Cardiac MRI in the isolated porcine heart reveals possible etiology of sudden right heart failure following heart transplantation

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Abstract

Occurrence of immediate post-transplant heart failure in the cardiac transplant recipient is typically attributed to elevated pulmonary vascular resistance, however other etiologies may play a role. At the completion of the transplant, free air, which has collected in the donor heart, is vented via an aortotomy. Free air may rise into the right coronary artery and obstruct reperfusion of the right ventricle. Cardiac perfusion MRI may offer a method of non-invasively determining the presence of air embolus. The objectives in the pilot study were to identify steps in the donor process where free air could enter into the aortic root causing obstruction of perfusion of the coronary arteries. A change in surgical technique could then be used to eliminate a portal of entry and cardiac perfusion MRI could validate the technique. Standard cardiectomy was compared to a variation in technique in two animals. Pulmonary vein ligation was completed in the experimental model before completion of cardiectomy. Both hearts were isolated and imaged using T1-weighted FLASH sequence and gadolinium contrast via the aortic root. Cardiac perfusion MRI imaging of the heart with the unligated pulmonary vein revealed evidence of air embolus and no perfusion of the right coronary artery compared to the ligated heart. Anatomically, the right coronary artery is anterior compared to the left coronary artery; and therefore we suspected that right ventricle complications were due to air emboli preferentially migrating to the right coronary artery. For our purposes, cardiac MRI was well suited to evaluate the donor heart because it allows assessment of perfusion before transplantation and may be used following transplantation in the clinical setting to assess not only perfusion, but also viability, and structure. With a high degree of specificity and sensitivity it replaces the functions of ultrasound, nuclear scanning, and computed tomography within one and the same setting. Furthermore, due to the absence of any toxic effects of standard gadolinium based MRI contrast agents; the

Introduction

This feasibility study was conducted to identify the origin of air embolus in porcine isolated hearts using cardiac MRI. Surgical steps in cardiectomy of the donor heart allowed our donor heart to collect air. As a result, air embolized to the right coronary artery created an obstruction to perfusion. Anatomically, the right coronary artery is anterior compared with the left coronary artery; and therefore we suspected that right ventricle complications were due to air emboli preferentially
pre-operative assessment by contrast-enhanced MRI would not compromise the condition of the donor heart [1].

Since the first cardiac transplant, complications with air embolus during cardiac transplantation have been more than a nuisance. Air emboli from cardiac surgery are traditionally associated with CNS stroke following open heart surgery. On the other hand, the etiology of right heart failure following transplantation has not always been easy to identify. Right heart failure has typically been associated with elevated pulmonary vascular resistance [2, 3]. Ultrasound, angiography, and pulmonary artery catheterization may not always identify the origin of right heart failure and fall short of the diagnostic ability of cardiac MRI and radionuclide ventriculography [4].

According to the registry from 1990 to 1999 Cardiac Transplant Research Database, early graft failure is the primary cause of mortality following transplantation, leading ahead of rejection, infection, neurologic, and other causes [5]. Early graft failure is a vague description of mortality due to relatively unknown causes and attributed to surgically related complications. However, a recent autopsy study identified right-sided heart failure as the primary cause of early death following transplantation [6]. This study highlights the importance of identifying the cause right heart failure leading to early graft failure. In our laboratory, we noticed that there may be a relation between air entry into the heart and air embolism to the anterior right coronary artery. Because of this relationship we set up an experiment to test our hypothesis.

Methods

Two adult domestic Yorkshire cross swine weighing 60–65 kg were used in the experiments A and B. The Institutional Animal Care and Use Committee a division of the Research Subjects’ Protection Program approved the two class B pigs for the completion of the experiments under a training protocol. All animals received humane care in compliance with the Guide for the Care and Use of Laboratory Animals formulated by the National Academy of Sciences (National Academy Press, 2101 Constitution Ave, NW, Washington, DC 20418, ISBN 0-309-053777-3).

The animals were brought to the pre-op area and induced with a combination of xylazine (500 mg), and Telazole (4 mg/kg) administered intramuscularly for anesthesia. Each animal was endotrachally intubated and ventilated with an ambu-bag. The animal’s thorax and abdomen was shaved and an intravenous line was established for fluid maintenance. The animals were then transported to the operating room. In the operating room, inhalational isoflurane (1.5–2.0 vol% in 41 O2) and supplemental 2:1 air and oxygen mixture was provided with mechanical ventilation to maintain an anesthetic depth of 1–1.3 MAC. Muscle relaxation was maintained with pancuronium bromide (0.1 mg/kg) administered intravenously. A midline incision was made and sternotomy completed. The innominate artery, and superior vena cava were dissected with a Metzenbaum scissors. The inferior vena cava was bluntly dissected free with a right angle. Umbilical tape was looped around the inferior and superior vena cava. Dissection was made in the plane between the pulmonary artery and the aorta. Heparin was administered (350 units/kg) and circulated for 5 min. Each heart was arrested with Beuthansia (1 ml/10 lbs.).

The pulmonary veins were then looped and ligated one animal (experiment A). Exsanguination was achieved by an inferior vena cava and pulmonary vein venotomies. The aorta was transected at the arch. The pulmonary artery, pulmonary veins, inferior and superior vena were transected and the heart was completely excised. The heart then was submerged in an ice/saline solution and the aorta was cannulated using a 3/8 in. adapter and cinched with a zip tie. Each heart was flushed with 1 l of Plegisol® at 245 ml/min using a peristaltic pump (Cardiovascular Instruments, Wakefield, MA) via the aortic root cannula as shown in Figure 1. The hearts were placed into an ice chest (Coleman, Wichita, KS) at 5–8 °C for 6 h 20 min (Experiment A) or 7 h (Experiment B) and subsequent images were acquired.

Image acquisition was performed in a 1.5 T system (Magnetom Sonata, Siemens Medical Systems, Islen, NJ) using a four-element phased array coil, with two anterior and two posterior coil