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Case Report

Dual Left Ventricular Outflow Tract Obstruction Presenting in Old Age

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Abstract
Hypertrophic obstructive cardiomyopathy with Rheumatic heart disease, severe aortic stenosis and severe mitral stenosis is an uncommon presentation. Diagnosis ofdual left ventricular outflow tract obstructive physiology, caused by severe symptomatic obstructive hypertrophic cardiomyopathy and concomitant moderate or severe aortic stenosis is of paramount importance for management. (myectomy and aortic valve replacement).
Our case, is a 67 years old hypertensive male presented with complain of chest pain, diffuse, radiating to left arm, associated with sweating and also with history of loss of consciousness.

Introduction

Hypertrophic cardiomyopathy and aortic stenosis are 2 conditions that cause obstruction to blood flow leaving the heart.

- Distinguishing feature:
  - HCM typically results in dynamic left ventricular outflow tract obstruction,
  - Severe AS results in fixed obstruction.

Simultaneous existence of both conditions in the same patient has been documented, although it is uncommon. Presence of sequential LVOT obstruction poses particular diagnostic challenges requiring meticulous imaging.Correct identification and quantification of this combined problem is crucial as it may necessitate a more complex invasive approach. Combination of surgical myectomy and AVR in patients who present with HOCM and severe AS is the treatment strategy.

Here, we report a rare case of Hypertrophic obstructive cardiomyopathy with Rheumatic heart disease, severe aortic stenosis and severe mitral stenosis with polycystic kidney disease, presenting for the first time at age of 67 years.

Case Report

A 67 years old hypertensive male, presented with complain of chest pain since 12 days, that increased in severity since 1 day, pain was diffuse, radiating to left arm, associated with sweating and numbness of left arm. Patient also had history of loss of consciousness and fall, 1month back.
Patient had no history of palpitation, dyspnoea, anasarca, gross hematuria, pain abdomen. No significant family history.

Examination

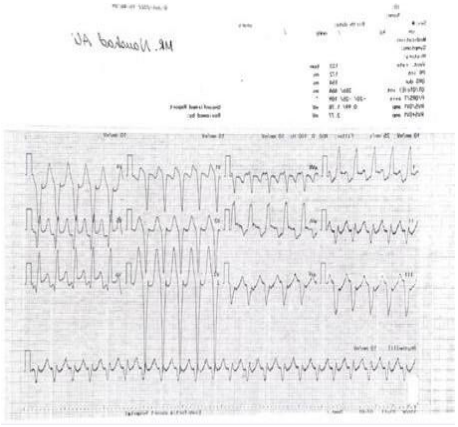
At the time of admission, the patient was conscious and fully oriented.

He was afebrile with pulse rate of 150 bpm, irregularly irregular and blood pressure was 98/60 mm Hg. Cardiac examination revealed mid systolic murmur (Levine III/IV) which significantly increased on standing up.

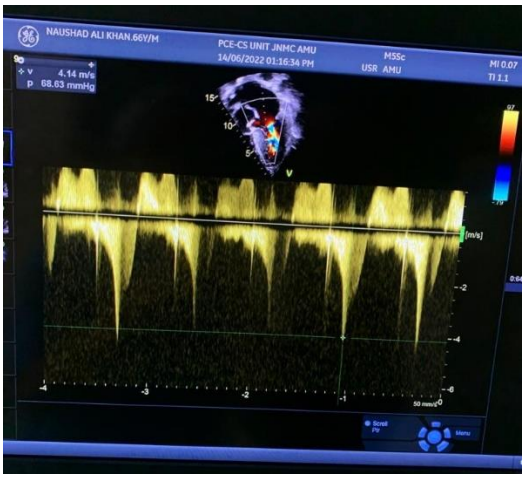
Investigations

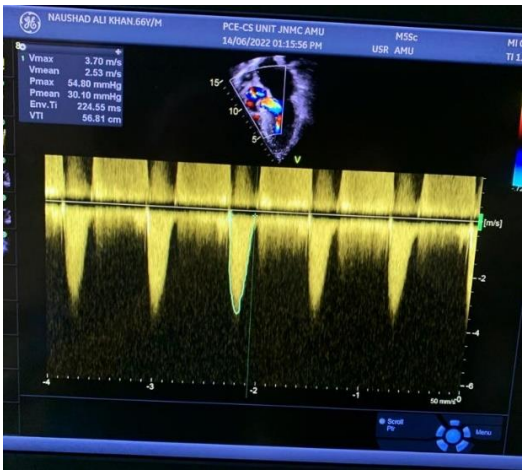
On biochemical evaluation renal function was deranged (Ur-52, cr- 1.7 mg/dl) and urine RM was normal. CBC, LFT, Trop I and NT-pro BNP were normal.

ECG: LVH with strain, AFib with FVR.



Transthoracic echocardiography: HOCM (LVOT GD- 69 MMHG and SAM) RHD, SEVERE AS with SEVERE MS with EF 70 %.





Coronary angiography: non critical CAD.

USG ABDO: bilateral bulky kidney with multiple variable size cyst, s/o PKD.

Management

Patient was admitted and stabilized for symptoms. Patient underwent DVR (AVR using #23 MERIL DAFODIL, MVR using #27 ST JUDE EPIC) bio-prosthetic valves, INTERVENTRICULAR SEPTAL MYECTOMY WITH LAA ligation.

Patient got discharged in stable condition from hospital and doing well post-surgery on medications.

Discussion

Hypertrophic cardiomyopathy is the most common of the genetic cardiovascular disease.HCM is characterized by a thickened but non dilated left ventricle in the absence of another cardiac or systemic condition.

Important to exclude Aortic valve stenosis, systemic hypertension, and some expressions of

physiologic athletes heart, capable of producing the magnitude of LV Hypertrophy evident.

The European Society of Cardiology guidelines recommend using a left ventricular wall thickness of ≥15 mm in the diagnostic criteria.[1] It has autosomal dominant pattern of inheritance.Among the known causal genes, *MYH7* and *MYBPC3* are the 2 most common, together being responsible for approximately half of the patients with familial HCM.[2]

While AS (valvular narrowing result in a fixed profile of obstruction), obstructive HCM (as a result of SAM of mitral valve and dynamic LVOT obstruction) can occur as a result of a complex interplay of basal septal hypertrophy, narrow LVOT, mitral valve/papillary muscle abnormalities, and a steeper LV inflow to outflow (aorto-LVOT) angle.[3]

It is not common to observe both AS and HCM simultaneously in the same patient, though there are patients in whom these conditions can coexist.[4]

The presence of sequential LVOT obstruction poses particular diagnostic challenges requiring meticulous imaging especially, Doppler echocardiography.

Correct identification of this dual obstructive physiology is also crucial for accurate diagnosis and has implications on family screening, follow-up recommendations, and therapeutic options.

There are currently no large-scale studies demonstrating that medical therapy is associated with a survival benefit in patients with obstructive HCM and, particularly, severe AS.

AVR and surgical myectomy are considered class 1 indications and definitive therapies to relieve fixed and dynamic obstruction in symptomatic patients with severe AS and obstructive HCM, respectively with excellent longer-term survival.[5]

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