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症　　例

Thrombosed Lillehei-Kaster Mitral Prosthesis

Yutaka Konishi, Katsuhiko Matsuda, Noboru Nishiwaki, Junichi Soneda, Ichiro Shimada, Yoshisada Kitao, Toshihiko Ban, Ario Yamasato*, Hiroyuki Fukumasu*, Norikazu Tatsuta** and Kazuaki Minami**

The Department of Cardiovascular Surgery, Faculty of Medicine, Kyoto University
*The Department of Cardiovascular Surgery, Takeda Hospital
**The Department of Cardiovascular Surgery, Otsu Red Cross Hospital
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Introduction

Prosthetic valve dysfunction due to thrombus is not rare. In contrast with slowly developing symptoms in patients with dysfunction of caged-ball valves, rapid clinical deterioration in those with dysfunction of tilting disc prostheses usually leads to sudden death or emergency surgery. However, the diagnosis of prosthetic thrombosis is not easy. Delayed diagnosis is fairly frequent and responsible for the high operative mortality rate (approximately 50%)[8].

This report describes two patients with late thrombosis of Lillehei-Kaster mitral valve prostheses. One was treated successfully with emergency surgery, though a definite diagnosis of thrombosed valve could not be made before surgery, and the other died before surgery. Some problems of prosthetic valve dysfunction are discussed.

Case 1.

A 38-year-old woman underwent mitral valve replacement with a 22-mm Lillehei-Kaster prosthesis for mitral stenoinsufficiency and tricuspid annuloplasty by DeVega's method for functional tricuspid insufficiency in 1979 at the age of 34. Cardiac catheterization performed one month after surgery revealed persistent moderate pulmonary hypertension and high pulmonary wedge pressure (Table 1). In addition, slight mitral regurgitation was demonstrated by left ventriculography as well as by a grade II/VI systolic murmur at the apex. However, clinical improvement was apparent, and it was decided to treat her with medications, including an anticoagulant. She had been well and worked as a charwoman until early January, 1984, when she developed dyspnea on exertion. Dyspnea became progressively more severe and she was admitted on February 1, 1984. On admission, she looked acutely ill and was orthopneic. Blood

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Present address: The Department of Cardiovascular Surgery, Faculty of Medicine, Kyoto University, Sakyo-ku, Kyoto, 606, Japan.

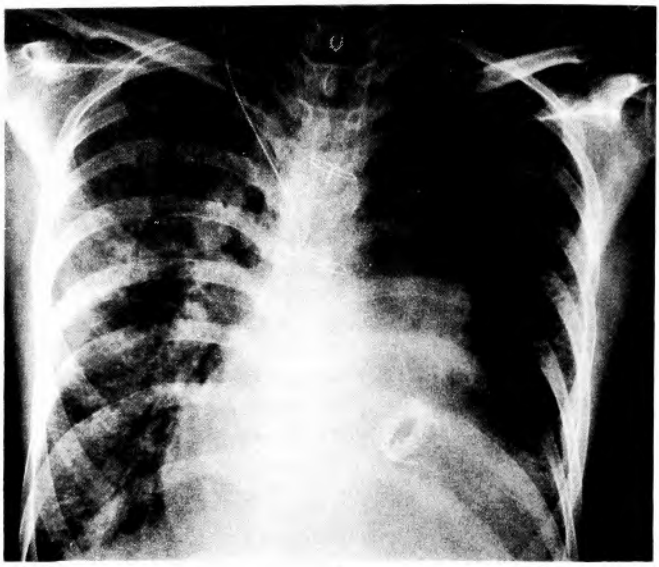

Fig. 1. Chest x-ray at the time of admission in Case 1. Severe pulmonary congestion is shown.

pressure was 110/80 mmHg. Her pulse was irregular (atrial fibrillation) at 120–130/min, and her respiratory rate was 30/min. Urine output was decreased (approximately 20 ml/hour). Prosthetic valve sounds were audible, and there was a grade II–III/VI systolic murmur from the

