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Obstructions of prosthetic heart valves: diagnosis and treatment considerations

Mehmet Nuri Karabulut[1], Rafet Günay[2], Mahmut Murat Demirtaş[3]

[1]Department of Cardiovascular Surgery, University of Health Sciences, Başakşehir Çam and Sakura City Hospital, İstanbul, Türkiye, [2]Department of Cardiovascular Surgery, University of Health Sciences, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Türkiye, [3]Retired Professor of Cardiovascular Surgery, İstanbul, Türkiye

ABSTRACT

Objectives: Since the first years of native heart valve replacement by - prosthetic valves; prosthesis thrombogenicity has kept its importance as a serious problem causing post-operative morbidities and mortality. This study aims to evaluate early postoperative morbidity and mortality of patients diagnosed with prosthetic valve thrombosis and treated surgically or non-surgically.

Methods: Thirty-one patients diagnosed with and treated for prosthetic valve thrombosis were evaluated retrospectively. The patients were followed up for 58 months.

Results: There were 24 females and 7 males. The mean patient age at the time of prosthetic valve thrombosis diagnosis was 40.7±11(range, 10-57) years. The mean duration between prosthetic valve replacement and the first signs of prosthetic valve thrombosis was 67. 67±66 (range, 1to 300) months. All patients presented with a functional capacity of NYHA Class III or IV. A total of 32 interventions; 27 surgical and 5 thrombolytic treatments due to elevated aortic prosthetic valve pressure gradient which did not improve with thrombolysis. Of 27 surgical interventions for thrombosed prosthetic valves, 21 involved mitral, 2 aortic, and 4 tricuspid positions. A total of 9 patients died during follow-up. The overall mortality rate was 29.03%. The mortality rate was 29.62 % after surgical interventions and 20% after thrombolytic treatment.

Conclusion: Currently prosthetic valve replacement is the basic palliation method in the management of patients with diseased native heart valves. In the majority of mechanical prosthetic valve obstructions, the main pathology is fibrous tissue proliferation-related to irregular warfarin usage, which in turn causes the development of acute symptoms secondary to acute valve thrombosis. The necessary treatment method for prosthetic valve obstructions should be either the use of thrombolytic agents or the replacement of the obstructed prosthetic valve with a new one.

Keywords: Obstructions, prosthetic heart valves, treatment

Since the first years of native heart valves' replacement by - prosthetic valves; prosthesis thrombogenicity has kept its importance as a serious problem causing post-operative morbidities and mortality. To eliminate the ball-cage valve and disc-cage valves' non-physiologic transprosthetic flow profiles; tilting-disc and bi-leaflet prosthetic valves have been developed [1]. However, bi-leaflet valves have

Corresponding author: Mehmet Nuri Karabulut, MD., Assist Prof., Phone: +90 212 909 60 00, E-mail: mnkarabulut@gmail.com

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surpassed other valve technologies and become the standard design with their anti-thrombogenic properties, flow dynamics close to physiological flow, easy implantability, and minimal contact with the subvalvular apparatus [2]. To reduce the thrombogenicity of the prosthetic materials to a minimum, the leaflets of the valves are manufactured with pyrolytic carbon. Besides the structural integrity of its own, pyrolytic carbon develops a biological adaptation to the living body by having a protein coating on its surface [3, 4, 5].

Prosthetic valve thrombosis has been described as occlusion of the prosthetic valve by a non-infective thrombotic material [6]. Prosthetic valve dysfunction because of valve size mismatch, infective vegetations, and restriction of the leaflet's moving parts by surgical suture materials are beyond this description. The risk of developing prosthetic valve thrombosis varies depending on the type of valve used, the position of the valve implanted, and the side of the heart. The risk of thrombosis is higher in mechanical valves compared to biological valves, in those implanted in the mitral position compared to those in the aortic position, and in prosthetic valves on the right side of the heart compared to those on the left side. This result develops with the interaction of the patient-related (coagulability, cardiac function, and cardiac morphology) and prosthesis-related multiple factors [7].

