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Case Report

From cold-blooded reptiles to embryological remnants: Persistent myocardial sinusoids ☆☆☆

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ABSTRACT

In nature, basically 2 types of myocardial vascular patterns exist: the sinusoidal and the coronary type. In the sinusoidal type, the sinusoid is completely fed by blood coming directly from the ventricle through a spongy sinusoidal network. This pattern is found in cold-blooded animals and in the early embryologic development of human (warm-blooded) hearts. A 61-year-old man with atrial fibrillation developed severe tachycardia with a severely reduced left-ventricular ejection fraction (LVEF) of 20%. The patient had no history of prior heart surgery or other cardiac interventions. He was referred for a computed tomography (CT) scan for assessment of pulmonary vein anatomy prior to their isolation. Incidentally, a focal myocardial defect of the midventricular infero-septal wall with tail-like extension into the right ventricular cavity was detected. In a cardiac magnetic resonance (CMR) scan there was no evidence of a myocardial infarction or fibrosis. In the absence of a ventricular septal defect by CT, CMR and echocardiography the diagnosis of a persistent myocardial sinusoid was evident. In this case, we used state-of-the-art methods for pathology visualization, illustrating the effectiveness of CT and CMR in the precise detection and differential diagnosis of myocardial anomalies including a multi-coloured 3D-printed model that may further enhance visuospatial appreciation of those anomalies.

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Abbreviations: LVEF, left-ventricular ejection fraction; CT, computed tomography; CMR, cardiac magnetic resonance; CABG, coronary artery bypass surgery; TMLR, transmyocardial laser revascularization therapy.

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Introduction

In nature, basically 2 types of myocardial vascular patterns exist: the sinusoidal and the coronary type with various transitional types in between. In the sinusoidal type, the sinusoid is completely fed by blood coming directly from the ventricle through a spongy sinusoidal network. This pattern is found in cold-blooded animals and – interestingly – in the early embryologic development of human (warm-blooded) hearts. The fetal myocardium undergoes compaction and is fed mainly and much more efficiently by epicardial coronary arteries. This is necessary to cope with higher metabolic demands [1]. In that sense, myocardial sinusoids represent an embryological remnant of human heart development.

Case report description

A 61-year-old man with atrial fibrillation developed severe tachycardiomyopathy with a severely reduced left-ventricular ejection fraction (LVEF) of 20%. The patient had no history of prior heart surgery or other cardiac interventions. He was referred for a computed tomography (CT) scan for assessment of pulmonary vein anatomy prior to their isolation. Incidentally, a focal myocardial defect of the midventricular infero-septal wall with tail-like extension into the right ventricular cavity was detected (Figs. 1 and 2, Videos 1 and 2, Supplemental Material 1). In a cardiac magnetic resonance (CMR) scan 6-months prior to the CT examination there was no evidence of a myocardial infarction or myocardial fibrosis (Fig. 3, Video 3). In the absence of a ventricular septal defect by CT, CMR and echocardiography the diagnosis of a persistent myocardial sinusoid was evident.

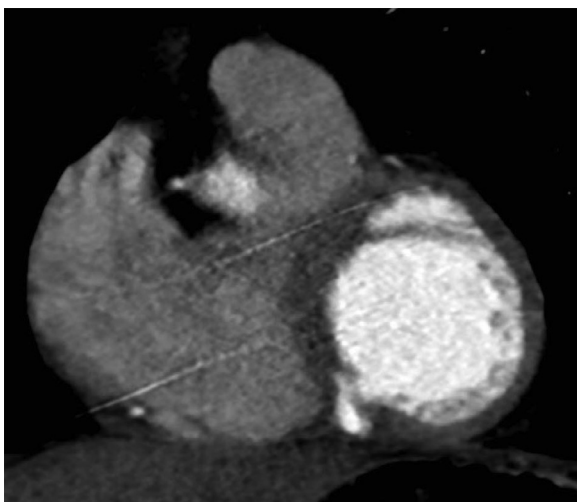


Fig. 1 – Contrast-enhanced computer tomography (CECT) image demonstrating the contrast-material filled defect in the left-ventricular, midventricular infero-septal wall with tail-like extension into the myocardium

