Idiopathic Perforation of a Porcine Aortic Bioprosthesis in the Aortic Position

Winston N. Bloch, Jr., M.D.;** Zeynel Karcioglu, M.D.;† Joel M. Felner, M.D.;** James S. Miller, M.D.;** Panagiotis N. Symbas, M.D., F.C.C.P.;‡ and Robert C. Schlant, M.D.;**

The report of a failure of a glutaraldehyde-preserved porcine aortic xenograft bioprosthesis in the aortic position after 13 months is presented. Severe aortic regurgitation resulted from three "idiopathic" perforations in one of the cusps, and a linear tear in another cusp. Light and electron microscopy showed generalized degeneration of collagen throughout the faulty valve. The absence of a platelet-fibrin coat on edges of the tear suggested a recent origin, compatible with cardiac catheter manipulation during unsuccessful attempts to cross the valve. The histopathologic data from this valve correlate with previously reported failures with formaldehyde-preserved xenograft valves.

Recent reports have documented the usefulness and suggested the superiority of the porcine aortic bioprosthesis for aortic valve replacement.1-3 As this application of the bioprosthesis has come into widespread use, relatively few complications have been reported.4,4 We will now describe idiopathic perforation of the porcine aortic bioprosthesis in the aortic position, a difficult-to-diagnose complication which has not been reported before.5,6 A similar type of perforation in a porcine aortic bioprosthesis in the mitral position has been attributed to rough handling (personal communication from N.E. Shumway, M.D.).

Case Report

On Sept 16, 1975, a 66-year-old white man underwent aortic valve replacement with a No. 23 Hancock* porcine aortic bioprosthesis as treatment for calcific aortic stenosis. Following surgery, his only cardiac murmur was a nonradiating spindle-shaped grade 1/6 early systolic murmur at the second right parasternal interspace.

He was subsequently lost to followup, and when next seen on Jan 19, 1976, he had a grade 2/6 early diastolic decrescendo blowing murmur along the mid-left sternal border, and blood pressure of 160/70 mm Hg. Echocardiography at that time revealed normal motion of the aortic bioprosthesis stent,8 but the porcine leaflets were not visualized. Chest x-ray film revealed a normal cardiac silhouette with a normal appearance of the porcine bioprosthetic annulus in the aortic position. Initial cardiac fluoroscopy showed

For editorial comment, see page 490

**From Emory University School of Medicine and Grady Memorial Hospital, Atlanta.
†Division of Cardiology, Department of Medicine.
‡Department of Pathology.
Reprint requests: Dr. Felner, Emory University School of Medicine, 69 Butler Street, SE, Atlanta 30303

Figure 1. (A, upper). Linear tear parallel to free margin of largest porcine leaflet. Hole produced by tear appears as triangular space in central upper part of picture of aortic side of valve. Cotton ball holds valve open, while paper triangle holds torn leaflet open. (B) Three fenestrations in one porcine leaflet, as seen at oblique angle from left ventricular side of bioprosthesis. Size of fenestrations progressively decreases from central fenestration to one near commissure. Smallest fenestration is continuous with linear tear, which extends to leaflet margin where it is adjacent to one end of linear tear in opposite (largest) leaflet. Note absence of valvular vegetations.

abnormal tilt of the aortic bioprosthetic annulus (7*).

On Sept 27, 1976, he reported two weeks of increased dyspnea on exertion and mild ankle edema. His blood pres-
sure was 180/60 mm Hg, and there was an increase in the
duration of the blowing decrescendo diastolic murmur
throughout diastole and in its intensity to grade 3/6. The
murmur also radiated to the cardiac apex, where a soft mid-
diastolic rumble was heard. The electrocardiogram was un-
changed from January, 1978, with sinus rhythm, atrial abnor-
mality and left ventricular hypertrophy. Chest x-ray film
revealed the changes of mild cardiac enlargement and in-
creased left ventricular prominence compared with Septem-
ber, 1975.