With this study, we aimed to report the findings and results of our patients diagnosed and treated with prosthetic valve thrombosis by comprehensively comparing them with the literature.

METHODS

This retrospective clinical study was performed at Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital. All patients admitted to the cardiac surgery center with Prosthetic Valve Obstruction were included in this study.

Thirty-one patients diagnosed with and treated for prosthetic valve thrombosis were retrospectively evaluated in a single center (24 females, 7 males). The patients were followed up for 58 months. When a prosthetic valve thrombosis diagnosis has been made.

An echocardiographic exam was also performed for every patient included in this study. Echocardiographic parameters including prosthetic valve gradients and functional status examined (Fig. 1). Pulmonary arterial and Pulmonary Capillary Wedge Pressures with the Central Venous Pressure recorded after Swan-Ganz Catheter insertion.

All patients presented with a functional capacity of New York Heart Association (NYHA) Class III or IV. Thrombolytic agents used to treat prosthetic valve thrombosis were Streptokinase (Kabikinase® Pharma-

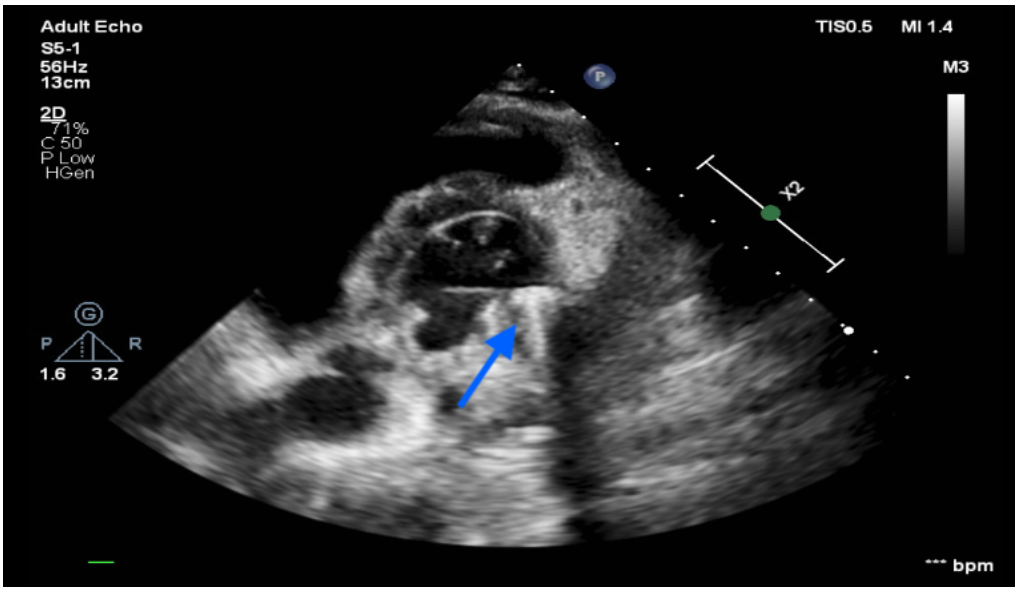

Fig. 1. Preoperative echocardiogram image demonstrating prosthetic valve thrombosis (blue arrow).

cia & Upjohn Sweden) and recombinant plasminogen activator (Actilyse®, Boehringer Ingelheim GmbH Germany).

The total dose of Streptokinase was 1. 500000 IU Per case with 250. 000 IU infused over 20 minutes and the rest of the total dose infused over 90 min. A total dose of recombinant plasminogen activator was 100 mg per case, with 10 mg infused in 2 minutes, an additional 40 mg infused in the first 1 hour, and the rest of the total dose infused in the first 2 hours.

All patients treated with thrombolytic agents received 25. 000 IU of unfractionated heparin in the first 24 hours of the treatment titrated by thromboplastin times kept at 2 times above the normal range (Table 1).

