



Manifestations of Atrial Tamponade in a Patient Implanted with SynCardia Total Artificial Heart

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Abstract

Implantation rates of the SynCardia Total Artificial Heart (TAH) system is growing owing to the increasing incidence of biventricular heart failure and the limited number of transplantable hearts. Among the complications associated with the device, atrial tamponade is an emergent condition that requires immediate attention. Atrial tamponade occurs when increasing pericardial hemorrhage leads to atrial compression that produces tamponade physiology and compromises cardiac output. Unfortunately to our knowledge, information concerning the early manifestations of tamponade in TAH patients is extremely scarce. Previous reports have focused on decreased cardiac output as an indicator of tamponade. However, other presentations including pulmonary decompensation are largely undescribed. This case provides a valuable account of the early manifestations of atrial tamponade observed prior to diminished cardiac output.

Key words: atrial tamponade; total artificial heart



Background

Congestive heart failure is a serious cause of morbidity and mortality that affects 1 to 2% of the developed world and 23 million people worldwide.^{1,2,3} Initial modalities of treating heart failure are focused primarily on pharmacological therapy.² However, the disease process is generally progressive and leads to worsening heart failure with symptoms refractory to medical management.² The growing advanced heart failure population has generated a need for additional therapies. Cardiac transplantation has been utilized as a definitive form of therapy for many years.^{1,2} Unfortunately, the number of available donor hearts is limited and the population of eligible transplant candidates is consistently rising.^{2,3} As a result, implantable devices have emerged as a viable option for severe biventricular heart failure. These devices have served as a bridge to cardiac transplantation and helped decrease the annual mortality of patients awaiting donor hearts.^{2,3} Devices have also been implanted in patients as destination therapy.² Currently, ventricular assist devices (VADs) and artificial hearts are the primary available options.^{2,3}

The SynCardia Total Artificial Heart (TAH) is the only implantable device approved by the United States Food and Drug Administration (FDA) for use in patients suffering from severe biventricular failure.⁴ The device functions by replacing the native ventricles with synthetic chambers that maintain pulsatile blood flow.⁴ Each synthetic chamber is composed of an air diaphragm and blood diaphragm separated by two dividing diaphragms. The inflation and deflation of the air chamber via the external console facilitates the emptying and filling of the blood diaphragm. This orchestrated activity functions to maintain pulsatile flow. The blood enters the ventricle from the atria passively via an inflow connector. Next, the blood exits the ventricle with inflation of the air diaphragm through the outflow connector. The outflow tracts attach to the pulmonary artery or aorta.⁴ Mechanical valves mounted within the inflow and outflow connectors maintain unidirectional flow. An external console delivers power and compressed air to the device through a driveline tunneled out of the anterior chest. The console monitors device function and initiates preprogrammed alarms during any malfunction.⁴

Among the possible complications arising from the use of the TAH, atrial tamponade is especially serious. It is a rare but potentially fatal occurrence in this fragile population.^{4,5} Prompt identification and intervention is crucial to prevent hemodynamic collapse.^{4,5,6} The SynCardia system has intrinsic alarms that sound in response to decreased cardiac output states, such as tamponade. Although these alarms are valuable tools to the clinician, they are not a substitute for clinical acumen. Literature evaluating the early manifestations of this phenomenon in patients implanted with TAHs is lacking. We present a case of atrial tamponade after TAH placement to emphasize the early clinical expression of this disease process and stress the value of clinical judgment.



Case Presentation

A 61-year-old man was transferred to our center with biventricular failure refractory to medical management. Upon arrival, the patient was alert, hypotensive (blood pressure 70/40 mmHg), tachycardic (110 beats per minute), tachypneic (25 respirations per minute), and hypoxic (oxygen saturation below 88% breathing room air). The patient required support with dopamine, neosynephrine, and oxygen supplementation. He had endured six months of worsening shortness of breath and pitting edema despite increased medical therapy. His medical history included obesity, diabetes mellitus, hyperlipidemia, hypertension, coronary artery bypass graft surgery, and automatic implantable cardioverter defibrillator placement for a depressed ejection fraction. There was no family or social history related to his severe heart failure.

