The Operated Cases of Mitral Valvular Disease

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I'M GOING TO report the unsuccessfully operated cases of mitral valvular disease which is the most common form of the acquired heart disease.

Case 1: A 44-year-old woman entered the hospital for dyspnea on exertion and compression sensation of the chest.

One year prior to this admission, she began to have the symptoms on sloping. Since then she had had episodes of cough and compression sensation in the forechest 2 to 3 times a month. No history of rheumatic fever was noted in the patient.

On admission a grade 3 diastolic murmur was audible at the apex and systolic murmur at the pulmonic area. The 2nd sound at the pulmonic area was accentuated. Phonocardiogram recorded an abnormal sound like the opening snap. But occasionally only a systolic murmur at the apex was audible. The left atrium was prominent in the postero-anterior, left anterior oblique and right anterior oblique chest X-ray films. Pressures of the pulmonary artery and capillary were elevated, 62 mmHg, 30 mmHg, respectively. Electrocardiogram was suggestive of the bilateral ventricular hypertrophy and mitral P-wave, but they were not so significant. On left atrial puncture the pressure was 20 mmHg which was slightly elevated and there was high V-wave (Fig. 1). Upon the diagnosis of mitral steno-insufficiency, the patient was operated upon for commissurotomy. On opening the chest, the left atrium was not so enlarged as was expected, and no hypertrophy

Fig. 1. Case 1, left atrial pressure tracing showing regurgitant V-wave.

of its wall was noted. There was found no thrombus in the blood flushed out through the appendage. There was no regurgitant jet of blood and thrombus in the left atrium on finger examination through the appendage, but a soft pigeon-egg sized tumor was palpable. It had a pedicle arising from the lateral wall of the left atrium near the valvular ring. This tumor covered the mitral orifice and made a finger impossible to get access to it. No abnormalities were found in the orifice itself. We know there is a case report¹⁾ in which a tumor was removed with closed method, but we decided to remove the tumor under direct vision because we were afraid of occurrence of cerebral embolism for its fragility.

The operation was finished without removing the tumor.

After the operation the patient remained unconscious and several hours later signs of cerebral embolism developed. The patient

died on the 3rd post-operative day.

At autopsy, there was a $5 \times 3 \times 2$ cm. myxoma in the left atrium and necrotic foci in the cerebrum and cerebellum. In the left common and internal carotid arteries were found thrombi in which no tumor cells were microscopically demonstrable. Microscopical examination of the tumor showed typical features of a benign myxoma (Fig. 2, and 3).

Fig. 2. Case 1, photograph of gross specimen of left atrial myxoma. Note the polypoid nature of the tumor.

Fig. 3. Case 1, photomicrograph (low power), showing diffuse scattering of stellate and spindle cells in amorphous stroma.

In this case there were no signs and symptoms suggesting the presense of embolism during the pre-operative period and the operation. As the prophylactic procedures to prevent the embolization, a gush of blood on opening the left atrium and compression of the carotid arteries in handling the left atrium, etc. were performed. In spite of these procedures the patient died because of the em-

bolism.

Tumor in the atrium should be operated under direct vision, but the patient was mistakenly operated upon with closed method because of the incorrect diagnosis of mitral valvular disease.

Whenever in making the diagnosis of a case, suspicious of mitral valvular disease, it should be emphasized to be aware of the presence of tumor arising from heart.

It is noted that the clinical pictures²⁾ of left atrial tumor are characterized by the sudden and intermittent onset of such symptoms and signs as the changeable murmurs and the others above mentioned. This hold true in our case.

Thrombosis of the left atrium is common in patients with mitral stenosis. It is conceivable that the cases with left atrial tumor may also have a formation of thrombi by blocking the mitral orifice with the tumor. There are many procedures to prevent embolism occurring during operation, but none of them is reliable. Among them Bailey's method³⁾ seems to be a relatively reliable one. But it is not available in all the cases because of its complicated processes. In this case it was not performed. Thrombi might be formed in remained parts of the appendage which has become a blind pouch after closure of the stump. It would seem this might be a cause of embolization in this case. The blood pressure became transiently lowered (80 mmHg) during the operation, which might give a clue to the formation of new thrombi in the auricle and to develop emboli in the carotid artery. The appendage should be ligated in the base not to make a blind pouch.

We had another case of mitral stenosis. The patient was 46-year-old man and died of cerebral embolism.

As the presence of thrombi was evident in the left atrium before the operation, a finger was inserted apart the lesion and commissurotomy was done. Of course, during this procedure the carotid arteries were compressed. In spite of the procedure embolism of the brain and left popliteal artery developed. So that embolectomy of the left popliteal artery

was immediately done and pulsation became palpable, but died on the 2nd post-operative day. At autopsy the emboli were demonstrated in the regions supplied by the middle cerebral arteries. There were mural thrombi in the atrium which might be responsible for the death.

