Croatia, only amphotericin B and imidazoles were registered, and we made a choice based on the medical literature. In this case, ketoconazole was successful. The problem is that there is no rule for how long antimycotic drugs should be prescribed for this kind of infection.[11] According to the previous reports, the best results were achieved with surgical extirpation and prolonged course (even 14 months) of high-dose parenteral miconazole (80 to 90 mg/kg/d).[12] Our patient was not a candidate for surgical treatment because of bilateral and disseminated pulmonary involvement. She developed such severe nausea while receiving a moderate dose of miconazole that the drug had to be discontinued and replaced by ketoconazole. The next problem is the combination of antifungal and antituberculosis drugs because both tend to be hepatotoxic. Besides that, rifampin (a potent inducer of hepatic enzymes) may accelerate the metabolizing of miconazole and consequently decrease its concentration in the serum under the therapeutic level. On the other hand, ketoconazole (a potent inhibitor of hepatic enzymes) may induce isoniazid-related toxicity. In the hope of avoiding that, we reduced the dose of isoniazid from 400 to 300 mg.

Most previously reported patients with invasive pulmonary pseudallescheriasis had a high degree of immunodeficiency. Based on laboratory findings, our patient was not demonstrably immunocompromised. However, following inhalation of fungal conidia, simultaneous tuberculous infection might impair the host's defenses and enable fungal invasion. Tuberculosis may impair the macrophage fungicidal mechanism. Chronic granulomatous disorders are known as a possible cause of dysfunction of the neutrophils, which is an additional risk for fungal infection.[2] Under certain conditions, fungal invasion may begin, and, consequently, it could be wrongly assumed that antituberculosis therapy had failed. On the other hand, in this case healing of tuberculosis had no influence on the course of the once-initiated fungal infection.

Fungal colonizing of healed tuberculous and sarcoid cavities is common and well documented, but coexisting tuberculosis and invasive pulmonary mycosis is a rare finding, especially in an apparently immunocompetent host. While coinfection with tuberculosis and invasive or semi-invasive aspergillosis[13,14] occasionally has been reported, tuberculosis associated with invasive pseudallescheriasis is an extremely rare finding. Rippon,[7] giving only a few details about the clinical course, noted a similar case in his textbook. Perusing the accessible literature on MEDLINE in the Varazdin Medical Center Library, we have not found any other reference of this kind in the last 13 years from 1983 through 1995.

References

1 Larone DH. Medically important fungi. Washington, DC: American Society for Microbiology, 1995; 132-33

2 Rubin RH. Fungal infection in the immunocompromised host. In: Fishman AP. Pulmonary diseases and disorders. New York: McGraw-Hill, 1988;1761-74

3 Travis LB, Roberts GD, Wilson WR. Clinical significance of *Pseudallescheria boydii*: a review of 10 years' experience. Mayo Clin Proc 1985; 60:531-37

4 Gueho E, De Hoog GS. Taxonomy of the medical species of Pseudallescheria and Scedosporium. J Mycol Med 1991; 118:3-9

5 Arnett JC, Hatch HB. Pulmonary allescheriasis. Arch Intern Med 1975; 135:1250-53

6 Kwong-Chung KJ, Bennett JE. *Pseudallescheria boydii*. In: Medical mycology. Philadelphia: Lea & Febiger, 1992;678-794

7 Rippon JW. Pseudallescheriasis. In: Medical mycology. Philadelphia: WB Saunders, 1988;651-80

8 Hung CC, Chang SC, Yang PC, et al. Invasive pulmonary pseudallescheriasis with direct invasion of the thoracic spine in an immunocompetent patient. Eur J Clin Microbiol Infect Dis 1994; 13:749-51

9 Montone KT, Litzky LA. Rapid method for detection of Aspergillus 5S ribosomal RNA using a genus-specific oligonucleotide probe. Am J Clin Pathol 1995; 103:48-51

10 Lake FR, Tribe AE, McAleer R, et al. Mixed allergic bronchopulmonary fungal disease due to *Pseudallescheria boydii* and Aspergillus. Thorax 1990; 45:489-91

11 Pluss JL, Opal SM. An additional case of pulmonary *Pseudallescheria boydii* improved with ketoconazole therapy [letter]. Chest 1985; 87:843

