Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Cardiology

Massive Restrictive Aortic Regurgitation Caused by Aortic Chordae Tendineae Strands: Unusual Links

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DOI: 10.36347/sjmcr.2024.v12i03.008 | **Received:** 15.12.2023 | **Accepted:** 19.01.2024 | **Published:** 12.03.2024

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Abstract **Case Report**

Determination of the etiology of a valvular heart disease is one of the major steps of every echocardiogram. Sometimes very unusual and rare etiologies can be found. In this article we report a case of very rare cause of aortic regurgitation, caused by the presence of aortic chordae tendineae strands leading to aortic cusps restriction in young asymptomatic athlete. In this case, we discuss two major problems. The first is the challenging diagnosis of this chordae tendineae strands, the second is the evaluation of an asymptomatic severe aortic regurgitation in a competitive athlete with an enlarged left ventricle.

Keywords: Chordae tendineae strands, aortic regurgitation.

INTRODUCTION

Aortic insufficiency is a very common finding in each echo-laboratory, with the main etiologies varying between degenerative, rheumatic disease and secondary to infective endocarditis. However, there are also more scarcest etiologies, very unusual, one of them being the restrictive aortic regurgitation caused by the presence of aortic chordae tendineae strands.

Learning Outcomes

- Discuss a very unusual etiology of aortic regurgitation
- Review echocardiogram features of severe aortic regurgitation
- Discuss the echo findings suggesting a restrictive aortic regurgitation by aortic chordae tendineae strands
- Discuss the 2021 ESC valve guideline pertaining to aortic regurgitation

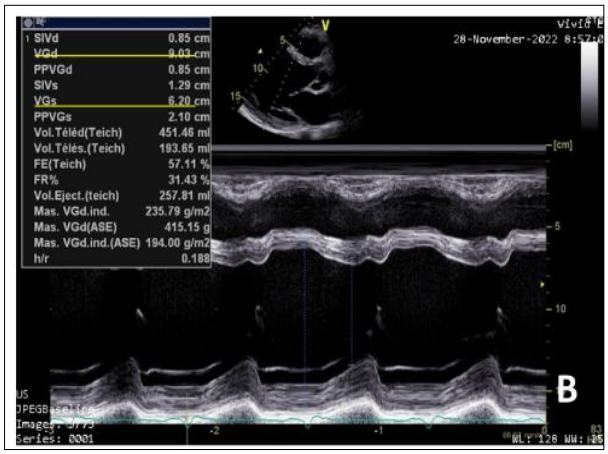
CASE PRESENTATION

A 31-year-old male patient, competitive athlete, basketball player, without any medical history was admitted to our department for the evaluation of an aortic regurgitation, newly discovered at a routine checkup; the first since a long period of inactivity, mainly due to the covid-19 pandemic.

He was totally asymptomatic. On physical examination, we noticed a pulsation of the carotid arteries and a widened pulse pressure with a systolic Blood pressure at 120 mmHg and a diastolic Blood pressure at 20 mmHg. The heart sounds were normal; a holodiastolic, II/IV grade murmur was perceived at the 3d and 4th left parasternal intercostal space.

