The Obstructed Path: Takotsubo Cardiomyopathy with Severe Mitral Regurgitation Due to Dynamic LVOT Obstruction

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Clinical Presentation

A 77-year-old woman with acute coronary syndrome developed hypotension requiring escalating norepinephrine doses and hypoxia despite stenting the "culprit" RCA.

Imaging Findings

TTE demonstrated normal LV size and EF of 35% with an akinetic apex and hypercontractile base, consistent with takotsubo cardiomyopathy (TCM). There was marked LV outflow tract obstruction (LVOTO) due to systolic anterior motion (SAM) of the mitral valve with resting gradient of 57 mmHg. There was severe MR secondary to SAM from LVOTO; mitral leaflets were structurally normal.

Summary/Discussion Points

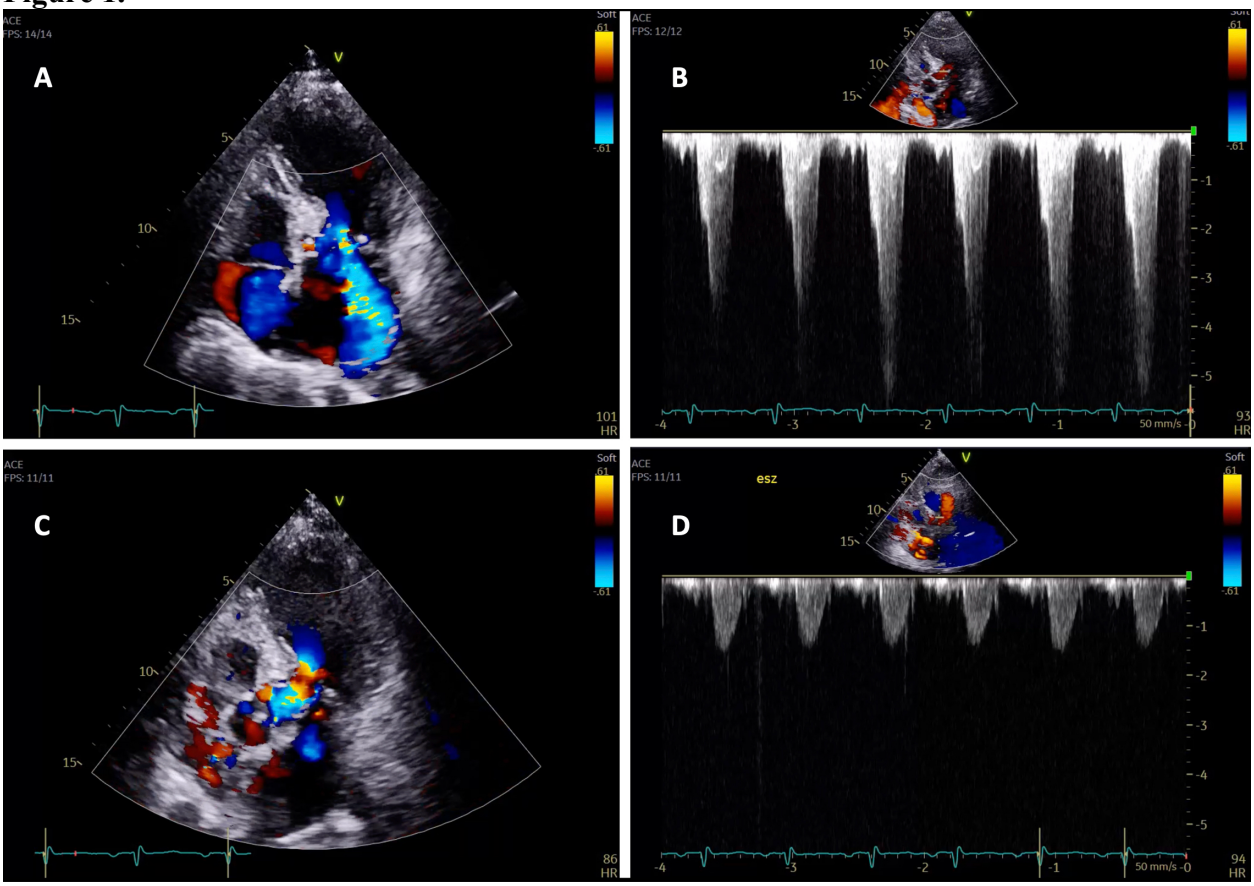
Norepinephrine was switched to phenylephrine, which abolished the resting LVOT gradient and decreased MR to mild. The patient stabilized, but continued to need high-flow oxygen. Attempts were made to wean phenylephrine under bedside TTE guidance to monitor LVOT gradient and MR in response to adjusting phenylephrine doses. Any attempt to wean phenylephrine resulted in worsening hypoxia, rising LVOT gradient, and severe MR. Esmolol was attempted, but was unsuccessful due to hypotension. Pulmonary artery catheter (PAC) revealed cardiac index of 2.65 L/min/m^2 (3-5 L/min/m^2), SVR of 1584 DS/cm^5 (770-1500 DS/cm^5), and borderline reduced filling pressures.

She received volume repletion, but despite improvement in CVP and PAWP, she remained phenylephrine dependent. She had normal thyroid and adrenal function, and was empirically treated for pneumonia with no improvement in hypotension.

One week post admission, while still phenylephrine-dependent, a PAC and TTE-guided attempt to wean phenylephrine was pursued. Phenylephrine dose was lowered and esmolol was added when LVOTO and severe MR recurred. Esmolol was gradually transition to metoprolol and phenylephrine was discontinued on post-admission day 15. Subsequent hospital stay was uncomplicated. Repeat TTE in 4 months showed normal LV function and wall motion, and no LVOTO, SAM, or MR.

While SAM and severe MR has been shown in TCM in small cohort studies, persistent association with over 2-week dependence on vasopressors has never been described. Our case serves as a reminder of the potential for TCM to cause cardiogenic shock secondary to severe LVOTO and severe MR in the context of pre-existing septal hypertrophy. Emergent TTE was essential to making the diagnosis and guiding management in this case.

Figure 1.



(A) Severe mitral regurgitation (MR) with posteriorly directed jet and **(B)** marked left ventricular outflow tract (LVOT) obstruction due to systolic anterior motion of mitral valve with resting gradient 57mmHg. With IV phenylephrine at 100mcg/min, **(C)** improvement of MR to mild and **(D)** abolished LVOT gradient.