The St. Jude Valve

Thrombolysis as the First Line of Therapy for Cardiac Valve Thrombosis

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**Background.** Thrombolytic therapy is a promising alternative to valve replacement in the management of prosthetic valve thrombosis. We sought to determine the short- and long-term results of treating thrombosed St. Jude heart valves with thrombolytic therapy as the primary treatment modality.

**Methods and Results.** Between March 1978 and December 1991, 988 patients underwent implantation of St. Jude prosthetic valves at our medical center, and all patients with thrombosed valves were identified prospectively. During this period, 17 patients (13 women; mean age, 66.8±19.0 years) developed prosthetic valve thrombosis (11 aortic, six mitral). In six patients, Coumadin was stopped in preparation for elective surgery. The clinical presentation was congestive heart failure in 13, syncope and fatigue in two, and a cerebrovascular accident in one; one patient was asymptomatic. The average duration of symptoms was 11.7±12.0 days (range, 1–45 days). Anticoagulation was subtherapeutic in all but one patient at the time of presentation. Cinefluoroscopy was the primary method used for diagnosis and was also used to follow the response to therapy. Twelve patients were treated medically (10 with thrombolytic therapy and two with heparin), three were treated surgically, and two were diagnosed at autopsy. Of the 12 medically treated patients, 10 had marked improvement in leaflet movement and symptoms within 12 hours. Thus, 10 of 12 patients (83%) had a satisfactory response to medical therapy alone. No medically treated patient died or had a major complication resulting in permanent damage. However, four of the 12 medically treated patients had minor complications, including a transient episode of facial weakness in one patient, hematomas in two, and epistaxis in one. Late rethrombosis recurred in two patients in the medically treated group and was successfully retreated with thrombolytic therapy. At 3 months, all patients were alive and well.

**Conclusions.** Thrombolytic therapy can be used as the first line of therapy for thrombosed St. Jude valves with a low risk of permanent side effects and excellent chances of success. In most cases, surgery can be reserved for patients who do not respond to thrombolytic therapy. (*Circulation* 1993;87:30–37)

**Keyword**s • thrombolysis • prosthetic valve • St. Jude heart valve • cinefluoroscopy • thrombosis

Valvular thrombosis, systemic emboli, and hemorrhage from anticoagulation are major complications of heart valve replacement with mechanical prostheses. The reported incidence of valvular thrombosis with currently available mechanical devices varies from 0.03% to 4.3% per year.1—10 Although the traditional treatment has been emergent reoperation, surgery is associated with the risk of operative death, significant morbidity, and high costs. Reported mortality rates for valve replacement in this setting vary from 4.5% to 20%.11—15 Surgical valve debridement is occasionally sufficient and may be associated with a lower operative mortality rate,16 although the rate of rethrombosis may be significantly higher.17

In 1971, Luluaga et al18 first reported successful treatment of a thrombosed Starr-Edwards prosthesis in the tricuspid position with thrombolytic therapy, and Baille et al19 subsequently reported the use of thrombolytic therapy for relief of left-sided prosthetic valve obstruction (a Cutter prosthetic valve in the aortic position) in 1974. Since these case reports, another 69 patients receiving thrombolytic therapy for cardiac valve thrombosis have been reported with satisfactory results.20—24 Thus, thrombolytic therapy is emerging as an alternative to surgery. However, the patients in these reported series have had a variety of different prosthetic valves, which makes it difficult to draw conclusions about the preferred treatment for thrombosis of a specific valve. Since our first experience with thrombolytic treatment of a thrombosed St. Jude aortic valve,24 we have used this approach as our primary mode of therapy and have collected data on our results prospectively. The purpose of this report is to analyze our experience with medical and surgical management of thrombosed St. Jude valves during a 13-year period.

**Methods**

**Patients**

From March 1978 to December 1991, 988 patients underwent heart valve replacement with a low-profile
FIGURE 1. Schematic representation of the St. Jude bileaflet valve, illustrating the opening angle (O), closing angle (C), the excursion of each leaflet (E₁, E₂), and the total leaflet excursion (E_total).

bileaflet St. Jude mechanical valve. Mitral valve replacement was performed in 401, aortic valve replacement in 458, and 129 had a double mitral and aortic valve replacement (both St. Jude valves). All such patients, regardless of age, risk status, or associated procedures, were included in this study. During this period, 17 patients (13 women; mean age, 66.8±19.0 years) developed prosthetic valve thrombosis (PVT) (11 aortic, six mitral). Of these, three patients initially underwent surgery elsewhere and were referred to our institution for treatment. Currently, we recommend that our mechanical valve patients be maintained on warfarin adjusted to maintain the prothrombin time at 1.5–2.0 times control values. Patients are also treated with dipyridamole (75 mg t.i.d.), if tolerated.

