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Thinner is not always better: Focal in-stent restenosis with stent deformation at coronary bend following ultrathin-strut drug-eluting stent implantation

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ABSTRACT

We describe a 64-year-old male who underwent percutaneous coronary intervention of an angulated right coronary artery lesion using overlapping ultrathin-strut everolimus-eluting stents. Although the initial angiographic result was optimal, the patient developed recurrent angina at six months, and repeat angiography revealed focal in-stent restenosis localised to the coronary bends with fluoroscopic evidence of stent deformation. The overlap segment was relatively spared. Repeat intervention with a conventional thin-strut DES resulted in excellent clinical and angiographic outcomes at one-year follow-up. This case underscores the importance of unfavourable outcome of ultrathin strut DES at coronary bends, possibly secondary to poor radial strength leading to stent deformation and in-stent restenosis.

1. Introduction

The introduction of drug-eluting stents (DES) has dramatically reduced the incidence of in-stent restenosis (ISR) compared with bare-metal stents. Ultrathin-strut DES (strut thickness less than 70 μm) further aim to improve procedural deliverability, vascular healing, and long-term outcomes by minimizing arterial injury and inflammation [1,2]. However, reducing strut thickness compromises the radial strength of the stent.

Vessel tortuosity and sharp angulation pose unique biomechanical challenges during stent implantation. Coronary bends are subjected to repetitive deformation during the cardiac cycle, creating localised stress concentrations that may influence stent integrity, drug distribution, and vascular response [3]. We report a case of focal ISR occurring at a coronary bend following implantation of an ultrathin-strut DES in the RCA, highlighting the interaction between stent design and lesion geometry.

2. Case presentation

A 64-year-old male with a prior history of long-standing tobacco smoking presented with effort angina for the previous 6 months. There was worsening in angina with minimal effort for the last few days. He underwent diagnostic coronary angiography, which revealed a diffuse, significant stenotic lesion involving the proximal to distal segment of the right coronary artery **(**Fig. 1a**)**. The lesion was located at an angulated segment of the vessel, without significant calcification or involvement of the bifurcation. There was mild non-obstructive atherosclerosis involving the left coronary artery. Percutaneous transluminal coronary angioplasty (PTCA) of the right coronary artery was performed using a standard technique **(**Fig. 1b-f**)**. After adequate lesion preparation (2.75 and 3.0 mm semicompliant balloon), two overlapped drug-eluting stents (3 × 37 mm and 3.5 × 40 mm) were deployed across the lesion. The stents were ultrathin strut (strut thickness 50 μm) cobalt-chromium everolimus eluting stent (EVERMINE 50, Meril Life Sciences). Post-dilatation was performed, and angiography demonstrated satisfactory stent expansion with no residual stenosis, dissection, or flow limitation. The patient was discharged on guideline-directed dual antiplatelet therapy.

Six months later, the patient presented with the recurrence of effort angina. He was compliant with drug therapy and smoking cessation. Repeat coronary angiography revealed significant in-stent restenosis, predominantly localised to the coronary bends **(**Fig. 2**)**. The overlap segment was relatively spared from restenosis. Fluoroscopic examination of the stents revealed a deformed stent at the coronary bends, which were not obvious at the time of stent implantation **(**Fig. 3**)**. A redo PTCA involving the restenosis segment was performed, deploying a standard