Statistical Analysis

Descriptive statistics were reported, including counts (n), percentages (%), mean±standard deviations, and median (minimum and maximum) values.

RESULTS

A total of 32 interventions; 27 surgical and 5 thrombolytic therapy, were performed on 31 patients. The mean patient age at the time of prosthetic valve thrombosis diagnosis was 40.7±11 (range, 10-57) years. The mean duration between prosthetic valve replacement and the first signs of prosthetic valve thrombosis was 67.67±66 (range, 1-300) months.

Patients had a mean left atrial diameter of 6.13±1.4 cm (range, 4. 3 to 10) by echocardiographic evaluation. Hemodynamic parameters of patients by Swan-Ganz catheter measurement were as follows; pulmonary capillary wedge pressure (PCWP): 26±8.3 mmHg, systolic pulmonary artery pressure (PAP systolic): 54±16 mmHg (range, 31 to 80), and diastolic pulmonary artery pressure (PAP diastolic): 27±10 mmHg (range, 13 to 48).

Of 31 patients, 26 had undergone single valve replacement, and 5 had undergone double valve replacement procedures during their initial heart valve operations. The transvalvular diastolic pressure gradient in patients with prosthetic mitral valve dysfunction was 19±9 mmHg (range, 8 to 45). In two patients with aortic prosthetic valve dysfunction transvalvular systolic pressure gradient was 77 and 88 mmHg. In four patients with prosthetic tricuspid valve dysfunction mean transvalvular diastolic pressure gradient was 13. 8 mmHg (range, 9 to 16) (Table 1).

One patient underwent surgery following thrombolytic treatment due to elevated aortic prosthetic valve pressure gradient which did not improve with thrombolysis. Of 27 surgical interventions for thrombosed prosthetic valves, 21 involved mitral, 2 aortic, and 4 tricuspid positions.

Mitral valve interventions included replacement of the dysfunctional prosthetic valve and thrombectomy and debridement of the malfunctional prosthesis in 17 patients. One patient with a dysfunctional prosthetic mitral valve died during the redo-sternotomy opening.

Primary aortic valve replacement was performed due to de novo native aortic valve disease during surgical management of prosthetic mitral valve dysfunction in 3 patients with prosthetic mitral valve thrombosis. In the same group, tricuspid valvular interventions were performed for concomitant tricuspid pathologies in five patients. Four of the tricuspid interventions were replacement of the native tricuspid valve and one was De Vega annuloplasty.

Of five patients diagnosed with prosthetic tricuspid valve dysfunction, four underwent surgical and one underwent thrombolytic treatment. Prosthetic valve re-replacement was performed on all patients who were surgically treated for tricuspid prosthetic valve dysfunction.

According to surgical explorative findings and pathologic examinations of dysfunctional prosthetic valves explanted from 27 patients who were surgically treated; the findings were pannus and thrombus in 16 cases, fresh thrombus in 7 cases, and biologic valve degeneration in 4 cases (Fig. 2).

Of five patients treated by a thrombolytic agent, thrombosed prosthetic valves were in mitral position in 3 patients, aortic position in one patient, and tricuspid position in one patient.

Seven patients died during the first 24 h. after interventions. 6 following surgical treatment and one following thrombolytic treatment. Surgically treated seven patients died in the early postoperative period.

Causes of mortality were peri-operative low cardiac output syndrome and; failure to wean from cardiopulmonary bypass for six patients and sepsis and