Diagnosis of PVT

Cinefluoroscopy is the preferred method for diagnosis and follow-up of St. Jude valve thrombosis at our institution. Since the St. Jude valve is only slightly radiopaque, a side or pivot view with the disks parallel to the x-ray beams 24–26 is required for optimal leaflet visualization and measurement of leaflet angles. Because of the surgeon’s individual variability in valve orientation, careful patient positioning is required for leaflet visualization, and optimal visualization frequently necessitates caudocranial angulation. Leaflet mobility and opening and closing angles (in degrees) were determined from frame-by-frame analysis of at least three cardiac cycles. The range of excursion of the leaflets (closing angle minus opening angle) was also calculated (Figure 1). Prior measurements by our group have shown the normal ranges for the measured opening angle to be 11±1° and the closing angle to be 120±2° for valve sizes 19–25 mm. 24 The closing angle for valve sizes 27–33 mm, according to the manufacturer’s specifications, is 130°. The cinefluoroscopy studies were initially performed before treatment was begun and at a minimum of every 24 hours afterward for assessment of the success of thrombolytic therapy.

Thrombolysis

Since June 1984, we have used thrombolytic therapy in every patient with St. Jude valve thrombosis, provided that the patient had no contraindications to thrombolytic therapy. The risks and benefits of both surgical valve replacement and thrombolytic therapy were discussed with each patient. All patients were required to sign an informed consent form before thrombolytic therapy was begun. The first two patients were treated with streptokinase (250,000 units bolus i.v. injection over 30 minutes followed by a maintenance infusion of 100,000 units/hr for 72 hours). However, the second patient had received streptokinase previously during a myocardial infarction and had a high antibody titer. This patient and subsequent patients were therefore treated with urokinase. Urokinase was given according to the same protocol recommended for treatment of pulmonary embolism: a bolus injection of 4,400 units/kg given over 10–15 minutes followed by a maintenance dose of 4,400 units·kg⁻¹·hr⁻¹. Two patients received only heparin as therapy. One of these patients presented with a cerebrovascular accident of embolic origin, and fibrinolytic agents were thought to be contraindicated.

Thrombolytic therapy was terminated if normalization of valve function was documented on cinefluoroscopy, if hemorrhagic complications prevented continuation of therapy, or after 24 hours if no improvement had occurred. After discontinuation of the thrombolytic agent, patients were heparinized when the partial thromboplastin time dropped to less than two times control, generally without a heparin bolus, and discharged on warfarin titrated to a prothrombin time of 1.5–2.0 times control and dipyridamole.

Follow-up

Follow-up was obtained annually by mailed questionnaire or by direct patient contact by a trained research nurse and was available in all patients treated medically for PVT.

Statistical Analysis

Data are expressed as the mean±SD. The statistical significance of the differences was analyzed by use of Student’s t test.

Results

Clinical Presentation

The clinical characteristics of the patients are presented in Table 1. Seventeen patients, 13 women and four men, with a mean age of 66.8±19.0 years (range, 12–91 years), were diagnosed as having PVT. The presenting symptom was pulmonary edema in four patients, heart failure or shortness of breath in nine patients, syncope in one, loss of balance in one, and cerebrovascular accident in one; one patient was asymptomatic and was diagnosed during routine checkup. Five of the patients with heart failure also had severe chest pain. Of the two patients diagnosed at autopsy, patient 16 had a prolonged hospitalization after surgery, and patient 17 died in the emergency room after presenting with pulmonary edema. There was no correlation between the severity of symptoms and the degree of limitation of leaflet motion. Neither did the type of symptom appear to correlate with the response to therapy.