Due to his decompensated state and need for cardiac support, a TandemHeart (CardiacAssist Inc.) peripheral ventricular assist device was utilized as a bridge to decision. The device facilitated weaning of vasopressor support and hemodynamic stabilization. Unfortunately, the patient could not be taken off of TandemHeart support as a result of biventricular failure. Furthermore, he did not meet eligibility for cardiac transplantation owing to worsening multi-organ system failure. The patient needed a bridge to facilitate clinical improvement before reevaluation for transplant candidacy. Twelve days after presentation, the patient underwent explantation of the TandemHeart and implantation with the SynCardia TAH that was intended as a bridge to definitive therapy.

The SynCardia device was implanted with a total cardiopulmonary bypass time of 178 minutes and an aortic crossclamp time of 148 minutes. His estimated blood loss was one liter and required transfusion with four units of packed red blood cells, three “jumbo” packs (400 mL each) of fresh frozen plasma, and one jumbo pack of platelets. The procedure was free of major complications and the patient was extubated 24 after surgical closure. His postoperative chest radiograph showed the SynCardia device in place with no pneumonic process (Figure 1). His chest tubes were removed four days after closure when the drainage significantly decreased and the air leaks had resolved. Additionally, lab values noted leukocytosis and anemia that was presumed to be the result of the extensive surgery and were appropriately managed. Improving the patient’s strength and ambulation became the focus of his postimplantation course.

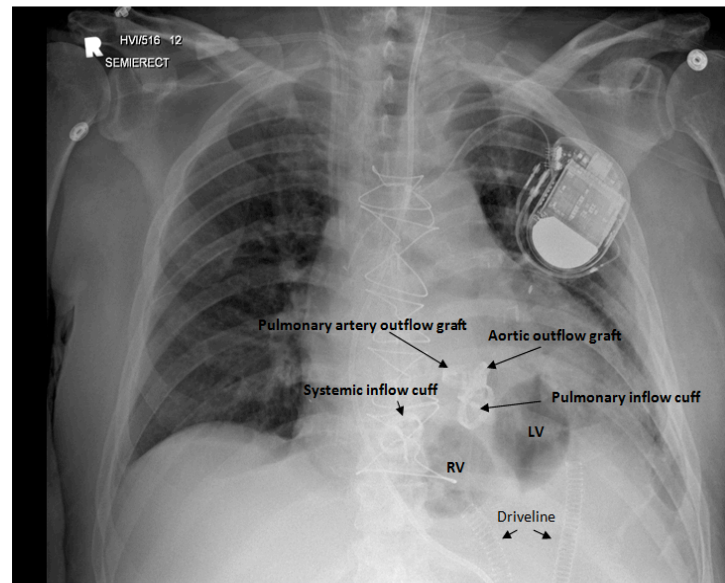


Figure 1: Chest radiograph post-operative day 3 after extubation.

Twelve days after device placement, the patient was noted to be pale, lethargic, and diaphoretic. No elevation in temperature was observed. Combined with these symptoms, the patient expressed feelings of clinical deterioration and increased shortness of breath. Assessment of vital signs revealed hypotension with a mean arterial pressure (MAP) of 45 mmHg and tachypnea with 25 respirations per minute. Laboratory evaluation revealed a lactate elevation suggesting insufficient end organ perfusion (Figure 2) and decreased hemoglobin concentrations, (Figure 3). However, the SynCardia module reported no alarms or reduction in cardiac output overnight or during the early morning. Because the clinical management team was concerned that there may have been an intrathoracic etiology for his acute clinical change, a chest radiograph was ordered. Alarms noting decreased fill volume and cardiac output from the SynCardia console were noted only after the patient clinically worsened and the chest radiograph was obtained. A new left pleural effusion (Figure 4) was identified, which prompted emergency chest tube placement and drainage of 700 milliliters of serosanguinous fluid. A computer tomography (CT) scan of the chest was ordered for further investigation into the cause of the pleural effusion. The CT scan revealed an aortic root graft aneurysm of 43 millimeters and increased complex mediastinal fluid with areas of attenuation indicative of possible hemorrhage and tamponade (Figures 6 and 7). A transesophageal echocardiogram was also obtained and confirmed the presence of atrial tamponade and noted left atrial compression (Figures 8 and 9).