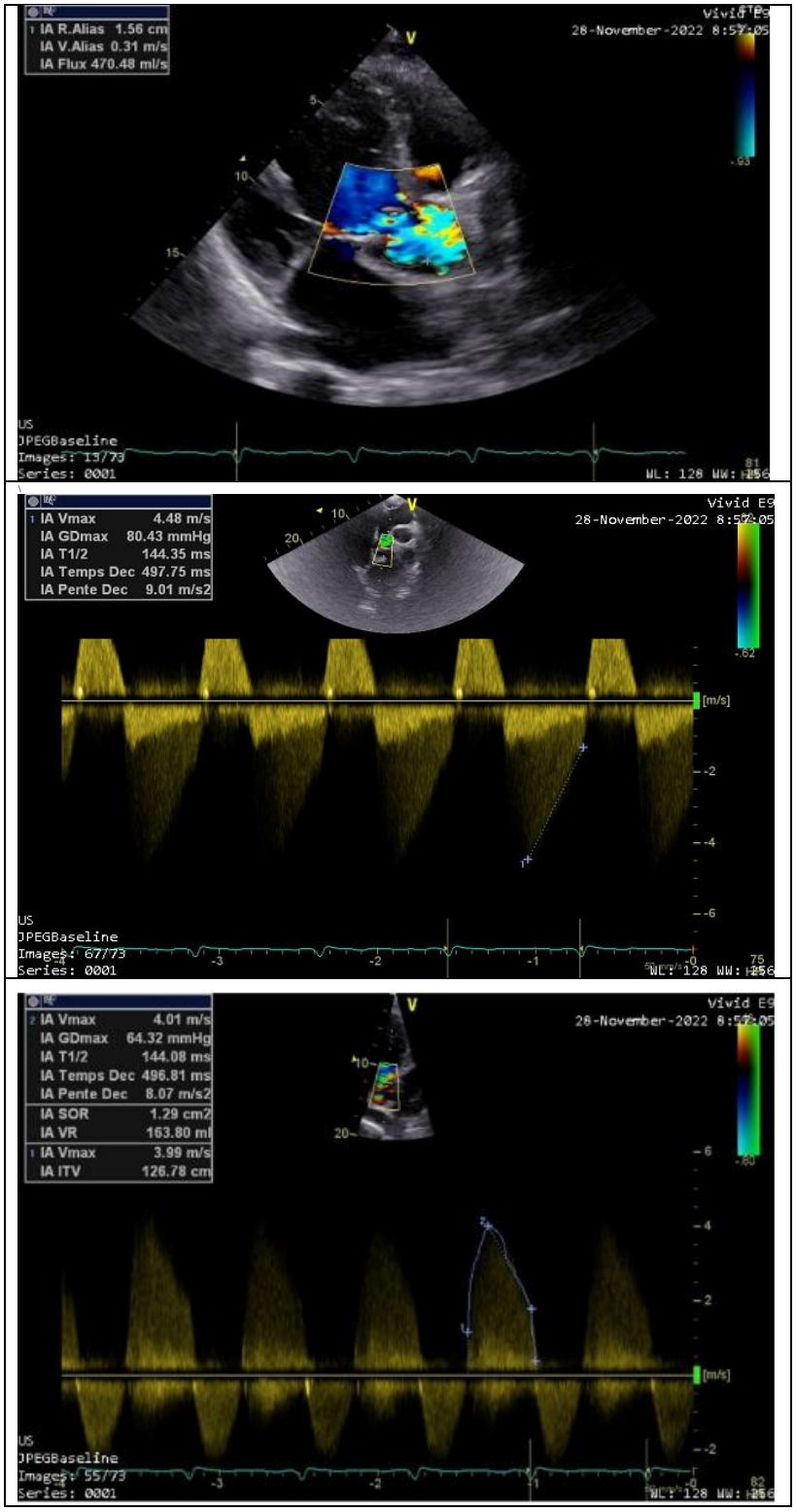
No fever or obvious inflammatory syndrome was present.

The electrocardiogram showed a sinus rhythm at a rate of 69 bpm, normal axis, left ventricular hypertrophy without repolarization abnormalities.



The patient underwent transthoracic echocardiography. The left ventricle was very dilated with a left ventricular end diastolic diameter (LVEDD) at 90 mm, a left ventricular end systolic diameter (LVESD) at 62 mm and an indexed LVESD 29 mm/m2, with normal wall thickness, and preserved global and segmental systolic function; ejection fraction at 50%.

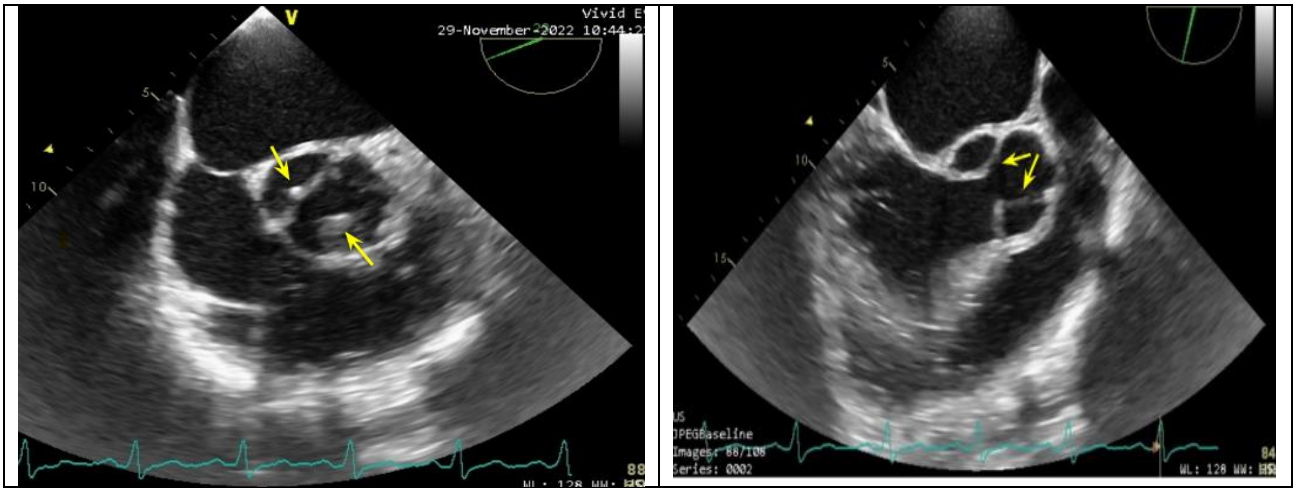
A massive aortic regurgitation was found, defined by an Effective regurgitant orifice area at 1.29 cm2 and a regurgitant volume at 171 ml, associated with a Pressure half time at 144 ms and a holodiastolic flow reversal in descending aorta with an end diastolic velocity at 55 cm/s.

