Journal of Athletic Training 2023;58(2):153–155
doi: 10.4085/1062-6050-0458.21

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Return to Sport From Viral Myocarditis in a Previously Healthy Collegiate Athlete: A Case Report

Abigail Wirtz, MS, LAT, ATC

Athletics and Recreation Department, Endicott College, Beverly, MA

The objective of this paper is to present the case of a healthy, 19-year-old female collegiate soccer player who developed acute pulmonary edema and acute heart failure in the recovery room after hip labral arthroscopic surgery. The patient's initial diagnosis, of negative pressure pulmonary edema in direct relation to extubation, was questioned when she became hemodynamically unstable. A cardiac biopsy revealed acute pulmonary edema and heart failure secondary to viral myocarditis. The patient was treated and discharged 10 days after admission. Specific and substantiated return-to-play guidelines after a cardiac event, specifically viral myocarditis, have been sparse. The interprofessional collaboration between athletic trainers and cardiologists is a key dynamic in the clinical decision-making process of a safe return to competitive athletic participation after a cardiac event.

Key Words: heart failure, cardiac rehabilitation, competitive sports

Key Points

- Viral myocarditis is linked to common "cold" viruses: enterovirus, adenovirus, and influenza.
- Cardiovascular complications from viral myocarditis may occur more frequently in the young, athletic population.
- Specific return-to-exercise protocols need to be created for each cardiac anomaly and cardiac event.
- Completion of an outpatient cardiac rehabilitation program should be considered, regardless of the patient's age and comorbidities, to allow for a safe return to competitive sport.

Viral myocarditis is an inflammation of the myocardium resulting from the invasion of a virus and its subsequent replication, leading to myocardial cytotoxicity and necrosis. Progression of the virus can result in dilated cardiomyopathy and cardiac dysfunction. Adenovirus and enterovirus are the leading causes of myocarditis in most cases.[1] Influenza viruses are another common cause, and cardiovascular complications are the second most frequent cause of death due to influenza.[2] Epidemiologic studies revealed that 90% of the population will become infected with one of these viruses in their lifetime[1]; however, as few as 5% may display clinical cardiac symptoms,[3] which include fatigue, dyspnea, palpitations, chest pain, and tachycardia.

Many cardiac conditions can appear in the young athletic population. Some conditions may be benign when recognized promptly and treated appropriately with the goal of preventing a catastrophic event. When a cardiac event does occur, the medical team must use an evidence-based progression for an athlete's safe return to sport. The lack of sound research outlining a safe, graded return-to-sport protocol is unsettling. The purpose of this paper was to present the case of a healthy collegiate soccer player who developed viral myocarditis unbeknown to her, her athletic trainer, or orthopaedic surgeon and to discuss the need for specific, research-based return-to-play protocols after a cardiac event.

Case Presentation

A 19-year-old White female soccer player underwent hip arthroscopy for a labral tear at an ambulatory surgery center. The patient was 1.65 m (5.4 ft) tall and weighed 72.5 kg (159.8 pounds), with a body mass index of 26.6 at the time of surgery. Her medical history was unremarkable. Specifically, she denied any history of cardiomyopathy, congestive heart failure, or valvular disease. The patient had not undergone any previous surgeries and had no experience with anesthesia.

No surgical complications were noted, and the patient was extubated in the operating room without evidence of laryngospasm. In the recovery room, she presented with dyspnea, labored breathing, and a productive cough of pink, frothy sputum; the oxygen saturation level was 85% with room air. The patient denied chest pain or palpitations, however. She was initially treated with 10 mg of furosemide and 100% oxygen via a nonrebreather mask. She was transferred to the emergency department to be evaluated and treated for pulmonary edema.

In the emergency department, the patient presented with a blood pressure of 73/50 mm Hg and a heart rate of 110 beats/minute. Her oxygen saturation had improved to 97% while en route to the hospital. A chest radiograph revealed bilateral, hazy, acinar opacification of the lungs, consistent with a clinical presentation of acute interstitial alveolar edema.[4] The patient's electrocardiogram demonstrated normal sinus rhythm at 91 beats/minute. Her arterial blood gas levels at the time were pH = 7.48, partial pressure of oxygen = 144 mm Hg, partial pressure of carbon dioxide = 25 mm Hg, serum bicarbonate = 19 mmol/L, and base = –3. Given the acute presentation and initial testing, her

diagnosis was negative pressure pulmonary edema related to extubation.