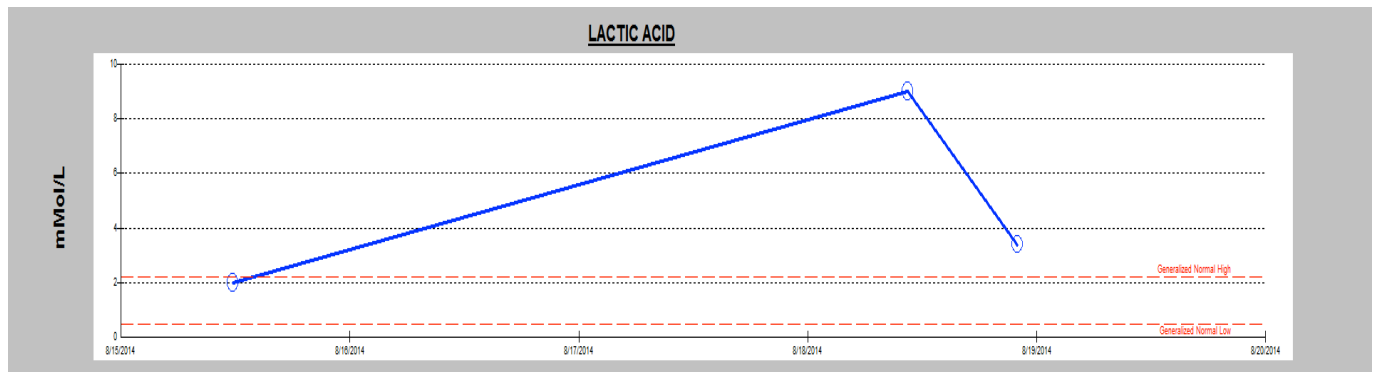


Figure 2: Trend of lactic acidosis on the day of atrial tamponade (8/18/14).

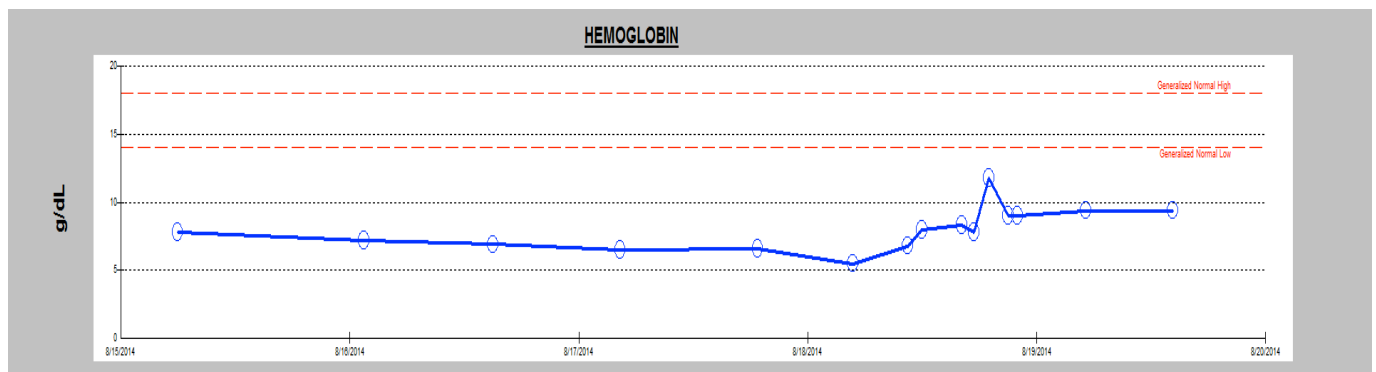


Figure 3: Trend of hemoglobin on the day of atrial tamponade (8/18/14).

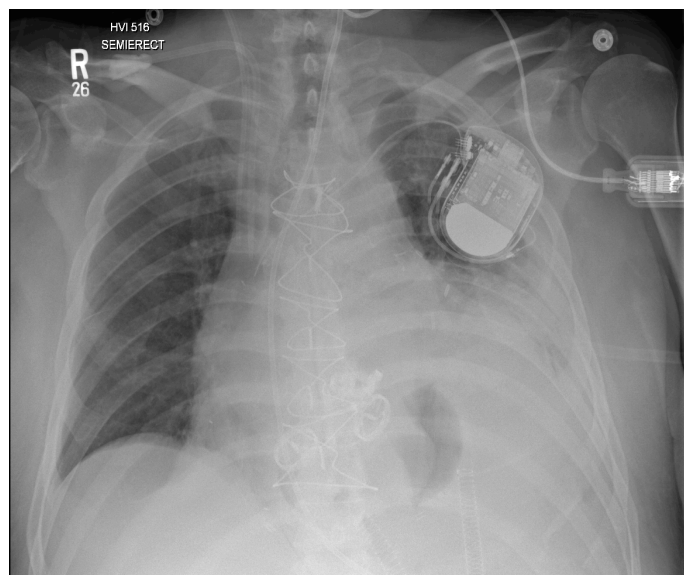


Figure 4: Portable chest radiograph of the patient on the day of atrial tamponade (8/18/14).



