or opportunistic infections. Also, leukemic infiltration should be considered in differential diagnosis of cardiac hypertrophy in this patient group. Repetitive cases with different clinical futures will increase our awareness and improve our understanding about leukemic heart disease. Prompt accurate diagnosis and urgent therapy may improve the clinical process of this disease.

Informed consent: An informed consent was obtained from the patient's wife.

Video 1. Parasternal long axis view illustrating severe myocardial hypertrophy, moderate pericardial effusion, and limitation of myocardial motion.

Video 2. Parasternal short-axis view showing extensive myocardial hypertrophy, moderate pericardial effusion, and systolic dysfunction.

Video 3. Apical four-chamber view displaying disproportionate myocardial hypertrophy, and limitation of diastolic expansion movement of the ventricles.

References

1. Clark WB, Strickland SA, Barrett AJ, Savani BN. Extramedullary relapses after allogeneic stem cell transplantation for acute myeloid leukemia and myelodysplastic syndrome. Haematologica 2010; 95: 860-3.
2. Jung MH, Lee YH, Lee KY, Jung HO, Youn HJ. Leukemic infiltration presenting as myocardial hypertrophy after complete remission of acute myeloid leukemia. Echocardiography 2017; 34: 136-8.
3. Facenda-Lorenzo M, Sánchez-Quintana A, Quijada-Fumero A, Laynez-Carnicero A, Breña-Atienza J, Poncela-Mireles FJ, et al. Cardiac Relapse of Acute Myeloid Leukemia after Allogeneic Hematopoietic Stem Cell Transplantation. Case Rep Oncol Med 2016; 2016: 5091021.
4. Chang K, Kim DY, Lee KH, Huh J, Kang JW, Shin DY, et al. An isolated cardiac relapse after allogeneic hematopoietic stem cell transplantation for acute lymphoblastic leukemia. Korean J Intern Med 2017; 32: 753-7.
5. Hori T, Suzuki N, Mizue N, Hatakeyama N, Takamuro M, Tsutsumi H. Relapse of T-cell all after stem cell trans- plant presenting as hypertrophic cardiomyopathy: the value of non-invasive diagnostic imaging in detecting cardiac leukemia. Pediatr Blood Cancer 2006; 46: 108-11.
6. Roberts WC, Bodey GP, Wertlake PT. The heart in acute leukemia: A study of 420 autopsy cases. Am J Cardiol 1968; 21: 388-412.
7. Barbaric D, Holley D, Lau KC, McCowage G. It is ALL in the heart: A patient with acute lymphoblastic leukemia and cardiac infiltration at time of diagnosis. Leuk Lymphoma 2002; 43: 2417-9.
8. Butany J, Nair V, Naseemuddin A, Nair GM, Catton C, Yau T. Cardiac tumours: diagnosis and management. Lancet Oncol 2005; 6: 219-28.
9. Makaryus AN, Tung F, Liu W, Mangion J, Kort S. Extensive neoplastic cardiac infiltration in a patient with acute myelogenous leukemia: role of echocardiography. Echocardiography 2003; 20: 539-44.
10. Mohty D, Damy T, Cosnay P, Echahidi N, Casset-Senon D, Virot P, et al. Cardiac amyloidosis: updates in diagnosis and management. Arch Cardiovasc Dis 2013; 106: 528-40.

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Echocardiographic imaging of systolic anterior motion caused by extremely elongated posterior mitral leaflet in hypertrophic cardiomyopathy

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Introduction

Systolic anterior motion is frequently observed in hypertrophic cardiomyopathy (HCM) and causes left ventricular outflow tract (LVOT) obstruction, and systolic anterior motion (SAM) is generally seen at the anterior mitral leaflet (AML) (1). However, an isolated SAM of the posterior mitral leaflet (PML) can be unusually seen. Full coaptation of mitral leaflets and an absence of mitral regurgitation are distinct features of this pattern, and elongation of PML is also essential. We present a case with this unusual pattern to increase awareness about this unique mechanism in HCM.

