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RIGHT CORONARY DISSECTION AND LEFT ANTERIOR DESCENDING THROMBUS: DUAL DILEMMA IN A YOUNG CARDIAC ARREST SURVIVOR

Holly Morgan, BSC, MB BCH, Michael McCann, BSC, MBBS(HONS), Alan Whelan, MBBS, FRACP, and Richard Clugston, MBBS, FRACP

Department of Cardiology, Fiona Stanley Hospital, Perth, Australia
Corresponding Address: Michael McCann, BSC, MBBS(HONS), Department of Cardiology, Fiona Stanley Hospital, 11 Robin Warren Drive, Murdoch, Perth 6150, Australia

□ **Abstract—Background: ST-elevation myocardial infarction (STEMI) leading to cardiac arrest is an exceptionally rare occurrence in young adults. Those affected tend to abuse sympathomimetic drugs, have strong family histories, or have a significant burden of cardiac risk factors. Another uncommon cause of STEMI is coronary artery dissection, which overwhelmingly affects middle- and older-aged women with few cardiac risk factors. Case Report: A 22-year-old athlete with no medical history was admitted to our institution post–cardiac arrest with an anterior STEMI and concomitant right coronary dissection. To our knowledge, this represents the first documented case of these simultaneous pathologies in a young cardiac arrest survivor. Why Should an Emergency Physician Be Aware of This?: Myocardial infarction is rare in young adults, and a diverse range of etiologies must be considered promptly to prevent delays in time-sensitive therapies, such as antiplatelet agents and revascularization. The emergency physician is most often the first point of contact in patients with acute coronary syndromes, and the failure to recognize it in young adults threatens them with premature death and potentially life-long disability.** © **2017 Elsevier Inc. All rights reserved.**

□ **Keywords—athlete; cardiac arrest; coronary dissection; myocardial infarction; STEMI; young**

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INTRODUCTION

ST-elevation myocardial infarction (STEMI) leading to cardiac arrest is an exceptionally rare occurrence in young adults. More common causes are the abuse of sympathomimetic drugs or myocardial infarction secondary to a significant premature burden of cardiac risk factors or inherited lipid disorders. Another rare cause of STEMI is coronary artery dissection, which overwhelmingly affects middle- and older-aged women with few cardiac risk factors. We describe the case of a 22-year-old athlete with no medical history, who was admitted to our institution post–cardiac arrest with an anterior STEMI and concomitant chronic right coronary dissection. Remarkably, the patient was resuscitated at the scene by his parents; to our knowledge, this represents the first documented case of these simultaneous pathologies in a young cardiac arrest survivor.

CASE REPORT

A 22-year-old previously healthy man was brought to our emergency department by ambulance after an out of hospital cardiac arrest. He had been complaining of "heartburn" that morning and was with his parents purchasing gastro-esophageal reflux medication when he collapsed. His mother and father—a family physician and surgeon, respectively—immediately began cardiopulmonary

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resuscitation. At the scene, defibrillator monitoring confirmed ventricular fibrillation, and he received two DC shocks with adjuvant adrenaline via an autoinjector device (EpiPen; Mylan, Canonsburg, PA). Return of spontaneous circulation was achieved before arrival of the ambulance and the patient was transferred to our hospital for assessment.

On arrival to the emergency department, his blood pressure was 138/77 mm Hg, his heart rate was 132 beats/min, and his oxygen saturation was 95% on a nonrebreathing oxygen mask at 15 L/min. He was breathing spontaneously with a Glasgow Coma Scale score of 8 (E1V2M5). An electrocardiogram in the emergency department (Figure 1) confirmed an anterior STEMI and he was subsequently taken for urgent coronary angiography, where an acute occlusion of the left anterior descending (LAD) artery was found (Figure 2). The LAD lesion was aspirated and a drug-eluting stent was deployed with complete restoration of normal coronary flow (Figure 3). Optical coherence tomography (OCT) was performed pre- and postpercutaneous intervention (PCI) and revealed residual thrombosis, with no evidence of LAD dissection (Figure 4). The right coronary artery (RCA) was dominant and showed an angiographic appearance consistent with coronary artery dissection, with moderate to severe stenosis involving the entire vessel (Figure 2). Retrograde filling of the RCA from left-sided collaterals was noted and left ventriculography revealed mild systolic dysfunction with apical akinesia. A decision was made not to percutaneously repair the RCA.