Table 1. **Patients demographic data, findings, and treatment options**

Number	Age	Gender	Time	Prosthetic valve type	Clinical Picture	NYHA class	Anticoagulation regimen	Rhythm	Pre-Op Echocardiography
1	46	F	1 month	MVR (29) – Carbomedics mechanical prosthetic valve	Pulmonary edema	IV	Irregular/ Insufficient	AF	Restricted leaflet movements in Prosthetic valve + Tricuspid stenosis
2	42	F	7 months	MVR (27) – Carbomedics mechanical prosthetic valve	Respiratory distress	III	Irregular/ Insufficient	NSR	Left atrial thrombus
3	43	M	24 months	MVR (31) – Sorin mechanical prosthetic valve	Pulmonary edema	IV	Irregular/ Insufficient	AF	Complete prosthetic valve Thrombosis+ 2 cm in diameter echo-dens thrombus above the valve
4	46	F	23 months	MVR (27) – Medtronic mechanical prosthetic valve+ Left atrial thrombectomy	Pulmonary edema	IV	Irregular/ Insufficient	AF	Restricted leaflet movements+Left atrial thrombus
5	30	M	30 months	MVR (27) - St.Jude mechanical prosthetic valve + Aortic valvotomy	Congestive heart failure	III	Irregular/ Insufficient	AF	Restricted leaflet movements+ Aortic regurgitation
6	39	F	9 months	MVR (29) – Carbomedics mechanical prosthetic valve	Pulmonary edema	IV	Irregular/ Insufficient	AF	Restricted leaflet movements
7	48	M	28 months	MVR (29) – Carbomedics mechanical prosthetic valve	Pulmonary edema	IV	Irregular/ Insufficient	AF	Restricted leaflet movements
8	52	F	20 months	MVR (29) – Sorin mechanical prosthetic valve + CABGx1	Pulmonary edema	IV	Irregular/ Insufficient	AF	Disc movements severely restricted
9	34	F	26 months	MVR (27) – Carbomedics mechanical prosthetic valve	Respiratory distress	III	Irregular/ Insufficient	AF	Restricted leaflet movements
10	38	F	8 months	MVR (29) – Carbomedics mechanical prosthetic valve	Respiratory distress	III	Regular/ Sufficient	AF	leaflet movements limited on anterior leaflet in the prosthetic valve, posterior leaflet is normal
11	48	F	42 months	MVR (29)+TVR (31) – Sorin mechanical prosthetic valves	Congestive heart failure	IV	Irregular/ Insufficient	AF	Disc movements severely restricted on the Tricuspid prosthetic valve
12	52	F	48 months	MVR (29) – Sorin mechanical prosthetic valve + TVR (31) Hancock Bioprosthetic valve	Congestive heart failure	III	Irregular/ Insufficient	AF	Restricted leaflet movements in Bioprosthetic Tricuspid valve
13	39	F	102 months	MVR (29) – Hall-Kaster mechanical prosthetic valve	Respiratory distress	III	Regular/ Sufficient	AF	Disc movements severely restricted
14	38	M	180 months	Starr-Edwards (Cage-Ball) mechanical prosthetic valve	Congestive heart failure	III	Regular/ Sufficient	AF	Stenotic prosthetic valve
15	53	F	300 months	Starr-Edwards (Cage-Ball) mechanical prosthetic valve	Congestive heart failure	III	Irregular/ Insufficient	AF	Stenotic prosthetic valve
16	48	F	60 months	MVR (31) – Omniscience mechanical prosthetic valve	Congestive heart failure	III	Irregular/ Insufficient	AF	Disc movements are severely restricted

