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IDENTICAL CRITICAL LEFT ANTERIOR DESCENDING ARTERY STENOSIS IN MONOZYGOTIC TWINS

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ABSTRACT

Introduction: Coronary artery disease (CAD) is a multifactorial condition influenced by both genetic predispositions and environmental exposures.

Case description: We present a pair of male monozygotic twins diagnosed with identical critical left anterior descending artery stenosis, suggesting a possible hereditary susceptibility involving this coronary segment. One twin presented with acute coronary syndrome while the other was diagnosed with CAD during screening prompted by his sibling's diagnosis.

Conclusion: This report underscores the importance of early screening among genetically related individuals and contributes to the understanding of phenotypic variability in genetically identical patients with CAD.

KEYWORDS

Coronary artery disease, monozygotic twins, hypercholesterolemia, smoking, genetic predisposition

LEARNING POINTS

- There is a strong genetic contribution to the anatomical distribution of CAD even when environmental risk factors differ between twins.
- Thorough cardiovascular risk assessment and management are warranted for asymptomatic identical twins when their co-twin presents with CAD. Early screening for subclinical disease and the consideration of advanced imaging modalities may be justified in this context.

INTRODUCTION

Coronary artery disease (CAD) is a leading cause of morbidity and mortality worldwide. Its aetiology is multifactorial involving modifiable risk factors such as smoking, hypertension, hyperlipidaemia, diabetes mellitus, obesity and sedentary lifestyle, and non-modifiable factors including age, gender, ethnicity and genetic predispositions[1]. Although environmental and modifiable

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risk factors significantly impact the onset and progression of CAD, an increasing body of evidence underscores the role of genetic predispositions as well[2].

Identical monozygotic twins offer a unique opportunity to study the heritability and phenotypic concordance of CAD due to their shared genetic makeup. In a systematic review of 31 cases of monozygotic twins with CAD, the mean age at CAD presentation was 45 years, most patients were male and had hyperlipidaemia. Interestingly, most twin pairs demonstrated concordant coronary anatomy suggesting a strong genetic influence on not only disease occurrence, but also on the pattern of coronary involvement[3].

We report on two identical monozygotic twins, one of whom presented with acute coronary syndrome. The other twin, initially asymptomatic, was also diagnosed with significant CAD following clinical screening. Both brothers shared a common site of left anterior descending (LAD) artery critical stenosis. This report emphasises the importance of early screening and preventive strategies in genetically at-risk individuals, even in the absence of symptoms or shared environmental risk factors.

CASE DESCRIPTION

A 43-year-old man (twin A) presented to the emergency department with chest pain and diaphoresis. He was a smoker and had hyperlipidaemia; his body mass index was 25 kg/m^2. His mother had CAD at 60 years of age. An echocardiogram (ECG) revealed anterior wall ST-elevation myocardial infarction; the troponin level was 40 ng/dl (normal <14 ng/dl), and Echocardiogram revealed left ventricular systolic dysfunction with anterior hypokinesis. He was immediately transferred to the catheterisation laboratory for percutaneous coronary intervention, which revealed a mid-LAD artery stenosis of 95%, distal LAD artery stenosis of 70%, distal left circumflex artery stenosis of 50%, first obtuse marginal artery stenosis of 80% and non-dominant right coronary artery (RCA) stenosis of 70%. A drug-eluting stent was inserted into the mid-LAD stenosis *(Fig. 1)*. During a subsequent percutaneous coronary intervention three weeks later, additional stents were inserted into the first obtuse marginal artery and into the distal LAD artery. Normal left ventricular function was observed on a subsequent Echocardiogram instead of ECG.

Consequently, his identical twin brother (twin B) was recommended to undergo a coronary evaluation. He had hyperlipidaemia, and his body mass index was 29 kg/m^2. Five weeks later, twin B underwent a coronary computed tomography (CT) which demonstrated a proximal LAD artery stenosis of 70–99% and first diagonal branch artery stenosis of 70–99%. The patient was asymptomatic, but due to the significant coronary CT findings, he was referred for further evaluation. His physical examination and blood tests were normal. His ECG showed a normal sinus rhythm without ischaemic signs, and did not reveal any pathological findings. A coronary angiogram revealed a proximal LAD artery stenosis of 99%, first diagonal artery stenosis of 90% and RCA stenosis of 30%. A drug-eluting stent was inserted into the critical stenosis of the proximal LAD artery *(Fig. 1)*.

DISCUSSION

Genetic predispositions to CAD are well recognised but often difficult to quantify. The definition of positive family history for CAD includes a first-generation relative aged ≤55 years for males or ≤60 years for females at the time of CAD diagnosis[4]. This definition is based on earlier studies on symptomatic CAD diagnosed by coronary angiography, whereas CAD is nowadays diagnosed frequently in asymptomatic patients using coronary CT.

