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CASE REPORT

INTERMEDIATE

CLINICAL CASE

Which One to Treat When Pannus and Thrombus Coexist in a Mechanical Aortic Valve?

An Equivocal Case

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ABSTRACT

The coexistence of pannus and thrombus is not uncommon. Accurate diagnosis of the etiology of prosthetic valve dysfunction (PVD) is of utmost importance in guiding adequate and rational therapy. We present a case of PVD in which computed tomography played a decisive role in guiding treatment. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2021;3:533-6) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 71-year-old woman presented to the cardiology clinic with progressive shortness of breath for 3 months. At admission, she was afebrile, nondyspneic without angina, blood pressure 110/70 mm Hg, heart rate 78 beats/min, New York Heart Association functional class II, and no significant findings on electrocardiogram. Results of routine biochemical tests including cardiac markers and hemogram were normal. International normalized ratio (INR) was 1.2, which was below the effective range.

LEARNING OBJECTIVES

- To be able to determine accurate etiology of prosthetic valve dysfunction in the aortic position using multimodality imaging.
- To understand the role of CT as a problem-solver for imaging in the aortic position.

MEDICAL HISTORY

The patient had undergone aortic valve replacement (19-mm Standard, St. Jude Medical, St. Paul, Minnesota) for rheumatic valve disease 5.5 years ago.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis consisted of prosthetic valve pannus or thrombosis.

INVESTIGATIONS

Transthoracic echocardiography (TTE) showed preserved ejection fraction (62%), moderate to severe transvalvular aortic regurgitation, and increased transprosthetic gradients. Maximum pressure gradient (PG) was 126 mm Hg, and mean PG was 74 mm Hg (**Figure 1**). A stuck disc without any discernible obstructive lesions was noted on fluoroscopy (Video 1). Transesophageal echocardiography