The level of anticoagulation on presentation was therapeutic (prothrombin time at least 1.5–2.0 times control values) in only one of our patients. The patient
with a therapeutic prothrombin time was also the only active smoker in the series. In all other patients, one or more prothrombin times were documented to be less than the target level within the preceding weeks. In 35% of the patients (six of 17), Coumadin was stopped <3 weeks earlier as preparation for elective surgery (pacemaker insertion, repair of femoral aneurysm, cholecystectomy, facial trauma, epidural injection, prostatectomy). Two patients were not being treated with Coumadin before the thrombolytic event because of previous life-threatening bleeding.

The mean time elapsed from valve replacement to the thrombotic event was 53.9±33.0 months (range, 4–108 months). The duration of symptoms ranged from 1 to 45 days, similar to the diverse mode of presentation of prosthetic valve thrombosis noted in other studies.9,13

**Outcome of Thrombolytic Treatment**

Of 12 initial events of valve thrombosis treated by thrombolytic agents in our hospital, both leaflets of the prosthetic valve were limited in motion in 10 patients (83%), and one leaflet was entirely immobilized in eight patients (66%). Treatment was defined as successful if the valve leaflets resumed more than 90% of the normal range of motion (Figure 2). In nine patients (75%), treatment was successful, and in one (8%) there was partial success. This patient's symptoms improved significantly, but cinefluoroscopy demonstrated residual limitation in leaflet motion. Two patients (17%) failed to demonstrate any change in leaflet motion; however, their clinical state was stabilized with conservative therapy. The opening angles before and after therapy were 60.8±19.3° and 28.8±19.1° (p<0.001), and the closing angles were 116.0±9.0° and 123.1±3.5° (p<0.01) before and after therapy, respectively (Table 2). The range of leaflet motion increased from 55.2±18.4° to 95.9±16.7° degrees (p<0.001) after treatment (Figure 3). All patients were released from the hospital and were followed by their cardiologists. None of these patients required subsequent surgery. At 3-month follow-up, all were alive and well.

**Patients With Recurrent Events**

Recurrent events are outlined in Table 3. During 17.2±17.6 months of follow-up (range, 1–65 months), two patients had cinefluoroscopic evidence of recurrent thrombosis. Patient 5 was initially successfully treated with urokinase, but 8 months later a second episode of thrombosis was diagnosed and again successfully treated with urokinase. The patient returned twice more with recurrent thrombus and was successfully treated 3 months and 7 months later with urokinase. This patient had metastatic breast cancer and was approaching senility, and her physicians had great difficulty keeping her Coumadin in a therapeutic range. At each admission, her prothrombin time was essentially normal (nontherapeutic). Twelve months after her last event, the patient died of heart failure after a prolonged hospitalization for significant gastrointestinal bleeding, during which her Coumadin therapy had been discontinued for at least three weeks before her death. Patient 10 had recurrent PVT 6

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**Table 1. Characteristics of Patients at Presentation**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Thrombosed valve/size (mm)</th>
<th>Time from surgery to event (months)</th>
<th>Duration of symptoms (days)</th>
<th>Clinical presentation</th>
<th>Adequate anticoagulation at presentation</th>
<th>Method of diagnosis</th>
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<td>1</td>
<td>79</td>
<td>F</td>
<td>Ao/21</td>
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<td>Syncope</td>
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<td>Cine</td>
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<td>2</td>
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<td>M</td>
<td>Mi/27*</td>
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<td>1</td>
<td>Fatigue</td>
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<td>Cine</td>
</tr>
<tr>
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<td>Ao/21</td>
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</tr>
<tr>
<td>4</td>
<td>66</td>
<td>F</td>
<td>Mi/31*</td>
<td>30</td>
<td>12</td>
<td>CHF/CP</td>
<td>No†</td>
<td>Cine</td>
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<td>77</td>
<td>F</td>
<td>Ao/23</td>
<td>38</td>
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<td>Cine</td>
</tr>
<tr>
<td>6</td>
<td>58</td>
<td>F</td>
<td>Ao/19</td>
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<tr>
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<td>17</td>
<td>64</td>
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<td>26</td>
<td>...</td>
<td>CHF</td>
<td>No†‡</td>
<td>Autopsy</td>
</tr>
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</table>

Mean: 66.8 ± 19.0  | SD: 53.9 ± 11.7  | Duration: 12.0

Ao, aortic; Mi, mitral; CVA, cerebrovascular accident; CHF, congestive heart failure; CP, chest pain; Cine, cinefluoroscopy.

