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Kasuistika | Case report

Stent thrombosis due to stent fracture in heavily calcified right coronary artery

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ABSTRACT

Coronary stent fracture is an often unrecognized cause of target vessel failure, however, it has been reported more frequently in the drug-eluting stent era. Clinical presentation of stent fracture may range from benign in-stent restenosis to potentially fatal acute stent thrombosis. Interventional treatment of stent thrombosis can be carried out by high pressure balloon dilatation or second stent implantation into the stented segment after thrombus aspiration. Intravascular ultrasound is mandatory in order to exclude mechanical problems in the background of the stent thrombosis and to achieve good final stent apposition and expansion. We report on a stent fracture induced stent thrombosis occurring in a highly calcified proximal right coronary artery. (Treated previously with rotational atherectomy in the middle part, but not in the aorto-ostial location.) Our case emphasizes the importance of opitimal plaque modification with rotational atherectomy in a calcified aorto-ostial segment of right coronary artery to prevent long term complications such as stent thrombosis or restenosis due to stent fracture.

Klíčová slova:
Fraktura stentu
Intravaskulární ultrazvuk
Rotační aterektomie
Trombóza stentu

SOUHRN

Často nerozpoznanou příčinou uzávěru koronární tepny je zlomení koronárního stentu; v éře lékových stentů je však tato příhoda stále častějším tématem článků. Klinicky se fraktura stentu projevuje různě – od benigní restenózy ve stentu až po potenciální fatální akutní trombózu stentu. Intervenčně lze trombózu stentu řešit dilatací balonku s použitím vysokého tlaku nebo implantací dalšího stentu do postiženého segmentu po odsátí trombu. K vyloučení mechanických příčin trombózy stentu a k zajištění potřebného usazení a roztažení stentu je naprosto nutné provést intravaskulární vyšetření ultrazvukem. V naší kasuistice popisujeme trombózu stentu vyvolanou frakturou stentu v těžce kalcifikovaném segmentu proximální pravé koronární tepny (již dříve řešenou rotační aterektomií ve středním úseku, ne však v oblasti odstupu koronárních tepen z aorty). Naše kasuistika podtrhuje význam optimálního zmenšení plátů rotační aterektomií v oblasti kalcifikovaného odstupu pravé věnčité tepny ve snaze zabránit vzniku dlouhodobých komplikací, např. trombózy stentu nebo restenózy v důsledku zlomení stentu.

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Introduction

Drug-eluting stents (DES) have mostly replaced bare-metal stents (BMS) as a safe and effective therapeutic modality for treating coronary stenosis. Recently, stent fracture (SF) has been suggested as one of the causes of restenosis (SR) [1] and stent thrombosis (ST) after DES placement [2]. As it is known from SIRIUS trial, patients with reported SFs have much higher lesion complexity, including extensive calcification, angulation ≥ 45°, lesion length ≥ 20 mm, proximal vessel tortuosity, total occlusions, and ostial location [3]. The presence of calcium deposits within the coronaries greatly increases the potential for complications during revascularization procedures such as coronary artery rupture, dissection and thrombosis [4]; however, coronary calcification in DES era is not associated with higher restenosis or stent thrombosis rate [5]. Calcified lesions can be treated with cutting balloon angioplasty (CBA) [6] and rotational atherectomy (RA) [7,8]. We report a case of SF which resulted in ST in a patient previously undergoing RA and stent implantation of two different segments in a highly calcified right coronary artery (RCA).

Case description

A 82-year-old male patient with hypercholesterolemia, hypertension and previous inferior myocardial infarction was referred to our University Hospital with unstable angina class IIC (classified by the Braunwald classification). Coronary angiography revealed a critical stenosis in the mid and a significant calcified lesion in the proximal and ostial part of the RCA (Fig. 1C). Left coronary artery showed significant calcified left anterior descendent (LAD) and intermedius artery (IM) stenosis (Fig. 1A). The patient was consulted for bypass grafting, which was declined due to distal coronary calcification. Due to therapy resistent rest pain, firstly the LAD lesion was dilated with acceptable result, but stent implantation was impossible due to severe coronary calcification (Fig. 1B). RCA stenosis was impossible to cross in the mid part with an 1.25×15 Ryujin ballon (Terumo, Japan) therefore the patient was called back for an RA. The RCA ostium was engaged with a 7 Fr guiding catheter (Mach1 FR 4.0, Boston Scientific Scimed, Maple Grove, MN) using the standard femoral approach. A Rotawire ES (Boston Scientific Scimed, Maple Grove, MN) was used to cross the lesion. RA was performed with an 1.5 and 1.75 Burr in the mid part of the RCA. After RA, two stents were implanted (one in the middle and one in the proximal RCA at high pressure [Taxus 3.5×32 and 3.5×24, Boston Scientific Scimed, Maple Grove, MN]) without covering the ostial segment. Final angiography showed no residual stenosis (Fig. 1D). Two years later the patient presented with unstable angina again. Repeated coronary angiography showed proximal LAD occlusion and significant ostial RCA stenosis (Figs. 2A, 2C). As cardiac surgery was still declined due to extreme calcification of the outflow tract, as an alternative option the LAD was stented with a Coroflex Blue stent 2.5×12 mm (Braun, Germany) (Fig. 2B) and the RCA ostium was stented with a Taxus 4×12 mm stent (Boston Scientific Scimed, Maple Grove, MN) after balloon predilatation (Fig. 2D). Twelve months later the patient presented once again with unstable angina. LAD was occluded below the diagonal branch while the previously deployed stent was patent. The proximal right coronary artery stent

