

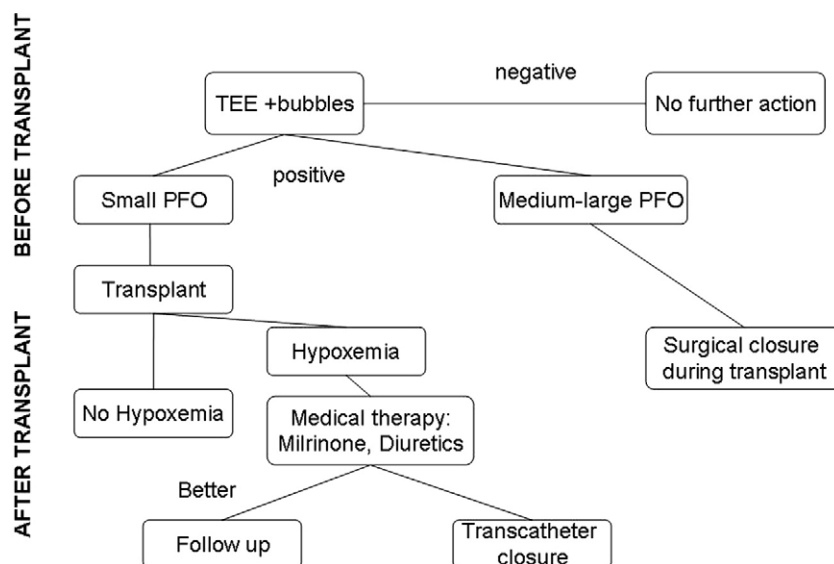


Figure 1. Management of PFO before and after heart transplantation.

sometimes be missed during this evaluation, suggesting that routine transesophageal echocardiography with a saline bubbles injection test may be useful in avoiding misdiagnosis. Treatment includes medical therapy with milrinone, diuretics and nitric oxide and emergent surgical closure² and transcatheter closure.³ Indications for a correct management of this clinical condition are still lacking, due to the anecdotal nature of the existing reports. We propose a simple algorithm (Figure 1) for diagnosis and therapy of patent foramen ovale before and after heart transplantation, based on the literature review and our own experience in managing PFO patients.^{4,5}

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ANAPHYLAXIS FROM APROTININ RE-EXPOSURE DURING HEART TRANSPLANTATION

To the Editor:

Aprotinin has been shown to reduce bleeding in open-heart surgery, including orthotopic heart transplantation (OHT), by providing an anti-fibrinolytic effect, preserving platelet function, and attenuating the inflammatory response to cardiopulmonary bypass (CPB). Aprotinin has been associated with hypersensitivity reactions, including anaphylaxis, but rarely results in death.¹

A 54-year-old man underwent coronary artery bypass surgery (CABG) in 1996 and again in 2005 when he received aprotinin. Seventeen months after his last CABG, he underwent OHT for end-stage heart failure. During induction of anesthesia the patient was hemodynamically stable. He developed acute desaturation, hypotension, bronchospasm and a diffuse erythematous rash after receiving a test dose of aprotinin (10,000 U or 1 ml). An anaphylactic reaction was suspected and immediate resuscitation was initiated with intravenous

hydrocortisone, adrenaline, fluids and vasopressors. Hypotension persisted and emergent femorofemoral CPB was instituted, followed by OHT. He developed coagulopathy requiring massive transfusions of blood products, protamin, desmopressin and activated Factor VII. Initially, the patient was weaned from CPB with good biventricular function. However, persistent pulmonary edema and the development of right-side failure required an extracorporeal membrane oxygenation device. Despite these aggressive measures, the patient remained vasoplegic, coagulopathic, developed ischemic leg and renal failure. He eventually died on Day 3 from multi-organ failure.

It has been demonstrated that there is a time-dependent decrease of IgG aprotinin-specific antibodies. Scheule et al demonstrated that 45% and 32% of patients were IgG-positive at median intervals of 3.7 and 13.3 months, respectively, after previous local and/or intravenous aprotinin exposure.²

A retrospective analysis of 240 patients re-exposed to aprotinin at a mean of 344 days after the first exposure revealed 7 adverse hypersensitivity reactions (2.8%).³ An interval between exposures of <6 months resulted in a significantly higher incidence of hypersensitivity reactions than in patients with a longer interval of re-exposure (4.5% vs 1.5%, $p < 0.05$).

Aprotinin is widely used in cardiac surgery, and anaphylactic reactions have been well reported. It is unclear whether pre-operative testing of IgG and IgE levels are of any value in predicting anaphylaxis.⁴

Serious concerns were raised in our fatal case of anaphylactic reaction to aprotinin, despite the remote exposure and administration of only the test dose. This leads us to conclude that aprotinin should not be given to patients with prior exposure.

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RESOLUTION OF PROTEIN-LOSING ENTEROPATHY AFTER SUCCESSFUL HEART TRANSPLANTATION IN AN ADULT PATIENT WITH ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY (ARVC)

To the Editor:

Protein-losing enteropathy (PLE) is the pathologic loss of plasma protein across the intestinal mucosa. The diagnosis of PLE is based on evidence of elevated α 1-anti-trypsin (A1AT) clearance and/or raised random fecal concentration along with serum hypoalbuminemia.¹ This condition may be caused by gastrointestinal, metabolic or cardiac diseases. The largest group of patients described with PLE resulting from a cardiac etiology are those treated with extracardiac or cavopulmonary anastomosis (Fontan procedure) for palliation of a univentricular heart. Heart transplantation in many pediatric cases has been reported to be successful in reversing the effects of PLE.²

A 62-year-old man with end-stage ARVC was referred to our tertiary heart failure and transplant unit for heart transplant assessment. At the time of referral in April 2006, he had developed biventricular heart failure with an echocardiogram revealing severely impaired right heart function, severe tricuspid regurgitation and mild left ventricle (LV) dilation with an LV ejection fraction estimated at 39% (normal >55%). He required frequent admissions for intravenous diuretic therapy. His severe right heart failure was complicated by PLE with a random fecal A1AT of 47.8 mg/g dry weight (normal: <1.2 mg/g) and consequent severe hypoalbuminemia (16 to 20 g/liter, normal: 35 to 50 g/liter).

In the course of transplant assessment, other causes of PLE and hypoalbuminemia were carefully excluded with an extensive gastrointestinal evaluation. He received fortnightly infusions of 100 ml of 20% albumin in an effort to maintain his albumin at >20 g/liter. His renal function was mildly impaired with serum creatinine of 123 μ mol/liter (normal: 70 to 120 μ mol/liter) and a glomerular filtration rate (GFR) of 55 ml/min/1.73 m² in the pre-transplant setting. He underwent orthotopic heart transplanta-