*Double valve replacement.
†Coumadin withheld for surgical intervention.
‡Treated only with aspirin+diprydamole.
months after her first episode, which was again successfully treated with urokinase. It is interesting that this patient had developed thrombosis of a Bjork-Shiley valve before implantation of her St. Jude valve. The patient is currently stable, with no evidence of PVT. Patient 3 failed thrombolytic therapy and was managed conservatively because of his high risk for reoperation. He died of progressive congestive heart failure.

**Table 2. Effect of Thrombolysis on Valve Function**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Thrombolytic agent</th>
<th>Opening angle</th>
<th>Closing angle</th>
<th>Leaflet motion</th>
<th>Outcome of therapy</th>
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<td></td>
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<td>Post</td>
<td>Pre</td>
<td>Post</td>
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<td>Streptokinase</td>
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<td>Urokinase</td>
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<td>Urokinase</td>
<td>52</td>
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</table>

Mean: 60.8 ± 28.8, 116.0 ± 123.1, 55.2 ± 95.9
SD: 19.3 ± 19.1, 9.0 ± 3.6, 18.4 ± 16.7

*p<0.001, p<0.02, p<0.0001*

Pre, before treatment; post, after treatment.
failure 24 months later at the age of 84. This patient also did not take his Coumadin regularly, and it is quite possible that his death was related to progressive PVT.

**Adverse Events**

No patient died as a result of thrombolytic therapy or suffered permanent major sequelae. However, four patients developed hemorrhagic complications requiring treatment. One patient (patient 4) developed a groin hematoma at the site of a recent catheterization that required surgical repair. This same patient also had the only thromboembolic event, consisting of transient left facial weakness lasting 2–3 hours, with no residual abnormality. A 91-year-old patient developed severe ecchymoses of both arms requiring a 2-unit blood transfusion. Thrombolytic therapy had to be modified in two patients, one for hemorrhathrosis of the shoulder and the second for epistaxis requiring nasal packing. The occurrence of complications was not clearly related to a specific thrombolytic agent.

**Surgical Treatment**

Three patients (patients 13, 14, and 15) underwent valve replacement for PVT (two aortic valve replacement, one mitral valve replacement). Patient 13 had surgery before our routine use of thrombolytic therapy for PVT. Patient 15 was suspected to have thrombus extending upward from a previous ventricular septal defect patch repair, a diagnosis that was confirmed surgically. Patient 14 presented 3 weeks after abdominal surgery (cholecystectomy), and thrombolytic therapy was thought to be contraindicated. All these patients had uneventful recoveries. One patient in this group died at home 23 months after surgery of a stroke after she had stopped taking her Coumadin regularly.

**Incidence of PVT**

To calculate the incidence of PVT, the total number of those at risk for this complication must be known; therefore, we used only patients whose original surgery was at our institution (n=988) to calculate the thrombosis rate. Of the 17 patients in this series, 14 patients were operated on here at Cedars-Sinai (nine aortic valve replacement, three mitral valve replacement, and two double mitral and aortic valve replacement). The mean interval from the time of surgery to the occurrence of thrombosis was 53.9 months (range, 1–108 months). This results in a linearized rate of PVT of 0.2% per patient-year, consistent with the literature.

**Discussion**

**Results of Thrombolytic Therapy for PVT**

During the past years, several reports of successful thrombolytic therapy of PVT in selected patients have appeared. This is the first study in which thrombolytic therapy has been studied prospectively as the first line of therapy for PVT in a single valve type in all patients who did not have a contraindication to thrombolytic therapy.