Table 1. ***Continued.***

Number	Age	Gender	Time	Prosthetic valve Type	Clinical picture	NYHA class	Anticoagulation regiment	Rythm	Pre-Op Echocardiography
17	32	F	12 months	MVR (27) – Carbomedics mechanical prosthetic valve	Pulmonary edema	IV	Irregular/ Insufficient	AF	Restricted leaflet movements
18	34	F	96 months	AVR (19) – Medtronic mechanical prosthetic valve	Chest pain at rest + ST changes on ECG	III	Irregular/ Insufficient	NSR	75 mmHg max gradient on mechanical prosthetic valve
19	34	F	96 months	AVR (19) – Medtronic mechanical prosthetic valve	Chest pain on exertion	II / III	Regular/ Sufficient	NSR	65 mmHg max gradient on mechanical prosthetic valve
20	47	M	42 months	MVR (29) – Sorin mechanical prosthetic valve + CABGx1	Shortness of breath	III	Irregular	NSR	thrombus image on leaflets
21	53	M	42 months	MVR (31) Carbomedics mechanical prosthetic valve	Shortness of breath	III	None	AF	thrombus image on leaflets
22	25	F	42 months	MVR (31) – St.Jude mechanical prosthetic valve	Pulmonary edema	IV	Irregular/ Insufficient	AF	Severely restricted leaflet movements
23	57	F	140 months	MVR (31) – Ionescu-Shiley mechanical prosthetic valve	Congestive heart failure	III	None	AF	Disc movements severely restricted in prosthetic mitral valve + Aortic Stenosis
24	46	F	125 months	MVR (29) + TVR (29) – Björk-Shiley mechanical prosthetic valve + Biocor Bioprosthetic valve	Congestive heart failure	III / IV	Irregular/ Insufficient	AF	Restricted leaflet movements in prosthetic Tricuspid valve
25	42	F	45 months	MVR (27) + TVR (27) – Carbomedics mechanical prosthetic valve + Biocor Bioprosthetic valve	Congestive heart failure	III / IV	Irregular/ Insufficient	AF	Restricted leaflet movements in prosthetic Tricuspid valve
26	54	F	54 months	MVR (31) – Carbomedics mechanical prosthetic valve	Shortness of breath	III	Regular/ Sufficient	AF	Paravalvular leakage +restricted leaflet movements
27	33	F	108 months	MVR (29) – Björk-Shiley mechanical prosthetic valve	Shortness of breath	III	Irregular/ Insufficient	AF	Restricted leaflet movements
28	38	F	204 months	AVR (27) – Carbomedics mechanical prosthetic valve	Chest pain + ST changes on ECG	III / IV	Irregular/ Insufficient	AF	Restricted leaflet movements
29	29	F	62 months	MVR (29) – Carbomedics mechanical prosthetic valve	Pulmonary edema	IV	Irregular/ Insufficient	AF	Restricted leaflet movements
30	21	F	60 months	MVR (31) + TVR (33) – Biocor Bioprosthetic valves	Congestive heart failure	III / IV	Irregular/ Insufficient	AF	Stenotic prosthetic tricuspid valve
31	10	M	48 months	MVR (25) –Biocor Bioprosthetic valve	Congestive heart failure	III / IV	None	NSR	Stenotic prosthetic mitral valve + Aortic regurgitation
32	31	F	110 months	MVR (27) – Biocor Bioprosthetic valve	Congestive heart failure	III / IV	None	AF	Stenotic prosthetic mitral valve

AF=Atrial fibrillation, CABG=Coronary artery bypass graft, ECG=Electrocardiography, AVR=Aortic valve replacement, MVR=Mitral valve replacement, TVR=Tricuspid valve replacement, NSR=Normal sinus rhythm

