapex. A 7×6 mm LV thrombus was attached to the apex (figure 2). Cardiac MRI suggested mixed aetiology of cardiomyopathy with features suggestive of a non-ischæmic dilated cardiomyopathy (DCM) in addition to the presence of ischaemic focal transmural myocardial infarction (figures 3–5, video 2). Cardiac catheterisation revealed non-obstructive coronary artery disease with 20% lesions in proximal and mid left anterior descending (LAD) coronary artery, as well as 40% diagonal branch disease with only mild luminal irregularities of the other vessels.

OUTCOME AND FOLLOW-UP

The patient was compliant with uptitration of lisinopril and carvedilol, quit smoking and was without cardiac symptoms at follow-up. Repeat echocardiogram at 2 months demonstrated normalisation of LV size, severe concentric hypertrophy with wall

Figure 1 Parasternal long-axis view demonstrating left ventricular (LV) dilatation, moderate LV hypertrophy.

To cite: Chockalingam A, Kumar SA. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2018-225044

Figure 2 Apical four chamber view demonstrating the 19×7 mm thrombus in the left ventricular apex.

thickness of 18 mm. Regional wall motion was normal, with EF improved to 55% and resolution of LV thrombus (figure 6, video 3). BP, which was in the normal range during the hospital course, was elevated again at 160/108 mm Hg. Warfarin and furosemide were discontinued, and hydrochlorothiazide was added for targeting optimal BP control <130/80 mm Hg with frequent home monitoring.

DISCUSSION

Current day clinical practice offers several imaging options to understand and optimally manage cardiac dysfunction and heart failure. This case illustrates several clinical challenges that remain despite using state-of-the-art imaging tools and invasive catheterisation. In retrospect, the most important clues to the underlying hypertensive cardiomyopathy were the history of 20 years of poor BP control, severe hypertension at presentation despite LV dysfunction, and ECG features of LV hypertrophy and secondary ST-T changes. Both echocardiogram and cardiac

Video 1 Parasternal long-axis view demonstrating left ventricular (LV) dilatation and moderate LV hypertrophy with severe LV dysfunction, ejection fraction 30%, global hypokinesia.

Figure 3 Cardiac MRI demonstrates the distal inferior wall focal transmural infarction and apical thrombus.

MRI demonstrated severe LV systolic dysfunction, global hypokinesia and only mild LV hypertrophy. The overall cardiac MRI findings were consistent with non-ischaemic DCM with superimposed embolic myocardial infarction at the watershed zone between LAD and right coronary artery territories.¹ The invasive coronary angiogram was also supportive of these cardiac MRI findings.

LV hypertrophy is widely recognised as an imaging marker of end-organ damage from hypertension. Doppler evidence for diastolic dysfunction and clinical heart failure with preserved EF are common manifestations in long-standing hypertension.² Nearly half of the cases of DCM are ischaemic in origin. Prior

Figure 4 Cardiac MRI demonstrates the right ventricular insertion site scar.

Figure 5 Cardiac MRI demonstrates the non-specific mid-lateral wall scar.

viral myocarditis, alcohol or hypertension are the common aetiologies in clinical practice for many of the patients manifesting with idiopathic cardiomyopathy.³ However, we did not come across any instance in the published literature where hypertension caused this type of clinical picture with LV remodelling and scarring pattern of DCM. Retrospectively, we believe our patient's hypertension caused severe concentric hypertrophy but when he developed heart failure, the picture was more suggestive

Video 2 Cardiac MRI showing severely reduced left ventricular systolic function with quantitative ejection fraction of 22% and severe global hypokinesis.

Figure 6 Parasternal long-axis view demonstrating normalisation of left ventricular (LV) size, severe LV hypertrophy.

of DCM with severe systolic dysfunction and delayed enhancement characteristics.

