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CASE REPORT

Enigma of Twins: Identical Presentation and Angiographic Lesion in Monozygotic Twins

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Abstract

We present a case of monozygotic identical twins presenting with coronary artery disease (CAD), there were striking similarities in the symptoms, coronary anatomy, and lesions.

Keywords: Coronary artery disease, Genetic, Monozygotic, Twins

1. Case report

1.1. History of presentation

Twin A was a 36-year male who was referred to our outpatient department with the presenting complaint of angina on exertion currently in New York Heart Association (NYHA) class III for two months. Twin B the monozygotic identical twin brother of twin A, had angina and dyspnea on exertion (NYHA class II) for 2 months, with a positive stress test.

1.2. Past medical history

Both the twins belong to the upper socioeconomic class, are well-educated, living in a joint family together. They have no history of addiction in form of substance abuse/smoking or tobacco consumption and both the twins were having a sedentary lifestyle. Twin A medical history included treatment for dyslipidemia and was on rosuvastatin (20 mg) along with aspirin (75 mg) and beta-blocker (metoprolol 25 mg) for 1 month. His family history was significant for an identical twin (twin B) who also has similar complaints of angina, dyspnea on exertion (NYHA class II) and was on treatment for dyslipidemia for 1 month including metoprolol (25 mg), aspirin (75 mg) & rosuvastatin (20 mg).

1.3. Investigations

The baseline ECG of both the twins was within normal limit however, stress evaluation test for Twin B had significant ST depression during the peak of exercise and in the recovery phase of TMT.

Both the twins underwent CT angiography showing significant lesions (Fig. 1), rest of all the investigations and their comparisons are depicted in Table 1.

1.4. Management

Twin A was treated with successful implantation of an everolimus-eluting stent in left circumflex to first OM (2.75 mm × 28 mm) (Fig. 2-II). While for twin B two everolimus-eluting stents in OM (2.75 mm × 28 mm) and PLV (2.5 mm × 36 mm) were implanted with satisfactory angiographic results (Fig. 2-IV; Fig. 3-III).

2. Discussion

Twins have been an interest in studies since antiquity. Twins' studies compare the similarity of a disease or a specific trait between twin pairs. As the

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pathogenesis of CAD is multifactorial with a conglomerate interaction between the genetic and environmental factors [1]. It has provided researchers with an invaluable tool to assess the effect of genetics and environmental factors independently in the development of CAD [2].

Although family history is an established risk factor for the development of CAD [3], twin studies prove to be more powerful due to inherent characteristics, such as similar exposure to pre-and post-natal environmental factors (e.g., lifestyle, diet, physical activity) as compared to the general population. These known and even unknown variables endow to the development of CAD [2], therefore attributing a phenotypic difference to genetic or environmental factors becomes easier with twin studies.

The idea of twins and cardiovascular similarity first began with studies for concordance of electrocardiograms [1], which revealed certain similarities, and more recently with angiographic concordance which has been shown only in a few case reports [4,5]. In the last two decades, several genetically defined risk factors have been established via twin studies as a causal factor for CAD including blood pressure, lipid levels [6], and smoking [7]. The incidence of monozygotic twins in India is estimated to be 3.67 per million [8]. In this context, our case provides comprehensive and detailed insights into CAD in monozygotic twins with similar risk factors.

The twins in our case are of interest due to several reasons: I) monozygotic; II) simultaneous and similar presentation (angina); III) relatively young age (mid-thirties); IV) similar predisposing risk factors (dyslipidemia); V) similar environmental factors; VI) no family history of CAD; VII) concordant angiographical lesions; IX) simultaneous and successful stent implantation in both the twins. Although we report a single pair of monozygotic twins, the evidence gained is suggestive of a genetic predisposition for the development of CAD in this specific subset of patients with no family history.

Earlier Frings et el [9]. showed that dominance of the coronary circulation was concordant in twins with inter-twin variability in coronary artery diameter and length. They showed that concordance of the lesion was only seen in 54% of the study group. However, the data is sparse and mainly represented by case reports, it is well documented that monozygotic twins have a similar subset of the lesion and premature CAD [8,10–12]. We like to point towards the fact that the evidence derived in favor of

Abbreviations

CAD	coronary artery disease
ECG	Electrocardiogram
TMT	Treadmill test
PLV	posterior left ventricular artery
RCA	right coronary artery
LAD	left anterior descending artery
LCx	left circumflex artery
LM	left main
OM	obtuse marginal artery
PCI	percutaneous coronary intervention