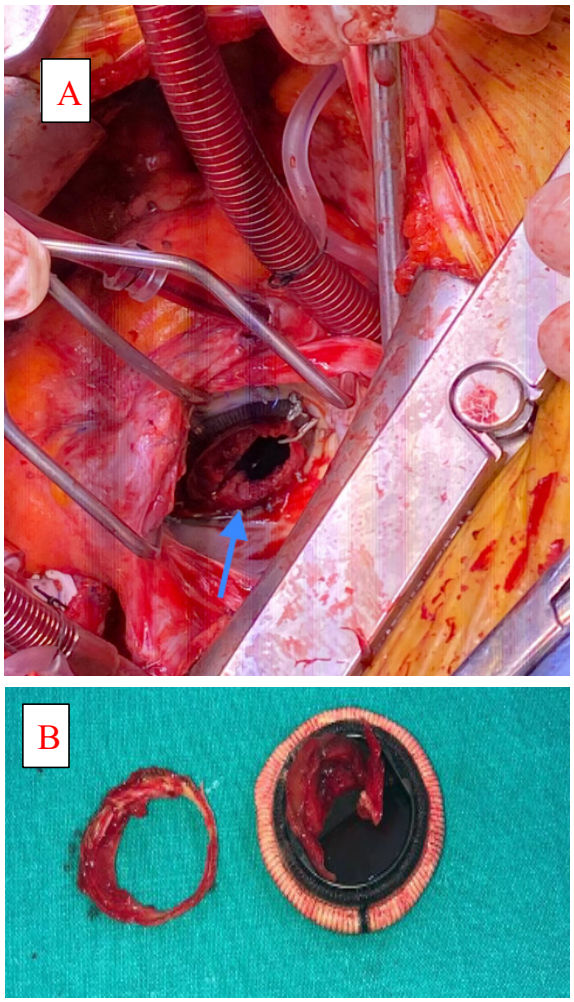

Fig. 2. (A) Intraoperative view of mechanical mitral valve thrombosis(arrow). (B) Mechanical mitral valve thrombosis and pannus.

multiorgan failure for two patients. A total of nine patients died during interventions or within the early postoperative period. Overall mortality was 29.0%, 29. 6% for the surgically treated group and 20. 0% for patients treated by thrombolytic therapy (Table 2).

DISCUSSION

The most frequent indication for prosthetic valve reoperations is prosthetic valve obstruction. Many researchers have reported that prosthetic valve obstruction develops as a consequence of many complex pathological mechanisms. The main component of these mechanisms is fibrous tissue proliferation associated with prosthesis thrombosis which disrupts normal prosthetic valve function [7, 8].

Fibrous tissue proliferation is responsible for prosthetic valve thrombosis in 80% of cases reported in the medical literature [9, 10]. The leading cause of tissue proliferation is still unknown. However, it has been argued that biocompatibility of the prosthetic valve material, endothelial damage which develops during surgical intervention, post-surgical low cardiac output, trans-prosthetic pressure gradients (especially at mitral position), pregnancy after valve implantation, prosthetic valve endocarditis and insufficient anticoagulant usage; all individually or interacting with each other trigger fibroblast proliferation [11-13].

Pannus formation was diagnosed in 59% of our surgically treated patients, including bioprosthetic valve obstructions. Thrombolytic treatment or simple debridement of the pannus tissue is still far from solving the underlying pathology. We treated aortic prosthetic valve obstruction in a patient by infusing a thrombolytic agent which failed to improve elevated trans-prosthetic valvular pressure gradients. The patient underwent, surgical intervention and debridement of the pannus formation. But surgical debridement would otherwise be unsuccessful or even potentially could cause harm because of the technical difficulty of removing pannus from both sides of the thrombosed prosthetic valve. Fresh thrombus (- primary prosthetic valve thrombosis) is the pathological diagnosis of 20% of surgically treated prosthetic valve obstruction cases in the literature. It was 26% in our series. Thrombosis mainly develops because of prosthetic valve thrombogenicity and ineffective anticoagulant usage [14, 15]. Thrombolytic agent usage or surgical thrombectomy are the available treatment options depending on the clinical condition [16].

In our series debridement and thrombectomy were applied in 3 of our surgically treated patients and re-replacement of the thrombosed prosthetic valve was the treatment of choice in of the cases. Prosthetic valve obstruction diagnosis depends on findings in physical examination, and fluoroscopic, echocardiographic, and hemodynamic studies. The nature of the prosthetic valve obstruction can be further assessed by transesophageal echocardiography in detail. Transthoracic echocardiographic evaluation was the main diagnostic tool in our study.

