

# Dynamic obstruction induced by systolic anterior motion of the mitral valve in a volume-depleted left ventricle: an unexpected cause of acute heart failure in a patient with chronic obstructive pulmonary disease

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**Abstract:** Systolic anterior motion (SAM) of the mitral valve (MV) and left ventricular outflow tract (LVOT) dynamic obstruction (DO) typically occur in hypertrophic cardiomyopathy; however, they can appear in an apparently normal heart in association with changes in cardiac loading conditions and/or hyperdynamic left ventricular (LV) performance. Meanwhile, chronic obstructive pulmonary disease (COPD) can impair LV filling by elevating pulmonary vascular resistance. The authors report a case of transient acute heart failure caused by LVOT DO resulting from SAM of the MV in a severely volume-depleted LV in a patient with acute COPD exacerbation.

**Keywords:** Chronic obstructive pulmonary disease (COPD); dynamic obstruction (DO); echocardiography; mitral valve (MV); natriuretic peptide

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## Introduction

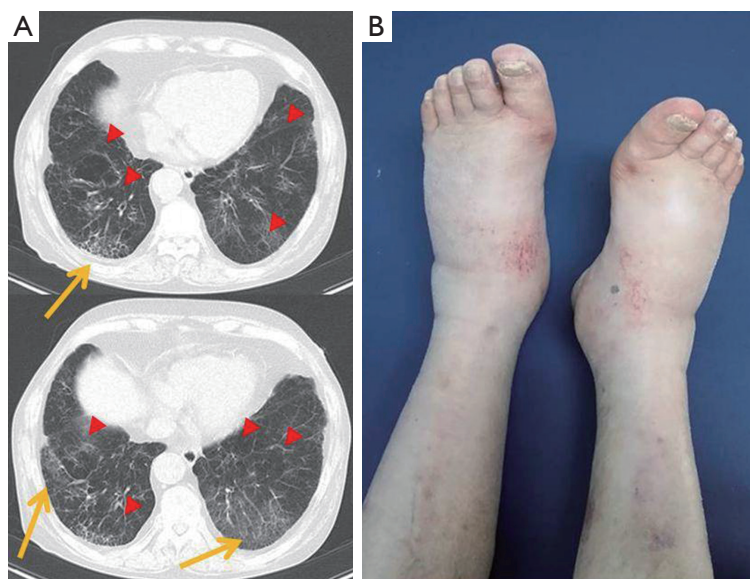
Left ventricular outflow tract (LVOT) dynamic obstruction (DO) resulting from systolic anterior motion (SAM) of the mitral valve (MV) is a typical feature of hypertrophic cardiomyopathy. However, this intriguing phenomenon can appear even in an apparently normal or near-normal heart under certain clinical conditions associated with hyperdynamic left ventricular (LV) contraction and/or LV volume depletion (1,2). In critically ill patients, the occurrence of LVOT DO caused by SAM of the MV is potentially serious, because it can significantly compromise cardiac output (2). Therefore, a clear understanding of intracardiac hemodynamics and early recognition of interplay between the MV and accelerated LVOT blood flow are of paramount importance.

Because of increased pulmonary vascular resistance, chronic obstructive pulmonary disease (COPD) can impair

LV filling; and thus, a decrease in LV volume can occur even before the development of overt cor pulmonale (3). Here, we report a case of transient acute heart failure caused by LVOT DO resulting from SAM of the MV in a severely volume-depleted LV in a patient who suffered from acute exacerbation of COPD. In addition, a diagnostic process with echocardiography, which played a unique role in explaining complex cardiac functional issues, and a patient-specific consideration for proper management will be discussed.