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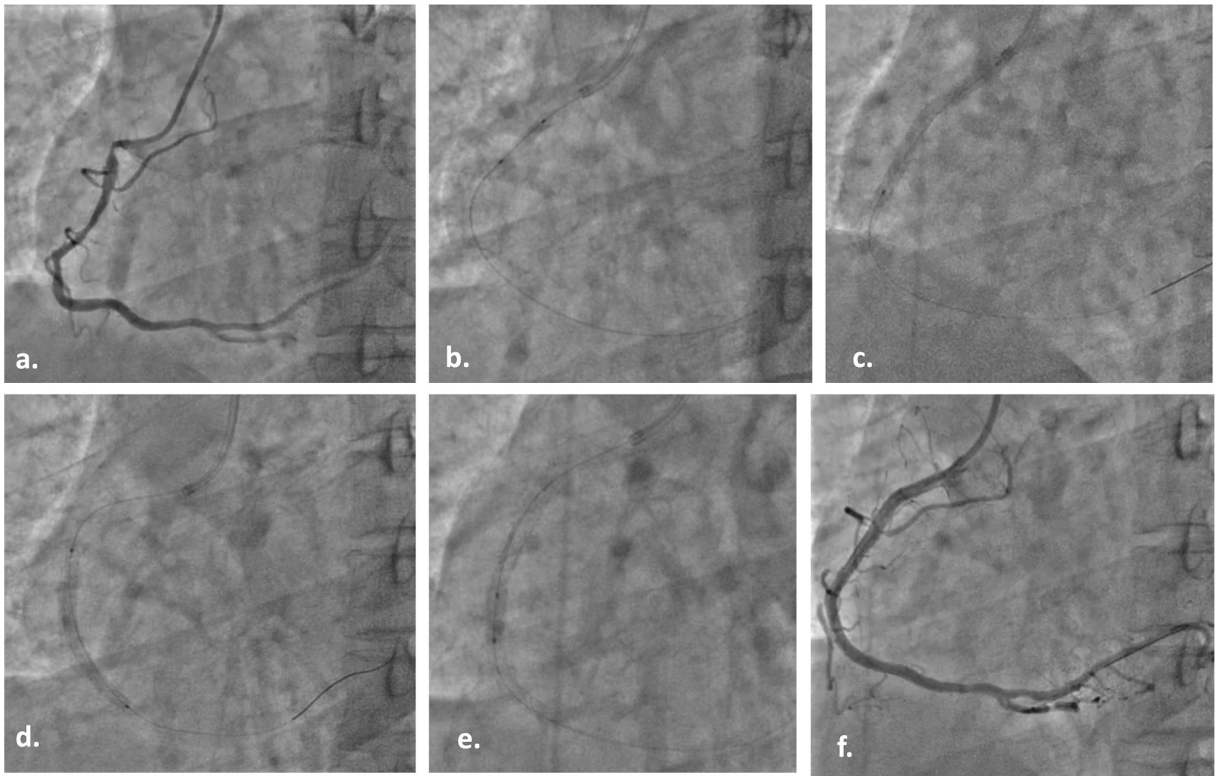

Fig. 1. Initial angiogram of the right coronary artery demonstrating a diffuse significant coronary stenosis from the proximal to distal segment (1a). PTCA procedure-predilation (1b), stent implantation (1c, d), post-dilation (1e) and final angiographic result (1f).

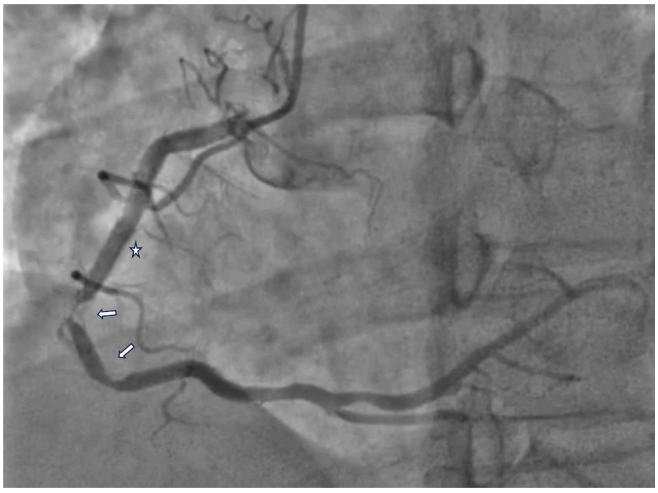

Fig. 2. Follow-up angiogram at 6 months showing focal in-stent restenosis involving the coronary bends (arrow) and relative sparing of the stent overlap segment (star).

DES (XIENCE Alpine 3 × 28 mm) after adequate high-pressure predilation (Fig. 4). Patient is doing well without any angina at 1-year follow-up following the repeat procedure.

3. Discussion

Although drug-eluting stents (DES) have significantly lowered the incidence of in-stent restenosis (ISR), this complication continues to occur in coronary lesions with complex anatomical and mechanical characteristics [1]. Among the various design characteristics of DES, stent strut thickness has emerged as a key determinant of vascular healing and long-term outcomes. Thinner struts are associated with reduced vessel injury, faster endothelialisation, and lower neointimal hyperplasia, translating into improved restenosis rates compared with earlier thick-strut platforms [2]. Advances in stent technology have led to the development of **ultrathin-strut DES**, typically defined by a strut thickness of <70 μm. These stents offer superior flexibility, enhanced deliverability, and improved conformability, particularly in tortuous coronary anatomy [3]. Meta-analyses and randomized trials have demonstrated favourable clinical outcomes with ultrathin-strut DES in routine practice, including lower target lesion failure and comparable or reduced rates of ISR when compared with conventional thin-strut DES [3,4]. Consequently, ultrathin-strut platforms are increasingly used across a broad spectrum of lesions.