Case Report

A 66-year-old asymptomatic male visited our clinic for a routine cardiac examination. He did not have any history of cardiac or systemic disease and had 3/6 systolic ejection murmur on the second intercostal space, which was heard on auscultation. Electrocardiography (ECG) showed nonspecific ST-T changes and left ventricular hypertrophy findings. His midesophageal, three-chamber view on transesophageal echocardiography showed extremely elongated PML (3.63 cm) (Fig. 1). Parasternal short-axis view demonstrated extensive hypertrophy of the left ventricle (Video 1). His interventricular septum thickness was 2.1 cm, and asymmetric septal hypertrophy can be seen in the parasternal long-axis view (Video 2). His LVOT gradient was 30 mm

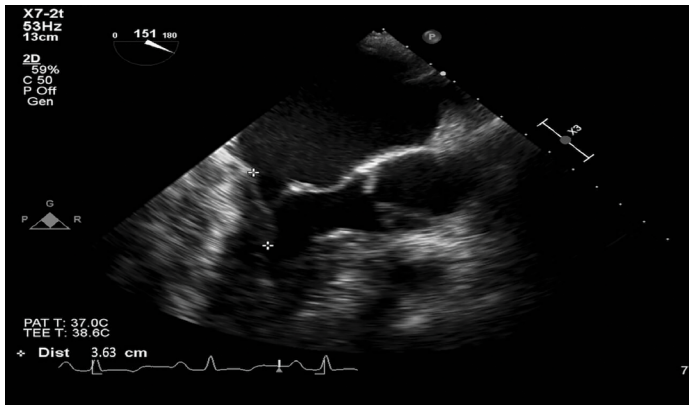

Figure 1. Length of the posterior mitral leaflet is measured as 3.7 centimeters

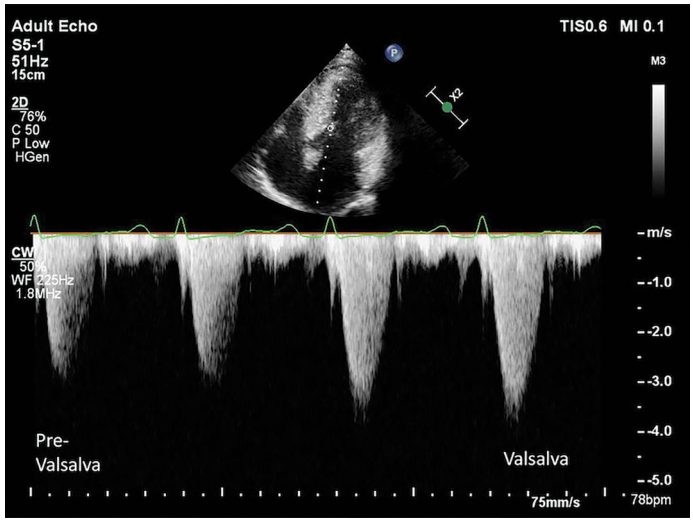

Figure 2. LVOT gradients before (30 mm Hg) and after (58 mm Hg) Valsalva maneuver