Cardiac catheterization on Oct 1, 1976, revealed severe
aortic regurgitation. Supravalvular cineangiography in the
left anterior oblique projection suggested that the aortic
regurgitant jet passed centrally through the bioprosthetic
leaflet. The bioprosthetic annulus lift was less than 10°.
Despite numerous attempts, the bioprosthetic valve could not
be crossed retrograde using either a No. 6.7 pigtail, or a No. 7
Gensini catheter.

Aortic valve replacement with a Hancock No. 23 porcine
aortic bioprosthesis was performed on Oct 26, 1976. No
paravalvular leakage was demonstrated around the porcine
bioprosthesis prior to its surgical removal.

There was a linear tear 2-3 mm inside the appositional
margin of the largest porcine leaflet, which paralleled the
leaflet edge along 8 mm of its 20 mm edge (Fig 1). An
adjacent leaflet had three oval fenestrations 1-2 mm in width
and 1 mm, 2 mm and 4 mm, respectively in length. (Fig 1).
The smallest fenestration was continuous with a linear tear
extending 4-5 mm to the free edge of the leaflet near a
commissure where it adjoined the linear tear in the adjacent
leaflet. The largest fenestration was located centrally in the
leaflet, with middle-sized fenestration located half-way be-
tween the two. Despite the defects in the leaflets, they
coapted appropriately without prolapse.

Each valve cusp of the failed aortic valve and of an
unimplanted control porcine heterograft aortic valve was
sectioned and stained by the hematoxylin and eosin, alcian
blue, Masson’s trichrome, and elastic von Gieson methods.
Transmission electron microscopic examination was also per-
formed on all cusps of both valves.

Light microscopy revealed a thin layer of platelets and
fibrin along most of the edge of each xenograft leaflet of the
patient’s aortic bioprosthesis, except along the edges of the
linear tear. No superficial bacteria, inflammation, calcium or
vascularization was found. The collagen fibers of the fibrous
(central) layer of all three valve leaflets were disorganized
and fragmented, suggesting fibrinoid necrosis with focal de-
naturation (Fig 2). In these areas, there was increased
cellularity, nuclear fragmentation and cellular debris. Such
changes were generalized and not confined to areas of leaflet
defects, but were, however, less striking in the one cusp with
no tear or perforation. Elastic fibers, however, appeared
normal.

Electron microscopy confirmed the general loss of struc-
tural integrity of collagen. Collagen fibers appeared dis-
rupted, with deposition of a finely granular electron-dense
material between the collagen fibers and the cells (Fig 3).

Figure 2. Transverse section of body of cusp of failed xenograft valve. Ar-
rows indicate focal hyaline degeneration of fibrosa layer (Masson’s tri-
chrome, ×125).

Figure 3. Electron microscopic picture of body of cusp of failed xenograft valve. There are irregular, electron dense deposits of degenerating collagen (DC) interspersed between intact collagen fibrils (C). V=vacuoles (×5650).

580 BLOCH ET AL

CHEST, 74: 5, NOVEMBER, 1978
xenograft agree with those by light microscopy in 11 of 11, and electron microscopy in 1 of 2 of the “failed” formaldehyde-preserved aortic xenograft valves previously reported.8

Our electron microscopy observations of electron-dense material merging with disrupted collagen fibers are in agreement with those of Rose,4 who also believed that it represented collagen degeneration in a xenograft valve. This speculation is partly supported by the presence of greater cellularity, fragmented nuclei, and cellular debris in the areas of fragmented collagen. We also believe that the amorphous granular deposits in the cusp fibrosa layers result from collagen disintegration. Similar light and electron microscopic findings of collagen degeneration were observed in ruptured chordae tendineae by Caulfield et al.9

It is significant that we have documented the same type of tissue degeneration in a glutaraldehyde-preserved porcine aortic xenograft as was previously found in formaldehyde-preserved porcine xenografts,8,10 which usually failed within several years of implantation.

Since there was a platelet-fibrin coat over much of the free edges of our patient’s valve cusps but none was present along the edge of the linear tear, we believe that the tear was a recent development. We postulate that repeated attempts to pass a cardiac catheter across the structurally weakened valve may have caused the linear tear.

ACKNOWLEDGMENT: We express our appreciation to Claudia Baste, Ph.D., Department of Anatomy, Emory University for her efforts at ultrastructural examination.

REFERENCES