Syncope and cardiogenic shock in an 80-year-old woman

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DESCRIPTION
An 80-year-old woman presented with a 2-day history of breathlessness and syncope. Her medical history included Parkinson’s disease and a recent diagnosis of myelodysplasia for which she had undergone a (painful) bone marrow aspiration 2 days earlier.

Presentation blood pressure (BP) was 69/38 mm Hg (and fell further to 59/40 mm Hg with impaired conscious level), heart rate 78, respiratory rate 17 and oxygen saturation 100% on air. Her presentation ECG revealed anteroseptal Q waves with 1 mm ST segment elevation (figure 1).

Emergency primary percutaneous coronary intervention was declined due to the probability of established myocardial infarction with consequent cardiogenic shock. Medical treatment and inotropic therapy with intra-aortic balloon pump (IABP) were directed. She underwent emergency portable echocardiography to understand the aetiology for cardiogenic shock.

The panel of images (figure 2A–C) reveals evidence of basal hyperkinesia, systolic anterior motion (SAM) of the anterior mitral valve leaflet (causing left ventricular outflow tract obstruction (LVOTO)) and severe mitral regurgitation (MR) which was the cause of profound hypotension. There was coexistent apical ballooning/akinesia of the left ventricular apex consistent with a diagnosis of Takotsubo cardiomyopathy.

A radial arterial line was inserted. Intravenous fluids combined with oral metoprolol (25 mg four times a day) resulted in gradual increase in systolic BP from 59 to 100 mm Hg over 12 hours. Repeat echocardiogram 72 hours later (figure 2D) revealed resolution of SAM and LVOTO with residual mild MR. Cardiac MRI (CMR) (figure 2E) 1 week after presentation revealed complete resolution of SAM, LVOTO and MR with no evidence of scarring or fibrosis, thus excluding myocardial infarction. Left ventricular function also had returned to normal (left ventricular ejection fraction >55%). Also, there was no evidence of proximal coronary artery occlusion on axial half-Fourier acquisition single-shot turbo spin-echo imaging.

Haemodynamic instability characterised by SAM of the mitral valve, LVOTO, severe MR and cardiogenic shock has previously been described in patients with Takotsubo’s syndrome.1 2 One of the largest series of Takotsubo cardiomyopathy described nine patients with LVOTO with marked reductions in gradients with intravenous esmolol.3 As far as we are aware, no previous description of dynamic LVOTO obstruction associated with Takotsubo has described BPs as low as in this case with altered conscious level. Second, the case is unique as it illustrates the rapidity of recovery with appropriate therapy. CMR confirmed absence of myocyte necrosis/infarction and complete resolution of SAM, LVOTO and MR and restoration of normal left ventricular systolic function within 7 days of presentation. Despite profound hypotension, this case highlights the effectiveness of beta blockade in reducing basal hyperkinesia and lessening LVOTO obstruction.

[Figure 1 A 12-lead ECG at presentation.]
It is important to avoid inotropic agents and IABP as LVOTO will be exacerbated.

Contributors TO and AK wrote the manuscript. TO undertook the literature search. NC was the sonographer who undertook the bedside echo, who selected the images and critiqued the manuscript. AK analysed the echo and directed treatment (as the consultant on call).

Competing interests AK has the following potential conflicts of interest: free-of-charge material from ROCHE for research; submissions pending for research funds from ROCHE and Abbott; funding for national conferences from Daiichi Sankyo and Bayer; funding for research from Bayer Pharmaceuticals; speaker and expert consultation fees from AstraZeneca, Menarini and Bayer.

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REFERENCES

Learning points
- Takotsubo cardiomyopathy can present with syncope and cardiogenic shock, particularly when associated with left ventricular outflow tract obstruction (LVOTO) and mitral regurgitation.
- Bedside echo can elucidate the aetiology and obviate potentially harmful treatment such as inotropic therapy and intra-aortic balloon pump which will exacerbate LVOTO.
- Despite profound hypotension, low-dose, titrated beta blockade can lessen LVOTO and improve haemodynamics.

and MR. It is important to avoid inotropic agents and IABP as LVOTO will be exacerbated.

Figure 2  (A) Still image of four-chamber transthoracic echocardiogram in systole. Arrow depicts systolic anterior motion of anterior mitral valve leaflet with narrowing of left ventricular outflow tract. (B) Identical still colour image of four-chamber transthoracic echocardiogram. Pathologies demonstrated include flow acceleration in left ventricular outflow tract (superior arrow) and severe mitral regurgitation (right lower arrow) and tricuspid regurgitation (left lower arrow).  (C) Pulsed wave Doppler trace of left ventricular outflow tract revealing severe obstruction (high velocities).  (D) Pulse wave Doppler signal of left ventricular outflow tract 72 hours after presentation revealing resolution of left ventricular outflow tract obstruction (normal velocities).  (E) Four-chamber cardiac MRI (1 week after presentation) late enhancement image 10 min after injection of 0.1 mmol/kg of Gadovist confirming absence of fibrosis or infarction of left ventricular myocardium.