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ABBREVIATIONS AND ACRONYMS

CT = computed tomography

EOA = effective orifice area

HU = Hounsfield unit

INR = international normalized ratio

PG = pressure gradient

PVD = prosthetic valve dysfunction

TEE = transesophageal echocardiography

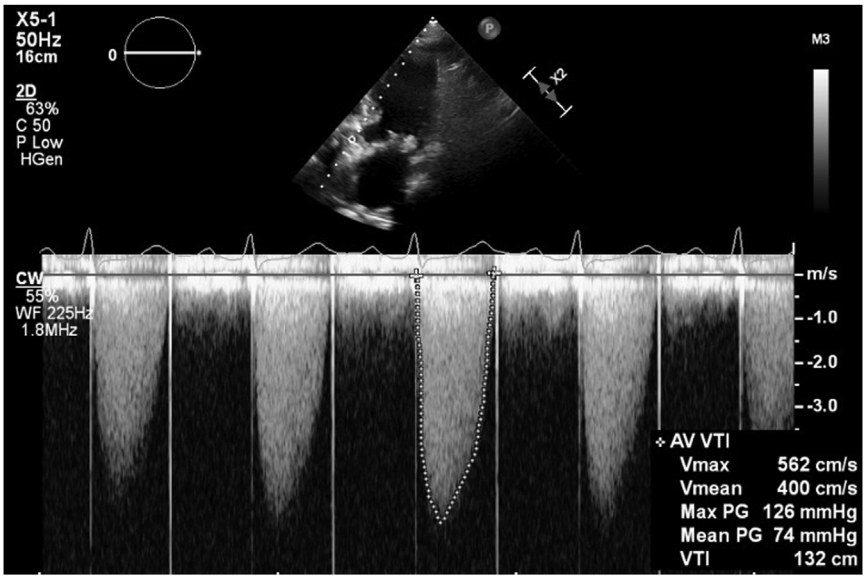
TTE = transthoracic echocardiography

(TEE) revealed significantly increased transprosthetic gradients (maximum PG 123 mm Hg; mean PG 75 mm Hg), prolonged acceleration time (109 ms), and decreased Doppler velocity index (0.21) and effective orifice area (EOA) (0.59 cm^2) (reference EOA 1.0 ± 0.2 cm^2). TEE confirmed moderate to severe transvalvular aortic regurgitation (Video 2). Leaflet mobility and the subvalvular region could not be evaluated by TEE due to acoustic shadowing on esophageal views (Video 3). An additional transgastric approach could not be obtained due to patient distress. Time in the therapeutic range was 35% for the last 6 months, which indicated probable prosthetic valve thrombosis. However, because the patient had undergone valve replacement more than 5 years ago and her symptoms were not definitively suggestive of acute valve dysfunction, the heart team excluded an obstructing pannus, which could not be excluded by echocardiography and fluoroscopy before thrombolytic therapy. Computed tomography (CT) was performed to aid in determining the etiology of prosthetic valve dysfunction (PVD). An opening angle of 62° and a closing angle of 95° with a stuck disc was apparent on cine images (**Figure 2**). CT revealed a low-density lesion (42 HU) measuring 8 × 6 mm (0.68 cm^2). The lesion was originating from the subvalvular region, growing into the ring housing, and interfering with the anterior hinge point, which resulted in the stuck disc. CT also showed a subvalvular nonobstructive pannus tissue (139 HU) that did not interfere with the prosthesis housing (**Figure 3**, Video 4).

MANAGEMENT

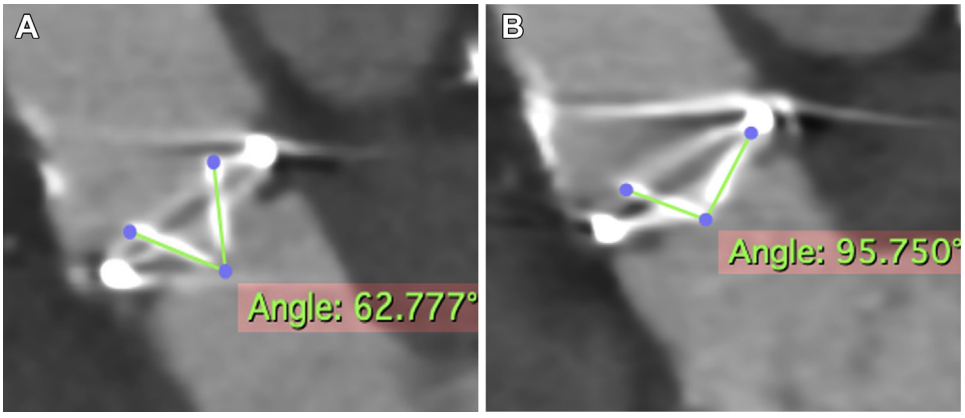
After considering the definitive findings obtained from CT, the heart team objectively justified the decision for thrombolytic therapy. After 6 h of infusion with 25 mg tPA tissue plasminogen activator, slight mobility was noted in the previously stuck disc, and TTE showed PG of 62/38 mm Hg. A double dose of tissue plasminogen activator (50 mg) was administered for the following 24 h. Control fluoroscopy revealed significantly reduced opening angle (Video 5). TTE showed significantly reduced transprosthetic gradient (48/20 mm Hg) and acceleration time (78 ms), and increased Doppler velocity index (0.34) and EOA (1.02 cm^2). Mild transvalvular aortic regurgitation was noted. The opening angle of the valve on fluoroscopy decreased to 24° from 45°. No hemorrhagic complications occurred. The patient was followed until INR reached an effective level and was discharged on warfarin therapy with regulation.

FIGURE 1 Transprosthetic Pressure Gradient on Transthoracic Echocardiography



Transthoracic echocardiography showing significantly increased transprosthetic pressure gradient (PG) (maximum PG 126 mm Hg; mean PG 74 mm Hg). VTI = velocity time integral.

FIGURE 2 Assessment of Prosthetic Valve Angles on Cardiac Computed Tomography



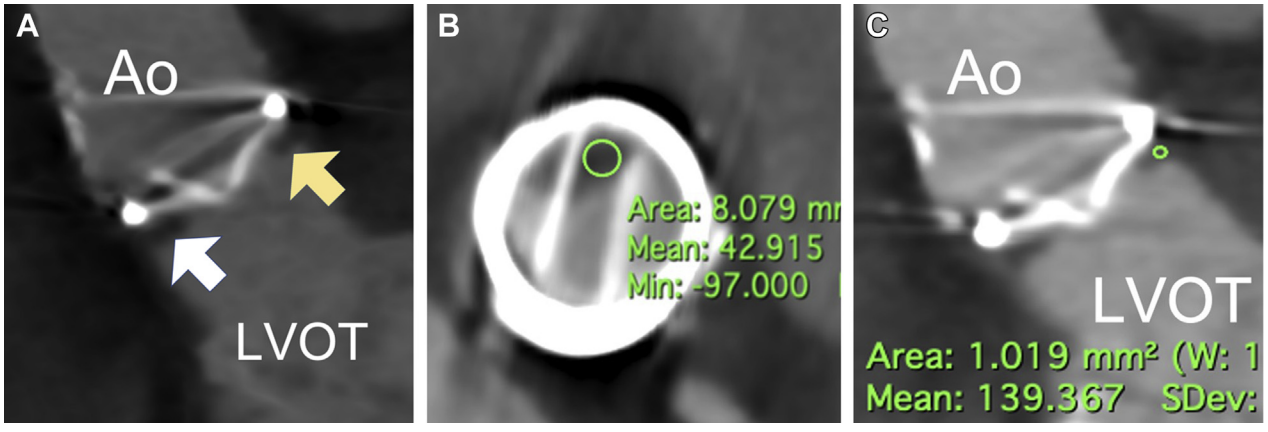
Computed tomography showing increased opening angle **(A)** and decreased closing angle **(B)** of the prosthetic discs.

DISCUSSION

Determination of valve dysfunction due to pannus versus thrombus is a demanding task when it is solely based on clinical presentation because both etiologies can have a similar presentation. The clinical spectrum ranges from mild dyspnea to acute pulmonary edema, which is not specific to the etiology (1). We acknowledge that thrombus is the probable cause given a history of insufficient anticoagulation, acute onset of valve dysfunction, and shorter duration between symptoms and surgery. Although our patient had inefficient anticoagulation, the long duration from surgery and the lack of signs for acute valve dysfunction raised concern for coexisting obstructing pannus because its presence could change management (2).

The success rate of thrombolytic therapy for prosthetic valve thrombosis has been reported to vary significantly from 62% to 82%, and the therapy is associated with risk of complications such as bleeding, embolization, and allergic reactions (3). The need for surgery is common when thrombolytic therapy fails because of large thrombi (>0.8 cm^2)

FIGURE 3 Assessment of Prosthetic Valve Lesions on Cardiac Computed Tomography



(A) Coronal reformatted computed tomography image depicting thrombus **(white arrow)** interfering with the hinge point of the aortic prosthetic valve, resulting in a stuck disc, and subvalvular pannus **(yellow arrow)** without housing ingrowth. **(B)** En face view showing the thrombus with low density (42 HU) in the valve housing. **(C)** Coronal view showing subvalvular pannus with high soft tissue density (139 HU). Ao = aorta; LVOT = left ventricular outflow tract.

and/or frequent coexistence of pannus (4). Thus, several tasks are essential before thrombolytic therapy: document the presence of the thrombus as the responsible pathology; quantify the thrombus; and determine the most efficient management (1,5,6). Echocardiography is highly prone to acoustic shadowing from metallic valve structures, especially in the aortic position. Three-dimensional TEE can clearly delineate vegetations, obstructive lesions, or paravalvular complications, but its ability to depict obstructing lesions in the aortic position may be significantly impeded by metallic artifacts (7,8). Cine fluoroscopy can provide angle measurements for the occluders before and after thrombolytic therapy, but it lacks the capacity for depicting the underlying etiology (9). CT has the advantage of distinguishing the most probable cause of PVD. Density measurement of the lesion can reliably distinguish between thrombus and pannus, and evaluation of the size and extent of the lesion can provide a rationale for thrombolytic therapy or surgery (1,5,6). Thrombolytic therapy not only is associated with the risk of bleeding or embolism, but it also has the probability of lower success in cases of large organizing thrombus or coexistence of pannus (4,10). These facets highlight the rationale for use of CT, and current guideline recommends the use of CT as class 1 evidence in suspected prosthetic valve thrombosis (1). In the multimodality imaging era, the decision-making process should not rely solely on conventional clinical manifestations or indirect imaging findings for aortic PVD. Rather, it should incorporate effective use CT, which can yield objective indications for appropriate treatment.

FOLLOW-UP

After 3 months, mean transaortic PG was 22 mm Hg, and INR rate was within the effective range. The patient was symptom-free.

CONCLUSIONS

We present a case of PVD in the aortic position with equivocal echocardiographic and clinical findings, but CT revealed valve thrombosis as the underlying etiology, thus providing the rationale for accurate treatment given the coexistence of pannus and thrombus. We highlighted that CT can be a problem-solver for imaging in the aortic position in case of PVD.

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KEY WORDS pannus, prosthetic valve dysfunction, thrombus

APPENDIX For supplemental videos, please see the online version of this paper.