Preserved leaflet or disc movement and echocardiographic images of thrombus are the echocardio-

Table 2. Type of intervention and results

Number	Age	Gender	Per-Op findings	Type of intervention	Results
1	46	F	Fresh thrombus in prosthetic valve orifice	MVR (31) + TVR (31) – Biocor Bioprosthetic valves	Low Cardiac out put+Per operative Exitus
2	42	F	Fresh thrombi in prosthetic valve orifice and left atrium	MVR (29) – St.Jude mechanical prosthetic valve + De Vega Ann.	Exitus at post-op day 17th.
3	43	M	Fresh thrombi in prosthetic valve orifice and left atrium	MVR (27) – Medtronic mechanical prosthetic valve + De Vega Ann.	Low Cardiac out put+Per operative Exitus
4	46	F	Pannus formation + fresh thrombus in the disc slot	MVR (27) – Medtronic mechanical prosthetic valve	Low Cardiac out put+Per operative Exitus
5	30	M	Pannus formation + thrombus in leaflet hinges	AVR (23) + MVR (29) – St.Jude mechanical prosthetic valves	Low Cardiac out put+Per operative Exitus
6	39	F	Fresh thrombi in valve leaflet hinges and left atrium	MVR (27) – St.Jude mechanical prosthetic valve	Complete recovery
7	48	M	Pannus formation + paravalvular leakage	MVR (27) – Carbomedics mechanical prosthetic valve	Exitus at post-op 2nd day (Cerebral edema)
8	52	F	Left atrial thrombus+ Complete obstruction above and below the valve (Thrombus + pannus formation)		Exitus during sternotomy
9	34	F	Pannus formation + fresh thrombus on leaflet hinges	MVR (31) – Hancock Bioprosthetic valve	Complete recovery
10	38	F	fresh thrombus on the anterior leaflet	Thrombectomy + Debridman	Complete recovery
11	48	F	Fresh thrombus in tricuspid prosthetic valve	TVR (31) – Hancock Bioprosthetic valve	Complete recovery
12	52	F	Thrombolytic treatment (r-tPA)		Complete recovery
13	39	F	Pannus formation + fresh thrombus in the disc slot	MVR (27) – Carbomedics mechanical prosthetic valve	Complete recovery
14	38	M	Pannus formation + thrombi in ball socket and cage	MVR (25) – St.Jude mechanical prosthetic valve	Complete recovery
15	53	F	Pannus formation + thrombi in ball socket and cage	MVR (31) – Carbomedics mechanical prosthetic valve + De Vega Ann.	Complete recovery
16	48	F	Pannus formation + fresh thrombus in the disc slot	MVR (29) – St.Jude mechanical prosthetic valve	Complete recovery

Table 2. ***Continued.***

Number	Age	Gender	Per-Op findings	Type of intervention	Results
17	32	F	Thrombolytic treatment (Streptokinase)		Exitus at 4th hour, (cerebral embolism)
18	34	F	Thrombolytic treatment (r-tPA)		Regressed symptoms
19	34	F	Pannus formation + fresh thrombus in the disc slot	Debridement + Aortoplasty	Complete recovery
20	47	M	Thrombolytic treatment (Streptokinase)		Complete recovery
21	53	M	Thrombolytic treatment (Streptokinase)		Complete recovery
22	25	F	fresh thrombus on leaflet hinges	MVR (29) – St.Jude mechanical prosthetic valve	Exitus at post-op day 24th (Sepsis)
23	57	F	Bioprosthetic valve degeneration + fresh thrombus on the valve	AVR (21) + MVR (29) – St.Jude mechanical prosthetic valves	Complete recovery
24	46	F	Bioprosthetic valve degeneration and fresh thrombus on tricuspid bioprosthetic valve	TVR (27) – St.Jude mechanical prosthetic valve	Complete recovery
25	42	F	Bioprosthetic valve degeneration + fresh thrombus on the valve	TVR (29) – Biocor Bioprosthetic valve	Complete recovery
26	54	F	fresh thrombus on leaflet hinges+ paravalvular separation from valve annulus+ left atrial thrombus	Paravalvular leakage repair + thrombectomy	Complete recovery
27	33	F	Pannus formation + thrombus in disc slot	MVR (29) -St.Jude mechanical prosthetic valve + De Vega Ann.	Complete recovery
28	38	F	Fresh thrombi on the leaflet hinges	AVR (25) – St.Jude mechanical prosthetic valve	Complete recovery
29	29	F	Fresh thrombi on the leaflet hinges	MVR (27) – Carbomedicsmechanical prosthetic valve	Complete recovery
30	21	F	Bioprosthetic valve degeneration + fresh thrombus on the valve	TVR (31) – Medtronic mechanical prosthetic valve	Complete recovery
31	10	M	Bioprosthetic valve degeneration and calcification + fresh thrombus on the valve	AVR (20) + MVR (25) – Carbomedics mechanical prosthetic valves	Complete recovery
32	31	F	Bioprosthetic valve degeneration + fresh thrombus on the valve	MVR (27) – Medtronicmechanical prosthetic valve+De Vega Ann.	Complete recovery

