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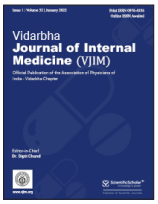

*Case Report*

# A Rare Presentation of Coronary Aneurysm following Bare-metal Stent

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## ABSTRACT

We present a case of post-percutaneous transluminal coronary angioplasty with a bare-metal stent to the right coronary artery (RCA). He presented with acute ST-elevation inferior wall myocardial infarction. Coronary angiography showed an aneurysm of proximal RCA extending from the proximal end of the stent to the distal end. There was a 90% discrete lesion beyond the stent with TIMI III flow. The development of coronary artery aneurysms (CAAs) was reported even before the era of stents. CAA can develop after plain balloon angioplasty, after directional coronary atherectomy, and after bare-metal and drug-eluting stents also but not after coronary angiography. Kachru *et al.* reported an incidence of 0.2% (4 of 2408) per DES without routine angiographic follow-up and Rha *et al.* reported an incidence of 1.7% (five out of 296) per patient at 6-month routine angiographic follow-up. CAA is an uncommon and often accidental finding, we report a case of CAA which presented as acute myocardial infarction mimicking stent thrombosis.

**Keywords:** Bare-metal stent, Coronary aneurysm, Drug-eluting stent, Percutaneous transluminal coronary angioplasty

## INTRODUCTION

The incidence of coronary artery aneurysms (CAAs) following coronary interventions is 0.3–6%. The development of CAA is a well-recognised complication following drug-eluting stent (DES) implantation. However, CAA following bare-metal stent (BMS) implantation is rarely reported.[1]

## CASE REPORT

We report a case of 70/M, DM and non-hypertensive who presented with NSTEIWMI. Coronary angiography (CAG) showed proximal right coronary artery (RCA) 80% discrete (non-culprit vessel), *de novo* lesion. He underwent percutaneous transluminal coronary angioplasty (PTCA) with BMS (3.25 × 20 from vascular concepts) as shown in [Figure 1]. BMS was used because of financial constraints. There was no complication during the first procedure.

After 45 days of the procedure, patient presented with acute ST-elevation inferior wall myocardial infarction [Figure 2]. (ECG showing acute ST-elevation inferior wall MI). He was suspected of subacute stent thrombosis and thrombolysed with 1.5 MU streptokinase at the peripheral centre. Check CAG showed aneurysmal dilation of proximal RCA extending from the proximal end of the stent to the distal end. There was a 90% discrete lesion beyond the stent with TIMI III flow [Figure 3] (showing an aneurysm).

He had no fever, and his TLC count and ESR were normal. This patient was not willing for further intervention and was lost to follow-up. This case is presented to discuss the rare presentation of aneurysm following BMS.

## DISCUSSION

Development of CAAs was reported even before the era of stents. In 1983, Holmes *et al.* reported the first case of coronary aneurysm following balloon angioplasty.[2] Bal *et al.* in 1991 reviewed 728 patients of plain balloon angioplasty with angiographic follow-up and found that the incidence of CAA was 3.9%. The predictive factor for the development of coronary aneurysms in their study was coronary dissection as stents were not much in use in the 1990s.[3]

Directional coronary atherectomy (DCA) became widespread in the USA after its approval by FDA in 1990. Various authors have reported an incidence of 0.5–10% in association with DCA.

In the BMS era, the development of CAAs was rarely noted and the incidence was not known exactly. However, with the

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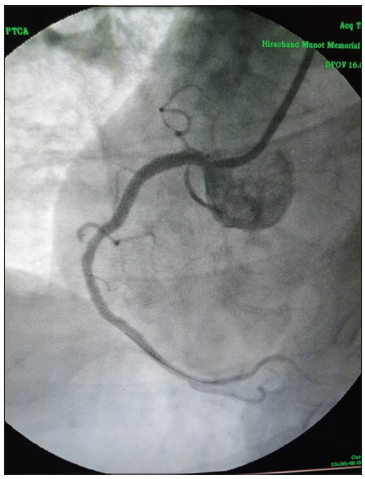