12 Dworzack DL, Clark RB, Borkowski WJ, et al. *Pseudallescheria boydii* brain abscess: association with near-drowning and efficacy of high-dose, prolonged miconazole therapy in patients with multiple abscesses. Medicine 1989; 68:218-24

13 Kueh YK, Chionh SB, Ti TY, et al. Tuberculosis and invasive aspergillosis in a young woman with myelodisplastic syndrome. Singapore Med J 1995; 36:107-09

14 Khono S, Hatano T, Yamada H, et al. Superinfection of chronic necrotizing pulmonary aspergillosis by *Mycobacterium tuberculosis*. Intern Med 1992; 31:540-43

Left Anterior Descending Coronary Artery Bridge*

A Cause of Early Death After Cardiac Transplantation

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Immediately following orthotopic transplantation, a patient suffered left pump failure, which resulted in death. Autopsy of the donor heart revealed a proximal left anterior descending artery bridge with a thrombus causing segmental distal anteroseptal infarction. In this case report, myocardial coronary bridges and their clinical implications are reviewed. Myocardial bridging and acute coronary obstruction should be considered in the differential diagnosis of

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patients with acute pump dysfunction following orthotopic cardiac transplantation.

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Key words: cardiac death; coronary artery bridge; thrombus; transplantation

Abbreviations: LAD=left anterior descending

The clinical importance of myocardial bridges has long been debated. Even though the long-term prognosis appears to be favorable,[1] several reports show an association of this condition with sudden death,[2] myocardial infarction,[3,4] coronary artery spasm,[5] ventricular arrhythmias, conduction disturbances, and chest pain syndromes indistinguishable from atherosclerotic heart disease.[6] We describe a case of immediate systolic dysfunction after transplantation that resulted from coronary artery thrombosis localized in the region of a myocardial bridge in the proximal left anterior descending (LAD) coronary artery.

Case Report

A 48-year-old man with ischemic cardiomyopathy and a nonreactive panel reactive antibody underwent orthotopic cardiac transplantation. The donor heart was obtained from a previously healthy 15-year-old boy with fatal trauma to the brain. The organ procurement and implantation were uneventful. Total ischemic time was 149 mins. Cardioplegia preservation technique was with Stanford/K^+ solution. Weaning from cardiopulmonary bypass could only be accomplished with aortic balloon counterpulsation due to persistent hypotension and low cardiac output. The patient required high doses of inotropic support including epinephrine, isoproterenol, and calcium due to profound left ventricular dysfunction, thought to be preservation-related. No obvious regional wall motion abnormality was noted but rather apparent left ventricular failure. Immediately after transplantation, the patient developed a new right bundle branch block with low QRS voltage and diffuse nonspecific ST-T segment changes. An echocardiogram revealed global hypokinesis of the left ventricle with distal and septal akinesis and marked reduction of the left ventricular ejection fraction. Hemodynamics were compatible with left ventricular failure, high left ventricular filling pressures, and severe low output state. Despite aggressive measures, the patient died on the third hospital day from persistent pump dysfunction. Coronary artery disease was not considered due to the donor's young age and apparent good health.

Postmortem examination revealed that the suture lines anchoring the donor's heart to the patient were intact; the coronary arteries had a right dominant circulation and were free of atherosclerosis. The LAD coronary artery contained a recent thrombus obstructing a 1-cm segment of the vessel immediately proximal to the point where the artery pierced the myocardium to follow an intramural course (Figs 1, 2). This myocardial bridge of the LAD coronary artery extended for a distance of 30 mm and had a maximum depth of 4 mm. Distal to the thrombus, there was an acute transmural anteroseptal myocardial infarction (Fig 3). The remainder of the myocardium, as well as the valves, showed no cellular or vascular rejection or other abnormalities.