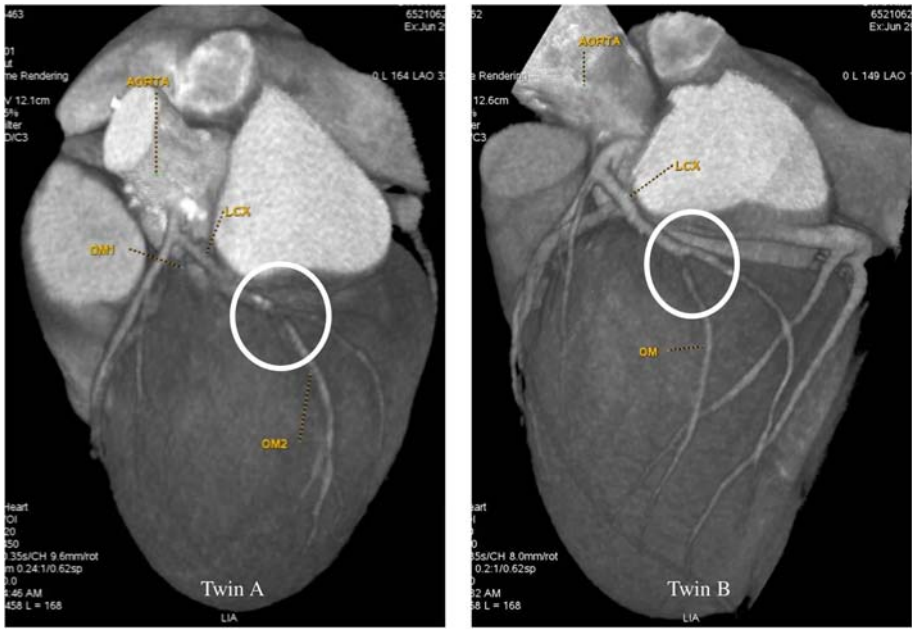

Fig. 1. 3D reconstruction of CT angiography showing similarity in lesion subset in both the twins (white circle-at LCx and OM bifurcation).

Table 1. Comparison of clinical and angiographic parameters between the two twins.

	Twin A	Twin B
BMI	23.437	21.875
Predisposing risk factor	Dyslipidemia	Dyslipidemia
TC[#] (mg/dl)	305↑	983↑
LDL[##] (mg/dl)	232 ↑	212↑
HDL[###](mg/dl)	55	41
TG[####] (mg/dl)	163	521↑
Biochemical Parameters		
• Hs-CRP *	4.37 (N)	1.99 (N)
• Homocysteine **	14.2 (N)	15.2 (N)
• Lipoprotein (a) ***	12.30 (N)	10.50 (N)
Coronary artery dominance	Right	Right
Coronary calcium score	65	22
Vessel Diameter (mm)		
• LM	4.00	4.5
• LAD	3.00	3.00
• LCx	2.75	2.75
• RCA	3.00	3.00
Coronary Lesion	95% lesion in OM (Fig. 2-I).	70% stenosis of the first OM (Fig. 2-III) and 90% stenosis of PLV (Fig. 3-II).

Serum Homocysteine range **(5–16 μmol/dl); Lipoprotein (a) range ***(<20 mg/dl).
BMI = body mass index (normal range 20–25 kg/m^2); N = normal value; ↑ =increased; TC = total cholesterol (#<200 mg/dl); TG = triglycerides (#### <165 mg/dl); HDL = high density lipoprotein (###>40 mg/dl); LDL = low density lipoprotein (##<100 mg/dl); Hs-CRP = high-sensitive C-reactive protein[*(<5.0 mg/dl)].

monozygotic twins is derived from case reports and are always subjected to publication bias where only the concordant features are reported and require larger multicentric studies for concrete evidence.

Although this is a single case report, certain conclusions can be drawn with our case: I) premature CAD in monozygotic twins; II) simultaneous similar presenting complaints; III) similar coronary anatomy along with lesion subset; IV) predominant play of genetic factor in the development of CAD. In a large Swedish study, the relative risk of death in the next 10 years was 8.1 for monozygotic twins when one of the