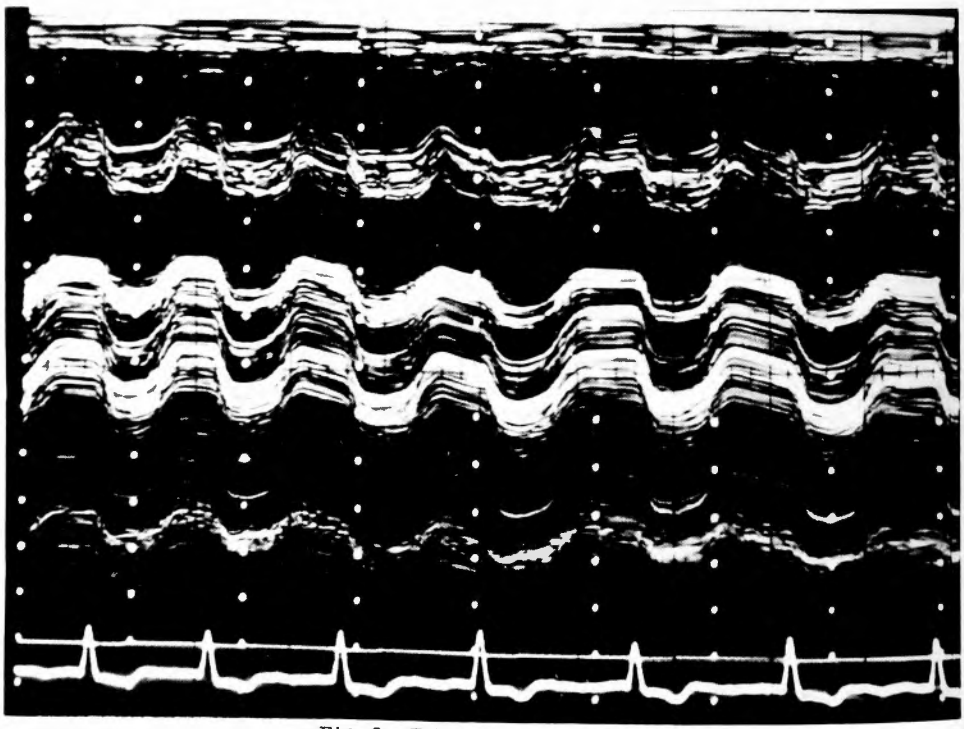

Fig. 2. Echocardiogram in Case 1.

Fig. 3. Lillehei-Kaster prosthesis removed from Case 1. The view of the prosthesis from the left ventricular aspect shows that a large pannus encroaches on the strut and impedes the disc motion.

left sternal border to the apex and a grade II/VI diastolic murmur at the apex. There were moist rales in both lungs and hepatomegaly, but no ascites or edema of the legs. A chest x-ray showed pulmonary congestion and cardiomegaly (Figure 1). An attempt was made to insert a Swan-Ganz catheter, but it could not reach the pulmonary artery, probably because of severe tricuspid regurgitation. The patient was treated intensively with dopamin, nitroglycerin and large doses of diuretics for one week. Her condition improved dramatically, and she could even walk by herself, although she sometimes complained of fatigue. The echocardiogram taken at this time demonstrated decreased opening and closing rates of disc motion and a reduced amplitude of excursion of the disc (Figure 2). However, the diagnosis of thrombosed mitral valve was not certain. On January 10, she developed the same symptoms again and her condition deteriorated progressively in spite of intensive medical treatment, including Urokinase in a dose of 60,000 units/24 hours, With a presumptive diagnosis of prosthetic valve thrombosis, surgical exploration was performed. At operation on February 17, 1984, a thrombus of recent origin was seen along the sewing ring, especially on the anterolateral aspect of the ring. On the ventricular side a pannus encroached around the anterior metal projection of the prosthesis, and the disc was fixed at an approximately 60 degree open position (Figure 3). Perivalvular leakage was not found. The prosthesis was replaced with a 25-mm Björk-Shiley prosthesis of the subannular type. The larger opening of the disc was oriented posteriorly, as the previous disc had

Table 1. Pre- and postoperative hemodynamic data.