**Figure 1:** Post-percutaneous transluminal coronary angioplasty with bare-metal stent.

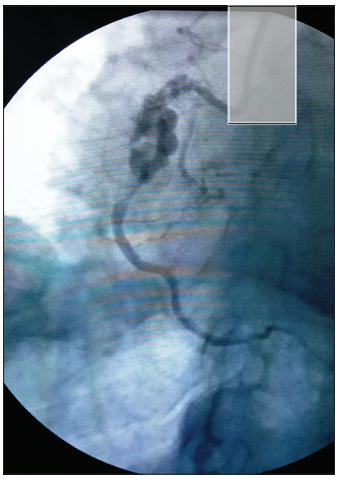

**Figure 3:** Coronary angiography showing an aneurysm in the proximal right coronary artery.

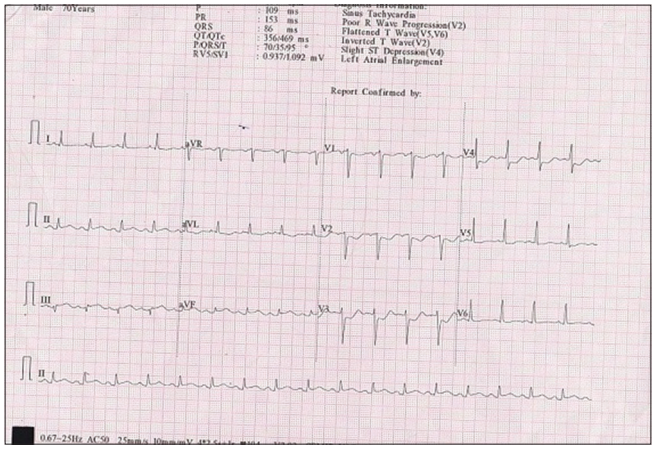


**Figure 2:** Acute ST-elevation inferior wall myocardial infarction.

increasing use of DESs and the mandatory use of follow-up angiograms at 6–9 months for the detection of restenosis in the various randomised trials, the development of CAAs has been highlighted.

### Incidence of aneurysms following stent implantation

The incidence varies according to the definition used for the diagnosis of the aneurysm. Coronary angiography the gold standard for the diagnosis of coronary aneurysms uses a luminal dilation of 50% larger than that of the adjacent reference segment for the diagnosis of the coronary aneurysm. However, in the pivotal DES trials, the definition of CAAs was vessel distension of 20% or more in diameter compared with the reference vessel at follow-up, a stricter definition compared with the definition used in the BMS era (vessel distension of >50%). An estimate of the actual incidence of CAA formation after BMS or DES implantation outside of randomised trials has been reported in only two abstracts. Kachru *et al*. reported an incidence of 0.2% (four of 2408) per DES without routine angiographic follow-up and Rha *et al*. reported an incidence of 1.7% (five out of 296) per patient at 6-month routine angiographic follow-up.

Classification of coronary aneurysms following stent implantation: Aoki[4] *et al*. have proposed a classification of aneurysms following stent implantation as follows:

Type I: This is the type of aneurysm that demonstrates rapid early growth with pseudo-aneurysm detected within 4 weeks. This type is typically complicated by clinical pericarditis and is related to the procedure-related arterial injury.

Type II: This aneurysm is typically subacute or chronic and is usually detected incidentally during angiography done for recurrence of symptoms or as part of a study protocol. Our patient falls into this category.

Type III: Mycotic or infectious aneurysm. Large mycotic aneurysms infected with *Staphylococcus aureus* after DES or BMS have been reported.

A similar type of stent aneurysm after BMS is described by Kalkan *et al*. in their case report of A cockscomb-like aneurysm of the RCA after BMS implantation. It was developed after 1 year.[5]

The exact mechanism of the development of CAA following BMS is not known but the proposed mechanisms are medial damage during original PTCA with either oversized balloon or stent or over-expansion of the same with high pressure.[3] Bal *et al*. reported dissections at the site of balloon dilation as a major predictor of the development of CAAs. In their study, PTCA complicated by dissection had a 9% incidence of CAA as compared to 2.9% without any dissection. These dissections

are usually not visible on routine coronary angiography and cause the formation of ulcers and microaneurysm.

The mechanisms of aneurysm formation following DES are different from above mentioned. The major cause is a hypersensitivity reaction to stent polymer which leads to an inflammatory response at the site of stent deployment which delayed the healing process. Virmani *et al*. documented that non-erodible polymer leads to low-grade chronic inflammation. Furthermore, in addition, drug elution slowly leads to antiproliferative and anti-migratory effects, leading to local disruption of elastic vascular intima and reduction in collagen concentration and changes that lead to the development of a coronary aneurysm.[6]

Late stent malapposition (LSM) is also one of the causes of CAA more often seen following DES. LSM is 4% after BMS implantation and 13.2% with sirolimus-eluting stents and 8.4% after paclitaxel-eluting stent implantation.[7] The incidence of LSM is more following primary angioplasty (54%).

## CONCLUSION

CAA is an uncommon and often accidental finding. Nevertheless, the exact mechanisms leading to CAA formation are still unclear. Unfortunately, the lack of specific prodromal symptoms or factors predisposing to the formation of CAAs significantly limits the diagnostic possibilities and, consequently, the therapeutic modalities.

### Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

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