



## Hemolytic Anemia 19 Years after Mitral Valve Replacement with A Porcine Bioprosthesis

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### Abstract

A 58-year-old man required replacement of a porcine mitral prosthesis because of severe hemolysis 19 years after implant. At operation the major pathologic finding was a perforation of one cusp with calcific deposition limited to the commissures. Clinically evident hemolytic anemia due to porcine valve dysfunction is rare particularly occurring very late postoperatively due to cusp perforation as in the present case.

### Keywords

Mitral valve replacement, Mitral valve reoperation, Porcine xenograft

### Introduction

Clinically evident hemolytic anemia due to porcine bioprosthetic dysfunction and leading to reoperation is a rare event. We report a patient who developed such complication late after mitral valve replacement (MVR) with a porcine valve.

### Case Report

A 58-year-old man had undergone mitral valve replacement with a 27 mm Carpentier-Edwards porcine bioprosthesis (Edwards Lifesciences, Irvine, USA) in 1993. Nineteen years later he presented with dark urine, fatigue and moderate dyspnea denying any history of infection. He was in atrial fibrillation with a systolic mitral regurgitant murmur. Hematologic evaluation was consistent with haemolytic anemia; particularly, reduced hemoglobin, hematocrit and haptoglobin (6.6 mg/dl), and increased total bilirubin and lactic dehydrogenase (752 U/L) with schistocytosis and reticulocytosis were observed. A 2D echocardiogram showed moderate mitral regurgitation and increased pulmonary artery pressure. At reoperation the mitral prosthesis was replaced with a bileaflet mechanical valve. At gross inspection the explant showed pliable leaflets with a small perforation of one cusp; on x-ray calcific spots were detected mainly at one commissure (Figure 1 and Figure 2). The patient recovered uneventfully and was discharged on postoperative day 8 when the haemoglobin and hematocrit levels had increased (12.8 g/dl and 37%, respectively), total bilirubin and haptoglobin were normal and lactic

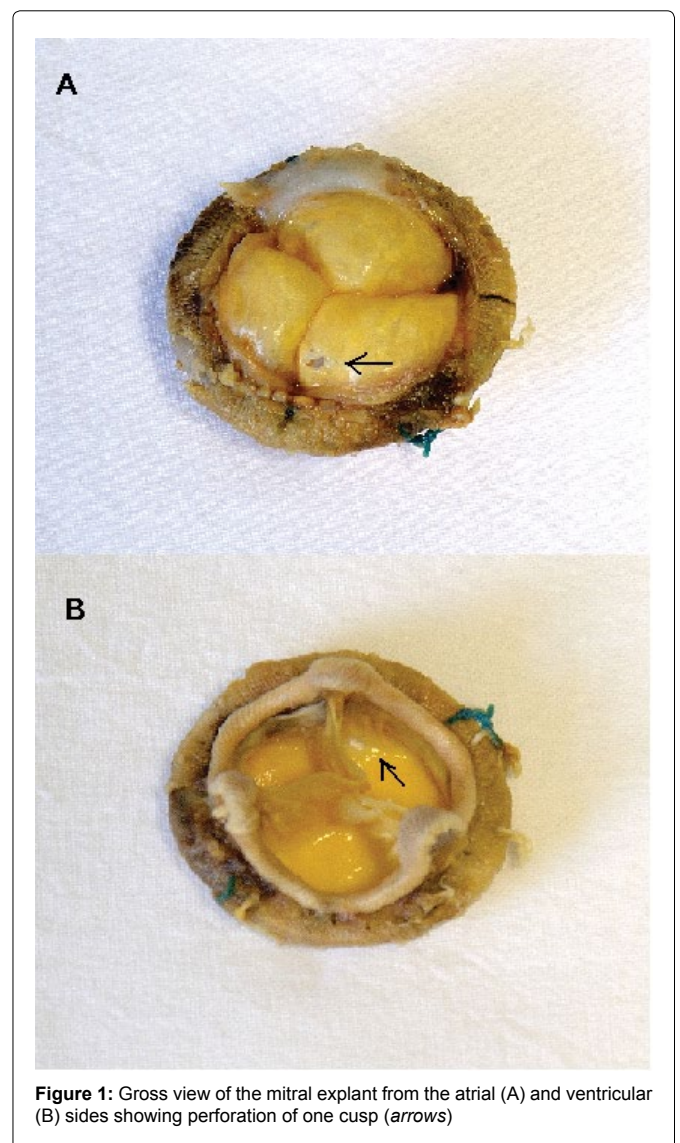
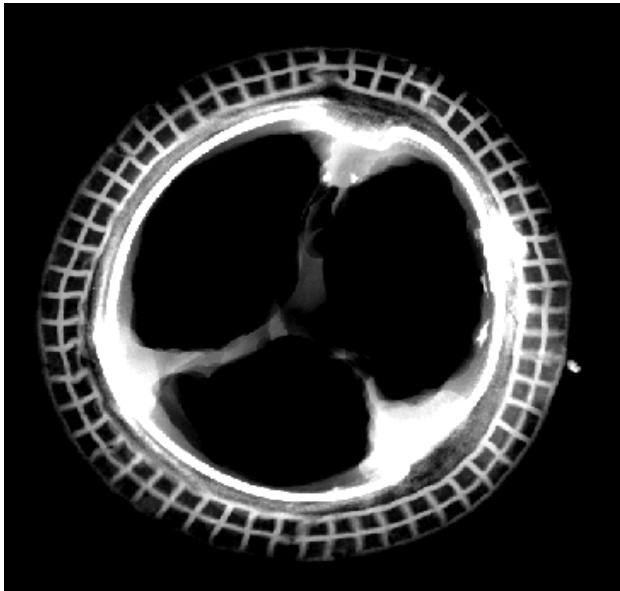


Figure 1: Gross view of the mitral explant from the atrial (A) and ventricular (B) sides showing perforation of one cusp (arrows)

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**Figure 2:** X-ray of the explant showing minimal calcific deposits

dehydrogenase was considerably reduced but still slightly increased (268 U/L).

### Comment

Subclinical hemolysis in normally functioning prosthetic valves is a frequent finding [1] but haemolytic anemia secondary to porcine valve dysfunction is rare. A limited number of cases of haemolytic anemia in tissue valve recipients has so far been reported all being related to severe bioprosthetic dysfunction requiring reoperation [2-5]. Conservative treatment of hemolytic anemia occurring in patients after MVR with either a porcine or pericardial bioprosthesis without evidence of hemodynamically significant mitral regurgitation was also

reported [6,7]. In 1990 Raymond et al. observed microangiopathic hemolytic anemia in a 59-year-old man who 10 years previously had undergone MVR with a porcine bioprosthesis [8]; at reoperation the cause of anemia was attributed to perforation of one cusp. Similarly, the peculiar finding in our case was the presence of clinically evident haemolytic anemia caused by a cusp perforation in a porcine xenograft who did not exhibit other major pathological changes, particularly in the absence of signs of infection. Interestingly enough in our case this complication occurred very late postoperatively, 19 years after MVR with a Carpentier-Edwards xenograft, indicating that first-generation porcine bioprostheses may have an extended durability in the individual patient.

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