AVR=Aortic valve replacement, MVR=Mitral valve replacement, TVR=Tricuspid valve replacement

graphic findings in primary prosthetic valve thrombosis besides prosthetic valve hemodynamic parameters such as trans-valvular pressure gradients. Observation of rapid clinical deterioration combined with echocardiographic findings renders primary prosthetic valve thrombosis the most probable diagnosis. Thrombolytic treatment would be the first option in the treatment of this fatal clinical condition in selected cases.

Thrombus images in echocardiographic studies were observed in patients who received thrombolytic treatment in our series (Fig. 1). One of those five patients died in the 4th hour of thrombolytic treatment because of cerebral thrombotic embolization. Risk of the cerebral embolization during thrombolytic treatment of left-sided prosthetic valve thrombosis was reported by several authors [15-20].

In the case of fibrous tissue proliferative invasion of the prosthetic valve orifices preventing leaflet or disc movement; fresh thrombosis is the final phase of the pathological process causing rapid clinical deterioration. In all surgically treated patients in our series, restriction of the leaflet or disc movement was the main echocardiographic finding and was confirmed by pannus determination in prosthetic valve orifices.

Treatment of prosthetic valve obstructions is a serious clinical entity with very high mortality rates approaching 44% [21-27].

The in-hospital mortality rate was 29.9% in our study. This result reflects the seriousness of the clinical symptoms of patients at hospital admission. Six patients admitted with pulmonary edema underwent surgical intervention and five of them died intraoperatively. But early intervention is very important and the overall in-hospital mortality rate shows its importance in our series. In all but four patients, anticoagulation titration was suboptimal. This is a result of inefficient cooperation between patients and healthcare providers.

Limitations

The most important limiting point of the study is the small number of patients. Multicenter studies are needed.

CONCLUSION

We present our clinical experience with a review of the available literature. Currently, prosthetic valve replacement is the basic palliation method in the management of patients with diseased native heart valves. Prosthetic valves carry an annual thrombosis risk of 0.03 to 4.3% under optimal conditions. Patient compliance with the anticoagulation regimen and its follow-up is our main problem. Echocardiographic evaluation and early detection of hemodynamic abnormalities, and fibrous tissue proliferation (pannus) during routine follow-ups are the key factors in the prevention of secondary prosthetic valve thrombosis. Transesophageal echocardiography is very important in differential diagnosis. In most cases, fibrous tissue proliferation is the main pathological process in the development of mechanical prosthetic heart valve obstructions. In this case replacement of the obstructed prosthetic valve with a new one is the only option in the treatment.

Ethical Approval

Our article titled "Obstructions of prosthetic heart valves: Diagnosis and treatment considerations" is an article derived from the thesis study. Ethics committee approval was not required at the time the article was edited.

Authors' Contribution

Study Conception: MMD; Study Design: MNK; Supervision: MMD; Funding: MNK; Materials: MMD; Data Collection and/or Processing: RG; Statistical Analysis and/or Data Interpretation: MNK; Literature Review: MNK; Manuscript Preparation: RG and Critical Review: MNK.

Conflict of interest

The authors disclosed no conflict of interest during the preparation or publication of this manuscript.

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