

A case of left bundle branch block with chest pain but negative troponin: a prompt for dilated cardiomyopathy?

Authors: Tha Htet Nyi^A and Hnin Zaw^A

Introduction

Chest pain with left bundle branch block (LBBB) on electrocardiography (ECG) is an alarming presentation in the acute medical unit, even without a rise in troponin. If acute coronary syndrome is unlikely, it is advised to consider common but overlooked differential diagnoses of LBBB such as dilated cardiomyopathy (DCM). The following case, which we came across in the acute medical unit, was a reminder to do so.

Case presentation

This previously well man in his mid-40s presented with a 1-week history of intermittent central chest pain, which was of a dull ache in nature with no radiation, exacerbated on exertion and by sitting forward. There was no report of preceding prodromal viral illness, no common cold- or flu-like symptoms and no shortness of breath. He had no previous cardiac problems and had never reported chest pain before. He was an ex-smoker and admitted to occasional use of recreational drugs, but no evidence of consistent exposure to cardiotoxic materials. There were no cardiac problems of note in his first-degree relatives and his exercise tolerance was then essentially normal. On clinical examination, he was relatively hypotensive and bradycardic, but results were otherwise unremarkable. An ECG revealed LBBB with sinus rhythm, no evidence of ischaemia. Troponin was negative two times and blood results were not remarkable, apart from a cholesterol of 5.6 mmol/L. A chest X-ray unveiled a borderline-enlarged heart and subsequent echocardiography revealed a severely dilated left ventricle (LV) with dimensions of 7.6 cm, along with a dilated left atrium and globally severely impaired left ventricular ejection fraction (LVEF) of 20%. For this, he was started on beta-blocker and angiotensin-converting enzyme (ACE) inhibitors. Echocardiography was repeated 1 month later, and showed no improvement in LV systolic function. Meanwhile, it was arranged for him to have a cardiac magnetic resonance imaging (MRI) to find out the underlying cause of DCM, and screening of his first-degree relatives was initiated.

Discussion

A 2016 cohort study illustrated that LBBB commonly presents with dyspnoea (30%) followed by chest pain (25%).¹ In addition, a quarter of patients were found to have DCM on echocardiography.¹ Evidence of LBBB having a negative impact on LVEF in the long term, subsequently leading to DCM, was also reviewed. A retrospective study revealed that almost 10% (9.8%) of patients with LBBB with preserved LVEF subsequently developed a significant decline in LV systolic function to an LVEF of approximately 32% over 4–5 years.² In terms of management of reduced LVEF in patients with LBBB, cardiac resynchronisation therapy (CRT) plays a more beneficial role (35% of patients in the study sample experienced an improved LVEF >50% post-CRT) compared with optimal medical therapy (only 6% of patients in the study sample benefited from improvement of LVEF after 3 months).³

Conclusion

Whenever ischaemic aetiology is unpromising, it is equally important to consider LBBB-associated non-ischaemic cardiomyopathies, as they can be overlooked and associated with underlying significant LV systolic dysfunction with subtle clinical features. Appropriate and timely specialist input will make a monumental difference to the patient's safety and quality of life, particularly in the long run. ■

Conflicts of interest

None declared.

References

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Authors: ^ANorfolk and Norwich University Hospitals NHS Foundation Trust, Norwich, UK