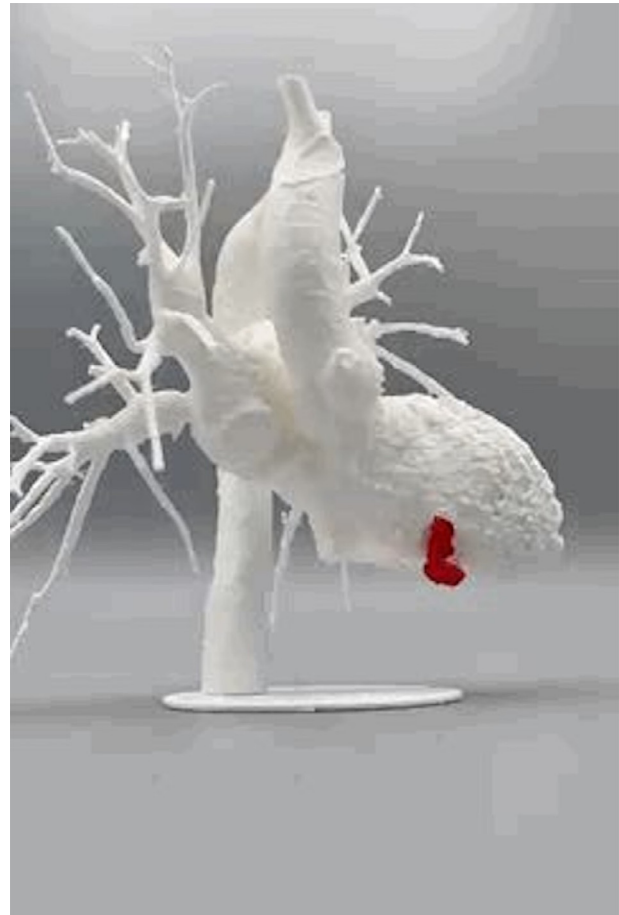


Fig. 2 – Image of an in-house 3D-printed model of the patient's heart with isolation of the left-sided heart. The myocardial sinusoid has been painted in dark red for better visualization

Discussion

Prior to modern coronary artery bypass surgery (CABG) and interventional revascularization, there had been attempts to augment blood supply to ischaemic myocardium by imitating the sinusoidal blood supply: Vineberg (1946) implanted the internal mammary artery into the myocardium next to small myocardial sinusoids [2] (Vineberg procedure, Fig. 4). Later attempts have been made to artificially create myocardial sinusoids by transmyocardial laser revascularization therapy (TMLR) [3,4]. In a review conducted by Briones et al., the authors found that TMLR significantly reduced the angina score as evaluated by the Canadian Cardiovascular Society (CCS) angina scale [5]. Both techniques, though, have always been subject to controversy due to contradictory results and never been broadly applied. According to AHA guidelines, a TMLR-CABG combination in selected patients may be superior to CABG alone in relieving angina; on the other hand, current ESC guidelines evaluated TMLR with a class IIIA recommendation.