Despite these advantages, concerns remain regarding the mechanical performance of ultrathin-strut DES in lesions exposed to high biomechanical stress, such as severe angulation, heavy calcification, and chronic total occlusions (CTO). The reduced strut thickness, while improving flexibility, may compromise radial strength and longitudinal integrity in rigid or dynamically stressed vascular segments [5]. The PRISON IV randomized trial highlighted this limitation, demonstrating higher late lumen loss and restenosis rates with an ultrathin-strut sirolimus-eluting stent compared with a conventional thin-strut everolimus-eluting stent in CTO lesions, underscoring the importance of adequate scaffold support in complex anatomy [6].

Coronary bends represent biomechanical "hinge points" subjected to repetitive flexion, torsion, and longitudinal deformation during the cardiac cycle [7]. Stent implantation across such regions may result in asymmetric expansion, non-uniform strut distribution, and focal malapposition, leading to disturbed wall shear stress—a recognised trigger for neointimal proliferation and ISR [8]. Experimental and bench studies have further shown that stents deployed in curved segments are prone to focal deformation and altered drug distribution, particularly when radial strength is limited [9].

In the present case, ISR was focal and localised to a sharp coronary bend, rather than diffuse, suggesting a predominantly geometry- and mechanics-driven mechanism rather than a systemic biological response. The angiographic appearance of stent deformation at the bend supports the hypothesis that reduced mechanical support from an ultrathin-strut platform at a hinge point may have contributed to localised restenosis. The right coronary artery, with its pronounced curvature and significant dynamic motion, is especially susceptible to such mechanical interactions and is a recognised site for stent-related complications [1,10]. Intravascular imaging would have offered valuable mechanistic insights; however, it could not be performed due to logistical constraints. Although the absence of intravascular imaging

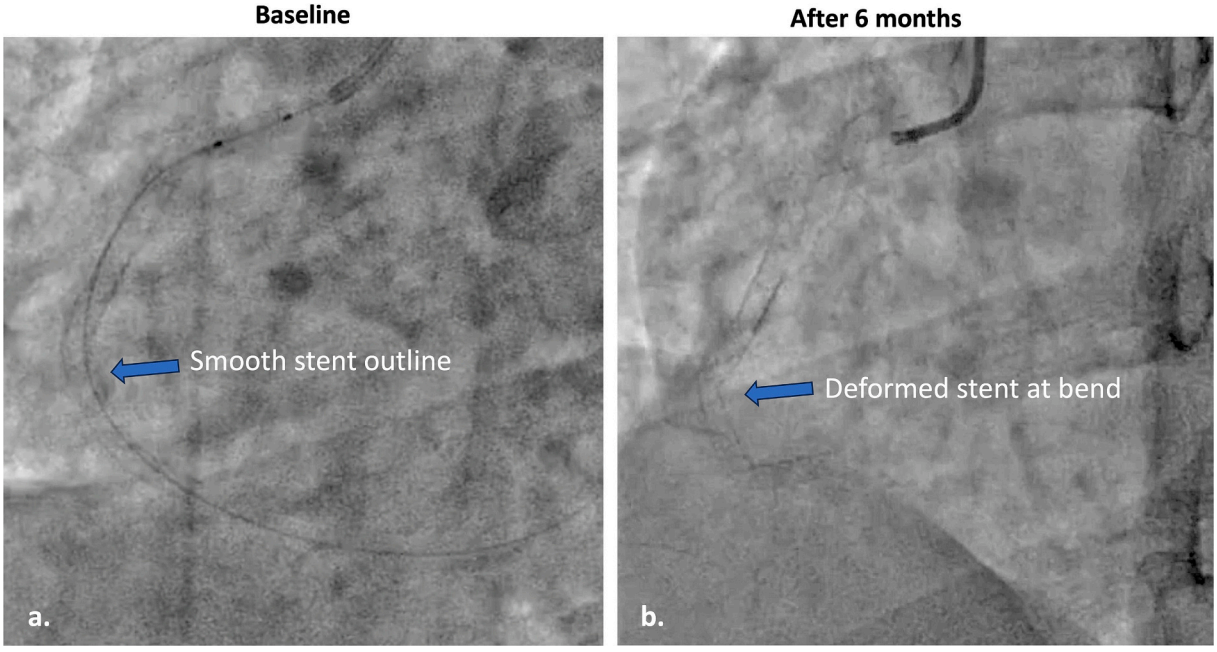

Fig. 3. Fluoroscopic image of the stents showing a deformed stent at the coronary bend (3b) compared to smooth stent counter during primary implantation (3a).