The presence of elevated troponin T levels in the patient's bloodwork initiated a more thorough evaluation from a cardiologist. A 2-dimensional transthoracic echocardiogram was performed, revealing a dilated left ventricle (LV), severely depressed LV systolic function with an ejection fraction of 25% to 30%, and trace mitral regurgitation. Endomyocardial biopsy is recommended for patients with the acute onset of heart failure, a dilated LV, and hemodynamic instability.[1] The biopsy revealed viral myocarditis.

The patient was prescribed dopamine and furosemide, which were administered concurrently to target hypotension and fluid overload, respectively. She responded well to this therapy, with her systolic blood pressure rising to 82 mm Hg 2 minutes after the administration of dopamine; her blood pressure rose to 100/55 mm Hg during the course of treatment. Bilevel positive airway pressure therapy was administered for additional supportive care to correct her arterial blood gas levels and improve dyspnea. The patient was ultimately transferred to a tertiary care center via helicopter. She was admitted to the intensive care unit for 10 days and received continuing cardiovascular treatment before being discharged.

The patient continued to have follow-up testing and appointments with her cardiologist, including repeat transthoracic echocardiogram, cardiac magnetic resonance imaging, bloodwork, and 3 months of monitoring via Holter monitor. Her ejection fraction normalized to 55% to 60% approximately 9 months after the initial event, permitting full clearance for competitive sports with no activity restriction**.** The patient returned for preseason camp, consisting of double sessions and team lifts, and stated she had been running during the summer months but not training specifically for soccer. The athletic training staff devised a plan to gradually return her to competitive soccer over a 3-week period, as the cardiologist provided no structured plan or algorithm. With a goal of not exceeding 75% of maximal exertion, the first week began with noncontact, technical soccer drills only and supervised conditioning on the sidelines by the athletic trainer. Week 2 continued with technical drills at 85% of maximal exertion with the addition of contact drills at 50% of maximal exertion and ongoing supervised conditioning. The goal for week 3 was 1 full practice participation each day at 75% of maximal exertion in contact drills. Exercise intensity was determined using the maximal heart rate formula (220 − age) and a heart rate monitoring device. The patient also used the rating of perceived exertion (RPE) scale as she often forgot her heart rate monitor.

During this period of training, she reported episodes of fatigue, shortness of breath, and dizziness. Unsure whether these symptoms were related to cardiovascular involvement or deconditioning, the athletic training staff was uncomfortable continuing her training or providing guidance without specific instructions from the cardiologist. The patient was questioned by her cardiologist at this time regarding her hydration status and if she was experiencing palpitations. She was told not to participate in "heavy" practices or games moving forward but was not provided with any concrete instructions defining "heavy" exercise.

Despite a slower progression and almost a year since the cardiac event, the patient continued to be hesitant to overexert herself on the field. She participated in practices and lifts sparsely or at a lower intensity than the rest of the team and did not play in competitions for the duration of the season. The patient ultimately returned to full participation the following season, nearly 2 years after the initial cardiac event.

DISCUSSION

With the high prevalence of adenovirus, enterovirus, and influenza on college campuses nationwide, it could be argued that viral myocarditis and the associated cardiac complications may occur more frequently in collegiate athletes than previously acknowledged. Having established protocols in place for a safe return to competitive sport after a cardiac event is extremely important. Unfortunately, in the areas of sports and cardiology, many clinical decisions are left to expert opinion[5] because specific guidelines are limited, resulting in much uncertainty and anxiety for the health care team responsible for creating and implementing exercise programs. A joint statement from the American Heart Association and American College of Cardiology[6] provided eligibility as well as disqualification guidelines for the clinician and patient when considering a return to competitive sport with cardiovascular abnormalities. However, this document does not outline which competitive sports are safe to resume, nor does the literature offer sound guidance on the progression (eg, mode, intensity, duration, and criteria for advancing stages). For individuals with myocarditis, resumption of competitive sport is allowed when systolic heart function has returned to normal and no arrhythmia is present at rest or with graded exercise.[6] Specific guidelines for a structured, graded return to sport for individuals who have recovered from viral myocarditis, pulmonary edema, or heart failure are not readily available.