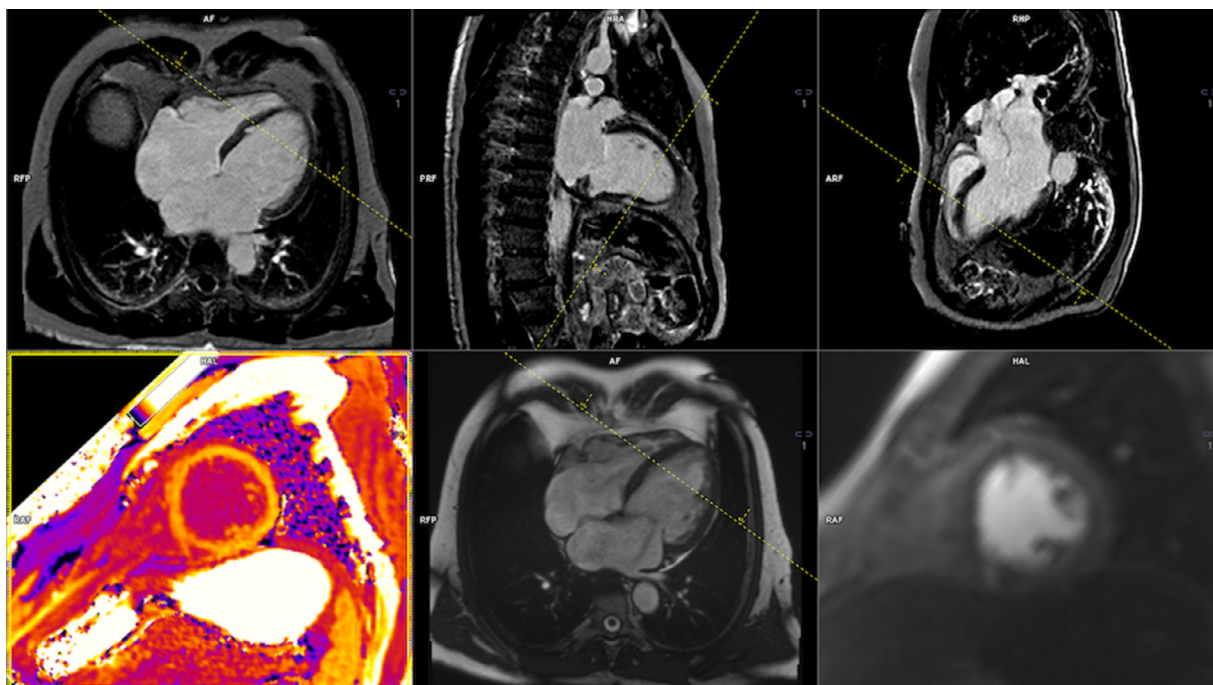


Fig. 3 – Images from a cardiovascular magnetic resonance examination of the patient. Upper row (from left to right): Late gadolinium enhancement (LGE) images demonstrating the absence of myocardial infarction. Lower row (from left to right): T1 map on the level of the myocardial sinusoid demonstrating blood-pool equivalent signal in the midventricular infero-septal wall. 4-chamber CINE image without evidence of a ventricular septum defect (VSD) on the level of the myocardial sinusoid. Stress perfusion image demonstrating again the presence of the myocardial sinusoid as a blood-filled structure that extends beyond the endocardium of midventricular septum (see also Video 3)

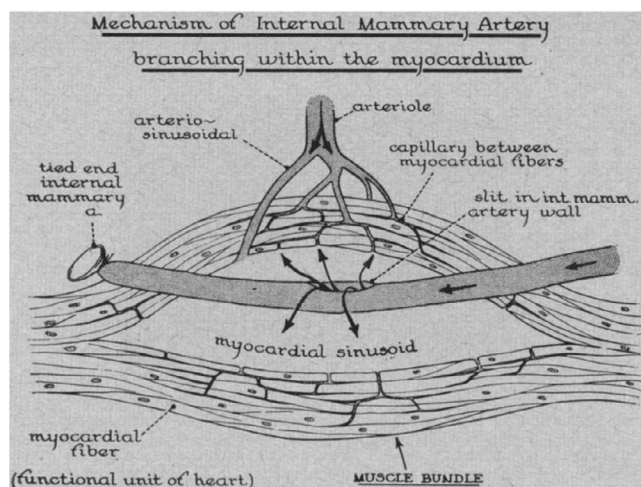


Fig. 6.—Drawing of implanted internal mammary artery in left ventricle with opening in side of vessel. Blood escapes from internal mammary artery into myocardial sinusoids, which is why the implanted vessel remains open until its own branches join the coronary arterioles.

Fig. 4 – From: Vineberg A. Coronary vascular anastomoses by internal mammary artery implantation. *Can Med Assoc J* 1958;78:871-9. According to Canadian Medical Association Journal's requirements, no reprint permission is necessary for using unmodified figures

Case reports regarding myocardial sinusoids are scarce and can affect both ventricles [6–8]. Since the number of cases is very low, it is unclear if isolated left-ventricular myocardial sinusoids are prone to formation of thrombi or have arrhythmogenic potential. Myocardial sinusoids need to be clearly differentiated from ventricular septal defect or post-ischaemic rupture as well as left-ventricular non-compaction cardiomyopathy. In this case, we used state-of-the-art methods for pathology visualization, illustrating the effectiveness of CT and CMR in the precise detection and differential diagnosis of myocardial anomalies including a multi-coloured 3D-printed model that may further enhance visuospatial appreciation of those anomalies.

Patient consent

The authors have declared that no patient consent.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.radcr.2021.11.057.

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