	Preop. July 20, '79	Op. Nov. 19, '79	After Op. Dec. 19, '79	Reop. Feb. 17, '84	After Reop June 13, '84
CI (l/min/M)	2.15		2.46		3.70
LVEF (%)	–		34		32
Pressure (mmHg)					
RA (mean)	5		8		11
RV (s/e)	63/5		62/11		62/11
PA (s/d)	59/29		66/27		59/25
PAW (mean)	–		24		–
LV (s/e)	93/4		128/16		114/18

CI Cardiac index, LVEF: Left ventricular ejection fraction,
RA Right atrium, RV: Right ventricle, PA: Pulmonary artery,
PAW: Pulmonary artery wedge, s/d(e) systole/diastole (end-diastole),
Op.: Operation

been. Tricuspid regurgitation was repaired by Carpentier's ring method. The postoperative course was stormy. The patient needed intra-aortic balloon pumping support for eight days. Cardiac catheterization performed four months after reoperation showed persistent moderate pulmonary hypertension and slightly impaired left ventricular function (Table 1). However, her symptoms resolved, and she was discharged on June 26, 1984.

Case 2.

A 44-year-old housewife with mitral stenoinsufficiency underwent mitral valve replacement with a 22-mm Lillehei-Kaster prosthesis in 1978 at the age of 38. She was on anticoagulant medication and did well until April, 1984, when she developed an upper respiratory infection and cough. A few days prior to admission, she complained of general malaise, dyspnea and palpitation. On April 29, 1984, she was admitted with increasing dyspnea. Blood pressure was 130/80 mmHg. Pulse was irregular (atrial fibrillation) at 130–150/min. On auscultation, there were gallop sounds and a grade II/VI systolic murmur at the apex, but the prosthetic sound was audible. There were moist rales in both lung fields, marked jugular vein distension and hepatomegaly. A chest x-ray showed marked pulmonary congestion and cardiomegaly (Figure 4). Approximately 12 hours after admission, the patient had sudden hypotension and bradycardia and lost consciousness. The presence of mitral prosthetic thrombosis was strongly suspected, and Urokinase was started at a rate of 45,000 units/hour. In spite of all efforts, the patient continued to deteriorate and died 12 hours later. Autopsy was not done.

Comment

Although prosthetic valves are associated with a considerable risk of thromboembolic accidents, particulary serious valve malfunctions are caused by thrombi that limit the movement

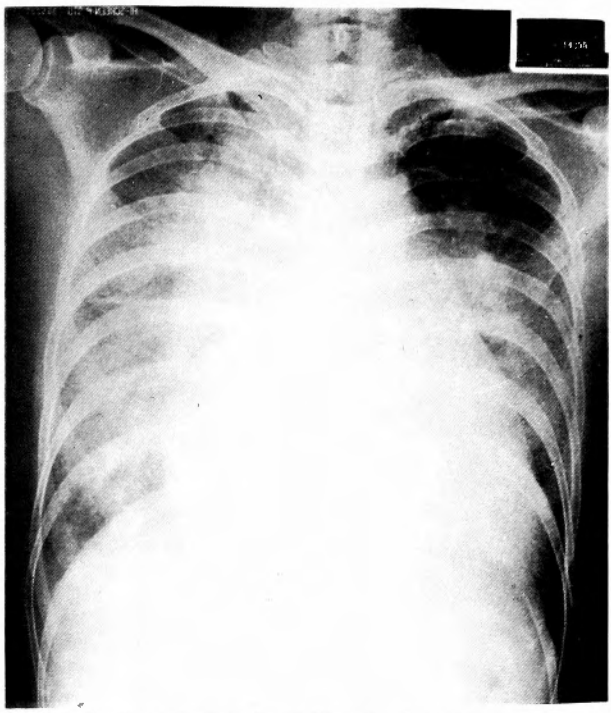

Fig. 4. Chest x-ray at the time of admission in Case 2. Severe pulmonary congestion is shown.

of the poppet. This complication is more frequent and severe with disc type prostheses, such as Björk-Shiley[3] or Lillehei-Kaster prostheses[9~12].

Both of our patients, though in one of them a final diagnosis could not be made, manifested unexplained rapid deterioration with severe dyspnea due to pulmonary edema. The first patient underwent successful surgery, but the second died of brain damage probably due to cerebral embolism.

