Korean J Thorac Cardiovasc Surg 2013;46:457-460 □ Case Report □
ISSN: 2233-601X (Print) ISSN: 2093-6516 (Online) http://dx.doi.org/10.5090/kjtcs.2013.46.6.457

The Unusual Suspect: Anemia-induced Systolic Anterior Motion of the Mitral Valve and Intraventricular Dynamic Obstruction in a Hyperdynamic Heart as Unexpected Causes of Exertional Dyspnea after Cardiac Surgery

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Dynamic left ventricular (LV) outflow tract obstruction is a characteristic feature of hypertrophic cardiomyopathy; however, it can also occur in association with hyperdynamic LV contraction and/or changes in the cardiac loading condition, even in a structurally normal or near-normal heart. Here, we report a case of anemia-induced systolic anterior motion of the mitral valve and the resultant intraventricular obstruction in a patient who underwent coronary artery bypass grafting and suffered from anemia associated with recurrent gastrointestinal bleeding.

Key words: 1. Anemia
2. Echocardiography
3. Mitral valve

CASE REPORT

Dynamic left ventricular (LV) outflow tract (LVOT) obstruction associated with the systolic anterior motion (SAM) of the mitral valve (MV) is a characteristic feature of hypertrophic cardiomyopathy. However, when certain conditions are met, this intriguing phenomenon can occur even in a structurally normal or near-normal heart. Here, we report a case of a transient intraventricular obstruction resulting from SAM of the MV in a patient who suffered from anemia due to gastrointestinal bleeding after coronary artery bypass grafting (CABG) surgery.

A 73-year-old Asian male presented with dyspnea (New York Heart Association class III/IV) on exertion and effort intolerance; the condition had developed over the course of several days. The patient had a medical history of radical subtotal gastrectomy (Billroth II) due to stomach cancer in 2003 and coronary artery bypass grafting (CABG) for treatment of stable angina with triple-vessel coronary artery disease in 2011. The result of CABG was successful, and thereafter, aspirin, angiotensin II receptor blocker, and statin were prescribed. The patient was monitored in the outpatient clinic after the operation without symptoms of angina pectoris or heart failure for two years. Recently, however, he had suffered from multiple episodes of gastrointestinal bleeding from small bowel angiodysplasia and peptic ulcer, which were treated with endoscopic Argon plasma coagulation and long-term gastric acid-suppressing therapy. During the episodes of

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Received: June 11, 2013, Revised: July 16, 2013, Accepted: July 22, 2013
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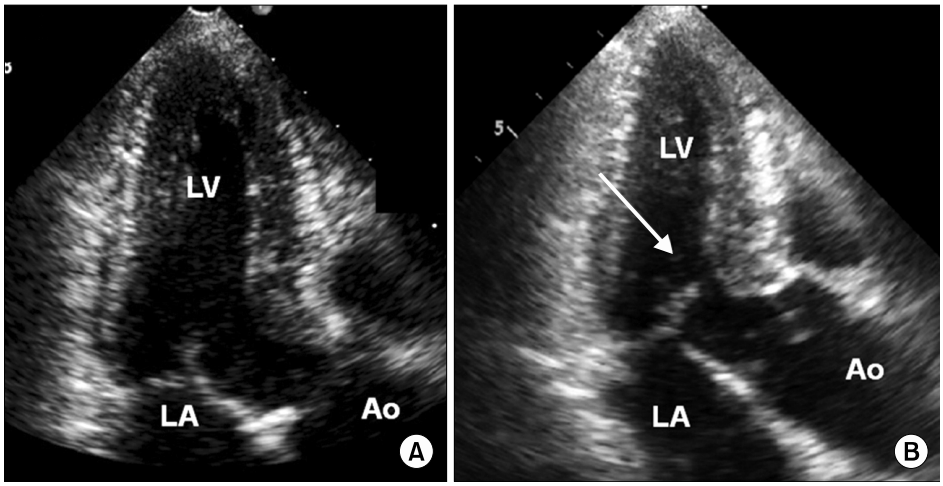

Fig. 1. Apical 3-chamber view showing the left ventricular (LV) outflow tract (LVOT) and MV during systole. (A) Apparently normal LV geometry and normal mitral valve (MV) before coronary artery bypass grafting. The LV ejection fraction was 60% with normal LV wall motion. At that time, the serum hemoglobin (Hb) level was 11.4 g/dL. (B) At the time of heart failure associated with anemia (Hb, 8.3 g/dL). The LV size was small, and LV contraction was markedly hyperdynamic (LV ejection fraction, 78%). Systolic anterior motion of the MV (arrow) was prominent, and LVOT was notably narrow. LA, left atrium; Ao, aorta.

acute hemorrhage, blood transfusion was performed at the attending physicians' discretion without any complications.