LVOT - left ventrcular outflow tract; mm Hg - millimeters of mercury

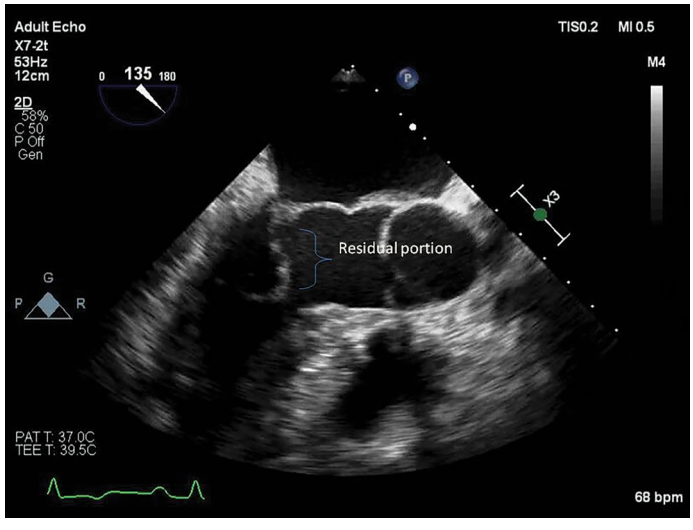

Figure 3. Coaptation of the anterior mitral leaflet with basal portion of the posterior mitral leaflet and systolic anterior motion of posterior mitral leaflet's residual portion

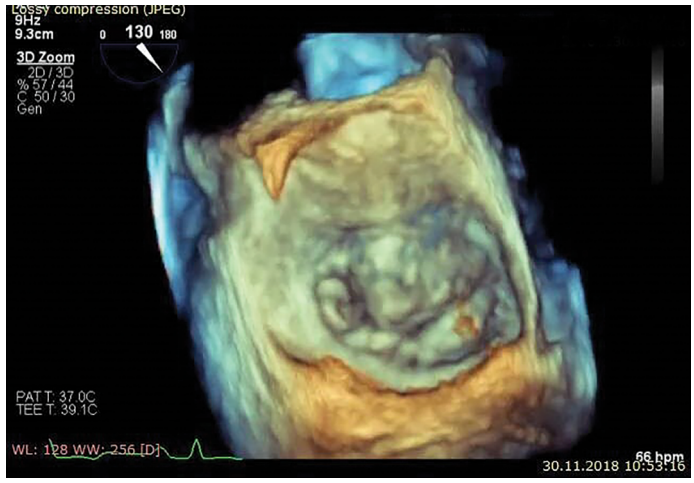

Figure 4. 3D imaging shows full coaptation of the anterior and posterior mitral leaflets. 3D-Three-dimensional

Hg at rest and 58 mm Hg with Valsalva maneuver (Fig. 2). Two-dimensional transthoracic echocardiographic images were taken with a Philips S5-1 ultrasound transducer probe (Philips Healthcare, Inc., Andover, MA, USA). Transesophageal echocardiography demonstrated SAM caused by extremely elongated PML with asymmetric left ventricular hypertrophy (Fig. 3, Videos 3 and 4). AML showed coaptation with the basal portion of the PML (Fig. 1-3, Videos 3 and 4). Color Doppler revealed systolic turbulence in LVOT at transesophageal 135° image (Video 5). Interestingly, mitral regurgitation was not observed in transthoracic and transesophageal echocardiography 135° images (Videos 5 and 6). Transesophageal 3D images demonstrated full coaptation of AML and PML (Fig. 4, Videos 7 and 8). Transesophageal echocardiographic images were obtained with a Philips X7-2 ultrasound transducer probe (Philips Healthcare, Inc., Andover, MA, USA).

Discussion

PML rarely causes SAM and LVOT obstruction in HCM (1). SAM can be seen in patients with HCM for various reasons such as elongated AML or PML, papillary muscle displacement, papillary muscle insertion anomaly, or calcification of mitral annulus or leaflets (2). SAM of elongated PML and hypertrophy of the left ventricle caused LVOT obstruction in our patient. The relationship between mitral leaflet elongation and HCM studied has been studied before (3). In HCM, mitral valve leaflets are elongated independently of other disease variables and are an important morphological abnormality responsible for LVOT obstruction (3). SAM and mitral leaflet elongation association also has been shown before (3). An experimental study showed that mitral leaflet elongation can play an important role in SAM in response to outflow forces (4). Isolated SAM of the PML with long residual portion is previously identified pattern in patients with HCM; in HCM, patients with isolated SAM of the PML is a unique and unusual pattern for dynamic LVOT obstruction, and clinicians should raise awareness about this pattern (1).

