Abstract: P1904

Hypertrophic cardiomyopathy and dynamic left ventricular outflow tract obstruction, a possible pathophysiological mechanism of injury in takotsubo cardiomyopathy.

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Topic(s):
Hypertrophic Cardiomyopathy

Citation:

Background

The aetiology of Takotsubo cardiomyopathy (TCM) is unclear and there have been case reports showing underlying features of hypertrophic cardiomyopathy (HCM) with left ventricular outflow tract obstruction is present in some cases of TCM. This has been postulated to be part of pathophysiological mechanism, as catecholamine driven left ventricular outflow tract obstruction, coupled with coronary microvascular spasm may contribute to myocardial stunning and apical ballooning seen in TCM. We report a case of underlying hypertrophic cardiomyopathy masked by Takotsubo cardiomyopathy.

Case

A 63 year old lady with a background of depression was admitted to hospital with severe chest pain that came on following the sudden onset of right calf pain. This was associated with sweating and nausea. She recently returned from America after visiting her son nine days ago. She did not report any recent bereavement. Pulmonary embolism was suspected.

Physical examination was normal. The admission ECG showed deep T wave inversion in anterolateral leads with a raised Troponin-T level of 492.9ng/L. Other blood tests including renal function, full blood count and c-reactive protein were normal, in particular D-Dimer 85 (normal range 0-229). Coronary angiogram showed unobstructed coronary arteries. Transthoracic Echo (TTE) demonstrated mid to apical akinesis of the left ventricle with preservation of basal contraction and an overall ejection fraction of (LVEF) 35-44%, features typical of Takotsubo when in the presence of unobstructed coronary arteries.

A cardiac MRI scan three months later demonstrated normal biventricular function with mild asymmetrical left ventricular hypertrophy with a basal septal bulge and mild chordal SAM suggestive of hypertrophic cardiomyopathy. Late gadolinium showed mild atypical enhancement in the basal to mid inferolateral segments. She did not have systemic hypertension.

Follow up TTE in a years’ time revealed normal left ventricular ejection fraction with mild hypertrophy of the basal septum and turbulent flow in the left ventricular outflow tract (LVOT), features consistent with HCM.

Conclusion

There have been previous case reports linking hypertrophic cardiomyopathy and Takotsubo cardiomyopathy, and this case adds to the growing evidence that basal septal hypertrophy and catecholamine driven LVOT obstruction may be part of the pathophysiological mechanism underlying Takotsubo cardiomyopathy. Therefore, we suggest a careful diagnostic approach and consideration of cardiac MRI to identify the concurrence of these two conditions.
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