Completing a formal outpatient cardiac rehabilitation program is a reasonable approach to a safe, graded, and supervised return to exercise and could also ease any anxiety the patient may be experiencing. However, if a cardiac rehabilitation program is not prescribed by the cardiologist, athletic trainers will need a starting point to safely return the patient to competitive sport.

In systematic reviews,[7,8] researchers have assessed the effects of a structured exercise program on individuals with symptomatic heart failure. Three studies yielded positive results regarding quality of life,[9,10] exercise capacity,[9–11] and recorded clinical events.[9,11] Lang et al[9] and Dalal et al[10] looked at exercise programs involving 3 sessions per week for a 12-week period, during which participants started at their own pace and used the RPE to determine intensity. Improved scores were seen for quality-of-life questionnaires and exercise capacity.[9,10] Reeves et al[11] combined endurance and strength training for a 12-week program in which the participants exercised for three 60-minute sessions per week. The intensity of the exercise was monitored and adjusted using the Borg RPE scale: participants started with low intensity and slowly increased to a rating of *somewhat hard*.[11] The results revealed a therapeutic effect on a timed repeated chair-rise exercise, as well as improvement in the quality-of-life score. Reeves et al[11] also reported a 29% lower rate of all-cause rehospitalizations for the intervention group.

Seo et al[12] conducted a systematic review to assess the effects an exercise-based program had on cardiovascular function and quality of life in individuals with cardiomyop-

athy and LV systolic dysfunction. The studies reviewed followed the FITT principle of frequency, intensity, time, and type and were consistent with general guidelines for healthy adults.[12] Specifically, Mehani[13] used a percentage of heart rate reserve as a measure of intensity and had participants exercising 3 sessions per week, whereas the methods of Holloway et al[14] were consistent with those of other studies in using RPE scales in the 12 to 14 or *somewhat hard* range to measure exercise intensity. In both investigations, functional and quality-of-life scores improved.[13,14] A review by Asplund and O'Connor[15] touched on the psychological readiness of the patient to return to sport after any major adverse medical event, which may further support the recommendation to complete an outpatient cardiac rehabilitation program.

The literature[7–14] showed positive results for patients exercising in the presence of cardiovascular deficits, and it could be argued that these factors would be favorable and a reasonable starting point for patients who have recovered from a cardiac event and are asymptomatic. When communication with the cardiologist is poor and the athletic trainer might not have access to results from a VO_2max test (maximal oxygen uptake), it is encouraging to know that the literature supports the use of RPE scales as an appropriate measure of intensity. To summarize, the literature[7–14] has shown positive outcomes when using the following ranges: 30 to 90 minutes of exercise, 2 to 5 sessions per week, and 40% to 85% of maximal heart rate or an RPE rating of 11 to 18.

Future researchers should investigate return-to-play progressions after a cardiac event to ease the transition to continued and sustained sport-specific exercise and training under the supervision of the patient's athletic trainer, strength and conditioning coach, team coach, and team physician.

Clinical Bottom Line

Athletic trainers bear a great deal of responsibility for not only preventing orthopaedic injuries in athletes but also preventing catastrophic injuries such as cardiac events. When cardiac events do occur in this patient population, athletic trainers need to be provided with a sound, research-based return-to-exercise protocol, including intensity and duration specifications and the criteria to advance stages. In addition, contraindications to exercise and red flags indicating when to discontinue exercise as the patient progresses would offer an appropriate way of adequately monitoring the patient. Interprofessional communication and cooperation among the athletic trainer, cardiologist, primary care physician, strength and conditioning coach, and team coach is paramount for a safe return to exercise.

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Address correspondence to Abigail Wirtz, Athletics and Recreation Department, Endicott College, 376 Hale Street, Beverly, MA 01915. Address email to awirtz@endicott.edu.