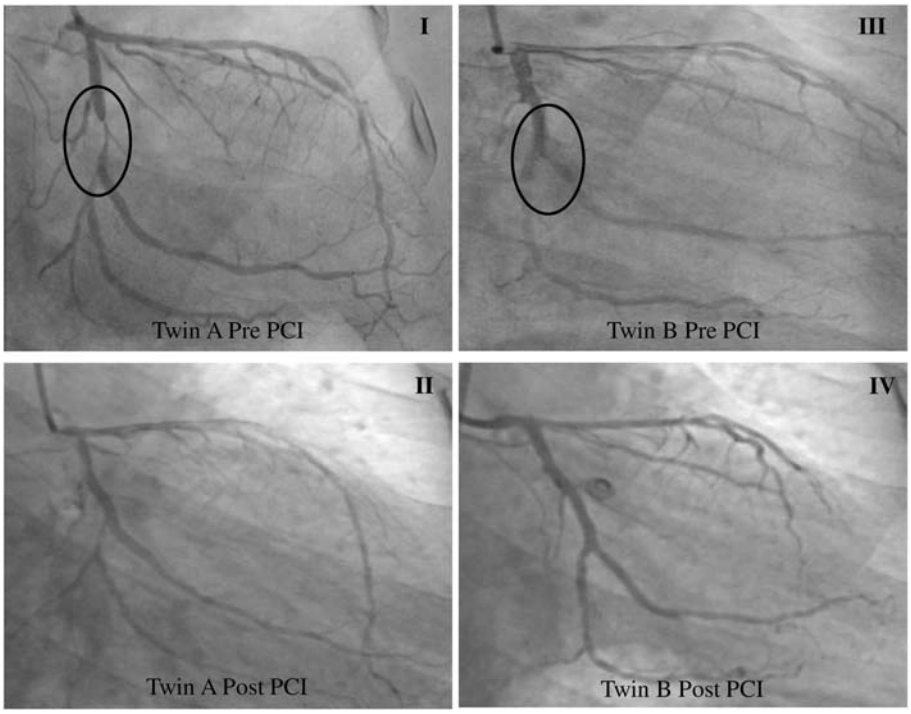

Fig. 2. Left coronary angiogram showing the baseline coronary lesion similarity between the presenting twins (I & III) and post PCI results (II & IV).

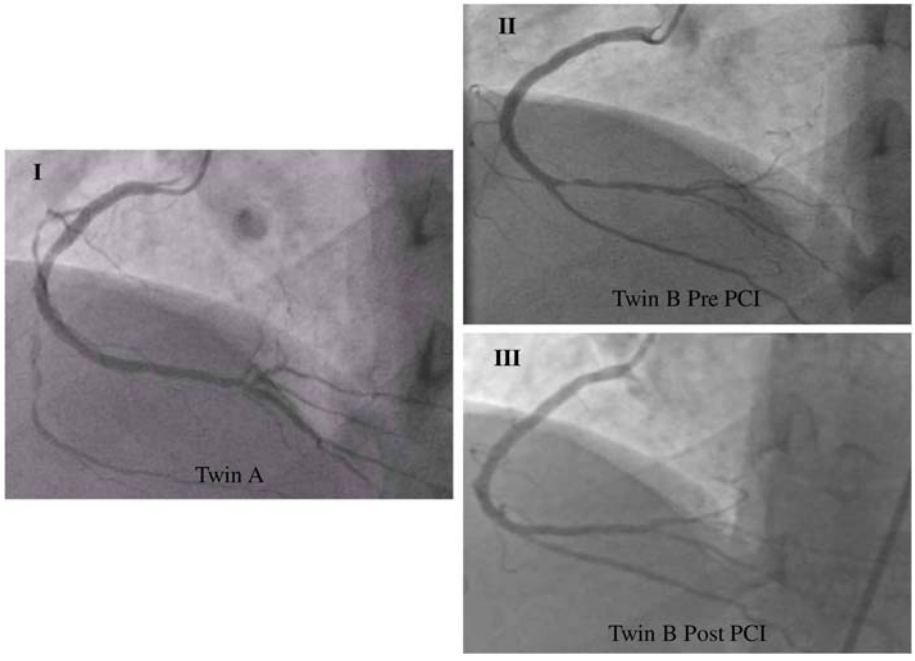

Fig. 3. Right coronary angiogram showing baseline coronary lesion (I & II) and post PCI result (III).

twins has expired because of CAD [5]. In a study by Samuels et al. [1] the likelihood of discovering occult CAD was as high as 50% in an asymptomatic twin of symptomatic pair. So, we conclude from our case that monozygotic twins have additional susceptibility to the development of CAD with concordant coronary artery lesions which need to be monitored aggressively and treated if required.

3. Conclusion

Reports such as these provide a concrete argument for a genetic role in the development of CAD. Based on our experience we propose strict surveillance of CAD among monozygotic twins regardless of young age, presence of risk factors, or limiting symptoms.

Learning objectives

1. There is a concordance of presentation as well as angiographic finding among monozygotic twins.
2. Premature coronary artery disease in monozygotic twins is a predominantly genetically determined entity.
3. This genetic predisposition gives us a chance for early prevention and robust surveillance among twins regardless of the presenting symptoms or traditional risk factors.

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Author contribution

Conception and design of Study: APS, AR. Literature review: APS, AR. Acquisition of data: APS, BB. Analysis and interpretation of data: APS. Research investigation and analysis: APS, AR, BB. Data collection: APS, APS. Drafting of manuscript: APS. Revising and editing the manuscript critically for important intellectual contents: APS, RKN. Data preparation and presentation: RKN. Supervision of the research: AR, RKN. Research coordination and management: RKN. Funding for the research: APS, AR.

Conflicts of interest

No relationships with industry.

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