Canadian Journal of Cardiology 35 (2019) 229.e7–229.e9 www.onlinecjc.ca

Case Report

Stress-Induced Cardiomyopathy Complicated by Dynamic Left Ventricular Outflow Obstruction, Cardiogenic Shock, and Ventricular Septal Rupture

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ABSTRACT

We describe the case of a 68-year-old woman presenting with stress cardiomyopathy (SCM), with concomitant cardiogenic shock, left ventricular outflow tract obstruction, and ventricular septal rupture. These complications have not simultaneously been reported in a single SCM case. The importance of early diagnosis of serial complications of SCM and using mechanical circulatory support as a treatment strategy are highlighted.

RÉSUMÉ

Nous décrivons le cas d'une femme âgée de 68 ans présentant une cardiomyopathie de stress (CMS) et, en concomitance, un choc cardiogénique, une obstruction de la voie d'éjection ventriculaire gauche et une rupture du septum interventriculaire. Ces complications n'avaient pas été rapportées simultanément dans un même cas de CMS. Nous insistons sur l'importance d'un diagnostic précoce des complications sérielles de la CMS et du recours à un soutien circulatoire mécanique à titre de stratégie de traitement.

Stress cardiomyopathy (SCM) is often characterized by chest pain, troponin elevation, and apical ballooning. It mimics acute myocardial infarction, but coronary angiography reveals nonobstructive disease. Although prognosis is favourable, rare but severe complications can occur, including heart failure, cardiogenic shock, left ventricular outflow obstruction, arrhythmias, valvulopathy, and ventricular rupture.

Case Report

A 68-year-old woman with hypertension and dyslipidemia, grieving her daughter's sudden death, presented with 1 hour of severe chest pain. Electrocardiogram showed anterolateral ST-segment elevation. Troponin T was 3.5 ng/mL. After administration of 35 mg tenecteplase, she was transferred to our center for right and left cardiac catheterization. This revealed minimal coronary disease, but an atypical flow pattern (Fig. 1, A and B); elevated left ventricular end-diastolic pressure (LVEDP = 28 mm Hg); and cardiogenic shock (cardiac index [CI] = 1.7 L/min/m^2 [height 165 cm, weight 70.9 kg, body surface area 1.80 m^2], pulmonary capillary wedge pressure = 27 mm Hg, and systemic vascular resistance of 2200 dyn·s/cm^5). An intra-aortic balloon pump (IABP) was placed intraprocedure. Milrinone and diuretics were initiated. Subsequent echocardiography showed reduced left ventricular ejection fraction (< 20%), apical ballooning, and a hyperdynamic base. Unexpectedly, severe dynamic left ventricular outflow tract (LVOT) obstruction (peak resting gradient = 94 mm Hg, LVOT diameter = 1.95 cm) with sinus tachycardia and systolic anterior motion of the mitral apparatus was noted (Fig. 1, C and D). IABP counterpulsation and milrinone were discontinued because both cause deleterious afterload reduction in this context. A trial of phenylephrine and

Received for publication May 15, 2017. Accepted December 5, 2018.

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See page 229.e9 for disclosure information.