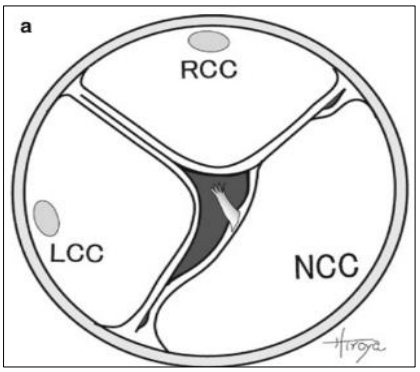


The tricuspid aortic valve is shown in figure ... The leaflet's motion was clearly restricted with a complete loss of coaptation contrasting with mild, marginal, thickening of the valve. We could also see abnormal structures: linear, non-mobile echos originating from the sinus of Valsalva.

There was a mild dilatation of the aortic root (diameter at sinus of Valsalva: 40 mm) with a normal annulus diameter.

A transesophageal echocardiogram revealed the presence of two strands, linking the non-coronary and the right coronary cusps to the aortic wall, at sinus of Valsalva, restricting their movement, and preventing their coaptation.

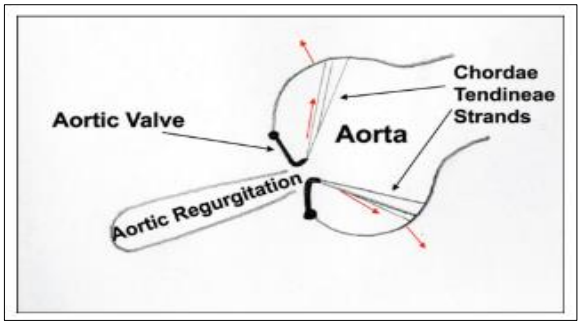




DISCUSSION

Aortic chordae tendineae strands are considered to be embryonic remnants of the cusp's formation process at an early stage of the aortic valve development. They can cause AR by two main mechanisms: restricted closure of the valve by the strands, and strand rupture between the aortic cusp and the aortic wall, typically associated with a dilated ascending aorta.

It is a very rare etiology, with only few cases reported; most of them were associated with the second mechanism, typically found in patients of advanced age, in whom rupture of strands can be caused by dilation of the ascending aorta and valve degeneration secondary to growth and aging, which makes our case even less common.



Our patient being totally asymptomatic, decision making about the management of his valvulopathy was based on the echocardiogram findings: the systolic function and the left ventricle (LV) enlargement, which have to been carefully evaluated in this young athlete.

Indeed, dilation of the LV is common and should be considered, to a certain point, as a normal finding in endurance Competitive athletes. In fact, several teams studied the exercise induced cardiac remodeling and reported a LVEDD > 55 mm in 45% of their athletes, 14% had a LVEDD > 60 mm, and a diameter > 70 mm was rarely found.

Similarly, LVESD may also be increased with athletic training with an upper limit at 49 mm.

Hence, athletes with severe AR and LVEDD or LVESD exceeding these values have a high likelihood that the regurgitation is contributing to the LV dilation. In our case, we were dealing with a severe, asymptomatic AR, with an ejection fraction at 50%, associated with a LV enlargement not solely explained by the physical training, which required a surgical treatment.

Aortic valve repair is an option only at centers with established expertise and experience and for patients with suitable anatomy; specific research is required to evaluate the clinical feasibility, the efficiency and the related complications of valve repair in restrictive AR with aortic strands.

Consequently, our patient was referred to surgery for aortic valve replacement.

CONCLUSION

Aortic regurgitation is a common valvular heart disease, with the degenerative etiology largely being the leading cause. However very unusual etiologies exist and thus require a careful evaluation. Our case highlights one of the rarest etiologies: aortic chordae tendineae strands causing restrictive aortic regurgitation, and emphasizes transoesophageal echocardiography which plays a central role in the assessment of severe aortic regurgitations with unexplained mechanism.

Surgical aortic valve replacement is for now, the main treatment for AR caused by aortic chordae tendineae strands, potentially associated with aortic root replacement if dilated.

Fundings: This work didn't receive any fundings.

Acknowledgments: None

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