Discussion

Myocardial bridges were first described in the modern pathologic literature in 1922. They are congenital aberrancies of the coronary artery, usually involving the LAD coronary

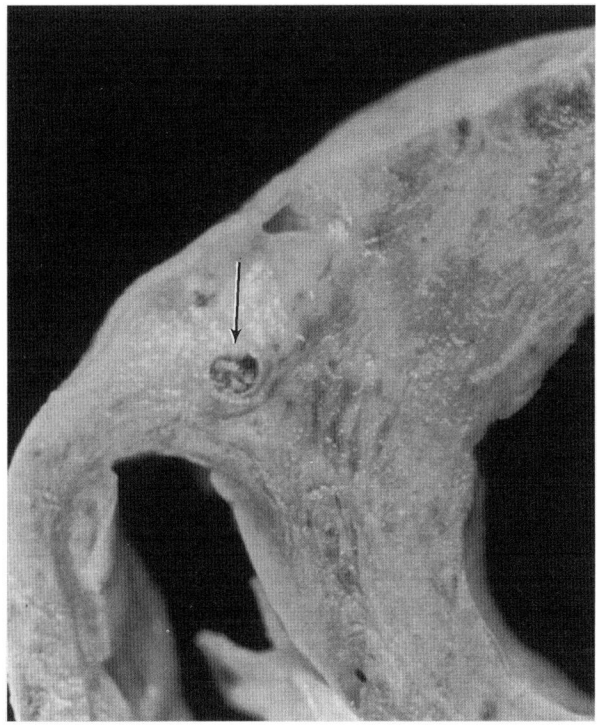

Figure 1. Gross photograph of the anteroseptal portion of the ventricle showing a recent thrombus (arrow) occluding the LAD coronary artery.

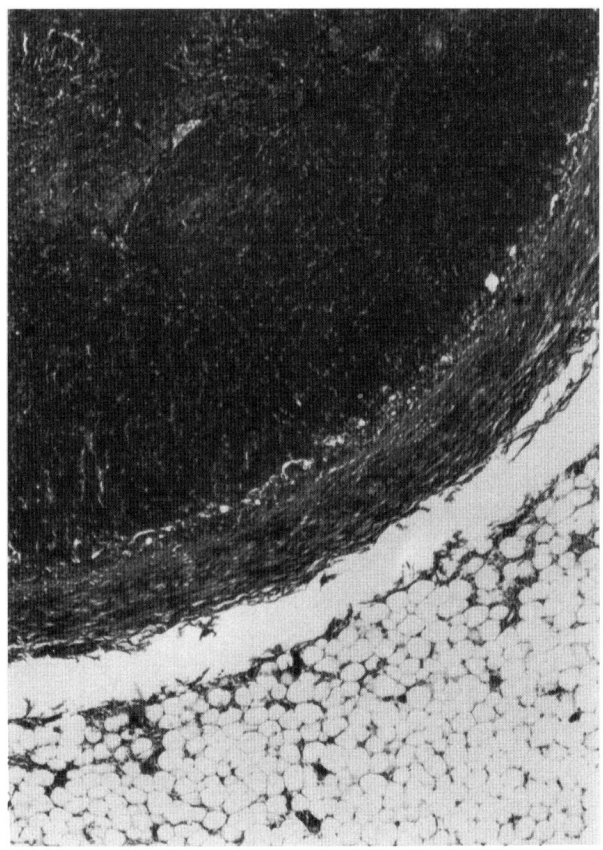

Figure 2. Photomicrograph of the thrombus and segment of LAD coronary artery wall. Early fibrin deposition distinguishes this thrombus from a postmortem clot.