We have published our experience with reversible cardiomyopathies in patients with critical illness due to stress cardiomyopathy (SC), inotropes, metabolic derangements and in postcardiac arrest situations.⁴ Irrespective of the wall motion abnormality, we have defined SC as acute reduction in cardiac function oftentimes due to mental or physical stress with spontaneous complete normalisation of cardiac function within days to weeks.⁵ We strongly believe several instances of SC in the critically ill manifest as global LV dysfunction. Global SC is not adequately represented in the international registries as catheterisation is not routinely performed in this group. If we consider hypertension as an acute stressor, treating LV dysfunction and heart failure with ACE inhibitors, beta blockers and diuretics all reduce BP and thereby relieve afterload stress. This patient arguably could have had hypertension-induced SC which normalised with adequate BP control.⁵

Video 3 Parasternal long-axis view demonstrating normalisation of left ventricular (LV) size, severe LV hypertrophy. LV ejection fraction improved to 55%.

This case illustrates the clinical challenges in determining aetiology and a definitive diagnosis despite echocardiogram, cardiac MRI and invasive cardiac catheterisation. Another consideration in this situation is acute myocarditis with transient LV dysfunction that later resolved. However, acute myocarditis is unlikely in this patient given that oedema would have further increased LV wall thickness compared with the subsequent echocardiogram; typically, there is insufficient time for ventricular dilatation which was seen in this patient; and the small extent of myocardial enhancement relative to the severity of LV dysfunction. As myocardial biopsy often does not change the clinical care in suspected myocarditis, it is deferred unless the patient fails to respond to appropriate therapy.⁶ Coronary angiography is a crucial step early in definitively excluding ischaemic aetiology of LV dysfunction.

In conclusion, we suggest high clinical suspicion for a wider differential diagnosis when patients initially present with LV

dysfunction and clinical heart failure. Serial echocardiograms may help minimise more invasive or extensive testing. Global variant SC is likely much more prevalent than widely recognised. Early invasive testing with coronary angiogram and cardiac MRI help optimise management in these complex situations.

Contributors AC: evaluated the patient, managed therapy and wrote the manuscript. SAK: suggested edits to the manuscript, helped in appropriately diagnosing this rare presentation and collected the key images for publication.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

© BMJ Publishing Group Ltd (unless otherwise stated in the text of the article) 2018. All rights reserved. No commercial use is permitted unless otherwise expressly granted.

Learning points

- ▶ Hypertension related left ventricular (LV) hypertrophy may be challenging to recognise when the LV dilates with systolic dysfunction.
- ▶ Cardiac MRI helps differentiate ischaemic and idiopathic cardiomyopathy, but may sometimes reveal a 'mixed' picture.
- ▶ Cardiac catheterisation remains the best option to definitively exclude coronary artery disease in patients with cardiomyopathy.
- ▶ Hypertensive LV dysfunction may be viewed as a global form of stress cardiomyopathy which can reverse with antihypertensives.

REFERENCES

- 1 Friedrich MG, Sechtem U, Schulz-Menger J, *et al.* Cardiovascular magnetic resonance in myocarditis: A JACC White Paper. *J Am Coll Cardiol* 2009;53:1475–87.
- 2 Lalande S, Johnson BD. Diastolic dysfunction: a link between hypertension and heart failure. *Drugs Today* 2008;44:503–13.
- 3 Yancy CW, Jessup M, Bozkurt B, *et al.* 2016 ACC/AHA/HFSA Focused Update on New Pharmacological Therapy for Heart Failure: An Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Failure Society of America. *J Am Coll Cardiol* 2016;68:1476–88.
- 4 Chockalingam A, Mehra A, Dorairajan S, *et al.* Acute left ventricular dysfunction in the critically ill. *Chest* 2010;138:198–207.
- 5 Chockalingam A. Stress cardiomyopathy of the critically ill: Spectrum of secondary, global, probable and subclinical forms. *Indian Heart J* 2018;70:177–84.
- 6 Yancy CW, Jessup M, Bozkurt B, *et al.* 2013 ACCF/AHA guideline for the management of heart failure: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. *Circulation* 2013;128:1810–52.

Copyright 2018 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow