



# Prosthetic Tricuspid Valve Thrombosis: Three Case Reports and Literature Review

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## Abstract

*A common complication of prosthetic heart valves is thrombosis. Although the incidence of prosthetic valve thrombosis (PVT) in the tricuspid position is high, there are not enough data on the management of it, in contrast to left-sided PVT. Here, we describe three cases of tricuspid PVT with three different management approaches: thrombolytic therapy; close observation with oral anticoagulants; and surgery. The first case was a woman who suffered from recurrent PVT, for which we successfully used Tenecteplase for second and third episodes. We employed Tenecteplase in this case for the first time in the therapy of tricuspid PVT. The second case had fixed leaflets in open position while being symptomless. At six months' follow-up, with the patient having taken oral anticoagulants, the motion of the leaflets was restricted and she was symptom-free. The last case was a woman who had a large thrombus in the right atrium immediately after mitral and tricuspid valvular replacement. The patient underwent re-replacement surgery and a new biological valve was implanted in the tricuspid position. Also, we review the literature on the pathology, signs and symptoms, diagnosis, and management of tricuspid PVT.*

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## Introduction

Since the 1950s, more than 80 models of the prosthetic heart valve have been developed and used.<sup>1</sup> Prosthetic valve thrombosis (PVT), however, remains a serious complication and can even prove lethal. Overall, the incidence of thrombosis is reported to be between 0.1% and 5.7% per patient-year.<sup>2</sup> The incidence is 0.5% to 6% in the aortic and/or mitral positions and up to 20% in the tricuspid position, whereas the risk of thrombosis in spite of adequate oral anticoagulation has been estimated at between 1% and

4% per year.<sup>3</sup> Although inadequate anticoagulant therapy remains the main cause of this complication, it seems that lower pressures on the right side of the heart with a slower blood flow across the tricuspid valve is the most important cause of higher risk of thrombus formation in prosthetic tricuspid valves.<sup>4,5</sup> In contrast to left-sided PVT, there is a paucity of data on the various aspects of tricuspid PVT. We herein present three cases of tricuspid PVT with different management approaches, namely thrombolytic therapy, conservative management, and re-replacement surgery, and then review the relevant literature.

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## Case Reports

### Case # 1

A 32-year-old woman was admitted to our hospital with the complaint of dyspnea (The New York Heart Association [NYHA] fractional class III) in January, 2004. Transthoracic echocardiography (TTE) showed left ventricular ejection fraction of 55%, severe mitral stenosis, mild mitral regurgitation, severe aortic regurgitation with moderate to severe aortic stenosis, and severe tricuspid regurgitation with moderate to severe tricuspid stenosis. In April 2004, the patient underwent three valves replacement surgery, during which she received a 24-mm St. Jude Valve (St. Jude Medical, Inc., St. Paul, MN, USA) in the mitral position, a 31-mm St. Jude Valve (St. Jude Medical, Inc., St. Paul, MN, USA) in the tricuspid position, and a 19-mm Regent Mechanical Prosthesis (St. Jude Medical, Inc., St. Paul, MN, USA) in the aortic position. Also, due to persistent atrial fibrillation with a slow ventricular response, she underwent permanent transvenous epicardial pacemaker placement during hospitalization. Twenty-one days after valvular surgery, TTE revealed that the function and gradients of the three prosthetic valves were within the acceptable range and the peak and mean gradients in the prosthetic tricuspid valve were 5 mm Hg and 3 mm Hg, respectively. At the time of discharge, the patient's international normalized ratio (INR) was 3.9. She was discharged from the hospital with the recommendation to use Warfarin (with goal INR 2.5 - 3.5) plus 80 mg Aspirin daily.

In February, 2006, the patient was re-admitted with the complaint of fatigue and palpitation. The pacemaker had a normal function. At the time of presentation, her INR was 2.5. TTE revealed that the prosthetic aortic and mitral valves had normal functions and gradients, whereas the prosthetic tricuspid valve had malfunction with high gradients. Fluoroscopic evaluation revealed that there was no motion in both leaflets of the prosthetic tricuspid valve, while the motion of both other prosthetic valves was complete and within the normal range. With the diagnosis of tricuspid PVT, the patient was prescribed 250,000 U of Streptokinase via a peripheral vein over thirty minutes, followed by an intravenous infusion of 100,000 U per hour of Streptokinase for forty-eight hours. On the next day, fluoroscopic evaluation showed no evidence of prosthetic tricuspid valve malfunction, and the mobility of both leaflets was completely restored. At discharge, the patient's INR was 2.6. She was discharged from the hospital with the recommendation to use Warfarin (with goal INR 3.0 - 3.5) plus 80 mg of Aspirin daily.

In November, 2006, the patient was re-admitted to our hospital with the complaint of atypical chest pain. At presentation, her INR was 1.8 and her other laboratory data and also electrocardiogram were unremarkable. TTE

revealed that the prosthetic aortic and mitral valves had normal functions and gradients, whereas the prosthetic tricuspid valve had malfunction with high gradients. Fluoroscopic evaluation revealed that both leaflets of the prosthetic tricuspid valve were fixed without any motion, whereas the motion of both other prosthetic valves was complete and within the normal range. With the diagnosis of recurrent tricuspid PVT, the patient was administered a total of 35 mg of Tenecteplase: 15 mg bolus, followed by 20 mg for four hours (the patient's weight was 64 kg). On the next day, fluoroscopic evaluation showed no evidence of prosthetic tricuspid valve malfunction, and the mobility of both leaflets was completely restored. Twelve days after the administration of Tenecteplase, TTE demonstrated that a significant reduction had occurred in the gradient across the prosthetic tricuspid valve (9.5 mm Hg peak gradient, 4.9 mm Hg mean gradient). At discharge, the patient's INR was 3.5. She was discharged from the hospital with the recommendation to use Warfarin (with goal INR 3.0 - 3.5) plus 80 mg of Aspirin daily.

Because of the battery depletion of the pacemaker, the patient was re-admitted for generator replacement on October 30, 2011. She had discontinued Aspirin two years previously due to gastrointestinal problems. She complained of atypical chest pain of two months' duration, but her myocardial perfusion scan was normal. TTE showed left ventricular ejection fraction of 55%, severe increased gradient in the prosthetic tricuspid valve with no paravalvular leakage, and normal function of the mitral and aortic prosthetic valves. Fluoroscopic evaluation of the prosthetic tricuspid valve revealed a severe drop in the motion of both leaflets. The patient was candidate for thrombolytic therapy with the diagnosis of recurrent tricuspid PVT. A single dose of 35 mg of Tenecteplase was administered via a peripheral vein, according to the dosing regimen used for acute myocardial infarction (the patient's weight was 65 kg). Fluoroscopic evaluation exhibited no evidence of prosthetic tricuspid valve malfunction, and the mobility of both leaflets was completely restored. Seven days later, TTE demonstrated that a significant reduction had occurred in the gradients across the prosthetic tricuspid valve (9 mm Hg peak gradient, 4 mm Hg mean gradient). She was discharged from the hospital with the recommendation to use Warfarin (with goal INR 3.0 - 3.5) plus 80 mg of Aspirin daily. Table 1 illustrates the three episodes of tricuspid PVT in our patient and the three successful methods for thrombolytic therapy.

### Case # 2

A 49-year-old woman was admitted to our hospital for the replacement of the generator of a pacemaker in May 2011. Thirteen years previously in another center, because of severe aortic regurgitation and severe tricuspid regurgitation, she



Table 1. Three episodes of tricuspid PVT in case 1 and successful thrombolytic therapy

Episode	Time from valve replacement	TTE findings in prosthetic tricuspid valve (before thrombolytic therapy)		Thrombolytic drug	Dosage
		Peak gradient	Mean gradient		
1	22 months	14 mm Hg	11 mm Hg	Streptokinase	250,000 U loading dose followed by 100,000 U/h for 48 hours
2	31 months	22 mm Hg	10 mm Hg	Tenecteplase	15 mg bolus, followed by 20 mg for 4 hours
3	93 months	17 mm Hg	8.5 mm Hg	Tenecteplase	35 mg bolus

PVT, Prosthetic valve thrombosis; TTE, Transthoracic echocardiogram

had undergone two valves replacement surgery, during which she received a 31-mm St. Jude Valve (St. Jude Medical, Inc., St. Paul, MN, USA) in the tricuspid position and a 21-mm St. Jude Valve (St. Jude Medical, Inc., St. Paul, MN, USA) in the aortic position. After surgery, due to the presence of complete heart block, she underwent permanent epicardial pacemaker placement during hospitalization. But during this period, she was admitted frequently because of infection at the site of the pacemaker. At the time of recent presentation, her INR was 1.9. TTE revealed that the prosthetic aortic valve had normal function and gradients, whereas the prosthetic tricuspid valve had malfunction with high gradients (15 mm Hg peak gradient, 10 mm Hg mean gradient). Fluoroscopic evaluation revealed that the motion of one leaflet of the prosthetic tricuspid valve was very restricted, while the other leaflet was fixed in an open position. The patient was symptomless. On physical examination, except for a holosystolic murmur at the left lower sternal border, there were no signs of heart failure. With the diagnosis of tricuspid PVT, the patient was administered 250,000 U of Streptokinase via a peripheral vein over thirty minutes, followed by an intravenous infusion of 100,000 U per hour of Streptokinase for forty-eight hours. Fluoroscopic evaluation showed no improvement in the motion of the leaflets. The patient was candidate for tricuspid valve replacement, but because of fungal infection in the pacemaker pocket and lead, replacement of the prosthetic valve or the generator was not performed. She was discharged from the hospital with the recommendation to use antibiotics with Warfarin (with goal INR 3.0 - 3.5) plus 80 mg of Aspirin daily. Six months later, she referred to our clinic; she had no signs or symptoms of heart failure. She was re-admitted for the replacement of the generator of the pacemaker. Before replacement, fluoroscopic evaluation showed no evidence of improvement in the motion of the prosthetic tricuspid valve leaflets. Because the patient was symptom-free, did not respond to thrombolytic therapy, and had a high risk for infection after valve replacement, surgery for valve replacement was not performed. After generator replacement, she was discharged with the recommendation to use Warfarin (with goal INR 3.0 - 3.5) plus 80 mg of Aspirin daily.

### Case # 3

A 28-year-old woman was admitted to our hospital with the complaint of orthopnea and dyspnea (NYHA fractional class III) in September, 2008. The electrocardiogram revealed atrial fibrillation. TTE showed left ventricular ejection fraction of 40%, severe mitral stenosis, severe tricuspid regurgitation, and a large mobile clot in the left atrium (2.7 × 2.0 cm). She underwent two valves replacement surgery, during which she received a 31-mm St. Jude Valve (St. Jude Medical, Inc., St. Paul, MN, USA) in the tricuspid position and a 29-mm St. Jude Valve (St. Jude Medical, Inc., St. Paul, MN, USA) in the mitral position. Also in the same section of surgery, TEE revealed a large mobile clot in the left atrium without any lesion in the other chambers; the large clot was removed. After surgery, she received an intravenous bolus dose of 5000 U of heparin, followed by intravenous heparin (20000 U per day in divided doses). Because of persistent atrial fibrillation with a low ventricular response after surgery, eleven days later, she underwent permanent transvenous epicardial pacemaker placement. However, during implantation, fluoroscopy revealed that the motion of one leaflet of the prosthetic tricuspid valve was very restricted. With the diagnosis of the malfunction of the prosthetic tricuspid valve, she was transferred to the operating room again. During the removal of the previous prosthetic tricuspid valve, a large clot was seen in the right atrium which was attached to the leaflet. Thus, all of the thrombus was removed and a new 29-mm Hancock II bioprosthesis (Medtronic Inc., Minneapolis, Minn.) was placed in the tricuspid position. Because of the displacement of the transvenous epicardial lead during surgery, a new epicardial lead was placed. After surgery, TEE demonstrated an acceptable gradient across the tricuspid valve (5 mm Hg peak gradient, 2 mm Hg mean gradient). She was discharged with the recommendation to use Warfarin (with goal INR 3.0 - 3.5) plus 80 mg of Aspirin daily.

## Discussion

### Pathogenesis

Many studies have demonstrated that the leading cause of PVT is subtherapeutic anticoagulation, which chimes in with the findings in our two cases.<sup>6-10</sup> This is most often due to either patient noncompliance or iatrogenic cessation of anticoagulants in preparation for another procedure. Furthermore, valve design and materials influence the incidence of thrombotic complications. Some mechanisms have a role in PVT formation such as molecular interactions and influence of transprosthetic blood flow. Molecular interaction occurs between corpuscular blood components, plasma, and artificial surfaces. The initial adsorption of plasma proteins (fibrinogen, fibronectin, von Willebrand factor, vitronectin, and thrombospondin) on the artificial surface is generally followed by platelet adhesion. The passage of blood through the prosthetic valve creates a turbulent flow with shear stress, which gives rise to a structurally and metabolically damaged endocardium and thus reduces its resistance to thrombosis. Also, subclinical hemolysis with the release of adenosine diphosphatase, platelet factor 4, beta-thromboglobulin, and other proteins triggers the activation of the plasma coagulation system. Other intrinsic factors can progress to thrombus formation; these factors include loss of active atrial contractions (atrial fibrillation), presence of some systemic diseases (e.g. systemic lupus erythematosus) or malignant tumors, and incomplete endothelialization of the sewing ring. Use of some drugs such as contraceptives leads to hypercoagulability state.<sup>3, 11-14</sup> Recently, Ricome et al. reported two cases of PVT secondary to heparin-induced thrombocytopenia.<sup>15</sup>

Type and position of the prosthetic valve and time from surgery can influence thrombus formation (Table 2).<sup>1, 13, 16, 17</sup> Additionally, some studies have indicated that season can be correlated with an increased risk for thrombotic events.<sup>18-20</sup> Piper et al. reported that PVT during winter months occurred more frequently than in the other seasons.<sup>13</sup>

### Signs and Symptoms

In contrast to the acute presentation of left-sided PVT, the onset of the symptoms of tricuspid PVT is usually insidious, and its diagnosis is often delayed. Sometimes symptoms are so slight that the patient is likely to have suffered from

them for months or even a year without feeling the need for referral to the hospital.<sup>4</sup> Sometimes, the patient even may have no symptoms related to the tricuspid valve thrombosis, and the thrombosis is detected only during routine clinical examination.<sup>4, 6</sup> However, the involvement of both leaflets is usually required to produce symptoms.<sup>6</sup> The most frequent symptoms related to tricuspid valve malfunction include dyspnea, ascites, peripheral edema, and systemic emboli.<sup>4, 6, 21, 22</sup> Also, sometimes the disappearance or attenuation of the prosthetic valve noise may be reported by the patient and/or relatives. Moreover, in some cases where an interatrial communication is present, a pulmonary embolus or a left-sided embolic event may be the presenting manifestation of tricuspid PVT.<sup>23, 24</sup>

It seems that physical examination may provide important clues for the diagnosis of tricuspid PVT, compared to the thrombosis of the left side, which normally has more severe symptoms.<sup>25</sup> Absence or muffling of prosthetic sounds in the tricuspid position might be noted. Other findings include auscultation of a new holosystolic murmur located at the left lower sternal border or in the subxiphoid region that may increase with inspiration or maneuvers that increase venous return (denoting the presence of tricuspid regurgitation), auscultation of a new murmur that is low in frequency, diastolic, located at the lower left sternal border or infraxiphoid area, and increases with inspiration and other maneuvers that increase tricuspid flow velocity (denoting tricuspid stenosis), or auscultation of a combination of murmurs that characterize both of these conditions. Sometimes mid-diastolic and/or pan-systolic murmurs can be heard in the tricuspid area (Zhang DY, Lozier J, Chang R, Sachdev V, Chen MY, Audibert JL, Horvath KA, Rosing DR. Case study and review: Treatment of tricuspid prosthetic valve thrombosis. *Int J Cardiol* 2011 Oct 14. [Epub ahead of print]).

### Diagnosis

The onset of the symptoms of tricuspid PVT is usually insidious and sometimes the patient has nonspecific symptoms or is even symptomless; therefore, suspicion of tricuspid PVT may be raised by physical findings, symptoms of heart failure, or rarely the diagnosis of embolization, especially in patients with poor anticoagulation therapy.<sup>8, 17, 25</sup> NYHA has classified PVT in functional classes I to IV. The non-obstructive forms of PVT (NYHA functional

Table 2. Thrombogenicity of mechanical prosthetic valve based on type, position, and time from surgery

	Lower	Higher
Valve type	Bileaflet-tilting-disk	Caged-ball single-tilting-disk
Position	Mitral or aortic	Tricuspid
Time from replacement	After 3 months	First 3 months



classes I or II) are usually incidental echocardiographic findings in patients with symptomless, nonspecific symptoms, or thromboembolic events. The obstructive forms (NYHA functional classes III or IV) correspond to obvious hemodynamic repercussions, sometimes including cardiogenic shock, and are often associated with cerebral or peripheral embolism.<sup>3, 11, 26</sup>

Imaging modalities in patients with suspected PVT are aimed to evaluate leaflet immobilization, cause of leaflet immobilization (thrombus versus pannus or both), and whether thrombolytic therapy attempt in the patient would be successful. Usually the first modality for detecting prosthetic tricuspid valve dysfunction is TTE. Montorsi et al. reported that sensitivity, specificity, and positive and negative predictive values for the diagnosis of PVT in the mitral or aortic position by TTE were 75%, 64%, 57%, and 78%, respectively. TTE, especially in experienced hands, can detect increased transvalvular gradients (mean of 6 mm Hg or higher, and peak of 15 mm Hg or higher), pressure half-time of 230 msec or higher, transvalvular gradients of 50% or higher than that observed before, wide intravalvular jet of tricuspid regurgitation, lower orifice area, visible thrombus on the prosthetic valve, and inability to demonstrate two different mobile echoes representing the valve leaflets in a high quality image. Indirect, nonspecific signs are an enlarged right atrium and engorged inferior vena cava.<sup>3, 6, 27</sup>

After performance of TTE, the diagnosis should be confirmed by more specific modalities, namely fluoroscopy or TEE.<sup>28</sup> Fluoroscopy is a non-invasive method for detecting PVT, especially in patients with bileaflet prosthetic valves, and have high clinical suspicious for PVT and normal Doppler study.<sup>29</sup> Sensitivity, specificity, and positive and negative predictive values for the diagnosis of PVT in the mitral or aortic position by fluoroscopy are 87%, 78%, 80%, and 91%.<sup>28</sup> Also, fluoroscopy has an important role for detecting the response to thrombolytic therapy. Thrombolysis significantly reduces the mean pressure gradient and improves valve leaflet opening angle. But some patients whose pressure gradient normalizes after thrombolytic infusion tend to continue to have concomitant abnormal leaflet motion at fluoroscopy, suggesting incomplete resolution of valve obstruction (pseudo responders). If lytic infusion is stopped at this time, the remaining thrombus could be the trigger for a late rethrombotic process. Thus, fluoroscopy should be carried out at regular intervals during therapy to confirm Doppler changes.<sup>29</sup>

TEE can correctly identify opening and closing angles in all patients, regardless of the prosthetic type.<sup>30</sup> TEE should be performed in selected patients even if fluoroscopy is negative because TEE is an invasive modality. On the other hand, fluoroscopy and TTE can correctly identify PVT in 85% of all cases. Thus, fluoroscopy and TTE are quick, effective, and complementary diagnostic tools for the diagnosis of PVT in most patients.<sup>28</sup> Despite the scarcity of

data on the role of TEE in diagnosing tricuspid PVT, it seems that if there is high clinical suspicion and other diagnostic modalities are not helpful, TEE will be help.<sup>27</sup> Furthermore, TEE is a superior modality for detecting the etiology of the obstruction (thrombus versus pannus), size, and location of the thrombus compared with TTE and fluoroscopy.<sup>31</sup>

Magnetic Resonance Imaging (MRI) and cardiac catheterization have limited diagnostic roles, because TEE and fluoroscopy can provide adequate data for decision-making. Since MRI is more expensive and time-consuming than echocardiography, it should be used only when prosthetic-valve regurgitation or paravalvular leakage is suspected but not adequately visualized by echocardiography.<sup>1</sup> In contrast, Cardiac Multi-Detector Computer Tomography can provide sharp images to characterize quantitatively the reduced mobility of prosthetic leaflets or even directly visualize and distinguish between thrombus and pannus.<sup>32-34</sup>

### Treatment

There are different therapeutic modalities available for PVT such as heparin treatment, thrombolysis, surgery, or even in some cases only watchful waiting. Selecting one of these modalities is largely influenced by the presence of valvular obstruction, valve location (left- or right-sided), and clinical status.<sup>6, 35</sup> Surgery is more frequently performed for the treatment of left-sided PVT, not least in patients with either NYHA functional class III–IV symptoms or a large clot burden<sup>36</sup> and thrombolytic therapy is more favorable for right-sided PVT, because the risk of systemic embolization and recurrence rate is high by thrombolytic therapy in left-sided PVT.<sup>37, 38</sup>

The conservative continued anticoagulation approach in patients with tricuspid PVT would only be appropriate if there is no significant hemodynamic compromise or a contraindication to either surgery or pharmacologic intervention is present. Shapira et al. reported that asymptomatic patients with tricuspid PVT who did not respond to thrombolytic therapy might be discharged from the hospital with long-term intensified anticoagulant therapy and close follow-up. The leaflet motion can be fully restored later.<sup>6</sup> However, Montorsi et al. proposed that leaflet mobility and duration of prosthetic valve symptoms were important factors in determining successful thrombolytic therapy. It may be because the amount of the thrombus that led to the stuck valve was minimal, thereby improving the chance of successful thrombolytic therapy.<sup>29</sup>

According the guidelines of the American Heart Association /American College of Cardiology (AHA/ACC) and the American College of Chest physicians (ACCP), in contrast to left-sided PVT, thrombolytic therapy is reasonable for right-sided PVT with NYHA functional class III-IV symptoms or a large clot burden.<sup>36, 39</sup> It is due to the high success rate and

low incidence of embolism compared to left-sided PVT. The European Society of Cardiology guideline also recommends thrombolytic therapy for tricuspid PVT, but this guideline does not mention the NYHA functional class or clot burden.<sup>40</sup> Long-standing symptoms even more than a month should not make one reluctant to use thrombolytic therapy.<sup>6</sup> If thrombolytic therapy fails, the presence of a large thrombus or pannus should be considered, which may require surgical intervention (thrombectomy or valve replacement).

In contrast to left-sided PVT, there is limited information about thrombolytic therapy for tricuspid PVT. Various thrombolytic agents have been used for PVT, including streptokinase, urokinases, and tissue-type plasminogen activator (tPA). The choice of the thrombolytic agent depends on several factors, including cost, time to attain maximal pharmacologic effect, half-life of the thrombolytic agent, and hemorrhagic complications. Amongst the above agents, streptokinase is cheaper and has lower cerebral hemorrhage rates. In contrast, tPA has a faster effect reversion and faster reach to maximal pharmacologic effect.<sup>3</sup> Roudaut et al. indicated that patients treated by streptokinase had a significantly full success rate compared to patients treated by tPA or urokinases (86%, 68%, and 59%, respectively). Nonetheless, combined therapy improved the results of thrombolytic therapy in all the groups.<sup>41</sup> Also, they concluded that full success by thrombolytic therapy was higher in patients in NYHA functional classes I or II; nevertheless, they did not find a significant difference between patients with tilting-discs and bileaflet valves, or between patients with first episode of thrombosis and recurrent thrombotic episodes groups.<sup>41</sup> The dosage and route of the administration of thrombolytic therapy are different in various studies. Hering et al. recommended using streptokinase by starting a bolus dose of 250,000 IU over thirty minutes, followed by an intravenous infusion of 100,000 IU per hour (same as the therapy of our patient in the first episode of PVT), urokinase by the same protocol used in patients with acute pulmonary embolism, and t-PA at a dosage of 100 mg given over a period of two to five hours.<sup>14</sup> Caceres-Loriga et al. recommended using streptokinase by starting a bolus dose of 250,000 IU over three minutes, followed by an intravenous infusion of 100,000 IU per hour (maximum duration of seventy-two hours), urokinase by starting a bolus dose of 4500 U/kg, followed by an intravenous infusion of 4500 U/kg/h (maximum duration of twenty-four to forty-eight hours), and tPA by starting a bolus dose of 15 mg over five minutes, followed by an intravenous infusion of 95 mg over ninety minutes.<sup>3</sup> Manteiga et al. used the short-course of thrombolytic therapy as a first line for PVT: streptokinase by starting a bolus dose of 250,000 IU over twenty minutes, followed by an intravenous infusion of 1,500,000 IU over ninety minutes, or tPA by starting a bolus dose of 10 mg, followed by an intravenous infusion of 90 mg over ninety minutes. They concluded that a successful rate by these

regimes was 82%.<sup>42</sup> However, Alpert recommended another dose for streptokinase in right-sided PVT (starting a dose of 500,000 IU over twenty minutes, followed by an intravenous infusion of 1,500,000 IU over ninety minutes).<sup>43</sup> Some other investigators have used direct intra-atrial infusion of thrombolytic for PVT.<sup>44, 45</sup> For the first time, Zhang et al. reported a case of tricuspid PVT, which was successfully treated by an intra-right atrium infusion of tPA.

Recently, Tenecteplase (a genetically engineered variant of tPA which has a longer half-life than tPA and is resistant to inactivation by plasminogen activator inhibitor-1<sup>46</sup>) has been utilized for PVT. Our literature search shows that Tenecteplase has been used in limited case reports for mitral or aortic PVT.<sup>47-52</sup> Although Tenecteplase has been prescribed in different doses and via different methods, we used this thrombolytic according to the dosing regimen employed for acute myocardial infarction. Data on Tenecteplase for the treatment of PVT are limited. Still, Melandri et al. in a review study about patients with acute myocardial infarction showed that this drug had some advantages compared with tPA. These advantages included being more fibrin-specific, usability in a single bolus dose, and having less non-cerebral bleeding. Be that as it may, mortality rates and intracranial hemorrhage rates were similar to those of tPA.<sup>46</sup> For the first time, we reported a successful use of Tenecteplase in our case 1 for the treatment of two episodes of recurrent tricuspid PVT. It seems that this drug might be a suitable alternative for the other types of tPA in the future.

If thrombolytic therapy is successful, a continuous infusion of unfractionated heparin is indicated and should be initiated. Moreover, activated partial thromboplastin time should be maintained at twofold the baseline values, followed by conversion to oral anticoagulation combined with Aspirin (50 to 100 mg per day).<sup>39</sup> In contrast to left-sided PVT, guidelines do not provide a recommended INR for prostheses in the tricuspid position.<sup>36, 39, 40</sup> For bileaflet prosthetic valves in the mitral position, a range of 2.5 - 3.5 is recommended, 2.0 - 3.0 in the aortic position for patients without additional risk factors for thromboembolism.<sup>39</sup> However, Shapira et al. recommended target INR levels of 3.5 - 4.0 for patients with tricuspid prostheses<sup>6</sup> and Zhang et al., in order to prevent future thrombotic complications of tricuspid PVT, considered target INR levels of 3.0 - 3.5.

### ***Recurrent tricuspid PVT***

A major disadvantage of thrombolytic therapy is the relatively high incidence of recurrent thrombosis during follow-up; however, data are limited about rethrombosis after thrombolytic therapy of tricuspid PVT. Recurrent rates after thrombolytic therapy vary from 11% to 31%.<sup>3, 53-58</sup> Overall, risk of recurrent thrombosis after thrombolytic therapy in left-sided prosthetic valves is higher than that in



the tricuspid position. A meta-analysis showed that while the incidence of recurrent thrombosis in left-sided prosthetic valves was 20%, it was 14% in the tricuspid position.<sup>14</sup> As was mentioned earlier, the etiology of recurrent thrombosis is the same as that of the first episode. Also, the coexistence of thrombus and pannus tissue on a prosthetic valve is another factor that can explain the recurrence of PVT after successful thrombolytic therapy in a subset of patients.<sup>53, 56</sup> However, some recurrences may be the result of an uncompleted resolution of the initial thrombotic process rather than the result of a new thrombosis. Thus, after successful thrombolytic therapy, it is very important to follow up patients with serial clinical and echocardiographic examinations.<sup>27</sup> The results of rethrombolysis after PVT recurrence are comparable to those obtained after the first thrombolytic therapy, which is concordant with our case 1.<sup>38, 41, 58-62</sup> Therefore, rethrombolysis is safe with a high successful rate and is recommended in patients with recurrent tricuspid PVT.

### ***Choose a prosthetic valve: mechanical or biological***

Tricuspid valve replacement is one of the most challenging operations of cardiac surgery. Most cardiac surgeons undertake tricuspid valve surgery infrequently and usually perform tricuspid valve repair. Incidence of tricuspid valve replacement is approximately 0.7% of all valve replacements.<sup>63</sup> Although many studies have been performed to determine the preference between mechanical or biological valves in the tricuspid position, they have not reached the same conclusion yet.<sup>63-69</sup> As was mentioned before, because the risk of thrombosis is high in the tricuspid position and thrombus formation is lower in biological valves,<sup>65, 66, 70</sup> we recommend the use of biological valves in the tricuspid position, which is similar to that in re-replacement valve surgery (the same as our case 3).

### ***Conclusion***

Thrombosis in tricuspid prosthetic valves is high and in some cases, patients are symptom-free or have a mild complaint. Thus, regular visits after valve replacement are reasonable and if there is suspicion of PVT, other modalities (first TTE) are recommended. Herein, we reported three cases of tricuspid PVT with different conditions. The main cause of PVT in our cases was subtherapeutic anticoagulation. The first case was a woman who suffered from recurrent PVT. In this case we successfully used for the first time Tenecteplase for second and third episodes. Given that this drug can be used in a single dose and has acceptable efficacy compared to the other conventional thrombolytic agents, we would recommend Tenecteplase as a good alternative for PVT

treatment. Also, this case shows us that thrombolytic therapy is a good option for recurrent tricuspid PVT, in contrast to left-sided PVT. The second case had fixed leaflets in open position, while the patient was symptomless. Thrombolytic therapy failed in this case; however, due to the patient's chronic infection, we could not replace her valve. At six months' follow-up, the motion of the leaflets was restricted and she was symptom-free. Thus, if thrombolytic therapy fails, surgery is not possible, the patient is symptom-free, and hemodynamic is stable, close observation with oral anticoagulant would be a reasonable course of action. The last case was a woman who had a large thrombus in the right atrium immediately after mitral and tricuspid valve replacement. We think that the cause of thrombus formation in this case was inadequate anticoagulation therapy. The patient underwent re-replacement surgery and a new biological valve was implanted in the tricuspid position.

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