Several rare monogenic causes have been described in the literature as genetic contributors to CAD. The most important example is familial hypercholesterolaemia

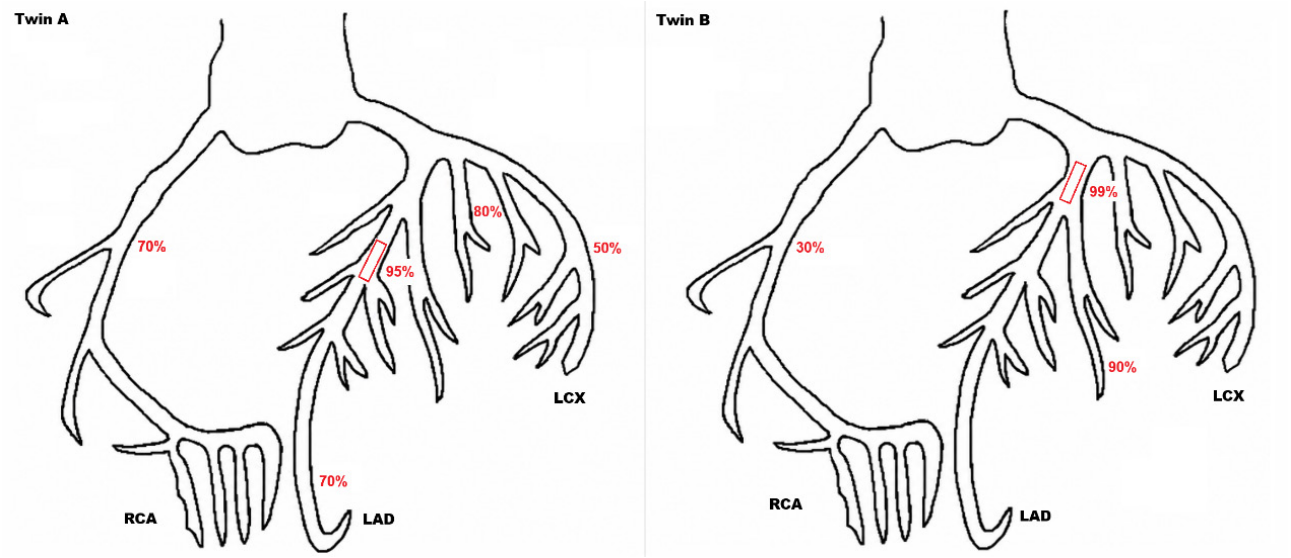


Figure 1. Coronary stenoses in both patients before and following stent insertion in the first coronary intervention. Abbreviations: LAD, left anterior descending; LCX, left circumflex; RCA, right coronary artery.

caused by loss-of-function of the low-density lipoprotein (LDL)-receptor consistent with reduced LDL clearance from the blood and LDL serum levels greater than 190 mg/dl[2]. The twin patients had lower LDL serum levels, and no clear monogenic disease was identifiable from their history. There are very few monogenic mutations associated with CAD, and they cannot explain the widely appreciated importance of family history of CAD. However, family history is difficult to evaluate due to reporting bias and incomplete documentation, and thus is not included in cardiovascular risk score calculators from the European Society of Cardiology[5] and the American Heart Association[6]. Some of the attributed risks associated with family history are mediated through the traditional cardiovascular risk factors (hypertension, hyperlipidaemia and diabetes mellitus) and social behaviours, and their effect may be diminished once adjusting for these risk factors. Evaluating genetic predispositions may overcome the difficulty of relying on family history.

In addition to monogenic changes, genome-wide association studies have identified more than 160 gene loci associated with CAD. These studies require large patient cohorts – typically more than one million – to identify a significant change and consist of single-nucleotide polymorphisms (SNPs). The mechanisms underlying these associations remain unclear since many of the loci are not in coding areas of the DNA, which underscores the difficulty of identifying genetic predisposition outside a monogenic setting. Nevertheless, polygenic risk scores have been developed, assessing the risk according to the number of SNPs a patient has. The risk scores show an added benefit especially in younger patients and in retrospective studies, but the applicability to the general population is not well defined. First, the risk scores were developed from European data. Moreover, interactions between SNPs and their combined biological effects remain poorly understood; monozygotic twins share the same DNA, thus presenting an opportunity to study polygenic risk scores. In a large Swedish study, the risk of cardiovascular death after one monozygotic twin dies prematurely from myocardial infarction was markedly elevated – higher than in dizygotic twins[7]. In a subanalysis, smoking was the only factor differentiating cardiovascular risk between monozygotic twins[8], but this was not observed in the reported twins.

In conclusion, these cases highlight the potential value of early coronary evaluation in monozygotic twins following a CAD diagnosis in one sibling and support the growing evidence for genetic influences on the anatomical distribution of CAD.

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