## Case report

A 72-year-old Asian male with a prior history of COPD and emphysema presented at our emergency department complaining of recently aggravated shortness of breath and purulent sputum. On physical examination, tachycardia



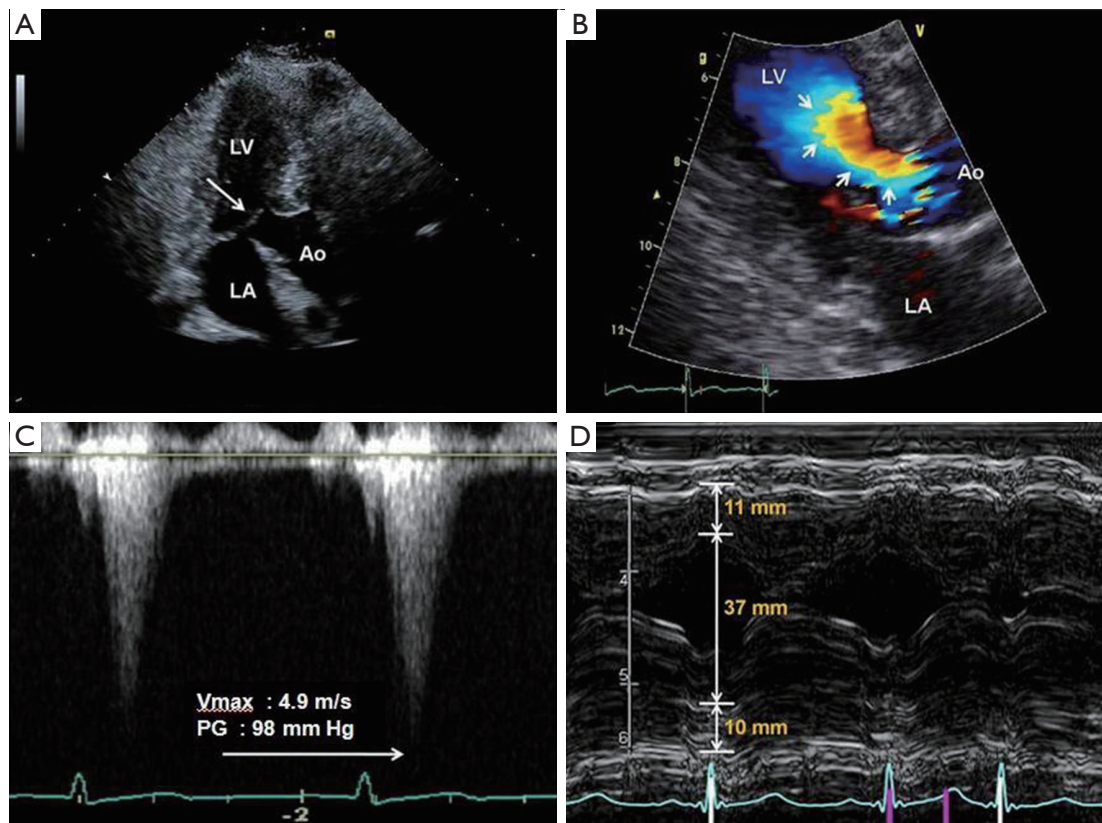
**Figure 1** (A) Chest computed tomographic images taken at time of presentation showing newly developed consolidation and ground glass opacity in both lower lung fields, suggesting pneumonia (arrows) superimposed on preexisting extensive emphysema (arrowheads); (B) generalized pitting edema on both lower legs suggesting right-heart failure associated with chronic obstructive pulmonary disease (COPD).

(heart rate: 110 beats/minute) and tachypnea (respiratory rate: 24 breaths/minute) were notable. The patient was agitated and showed cold, clammy skin, sweating, external jugular venous distension, and peripheral pitting edema (*Figure 1*). On auscultation, coarse breath sounds with rales were audible in both lower lung fields, and unexpectedly, systolic murmur (grade IV/VI) was prominent at the left sternal border. Twelve-lead electrocardiography showed sinus tachycardia and non-specific ST-segment/T-wave changes. Chest computed tomography depicted newly developed multifocal ground glass opacity in both lungs, suggesting pneumonia superimposed on preexisting extensive emphysema (*Figure 1*). Laboratory findings revealed hyponatremia (121 mEq/L) and elevated pro-brain natriuretic peptide (pro-BNP) level (2,515 pg/mL). Blood urea nitrogen was 28 mg/dL, serum creatinine was 1.6 mg/dL, serum protein was 7.4 g/dL, and serum albumin was 4.3 g/dL. Leukocytosis (white blood cells:  $23,140/\text{mm}^3$ , segment neutrophils: 93%) and elevated high sensitivity C-reactive protein (15.2 mg/dL) were also reported. Arterial blood gas analysis revealed respiratory alkalosis (pH: 7.49,  $\text{PaCO}_2$ : 21 mmHg,  $\text{PaO}_2$ : 76 mmHg,  $\text{HCO}_3^-$ : 21,  $\text{SaO}_2$ : 96%), which was apparently associated with tachypnea.