Our patient is a remarkable example of this pattern in patients with HCM, and we used different types of echocardiography to demonstrate the relationship between PML elongation and SAM. An absence of mitral regurgitation is also an interesting finding in presence of SAM. Mitral leaflet anomalies are important in HCM pathophysiology, for example, USA national outcome study showed the need to increase experience about mitral valve surgery with concomitant septal myectomy to treat patients with HCM because nearly a quarter of septal myectomy patients underwent concomitant mitral valve surgery (5). This paper underlines the importance of mitral valve mechanics in HCM to understand the pathophysiology and manage patients. We presented a unique pattern of mitral valve abnormality in a patient with HCM using multiple echocardiographic images.

Conclusion

HCM includes diverse pathophysiological mechanisms with clinical outcomes. Obstructive HCM is usually identified with asymmetric septal hypertrophy and anterior motion of AML during systole. However, different pathologic appearances are possible in HCM. We showed a unique pattern in a patient with HCM who had extremely elongated PML and isolated SAM of this leaflet using different types of echocardioraphy. An absence of mitral regurgitation is also a distinctive feature of this pattern. Understanding this pattern and increasing awareness will provide diagnostic and therapeutic benefits for similar patients. Our patient is a good representative of this unique pattern with demonstrative images.

Informed consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying videos and images.

Video 1. Parasternal short-axis view of the hypertrophic left ventricle.

Video 2. Parasternal long-axis view of systolic anterior motion of the posterior mitral leaflet and coaptation of the anterior mitral leaflet with basal portion of the posterior mitral leaflet.

Video 3. Transesophageal 135° midoesophageal view of the mitral leaflets. The movie shows systolic anterior motion of the posterior mitral leaflet and coaptation of the anterior and posterior mitral leaflets.

Video 4. Transoesophageal 0° midoesophageal view of the mitral leaflets. The movie shows systolic anterior motion (SAM) of the posterior mitral leaflet and coaptation of the anterior and posterior mitral leaflets.

Video 5. Transesophageal color Doppler view of the absence of the mitral regurgitation and presence of left ventricular outflow tract obstruction.

Video 6. Absence of the mitral regurgitation in apical 2-chamber view of transthoracic echocardiography.

Video 7. Full coaptation of mitral leaflets showed by 3D imaging. 3D-Three-dimensional.

Video 8. Full coaptation of mitral leaflets showed by 3D imaging. 3D-Three-dimensional.

References

1. Maron BJ, Harding AM, Spirito P, Roberts WC, Waller BF. Systolic anterior motion of the posterior mitral leaflet: a previously unrecognized cause of dynamic subaortic obstruction in patients with hypertrophic cardiomyopathy. Circulation 1983; 68: 282-93. [CrossRef]
2. Sherrid MV, Balaram S, Kim B, Axel L, Swistel DG. The Mitral Valve in Obstructive Hypertrophic Cardiomyopathy: A Test in Context. J Am Coll Cardiol 2016; 67: 1846-58. [CrossRef]
3. Maron MS, Olivotto I, Harrigan C, Appelbaum E, Gibson CM, Lesser JR, et al. Mitral valve abnormalities identified by cardiovascular magnetic resonance represent a primary phenotypic expression of hypertrophic cardiomyopathy. Circulation 2011; 124: 40-7. [CrossRef]
4. He S, Hopmeyer J, Lefebvre XP, Schwammenthal E, Yoganathan AP, Levine RA. Importance of leaflet elongation in causing systolic anterior motion of the mitral valve. J Heart Valve Dis 1997; 6: 149-59.
5. Holst KA, Hanson KT, Ommen SR, Nishimura RA, Habermann EB, Schaff HV. Septal Myectomy in Hypertrophic Cardiomyopathy: National Outcomes of Concomitant Mitral Surgery. Mayo Clin Proc 2019; 94: 66-73. [CrossRef]

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