Incidence of the embolism, in patients with mitral stenosis, varies from more than 10% to less than 1%, depending upon the investigators. As the embolism is very often fatal, all patients, especially having history suggestive of embolism or thrombi recognizable through the atrial wall on operation, should be operated on under direct vision. Unfortunately, if cerebral embolism develop, angiographic study would be helpful to visualizing its site and the treatment of it in such a patient as case 1. Removal of emboli from the carotid artery is not simple, but aspiration of emboli through a catheter inserted into the artery is occasionally helpful.⁴⁾

Case 2: A 37-year-old man have had mitral stenosis with pulmonary hypertension for four years. A grade 3 diastolic murmur at the apex was audible. Chest X-ray films showed a enlarged left atrium. Pulmonary artery pressure was 80mmHg, pulmonary capillary 52mmHg.

At operation, the left atrium was distended and enlarged. Its wall was hard on palpation. A purse-string suture was very carefully done, through which a finger was inserted. It was evident that the mitral orifice was narrowed to approximately 0.5 cm² and a calcification of the valve marked. Commissurotomy with a finger and then with a knife was failed to be done. A laceration in the left atrium and hemorrhage occurred on removing the inserted knife from the left atrium (Fig. 4). For the hemostasis, a large purse-string suture and stay sutures around the laceration were applied. On tying these sutures a part of the atrial wall was torn for fragility which made the laceration larger and bleeding became more massive and finally it became impossible. For control of lacerated atrium, tamponade with fingers, one inside the atrium, the other outside, is one of the most

Fig. 4. Case 2, laceration of the left atrium (schematic).

reliable procedures, but its efficacy is not always sure. Another reliable procedure is to induce artificial cardiac arrest, and then to interrupt blood circulation temporarily by clamping efferent blood vessels, that is, aorta and pulmonary artery at their origin. To clamp afferent vessels is anatomically difficult. Duration of the interruption at normothermia is limited for 4 min. which is long enough to control bleeding in some cases. This procedure is worthy of resuscitating a patient, he will otherwise die. Many method for inducing cardiac arrest have been recommended, one of which is to make the heart fibrillated by weak electric stimuli⁵⁾. After establishment of hemostasis defibrillation of the heart is usually easy. Of course, rapid replacement of the blood loss is essential.

Case 3: A 39-year old man have had mitral stenosis with atrial fibrillation for 3 years.

Phonocardiogram revealed presystolic murmur. Chest X-ray films showed the prominent left atrium. Pulmonary capillary pressure was 45 mmHg. On left atrial puncture the pressure was 40 mmHg and pressure tracing was of stenotic type.

On operation, the mitral orifice was stenotic

to approximately 1cm², presenting an absence of the jet, and the valve leaflets were markedly calcified. Mitral orifice was dilated to 1.5 finger-breadths with digital commissurotomy.

The patient died of cardiac failure on the 42th post-operative day with a course of

high hectic fever (39° to 40°C), starting from the 2nd week following operation. Laboratory findings including blood culture, failed to detect a cause of fever, which was intractable to all available medicine, including antibiotics (Table I).

TABLE I CASE 3, CHART OF THE POSTOPERATIVE COURSE

		Op	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																											
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Note	Erythromycin 1.0 gm. i.v.				Erythromycin 1.0 gm. oral										Chloromycetin 1.0 gm. oral				Kanamycin 1.0 gm. i.m.				Chloromycetin 2.0 gm. oral										Predonin 10 mg. oral						Penicillin 3 million i.u. i.m.																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																
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Although, it is likely to deem its cause to be a recurrence of rheumatic fever, this patient had not have the history, nor the histological findings of the removed appendage showed the evidence. However, the rheumatic fever can not be ruled out altogether in this case. Steroid hormones were administered for 1 week starting from the 2nd week after the operation.

There was a report on candidiasis⁶⁾ following the heart surgery, but laboratory tests for it were not done in this case.

Post-commissurotomy syndrom, which is due to reaction after trauma or inflammation, might be responsible for the high hectic and intractable fever.

Accumulation of the blood in the pericardiac and/or pleural cavity might be also responsible. In this case, the appendage was closed with interrupted sutures after single

ligation with two drains inserted into the chest, ensuing a possible secondary hemorrhage from the stump. It is presumed that the febrile reaction might represent the effect of absorption of the blood accumulating in the pericardiac sac.

In retrospect, the pericardiac sac should have been opened to the pleural cavity in this particular case.

I am pleased to have any information about the cause of this fever.

Today I presented the cases of mitral stenosis which were operated without favorable results.

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