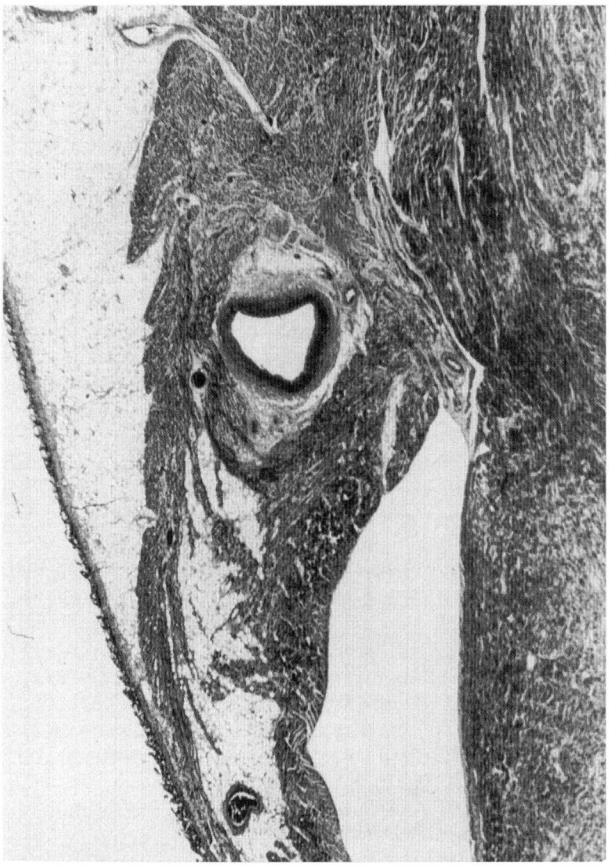

FIGURE 3. Photomicrograph of the anterior portion of the ventricular septum and right anterior ventricular wall. A structurally normal left anterior coronary artery is bridged by the myocardium. The thrombus shown in the previous two illustrations was located immediately proximal to this bridge (Masson's trichrome stain, original ×10).

artery. The incidence of myocardial bridges is variable but is higher in autopsy series than in angiographic series.

Several pharmacologic agents augment the degree of myocardial bridge constriction observed during angiography; these include nitroglycerin, epinephrine, and isoproterenol. With rapid arterial pacing, chest pain, and ST segment depression as well as abnormal coronary sinus lactate extraction has been found in patients with angiographically documented myocardial bridges. Although rarely performed, in severe cases supraarterial myotomy has been shown to reverse wall motion abnormalities and improves diastolic coronary blood flow.

In our patient, the hyperadrenergic postoperative state, as well as the use of high doses of sympathomimetic agents, might have augmented the systolic arterial narrowing produced by the myocardial bridge. Likewise, sinus tachycardia might further compromise diastolic coronary blood flow. The postoperative hypercoagulable state might have promoted thrombus formation in a segment proximal to the myocardial bridge.

Conditions that result in early graft failure include hyperacute rejection, inadequate organ preservation, or prolonged ischemic time, increased pulmonary vascular resistance with failure of the right side of the heart, and mechanical problems such as pulmonary artery torsion.[7]

To our knowledge, this is the first case report of an occlusive coronary thrombus forming in the region of a myocardial bridge leading to massive infarction and systolic failure after cardiac transplantation.

Immediate coronary angiography should be considered in the evaluation of patients with perioperative acute pump dysfunction following orthotopic cardiac transplantation. Early identification of an occlusive myocardial bridge may allow for revascularization and minimize left ventricular damage.

REFERENCES

1 Kramer JR, Kitazume H, Proudfit WL, et al. Clinical significance of isolated coronary bridges: benign and frequent condition involving the left anterior descending coronary artery. Am Heart J 1982; 103:283-88

2 Morales AR, Romanelli R, Boucek RJ. The mural left anterior descending coronary artery, strenuous exercise and sudden death. Circulation 1980; 62:230-37

3 Vasan RS, Bahl VK, Rajani M. Myocardial infarction associated with a myocardial bridge. Int J Cardiol 1989; 25:240-41

4 VanBrussel BL, VanTellingen C, Ernst SMPG, et al. Myocardial bridging: a cause of myocardial infarction? Int J Cardiol 1984; 6:78-82

5 Ciampricotti R, El Gamal M. Vasospastic coronary occlusion associated with a myocardial bridge. Cathet Cardiovasc Diag 1988; 14:118-20

6 Morales AR, Romanelli R, Tate LG, et al. Intramural left anterior descending coronary artery: significance of the depth of the muscular tunnel. Hum Pathol 1993; 24:693-701

7 de Marchena E, Futterman L, Wozniak P, et al. Pulmonary artery torsion: a potentially lethal complication following orthotopic cardiac transplantation. J Heart Transp 1989; 8:499-502

Dextran Syndrome*

Acute Hypotension, Noncardiogenic Pulmonary Edema, Anemia, and Coagulopathy Following Hysteroscopic Surgery Using 32% Dextran 70

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Dextran solutions are favored distending media for many hysteroscopic procedures because they are easy to administer, distribute uniformly within the uterine cavity, and are relatively nontoxic. We present the case of a 26-year-old woman who developed hypotension, noncardiogenic pulmonary

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