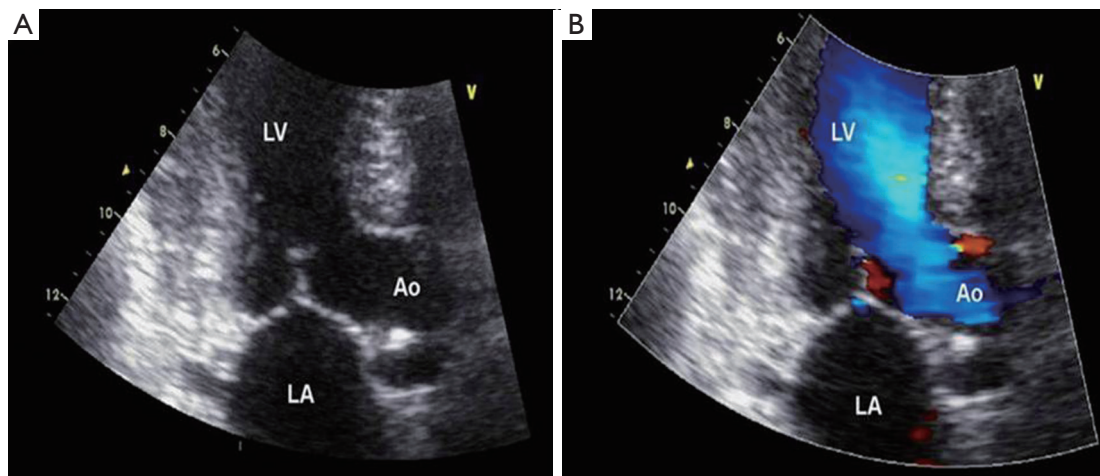
Transthoracic echocardiography was performed to evaluate the nature of the systolic murmur, and the results

showed a small LV cavity (LV end-diastolic dimension, 37 mm) with vigorous LV contraction (LV ejection fraction, 80%) suggesting LV volume depletion and a hyperdynamic heart (*Figure 2*). The right ventricle was not dilated and there was no evidence of overt cor pulmonale. Most notably, SAM of the MV was observed and flow acceleration across the LVOT by color Doppler with a late-peaking flow profile by continuous Doppler was remarkable, all of which indicated LVOT DO (*Figure 2*).

Under a diagnosis of acute exacerbation of COPD due to pneumonia, oxygen therapy was started to relieve breathlessness and to reduce agitation, and an oral antibiotic (levofloxacin 500 mg daily) was prescribed. Tailored management of the coexisting acute heart failure resulting from LVOT DO was given to patient with specific consideration, and a cardio-selective beta-blocker (bisoprolol 5 mg daily) and intravenous crystalloid were administered to improve hyperdynamic LV contraction and left-heart oligemia, respectively. Two days after initiating treatments, major symptoms showed dramatic improvements and the systolic murmur was no longer audible. In follow-up echocardiography conducted after the initial treatment, SAM of the MV had completely disappeared and low velocity laminar LVOT blood flow was observed, suggesting that the LVOT DO had fully resolved (*Figure 3*). Laboratory findings supported this improvement;



**Figure 2** Two-dimensional transthoracic echocardiographic evaluation was performed at time of presentation when the patient was dyspneic and agitated. (A) Systolic anterior motion (SAM) of mitral leaflets (arrow) in the volume-depleted left ventricle; (B) color Doppler image showing aliasing at the region between the mitral valve (MV) and the interventricular septum, suggestive of flow acceleration at the left ventricle outflow tract (LVOT) (arrows); (C) a continuous wave Doppler image showing a late peaking ("Dagger shaped") profile of LVOT flow with a maximal pressure gradient of 98 mmHg, suggesting dynamic obstruction (DO); (D) M-mode image at the mid-ventricular level showing a small left ventricular (LV) cavity and normal wall thickness.