Ten of 12 initial events (83%) responded to thrombolytic therapy. The age of patients, anatomic location of the prosthetic valve, and duration of symptoms did not predict the success of therapy. Within the first 6 months of follow-up, there were no episodes of rethrombosis. All patients, including the two who did not respond, were managed conservatively during the follow-up period. Our success rate is similar to the expected incidence of thrombus as the main cause of valve obstruction (70%) as described by Deviri et al. This suggests that failure or only partial success of thrombolytic therapy may be a result of pannus formation. Deviri reported pannus formation to be the sole cause of obstruction in 11% of valves and to be present in combination with thrombus in another 12% of valves. Most patients could be stabilized hemodynamically, probably because the St. Jude valve is a bileaflet tilting disk and thrombus formation can totally occlude motion of one disk while continuing to allow flow through the other. The mechanism for restricted opening of both
leaflets is not clear and may involve larger or multiple thrombi or could result from thrombosis of one leaflet only, with restriction of the second leaflet resulting solely from hemodynamic factors (i.e., alteration of the normal flow patterns resulting in incomplete leaflet opening). The low incidence of embolization in this series may result from preferential formation of thrombus at the disk pivot points in St. Jude valves. Formation of pivot point thrombus means that relatively small amounts of thrombus can result in restriction of leaflet opening. These relatively small amounts of thrombus may dissolve completely or substantially during thrombolytic therapy with a lower risk of embolization. This may result in a larger safety margin for thrombosed dual-disk valves compared with single-tilting-disk valves, in which larger amounts of thrombus may be required to obstruct the valve. It should be noted that some patients may demonstrate only partial or minimal improvements in opening angles but have a good clinical response (resolution of heart failure, etc.). It is possible that those patients may have had thrombus obstructing the valve orifice that was lysed by thrombolytic therapy. Thus, it is conceivable that hemodynamic and clinical improvement could occur despite persistent restriction of leaflet motion.

**Embolization**

Only one patient had a transient ischemic attack related to embolization, which resolved completely within 2 hours. This experience is similar to that of Kurzrok et al., who found that all manifestation of embolization disappeared within 2 days. Thus, it seems that after thrombolytic therapy, the majority of patients with a thrombosed prosthesis in the left side of the heart will not develop permanent neurological sequelae caused by systemic embolization. These data are also in accordance with observations of patients receiving urokinase for left ventricular thrombus after myocardial infarction.

**Choice of Thrombolytic Agents**

To decrease the risk of embolization, we believe a less intense but more prolonged thrombolytic state is desirable than is used for treatment of acute myocardial infarction. This is the primary reason that we have selected the urokinase protocol discussed in the “Methods” section. Urokinase has been used successfully in this manner for dissolution of peripheral arterial as well as pulmonary artery thrombi and also has the advantage of a lack of hemodynamic and allergenic effects. In two of our patients, heparin alone resulted in significant improvement, and its value as sole agent of treatment should be evaluated in further studies.

**Role of Cinefluoroscopy and Doppler Echocardiography**

In our experience, cinefluoroscopy is a simple and accurate method both for diagnosis of St. Jude PVT and to follow the response to thrombolytic treatment. Doppler echocardiography may also have an important role, provided that accurate measurements of gradients and valve area can be made. Doppler gradients are not sufficient for diagnosis unless they are very high, and serial changes in gradient may not be useful, because they may be caused by changes in cardiac output and hemodynamic state. In addition, Doppler gradients may be significantly higher than catheter gradients in St. Jude valves because of the phenomena of pressure recovery and high localized gradients in the St. Jude valve. However, Doppler echocardiography can provide important hemodynamic information if valve areas are assessed.

A further factor complicating the use of Doppler measured gradients and valve areas is the possibility that the relation between Doppler and catheter gradients may change as valve function returns to normal. Preliminary in vitro results from our lab suggest that the phenomena of pressure recovery and high localized gradients do not occur in thrombosed St. Jude valves. As valves become progressively more stenotic, flow through the valve becomes more turbulent, and less pressure recovery occurs. Thus, the discrepancy between Doppler and catheter gradients may progressively disappear as a valve becomes stenotic. This raises the possibility that when one measures a high Doppler gradient in a St. Jude valve, it may be a result of either a true high gradient in a stenotic valve or the recognized Doppler overestimation of gradient because of pressure recovery in a normally functioning valve prosthesis.

In assessing the relative roles of cinefluoroscopy and Doppler, it must be remembered that each method provides different kinds of information. Cinefluoroscopy provides information solely about restriction of leaflet motion. In contrast, Doppler echocardiography provides hemodynamic information about pressure gradients and valve areas. Therefore, Doppler echocardiography and cinefluoroscopy may have complementary roles in assessing prosthetic valve function. Further studies are needed to establish the preferred method for diagnosis and follow-up in patients with thrombosed prosthetic valves.

**Incidence and Characteristics of PVT**

The linearized incidence of St. Jude PVT for patients operated on in our institution is low (0.2% per patient-year). It is important to note that in all but one of the cases, PVT occurred in association with subtherapeutic anticoagulation (prothrombin time <1.2 times control) or nonuse of warfarin (two patients). In six of 17 (35%) of these patients, Coumadin treatment was stopped or modified because of elective surgical intervention. Elective surgery therefore emerges as a major risk factor for PVT in our study population. Although 13 of 17 patients with PVT were female, we cannot conclude that women are at a higher risk for developing PVT because of the small number of patients studied.