Figure 5: Syncardia console measurements on day of atrial tamponade.

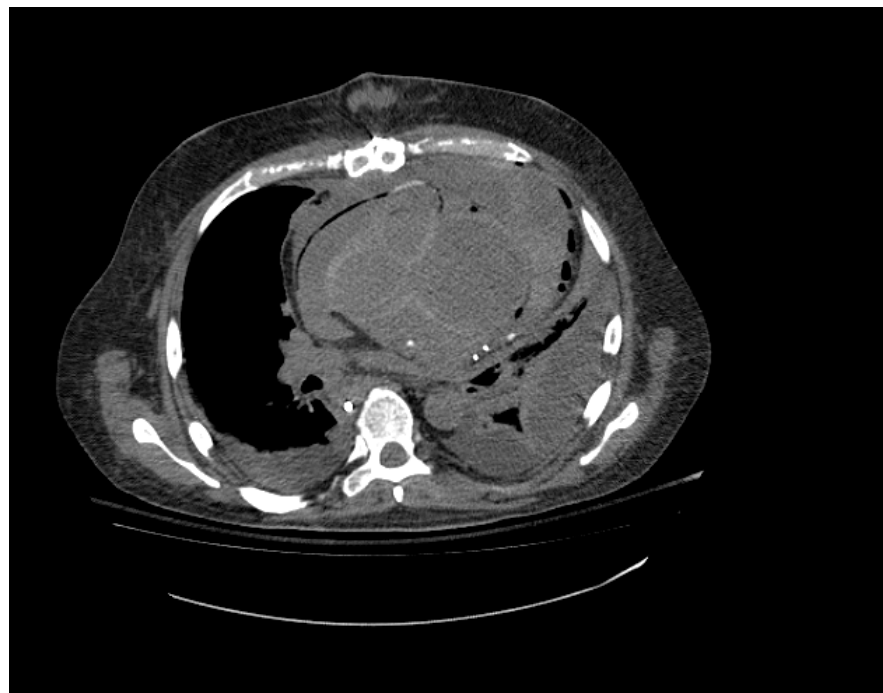


Figure 6. Computed tomography scan showing hemorrhage and pericardial effusion in transverse plane.



Figure 7. Computed tomography scan showing hemorrhage and pericardial effusion in coronal plane.

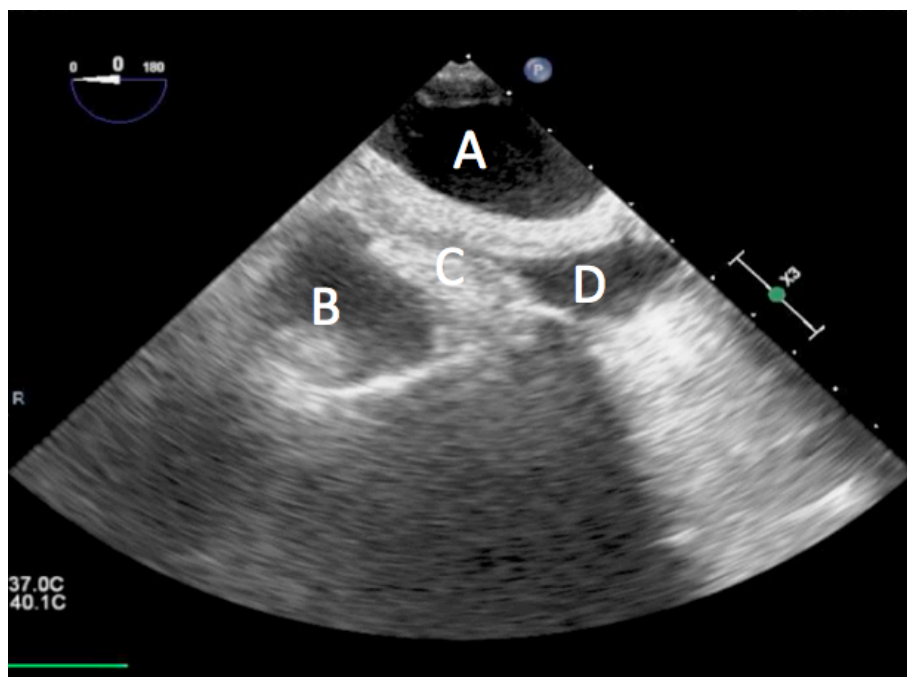




Figure 8. Trasesophageal echocardiogram showing left atrial compression from atrial tamponade. A: Blood in the pericardium. B: Right atria. C: Compressed left atrium. D: Pulmonary vein.