At the time of the current presentation, vital signs were not remarkable (blood pressure, 110/70 mmHg; heart rate, 72 beats/min). A physical examination revealed that he was pale in appearance and his extremities were rather cold, with slightly decreased skin turgor and a dry tongue. Of note, on auscultation, a systolic murmur (grade IV/VI) was audible at the left lower sternal border. Chest radiography was not remarkable, except mild cardiomegaly. Findings of a 12-lead electrocardiography examination showed a sinus rhythm and nonspecific changes in the ST-segment and T-wave. Laboratory findings indicated normocytic and normochromic anemia (haemoglobin, 8.3 g/dL; [normal range, 13 to 17 g/dL]; mean cell volume, 86.6 fL; [normal range, 79 to 96 fL]; and mean cell hemoglobin, 28.4 pg; [normal range, 27 to 31 pg]). The blood urea nitrogen was 11.4 mg/dL, and the creatinine level was 0.6 mg/dL. The serum protein level was 6.2 g/dL, and the albumin level was 3.2 g/dL. The pro-brain natriuretic peptide level was 228.8 pg/mL.

We performed transthoracic echocardiography for a further evaluation of the systolic murmur and cardiac pathology potentially associated with symptoms of heart failure. Results showed the newly developed systolic anterior motion (SAM) of the mitral valve (MV) (Fig. 1). The left ventricular (LV) cavity size was small (LV end-diastolic dimension, 41 mm) with remarkable hyperdynamic LV contraction (LV ejection fraction, 78%), suggesting intracardiac volume depletion. The patient's heart was essentially free from myocardial or valvular abnormality, except concentric LV remodeling (relative wall thickness, 0.44; LV mass index, 73 g/m^2). A color Doppler evaluation showed aliasing at the portion between the MV and the interventricular septum during the systolic phase, suggesting flow acceleration across the LV outflow tract (LVOT). A continuous Doppler study of the LVOT demonstrated a late-peaking profile of the flow suggestive of dynamic intraventricular obstruction. The maximal pressure gradients were 54 mmHg at rest and 65 mmHg during the strain phase of the Valsalva maneuver estimated by the systolic blood flow velocity. This was suggestive of significant dynamic LVOT obstruction, which was aggravated during preload reduction (Fig. 2). Meanwhile, the early diastolic mitral inflow velocity (E) was 0.4 m/sec, and the early diastolic mitral annulus velocity (E') at the septal corner was 0.05 m/sec (E over E' ratio, 8). The inferior vena cava diameter was 1.1 cm and showed good collapsibility (respiratory variability was greater than 50%).

The patient was relatively stable and showed no signs of

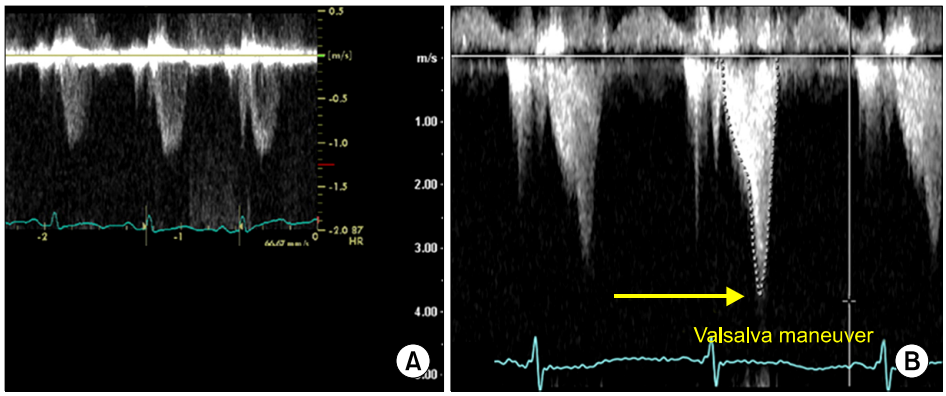

Fig. 2. The change in the Dopper contour at the left ventricular outflow tract (LVOT). (A) The seemingly normal contour of the LVOT pulsed-wave Doppler image with normal velocity (1.2 m/sec) before coronary artery bypass grafting. (B) Echo performed when the patient suffered from anemia (hemoglobin, 8.3 g/dL). Dagger-shaped continuous-wave Doppler contour across the LVOT during the strain phase of the Valsalva maneuver. The peak velocity of the flow was more than 4 m/sec, and the pressure gradient was estimated to be 65 mmHg, suggesting significant subaortic obstruction. The patient's aortic valve was normal, and there was no evidence of a fixed obstruction at the aortic level, such as aortic stenosis or subaortic web.