These statistics also highlight a major rationale for using thrombolytic therapy instead of surgical valve replacement. In all but one case, there was a reason for PVT related to the patient (i.e., a nontherapeutic prothrombin time). Thus, in each of these cases, there is no reason to suspect that there is a problem intrinsic to the prosthetic valve. If clot lysis can be achieved and the patient maintained on therapeutic Coumadin levels, there should be little likelihood of recurrent PVT. Therefore, we believe that in cases in which there is a clearly defined episode of subtherapeutic anticoagulation, thrombolytic therapy should be considered as initial therapy.
FIGURE 4. Flow chart showing treatment strategy for thrombosed St. Jude valves (see text for details).

Limitations of Study

In this study, no patients with a thrombosed tricuspid valve were identified, and we therefore cannot draw conclusions about management of these patients. Because of the small number of events, we also could not assess the roles of atrial fibrillation or smoking as risk factors for PVT, nor could we compare the efficacy of different thrombolytic agents. Our estimate of the incidence of thrombosis is probably an underestimate, because it is possible that some patients may have been treated elsewhere or may have died suddenly before reaching medical care. Finally, it should be stressed that our low thromboembolic rate may be valid only for the St. Jude valve. It is possible that caged-ball and single-disk valves may have a different incidence of thromboembolism or rate of successful thrombolysis. We would be cautious in extrapolating these results to other types of prosthetic valves.

Clinical Implications

Our results suggest that thrombolytic therapy should be the first line of treatment for thrombosed St. Jude valves. Medical treatment of thrombosed St. Jude valves is safe, has a low complication rate, and, in this series, resulted in no deaths related to treatment compared with the reported 4.7–20% perioperative mortality. Figure 4 outlines our present protocol for managing patients with suspected thrombosis of a St. Jude valve. Initial diagnosis is by cinefluoroscopy but might also be done by echocardiography. The key to diagnosis is restriction of leaflet motion, however, and not the demonstration of high gradients, because Doppler echocardiography may overestimate gradients in St. Jude valves as well as underestimate valve areas.32

Once significant leaflet obstruction is confirmed, patients without contraindications should receive thrombolytic therapy. Patients should provide informed consent and be aware of the risks of embolism and hemorrhage as well as the alternative treatments and their risks (i.e., surgery). After thrombolytic therapy is begun, cinefluoroscopy (or possibly Doppler echocardiography) should be performed daily to evaluate the improvement in valve function. If “normalization” of valve function occurs (defined as either normal opening and closing angles or significant improvement in the opening angles as defined in the “Methods” section), patients should be switched to heparin and then Coumadin and dipryidamole for the long term. Patients who do not respond within 48–72 hours should be referred for surgery, because these patients may have tissue ingrowth obstructing the valve (pannus formation) and probably will not respond to continued thrombolytic treatment. Gratifyingly, none of the patients receiving thrombolytic therapy in our series required surgery (although it was offered to one), and their clinical status greatly improved. If surgery is needed for PVT, it is also possible that thrombolytic therapy may allow surgery in a more stable clinical setting, although the improvement in hemodynamics must be balanced against the increased risk of hemorrhage perioperatively. Patients who return with repeated episodes of thrombosis can usually be treated successfully with thrombolytic therapy. However, these patients may be at a continuing risk for further thrombotic episodes, probably related to patient factors (senility, noncompliance, etc.), and consideration should also be given to prosthetic valve replacement with a tissue valve. Patients with residual limitation of leaflet motion after thrombolytic therapy remain a clinical challenge. In our experience, they may be managed conservatively, but this decision should be individualized.

Conclusions

Thrombosis of a St. Jude prothetic valve is a rare but potentially lethal clinical situation. The clinical presentation varies from minimal or absent symptoms to circulatory collapse, and prompt diagnosis is essential. Many patients with PVT give a recent history of stopping anticoagulation to undergo surgery. Thrombolysis may be used as the first line of therapy in patients with thrombosed St. Jude valves and appears to be an effective and safe treatment. We believe that surgery should be reserved for patients who cannot be stabilized medically and for patients who have contraindications to thrombolytic therapy.

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