Transesophageal Echocardiography in the Detection of Prosthetic Mitral Valve Thrombosis Caused by Inadequate Anticoagulation

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Intracranial hemorrhage resulting from oral anticoagulation in patients with prosthetic heart valves is rare. If it occurs, however, the anticoagulation treatment should be immediately reversed. The difficulty is to decide when to resume treatment. Without anticoagulation, the patient is at high risk of valve thrombosis and systemic embolization. We report a patient with a prosthetic mitral valve who developed intracranial hemorrhage as a complication of anticoagulation. Reinstitution of warfarin was delayed, and she developed severe valvular thrombosis with pulmonary edema. This was promptly diagnosed using bedside transesophageal echocardiography, and surgery was successfully carried out. Warfarin was restarted 4 days after surgery without further sequelaes.

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KEY WORDS: • transesophageal echocardiography • TEE • intracranial hemorrhage • anticoagulation • prosthetic heart valve • thrombosis • embolism

CASE REPORT

A 49-year-old woman with rheumatic heart disease and mitral stenosis had had mechanical valve replacement 5 years previously. Before surgery, she had had functional class II congestive heart failure. There was no history of hypertension. She received oral anticoagulation (warfarin) with careful monitoring of the prothrombin time to maintain an international normalized ratio (INR) of between 2 and 3.

She arrived in our emergency department after sudden loss of consciousness at home. Brain computed tomography without contrast revealed a huge intracranial hemorrhage (ICH) in the left hemisphere (Fig. 1). A review of her history indicated that she had been taking an irregular dose of warfarin and that she had not attended follow-up clinics in the previous month. Her INR was found to be elevated (37.7). The anticoagulation was reversed and she successfully underwent craniotomy for evacuation of the hemorrhage. No further intracranial bleeding occurred after surgery. A transthoracic echocardiogram (TTE) performed just after surgery revealed a mildly enlarged left atrium (42 mm) and a left ventricular ejection fraction of 70%. The mitral valve
orifice measured about 3 cm to 3.5 cm in diameter, and the pressure gradient was 9.8 mmHg.

Approximately 6 weeks after surgery, she developed dyspnea. Arterial blood gases showed severe hypoxemia (PaO₂: 51.4%) with respiratory alkalosis. Chest radiography after intubation and mechanical ventilation revealed bilateral homogeneous alveolar infiltrates. There was mild leukocytosis, but she was afebrile. An electrocardiogram showed sinus tachycardia with low QRS voltage in the limb leads. Swan-Ganz data indicated elevated pulmonary arterial pressure (77 mmHg) and pulmonary arterial wedge pressure (42 mmHg). A repeat TTE demonstrated a diminished mitral valve orifice of about 1.5 cm in diameter, by continuity equation, with abnormal echodense material obstructing the mitral orifice during the diastolic phase. An elevated transmural pressure gradient of about 46 mmHg and severe pulmonary hypertension estimated to be 77 mmHg were also observed. There was paradoxical septal wall motion and right ventricular enlargement (28 mmHg), but no obvious wall-motion abnormality. In order to obtain a better picture of the valve, transesophageal echocardiography (TEE) was arranged. This demonstrated the adhesion on the left atrial side of a large organized thrombus to the anterior mechanical mitral leaflet. There was no disc motion interference, but left ventricular diastolic filling was interrupted. In addition, a diminished central jet was seen from the mid-esophageal long-axis view (Figs. 2–4). Posterior leaflet movement was intermittently limited by a small thrombus obstructing the inflow orifice of the mitral area.

Fig. 1. An area of hyperdensity in the left cerebral hemisphere demonstrates intracranial hemorrhage on computed tomography with mass effect and subsequent midline shift.

Fig. 2. The white arrow indicates a large thrombus over the anterior mechanical mitral valve with obstruction of flow. The black arrow shows the original intact mechanical valve leaflet.
Emergency mitral valve replacement was performed with cardiopulmonary bypass. A large thrombus was found on the anterior leaflet (Fig. 5). The patient recovered well from surgery and was successfully extubated soon after the operation. Chest radiography 2 days later revealed rapid improvement of the pulmonary edema. Oral anticoagulation was restarted 4 days after surgery. There was no further intracranial bleeding, and the patient was discharged 2 weeks after the operation.

**DISCUSSION**

Acute dysfunction of the prosthetic valve, either because of annular dehiscence with valvular regurgitation or leaflet thrombosis with acute occlusion, may lead to an abrupt reduction in cardiac output, refractory cardiogenic shock, and/or pulmonary edema.

On echocardiography, our patient had a demonstrable mitral valve thrombosis with obstruction of flow, an elevated transvalvular gradient, no wall-motion abnormality, severe pulmonary hypertension with paradoxical septal wall motion, and right ventricular enlargement. These findings were all consistent with an obstructing valve thrombus being the sole cause of a sudden, severe decrease in cardiac output with a compensatory tachycardia.

Echocardiography performed in such situations is quite helpful and is non-invasive. TEE, as this case demonstrates, plays a more important role in the greater understanding of cause than TTE. The traditional measurement of transvalvular velocities across prosthetic valves is not always accurate, as different volume flow rates reasonably lead to different estimations of the stenotic prosthetic valve area. The pressure half-time method under these conditions may be related to different flow rates and is, thus, not independent. Use of the continuity equation method in the evaluation of prosthetic valve area.
may be more accurate and practical because it is relatively more independent of the volume flow rate being affected by various pathophysiologic conditions. It assumes that the forward stroke volume throughout the heart is equal, and a given cross-section area times regional velocity determines the same flow rate anywhere.

The recommended INR to prevent thromboembolism with prosthetic heart valves (PHVs) is between 3.5 and 4.5 [1]. However, this confers a 4- to 10-fold increased risk of ICH as a potentially fatal complication. It is reported to occur with a frequency of 1% per patient-year [2]. Profound prolongation of the INR (37.7) was the most likely cause of the ICH in our patient, as she had neither a history of hypertension nor an elevated blood pressure on admission for her ICH [3].

No large-scale studies have yet addressed the question of how long anticoagulant treatment can be safely withheld in a patient with a PHV [4]. According to Cannegieter et al, the risk of embolism from PHVs resulting in major stroke or death is 4% a year, and the risk of valve thrombosis is 1.8% a year [5]. Crawley et al gave estimates for risk assessment when anticoagulation is withheld [6]. Stopping anticoagulation for 6 weeks, as in our case, would theoretically carry a risk of embolism of about 0.67%, with a daily risk of 0.016%. Butler and Tait studied 35 patients with oral anticoagulant-related ICH and concluded that temporary cessation of anticoagulation therapy (mean, 7 days) is safe [7]. Our patient was off warfarin for much longer than this, which undoubtedly increased her risk.

The exact cause of the acute pulmonary edema was promptly identified by use of bedside TEE, and early surgery provided a better outcome. TEE enabled specific and precise morphologic observation and delineation of the mitral valve structure. This is especially relevant when a prosthetic valve implant causes reverberation and acoustic shadow, obscuring what may be seen on traditional TTE.

**CONCLUSION**

We suggest the need for large prospective trials to help resolve the problem of when anticoagulation treatment should be restarted after neurosurgery in a patient with a prosthetic mitral valve. Focus should be on the proper and accurate findings of intracardiac structure and morphology, enabling full diagnosis and the opportunity for rapid management. While traditional TTE offers useful information, early intervention with TEE, with its precise anatomic resolution, saves time and may save a life.

**REFERENCES**