Evaluation of the Lillehei-Kaster prosthesis is sometimes difficult. Valve dysfunction is not easily detected by auscultation because the opening click is commonly absent and apical mid-diastolic as well as systolic murmurs are often heard without valve dysfunction[1,6]. In addition, fluoroscopy is not helpful in the assessment of function of this valve because of its radiolucence. At present, echocardiography combined with phonocardiography seems to be the most reliable technic for measuring prosthetic valve function, but again, several parameters in echocardiography are easily influenced by left ventricular function, heart rate, atrial fibrillation and any changes in cardiac output[1,4] In our first patient, decreased velocity of opening and closing of the poppet and decreased excursion of the disc suggested a thrombosed valve. However, we waited about two weeks before deciding to perform surgical exploration partly because our experience with echocardiographic findings of the Lillehei-Kaster valve was limited and no preceding recordings were available for comparison and partly because the patient's condition was temporarily improved with intensive medical treatment which suggested the presence of left ventricular dysfunction. It may be difficult to distinguish prosthetic valve dysfunction from left

ventricular dysfunction in patients with congestive heart failure after valve replacement. The abrupt recurrence of symptoms after a prolonged period of improvement often indicates prosthetic valve dysfunction and is rare in myocardial failure unless the history suggests the onset of a new disease[12]. However, the final diagnosis can be made only by cardiac catheterization and left ventriculography[1,12], although in most instances the patient's condition is too severe to perform these studies, as was true in our patients.

There are two opinions on the etiology of thrombosed valves despite adequate anticoagulation; one is related to surgical techniques regardless of the type of prosthesis, such as implantation of a prosthesis that is too large[5], use of Teflon felt pledgets to suture a prosthesis[11] or orientation of a prosthesis[2], and the other is related to the design or materials of the prosthesis itself. The close similarity between thromboses on Björk-Shiley and Lillehei-Kaster prostheses indicates that these tilting valves have the same fundamental weakness. That is, the contiguous association between a mobile lightweight poppet and a thrombogenic valve ring can easily cause disturbances of valve function even with a small thrombus[8]. In our patients, a 22-mm prosthesis was not too large, but pannus formation was seen around the anteriorly seated metal strut, suggesting that the strut could become embedded in the adjacent endocardium of the left ventricle and that tissue ingrowth was provoked and could subsequently impede disc motion.

There have been several reports on the beneficial effects of fibrinolytic therapy with a large dose of Streptokinase or Urokinase (100,000–200,000 units/hour) when valve thrombosis was suspected[7,13]. However, hemorrhagic complications and the risk of embolic migration after thrombolysis have also been reported. Therefore, fibrinolysis should be reserved for patients with severe clinical symptoms[7].

In conclusion, the diagnosis of a malfunctioning prosthesis is not easy. It is essential, therefore, to perform postoperative serial phono- and echocardiography because these tests are not very specific for thrombosis and are helpful only when compared sequentially[4].

Summary

Two patients had late thrombosis of Lillehei-Kaster mitral prostheses. Both had unexplained rapid deterioration with severe dyspnea and pulmonary edema late after mitral valve replacement with Lillehei-Kaster prostheses. The first patient, a 38-year-old woman, had emergency reoperation, although a definite diagnosis of thrombosed valve could not be made before surgery. At operation, a thrombosis of recent origin was seen along the sewing ring of the prosthesis, and on the ventricular side a pannus encroached around the metal strut so as to impede disc motion. The prosthesis was successfully replaced with a Björk-Shiley prosthesis. The second patient, a 44-year-old woman, died before surgery.

Evaluation of a thrombosed valve is not easy by auscultation, phonocardiography or even by echocardiography. The diagnosis may be possible only when postoperative serial phono- and echocardiograms are available for comparison.

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和文抄録

Lillehei-kaster 僧帽弁による血栓弁

京都大学医学部心臓血管外科

小西　裕，松田　捷彦，西脇　登，曽根田純一

嶋田　一郎，北尾　義実，伴　敏彦

武田病院心臓血管外科

山里　有男，福増　広幸

大津赤十字病院心臓血管外科

龍田　憲和，南　一明

Lillehei-Kaster 僧帽弁置換術後，遠隔期において発生した血栓性弁機能不全症の2例を報告した．1例（38歳，女性，術後4年）は緊急再手術により救命しえたが，他の1例（44歳，女性，術後6年）は脳障害を併発して再手術を受けることなく死亡した．血栓による人工弁機能不全の問題を考察し、とくにその診断は容易でないことを強調した．術後定期的に心音図，心エコー図など施行して，それらのデータを経時的に比較検討することが診断上最も重要なことである．