A former elite swimmer, the patient was an active individual who attended the gym regularly, with a muscular build and height of 175 cm. He denied any previous episodes of chest pain or shortness of breath on exertion. He described a short history of intermittent dyspepsia, usually after meals and not associated with exertion. He had no medical history and no history of childhood illness, such as Kawasaki disease. The patient did not drink alcohol and there was no history of recreational or anabolic drug use. There was no family history of cardiac disease and no clinical features to suggest connective tissue disease.

During his admission to the coronary care unit he underwent chest, abdomen, and head/neck computed tomography angiography that revealed no evidence of large vessel disease, dissection, or stenosis and no evidence of fibromuscular dysplasia (FMD). Echocardiography showed normal ventricular size, normal wall thickness with mild segmental systolic dysfunction, and an ejection fraction of 52%. Hs-troponin T levels peaked at 3840 ng/L and his creatinine kinase levels were 1550 U/L, with other blood work, including lipids, hemoglobin A1C, electrolytes, and hematology all unremarkable. Serum testosterone levels were not suggestive of anabolic steroid abuse, and drug screening was negative. Dual antiplatelet therapy via aspirin 100 mg daily and ticagrelor 90 mg twice daily was commenced, with metoprolol 50 mg twice daily, atorvastatin 40 mg, and ramipril 5 mg daily also initiated. At day 5 he was discharged home without any residual neurologic deficits and remains under the care of a specialist cardiology and hematology service. Extensive outpatient testing has failed to yield a unifying diagnosis 1 year after his arrest; given the absence of family history or clinical evidence of

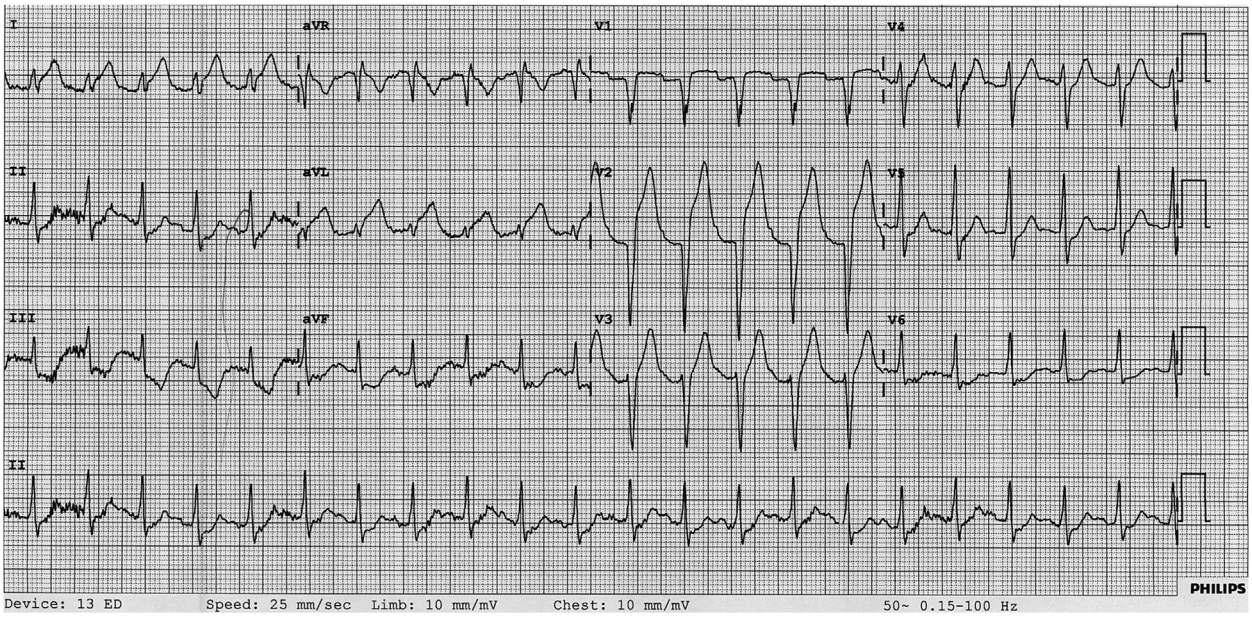

Figure 1. Presenting electrocardiogram, showing anterior ST elevation and reciprocal inferior ST depression.