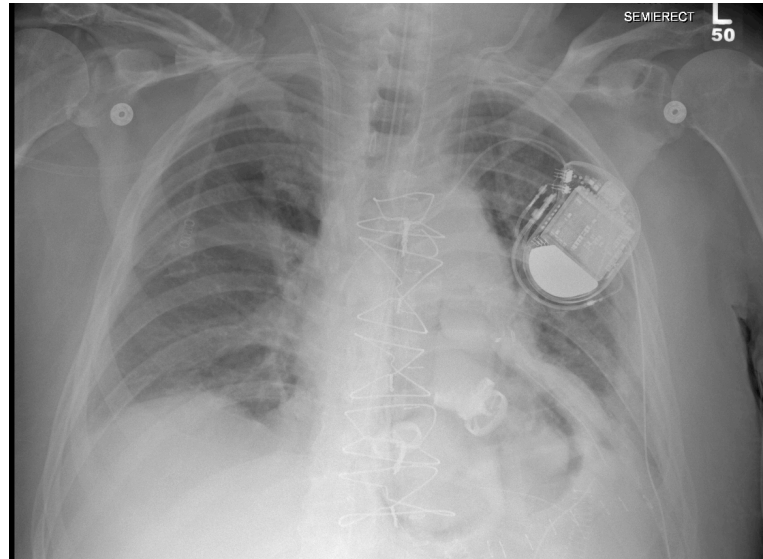


Figure 9. Chest radiograph after surgical evacuation of pericardial effusion and control of intrathoracic bleeding.

The patient was promptly taken to the operating theater for evacuation of any pericardial effusion. Blunt dissection identified the effusion and prompted surgical release of the large hemorrhagic collection. Multiple pericardial clots were removed, revealing evidence of ongoing bleeding from the aortic suture line to the SynCardia device. This area was noted to be oozing in several places on the posterior aspect of the heart. Bleeding was controlled with several interrupted sutures and the aortic graft was collared with a second Dacron graft. This collaring approach served to create two bands around the distal aorto-graft anastomosis. The patient tolerated the procedure well with resolution of atrial tamponade and pleural effusion.

Discussion

SynCardia TAH use in patients suffering from severe biventricular failure has steadily increased.^{7,8,9} Atrial tamponade is a serious complication that inhibits cardiac output by preventing atrial filling. The lack of ventricular preload due to atrial collapse results in circulatory compromise. This condition requires immediate identification and intervention to prevent catastrophic ramifications.^{10,11} In the event of tamponade inducing cardiac arrest in patients implanted with a TAH, the primary means of stabilization requires surgical evacuation of the pericardial effusion. Chest compressions will not benefit patients with atrial tamponade in the presence of a TAH since external force will not compress the artificial ventricle.⁴ Literature concerning the early manifestations of tamponade in TAH implanted patients is largely absent. Previous reports have indicated that decreased cardiac output is the primary



symptom^{4,5,6} Our case report demonstrates the immense value of physical exam and clinical acumen in identifying and treating this complication. Tachycardia, hypotension, tachypnea, and increased difficulty breathing were the first symptoms present in our patient. These findings launched further investigations before console alarms noted decreased cardiac output. The early identification of thoracic hemorrhage that resulted in a left pleural effusion and atrial tamponade allowed our team to intervene and maximize the potential for recovery. The evolution of a graft leak to cardiac compromise suggests that there is a spectrum of clinical manifestations that may facilitate early tamponade identification. Even with the implementation of cutting-edge technology, the fundamentals of good clinical practice are invaluable assets in optimizing patient care. This case reports early manifestations of atrial tamponade in the presence of SynCardia TAH to offer a clinical experience missing in current literature.

Learning points

- Atrial tamponade in patients with total artificial hearts can be lethal.
- Prompt identification and surgical evacuation decrease morbidity and mortality.
- Clinical signs of acute decompensation may precede device alarms.
- Physical evaluation of the patient cannot be substituted by technological applications.

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