https://doi.org/10.1016/j.cjca.2018.12.005

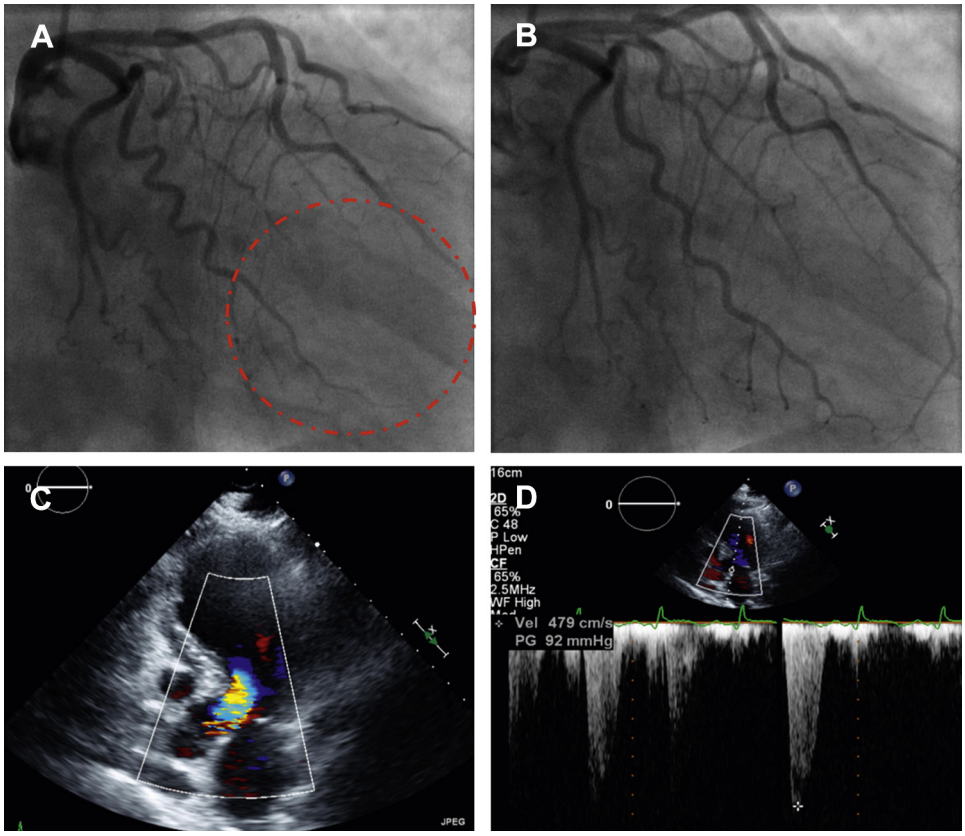

Figure 1. (**A**) Left coronary angiography demonstrating impaired diastolic apical coronary flow (**red circle**). (**B**) Angiographic image within 2 frames of image (**A**) demonstrating patency and complete systolic coronary filling. (**C**) Transthoracic echocardiogram (TTE) spectral Doppler tracing demonstrating high-velocity left ventricular outflow tract (LVOT) flow. (**D**) TTE color Doppler image demonstrating LVOT flow acceleration and apical ballooning.

esmolol to relieve LVOT obstruction was unsuccessful. Hemodynamic optimization was ultimately achieved with percutaneous mechanical circulatory support (PMCS) using an Impella CP (ABIOMED, Danvers, MA) (placed by standard right femoral artery cannulation with a 14F peel-away catheter). Device flow rates were iteratively adjusted to optimize CI; at the P4 setting, the outflow gradient was reduced to 15 mm Hg, the CI improved to 2.6 L/min/m^2, and the lactate normalized (Fig. 2A). Serial bedside echocardiography performed multiple times per day confirmed stable positioning of the Impella device 3.6 to 4.0 cm from the aortic valve, and no adjustment was necessary. On day 4 of hospitalization, the patient became acutely tachycardic and dyspneic. Mixed venous saturation drawn from a pulmonary artery catheter was noted to have abruptly increased compared with prior stable measurements (48% to 78%). Ventricular septal rupture (VSR) was suspected, and a small apical rupture was confirmed by echocardiography (Fig. 2, B-D). Surgical closure was deemed to be of prohibitive risk given worsening shock and multiorgan dysfunction despite maximum vasopressor support and escalation of Impella support from the P4 to the P8 setting, which achieved 2.6 L/min of maximum flow. Venoarterial extracorporeal membrane oxygenation was initiated by right femoral arterial and venous cut-down, and she was transferred to a heart transplant center, but her condition worsened and she died several days later.

Discussion

The estimated prevalence of severe LVOT obstruction, cardiogenic shock, and VSR in SCM is 20%,[1,2] 15% to 23%,[3,4] and 0.2%,[5] respectively. To our knowledge, concurrent occurrence of this triad has never been reported in the same patient.

In this case, we postulate that increased LVEDP in the setting of severely reduced ejection fraction and LVOT obstruction led to increased wall stress. This likely yielded impaired microvascular flow despite epicardial patency, triggering coagulative necrosis and VSR at the thinnest portion of the myocardium, where tension is greatest. Implication of thrombolytics in pathologic progression to VSR would be speculative.

The interplay of advanced cardiogenic shock (treated initially with inotropes, diuretics, and IABP) and LVOT obstruction (β-blockade and α-agonism) made establishing an effective treatment strategy challenging. There are no treatment guidelines for SCM complicated by both processes.

Anticipated reduction in obstruction, LVEDP, and hyperadrenergic tone using β-blockers and diuretics may be overwhelmed by advanced shock, thus requiring PMCS. Although our patient responded well to the Impella CP, her system could not tolerate the added insult of VSR. It is possible that an Impella 5.0 device, capable of generating greater flow rates (but unavailable at our institution at the time), could have been more efficacious. It is unknown if early use of PMCS could prevent a mechanical complication such as VSR.

