Déjà vu: coronary artery disease in monozygotic twins

Włodzimierz Grabowicz, Konrad Masiarek, Izabela Warchoł, Tomasz Górnik, Andrzej Lubiński

Department of Interventional Cardiology and Cardiac Arrhythmias, Medical University of Lodz, Łódź, Poland

Coronary artery disease (CAD), the leading cause of death in Poland, originates in early life.¹ The lack of fully satisfying treatment strategies caused researchers to look at CAD from a different angle. Consequently, it was postulated that epigenetic factors play an important role in the early-life programming of adult health and diseases.¹ Due to the fact that twin pairs may experience different intrauterine environments, studies on monozygotic twins with discordant phenotypes are one of the most useful study designs in epigenetic epidemiology.¹

The largest prospective study in a twin cohort was conducted by Silventoinen et al² and was based on pooled twin data from Denmark, Finland, and Sweden. The authors acknowledged the existence of a clear aggregation of CAD mortality in twins, and especially in monozygotic twins (genetically identical). Similarly, previous studies have shown concordance in symptoms of angina pectoris in twins.² Subsequently, Hjelmborg et al³ indicated that studies in twin cohorts seem to be vital to ensure the representativeness to the general population.

Our case is an example of a monozygotic twin pair presenting with a similar lesion location in the left anterior descending artery (LAD) and an identical risk factor profile.

Twin A, a 53-year-old man, was diagnosed with non–ST-segment elevation acute myocardial infarction and was successfully treated with the deployment of 3 sirolimus-eluting stents (CRE8, 3.5×16 mm; CRE8, 3.0×21 mm; and CRE8, 2.75×16 mm [CID S.p.A., Saluggia, Italy]) in the proximal and medial portions of the LAD (FIGURE 1A). The patient was subsequently referred to our institute for further management. The following risk factors were not present: obesity,

diabetes mellitus, hypertension, or smoking. His only risk factor was dyslipidemia. Coronary angiography revealed a triple-vessel disease with subtotal (90%) occlusion in the proximal portion of I marginal branch and an 80% discrete lesion in the distal portion of the left circumflex artery (LCX) (FIGURE 18). The patient was treated successfully with the deployment of 2 drug-eluting stents in the LCX/I marginal (bifurcation, CRE8, 3. 5×16 mm) and in the distal LCX (CRE8, 3.0×16 mm) lesions. Transthoracic echocardiography revealed normal chamber diameters and left ventricular function with preserved ejection fraction (60%).

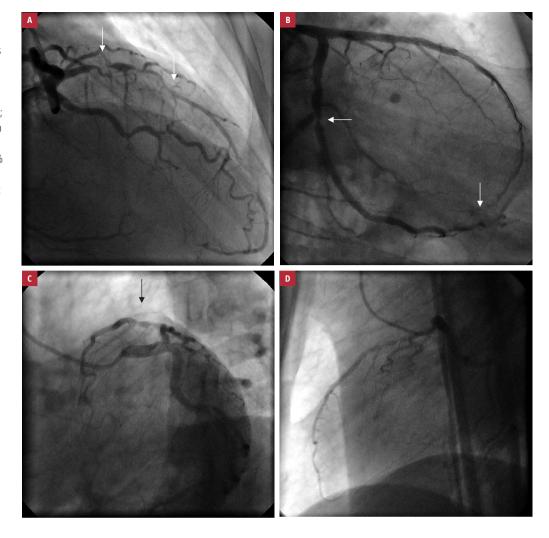
Twin B, the monozygotic twin brother of twin A, a 53-year-old man, was convinced to undergo exercise ECG treadmill test, as he was experiencing dyspnea upon exertion. His only prevalent risk factor was dyslipidemia, similarly to his brother. The exercise test yielded postive results on ECG, so he underwent coronary angiography, which revealed a single-vessel disease with a 95% lesion at the seventh segment of the LAD (FIGURES 1C and 1D). The right coronary artery and LCX were normal. A sirolimus-eluting stent was later successfully implanted in the LAD. Transthoracic echocardiography revealed normal chamber diameters with hypokinesis of the anterior wall and a preserved ejection fraction (50%).

Little is known about angiographic findings in twin pairs with CAD. Although previous observational studies have claimed that the location of coronary lesions is not hereditary, the similar location of stenosis in the LAD, age at first cardiac event, and risk factor profiles show concordance in this pair of identical twins.

In conclusion, we suggest that when one twin presents with CAD, the second twin should be under closer medical surveillance.

Correspondence to:
Izabela Warchoł, MD, Department
of Interventional Cardiology
and Cardiac Arrhythmias,
Medical University of Lodz,
ul. Żeromskiego 113, 90-549 Łódź,
Poland, phone: +4842 63 93563,
email: izabelaritawarchol@gmail.com
Received: April 24, 2019.
Revision accepted: July 23, 2019.
Published online: July 25, 2019.
Kardiol Pol. 2019; 77 (9): 886-887
doi:10.33963/KP.14910
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FIGURE 1 Coronary angiography of the left anterior descending arteries (LADs) of the twin pair. Twin A: A - lesions in the proximal and medial portions of the LAD (arrows); **B** – subtotal (90%) occlusion in the proximal portion of I marginal branch and an 80% discrete lesion in the distal portion of the left circumflex artery (arrows). Twin B: C, D – single-vessel disease with a 95% lesion at the seventh segment of the LAD (arrow)



ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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HOW TO CITE Grabowicz W, Masiarek K, Warchoł I, et al. Déjà vu: coronary artery disease in monozygotic twins. Kardiol Pol. 2019; 77: 886-887. doi:10.33963/KP.14910

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