**Figure 3** Follow-up echocardiography after the patient was stabilized after cardio-selective beta-blocker administration and careful volume restoration therapy. (A) It shows complete absence of systolic anterior motion (SAM) of the mitral valve (MV); (B) color flow image shows full resolution of flow acceleration and normal low-velocity laminar flow at the left ventricular outflow tract (LVOT).



pro-BNP level had dropped to 369 pg/mL and plasma sodium level increased to 139 mEq/L.

After 3 days of initial treatment targeting left-heart failure, intravenous hydration was stopped and diuretics (torsemide 10 mg daily) were prescribed to address the peripheral edema resulting from right-heart failure associated with COPD. However, two days later, dyspnea showed temporary deterioration, and an echocardiographic assessment revealed reappearance of SAM of the MV and LVOT DO. In addition, at this time plasma pro-BNP had increased to 620 pg/mL. Hence, the diuretic was discontinued and the patient was encouraged to maintain careful oral hydration. After several days of treatment, the patient was restabilized, the pneumonia superimposed on COPD improved, and the acute heart failure resulting from LVOT DO induced by LV volume depletion subsided. The patient was discharged on beta-blocker.

## Discussion

Our patient had a unique presentation in that: (I) acute exacerbation of COPD was accompanied by transient acute heart failure; (II) the heart failure was induced by LVOT DO resulting from SAM of the MV in an apparently normal heart; and (III) left-heart volume decrement, resulting from LV filling impairment due to severe lung disease, generated the LVOT DO. This case provides a valuable experience regarding the treatment of patients with COPD who showed changes in intracardiac volume distribution and alterations in cardiac performance. From that perspective, timely performed echocardiographic evaluations were invaluable for accurate diagnosis of the unexpected left-heart problem, which might have contributed to the patient's dyspnea, and provided us with helpful insight for better understanding of the patient's clinical course in which we practiced a patient-specific management.

Barr *et al.* (3) conducted a population-based study using cardiac magnetic resonance imaging, and reported that emphysema was associated with smaller LV volume in the setting of COPD in the absence of clinical cardiovascular disease. Proposed mechanisms for this observation included loss of pulmonary vascular capacity resulting from parenchymal destruction, ventricular interdependence, hypoxic pulmonary arterial vasoconstriction, and pulmonary hyperinflation resulting in elevated intrathoracic pressure exceeding venous pressure (3). Our patient suffered from acute exacerbation of COPD, which might have caused a considerable increase in pulmonary vascular resistance,

impaired LV filling, and eventually led to LV volume reduction. Sequentially, SAM of the MV and concomitant LVOT DO occurred in a volume-depleted but structurally normal LV. As was further evidenced by an elevated pro-BNP level, these phenomena caused transient acute heart failure superimposed on COPD in our patient. In fact, these events can occur in a structurally normal heart in association with severe dehydration, massive bleeding, catecholamine usage, sepsis, etc., which can induce hyperdynamic cardiac performance and changes in LV loading conditions (1,2).

Along with conventional management for COPD and pneumonia, we initiated tailored treatment for the unusual cardiac functional abnormality. After the administration of beta-blocker and careful hydration to relieve SAM of the MV, symptoms showed dramatic improvements, in particular, LVOT DO was absent, and notably, pro-BNP levels concomitantly decreased. More interestingly, when we prescribed diuretics to manage peripheral edema, breathlessness was temporarily aggravated, SAM of the MV reappeared and pro-BNP level re-increased. Plasma pro-BNP levels increase during volume- and/or pressure-overload of the heart (4). Briguori *et al.* (5) reported an association between LVOT pressure gradients and BNP plasma levels in hypertrophic cardiomyopathy patients, and added elevated LV systolic wall stress induced by LVOT DO appeared to be the principal stimulus for the secretion of the natriuretic peptide. Although the question as to whether BNP level is correlated with LVOT DO in subjects free from hypertrophic cardiomyopathy remains to be answered, the suggestions made by Briguori *et al.*, could explain trends in plasma natriuretic peptide levels observed in our patient.

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## Footnote

**Conflicts of Interest:** The authors have no conflicts of interest to declare.

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