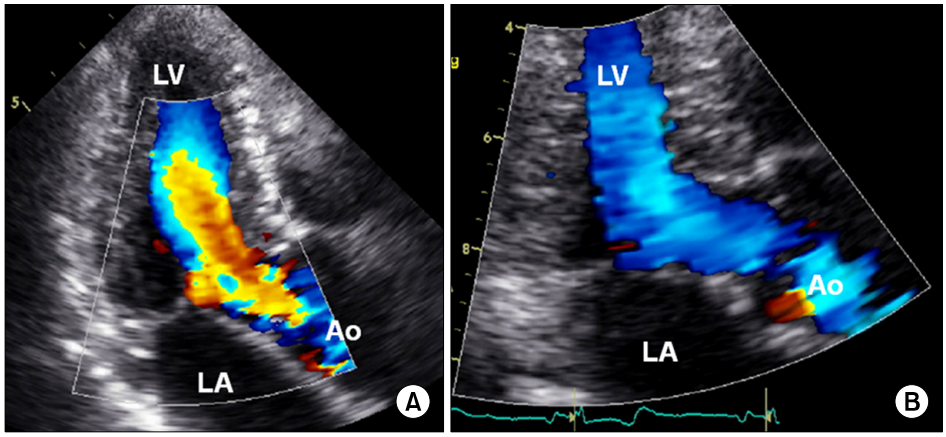

Fig. 3. (A) Color Doppler image taken at the time of anemia (hemoglobin, 8.3 g/dL). Note the aliasing of color Doppler from the mid-level of left ventricular (LV) to LV outflow tract LVOT, suggesting flow acceleration and dynamic obstruction. (B) A color Doppler image at the LVOT after treatment. The systolic anterior motion of the mitral valve was abolished. Concurrently, this image shows a low-velocity laminar LVOT blood flow. The patient's symptoms were improved dramatically. LA, left atrium; Ao, aorta.

active gastrointestinal bleeding or hemodynamic deterioration at the time of presentation; therefore, he was treated with a chronic oral iron supplement rather than further blood transfusion. In addition, based on the result of the echocardiographic evaluation, the angiotensin II receptor blocker was discontinued; instead, beta-blocker (bisoprolol 5 mg daily) and oral hydration were encouraged in order to minimize LV hypercontractility. One week after treatment, the patient was free from previous symptoms of heart failure, and the systolic murmur was no longer detected. In the follow-up echocardiographic evaluation, the SAM of the MV was fully abolished, and a laminar LVOT blood flow was observed (Fig. 3). Hence, we decided to continue the altered medication for the time being and instructed the patient to avoid dehydration and physical overexertion.

DISCUSSION

Intraventricular obstruction resulting from the SAM of the MV is typically observed in hypertrophic cardiomyopathy due

to the asymmetrical bulging of the interventricular septum, which causes narrowing of the LVOT. However, the interesting 'malfunction' of the seemingly normal MV is observed in certain clinical settings, even in an apparently normal or minimally abnormal heart, particularly in critically ill patients. In fact, dynamic intraventricular obstruction due to the SAM of the MV, sometimes accompanying significant mitral regurgitation, has been reported in the case of an acute coronary syndrome [1], after valve surgery [2], and in the case of an apical ballooning syndrome [3] when the LV geometry is altered. In addition, dynamic obstruction of the LVOT can occur upon vigorous contraction of the LV in patients without organic heart disease, such as sepsis [4], and under the influence of catecholamine [5]. In the current case, anemia was the precipitating factor for hyperdynamic LV contraction during physical effort, which induced the SAM of the MV with intracardiac obstruction. We reckon that because the SAM of the MV tends to become aggravated during exercise, a transient alteration of the MV movement, along with anemia, served as the cause of effort intolerance in our patient.

SAM of the MV can occur in various situations; however, clinicians tend not to pay attention to this intriguing phenomenon, and thus, it tends to be undiagnosed and ignored. Our patient complained of shortness of breath mainly during exercise, and the most plausible clinical suspicions included diastolic dysfunction, postoperative constriction [6], and/or graft failure after CABG. Of note, with timely echocardiography, we were able to correctly diagnose subaortic obstruction due to the SAM of the MV induced by anemia; this made the tailored management possible. We attempted to correct the anemia, restored the intracardiac volume with hydration, and prescribed a beta-blocker, all of which alleviated the patient's symptoms. In conclusion, intracardiac dynamic obstruction due to the SAM of the MV can occur in association with acute changes in the afterload [4], preload [7], LV, and/or MV apparatus geometry [1,3], and ventricular contractility [4]; therefore, it should be suspected as a potential cause for heart failure even in patients without hypertrophic cardiomyopathy. From this perspective, an insightful echocardiographic evaluation is invaluable.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

ACKNOWLEDGMENTS

This work was supported by the Gachon University Gil Medical Center (grant number: 2013-10) and by Boryung Pharmaceutical Company, Republic of Korea.

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