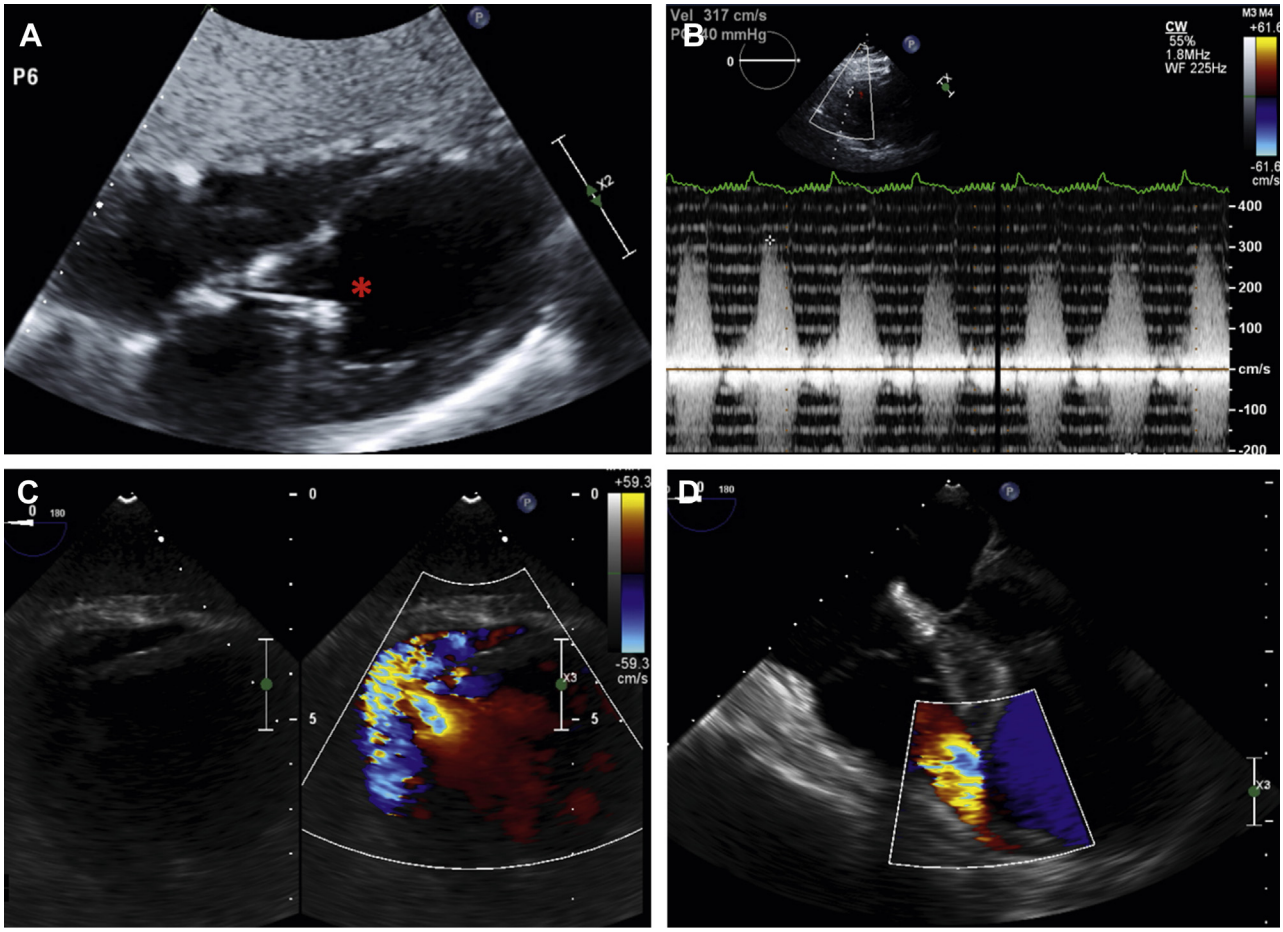

Figure 2. (**A**) Subcostal TTE image of the Impella CP (ABIOMED, Danvers, MA) in the LVOT (**asterisk**). (**B**) TTE spectral Doppler tracing demonstrating ventricular septal defect flow; background interference artifact from Impella device. (**C**) Trans-gastric transesophageal echocardiogram view demonstrating color Doppler flow through apical ventricular septal defect. (**D**) Four-chamber transesophageal echocardiogram view demonstrating color Doppler flow through apical ventricular septal defect.

Conclusion

SCM can end catastrophically because of compounding of rare complications. Early screening and detection of outflow obstruction may modify management strategy, including use of PMCS to off-load the ventricle and reduce wall tension.

Disclosures

The authors have no conflicts of interest to disclose.

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