A 64-year-old man was admitted to a community hospital for evaluation of fever of unknown origin of 2 weeks’ duration. The patient had also sustained a 5-kg weight loss during the previous 3 weeks. Physical examination was unremarkable; in particular, no heart murmur could be detected. On admission, his leukocyte count was 20,880 cells/µL; C-reactive protein was 199 mg/L; and 4 blood cultures were positive for *Enterococcus faecalis*. Chest x-ray and abdominal and thoracic computed tomography scans were normal. Bone marrow biopsy was consistent with nonspecific inflammation. Follow-up chest x-ray examination, however, showed a lesion in the lower right lung field; this lesion appeared to be an abscess on computed tomography. Three different transesophageal echocardiograms indicated no pathological findings; in particular, there was no evidence of valvular vegetations.

The patient received intravenous antibiotic therapy for 3 weeks. Fever and inflammation parameters declined to normal values and, after an interval of 8 days free of fever, the patient was discharged. Five days later, fever recurred, leading to readmission.

On readmission, the total leukocyte count was 19,000 cells/µL, and blood cultures were positive for *Enterococcus faecalis*. Two further transesophageal echocardiograms, however, were negative for endocarditis. When fever recurred, the patient was referred to our hospital for evaluation.

On admission, there was a 3/6 systolic murmur in the left parasternal area. Transthoracic echocardiography revealed severe pulmonary regurgitation due to partial destruction of the valve by a large vegetation (Figures 1 and 2). Diagnosis of pulmonary valve endocarditis was confirmed by transesophageal echocardiogram. In addition, a transthoracic echocardiogram showed right heart enlargement, and abdominal ultrasound revealed signs of right heart failure. After 2 additional weeks of antibiotic therapy, the patient underwent uneventful pulmonary valve replacement (25-mm homograft) and coronary bypass grafting (right internal mammary artery to the right coronary artery) (Figure 3). The further clinical course was uneventful.
Figure 3. Pulmonary valve and vegetation after excision.
Pulmonary Valve Endocarditis
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