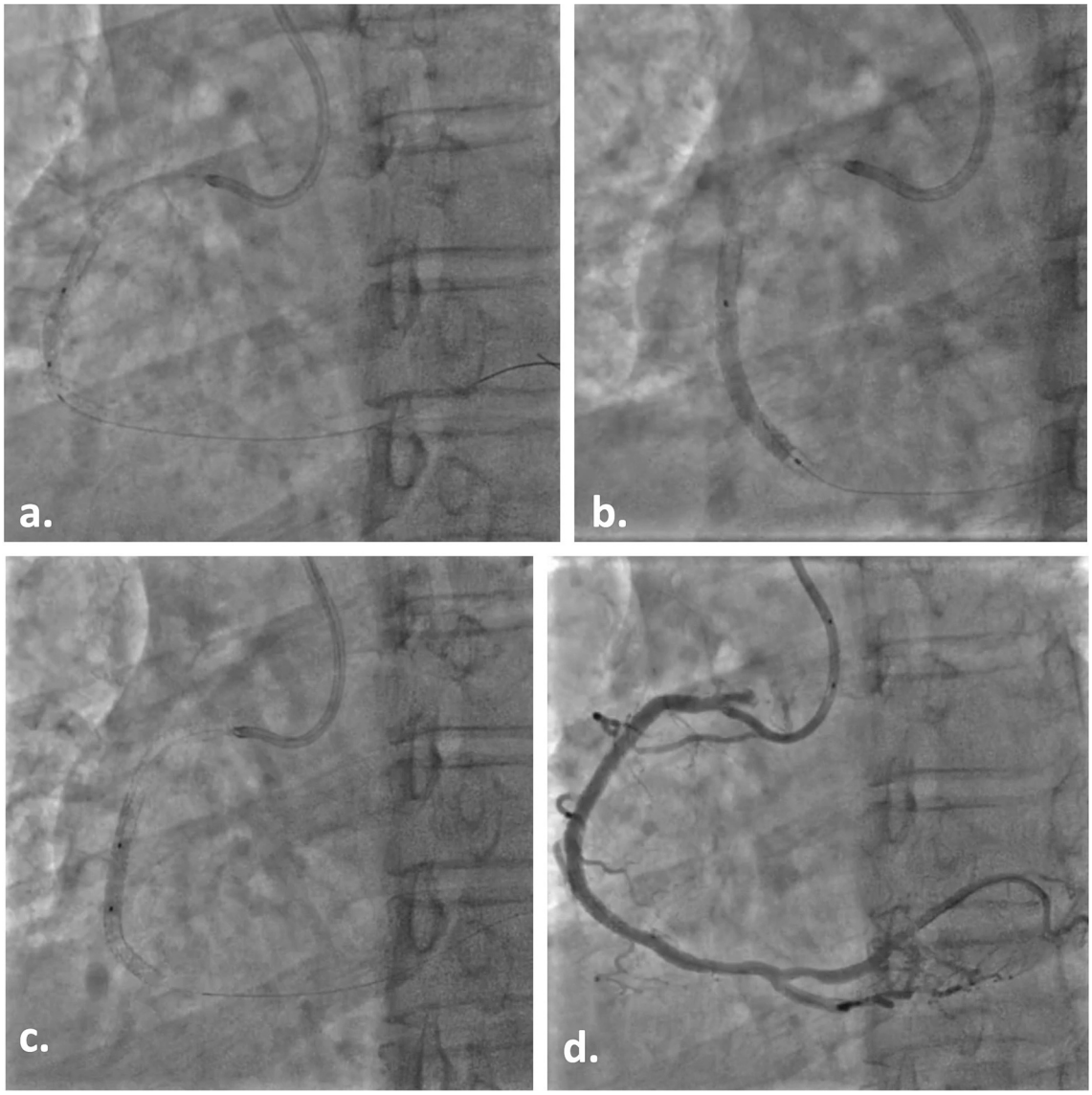

Fig. 4. Redo PTCA procedure: high-pressure predilation (4a), stenting across the ISR segment (4b), postdilation (4c) and final angiographic result (4d).

represents a limitation of this case report, the fluoroscopic images convincingly demonstrate stent deformation at the coronary bend.

Overall, this case reinforces the concept that while ultrathin-strut DES offer important advantages, lesion-specific mechanical demands should guide stent selection, particularly in sharply angulated or highly mobile coronary segments.

4. Conclusion

This case illustrates significant focal in-stent restenosis occurring at a coronary bend following implantation of an ultrathin-strut DES in the right coronary artery. While ultrathin-strut stents offer substantial advantages, their performance may be influenced by complex coronary geometry. Careful lesion assessment, appropriate stent selection, and adjunctive intravascular imaging may be particularly important when treating sharply angulated coronary segments.

CRediT authorship contribution statement

Asharam Panda: Conceptualization. **Sumit Kumar:** Data curation. **S.S. Mishra:** Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

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