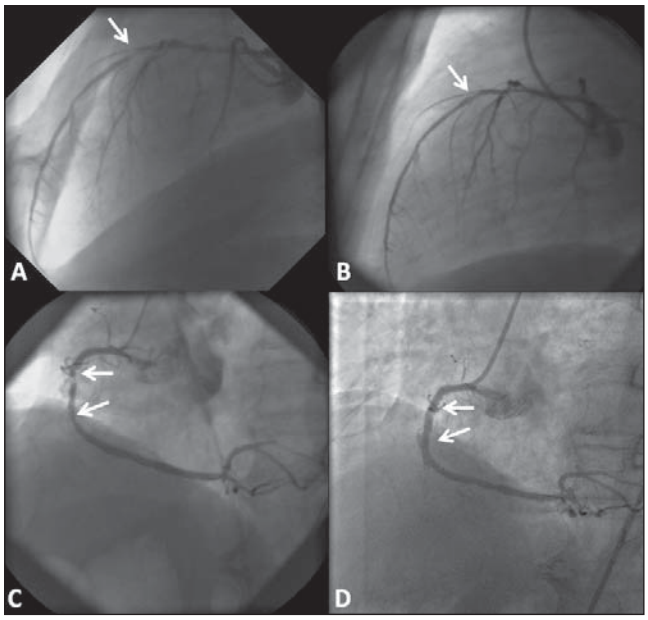

Fig. 1 – Coronary angiography revealed a calcified significant stenosis in the proximal LAD and intermedius artery (white arrows) (A). Coronary angiography after high pressure balloon angioplasty in the proximal LAD (B). Coronary angiography showed a critical, heavily calcified stenosis in the proximal and a significant stenosis in the mid segment of the dominant RCA (white arrows) (C). Coronary angiography after RCA rotational atherectomy and dual stenting (white arrows) (D).

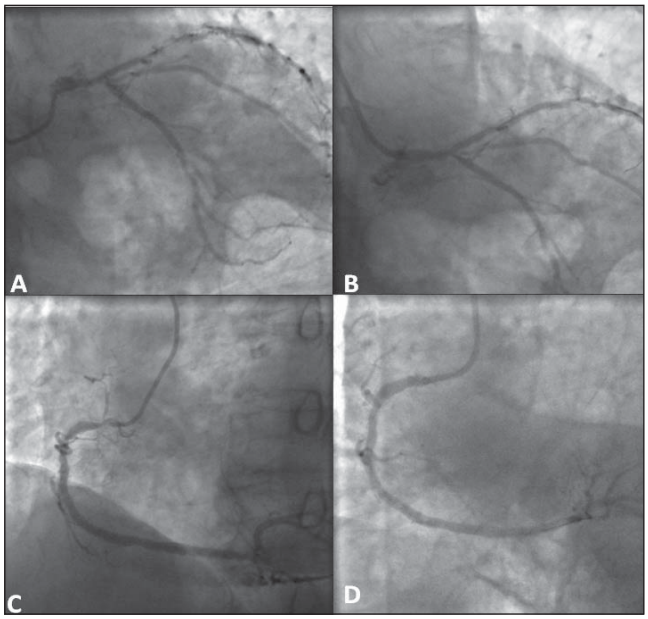

Fig. 2 – Left coronary angiography before and after LAD balloon angioplasty (A, B). Right coronary angiography before and after second stent implantation (C, D).

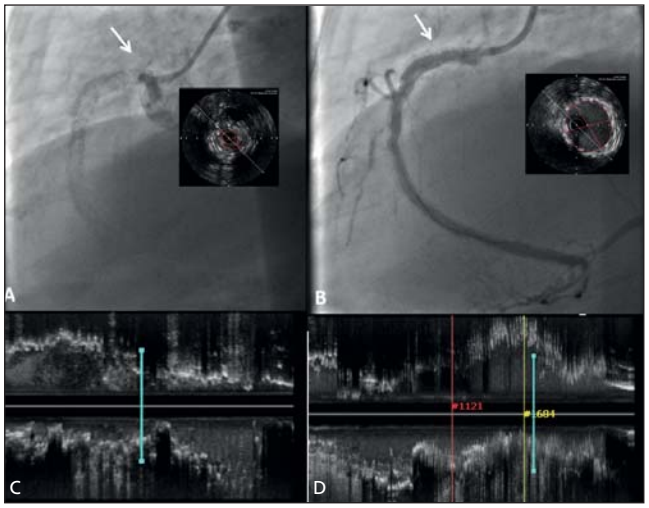

Fig. 3 – Right coronary angiography: complete occlusion with TIMI 0 flow; IVUS longitudinal and cross section pictures: stent fracture in the proximal stent (A). (D) shows TIMI 3 flow with no residual stenosis after thrombus aspiration, high pressure balloon angioplasty and stenting. IVUS picture shows good stent apposition and expansion in the proximal RCA (Stent CSA: 17 mm^2) (Fig. 4B).

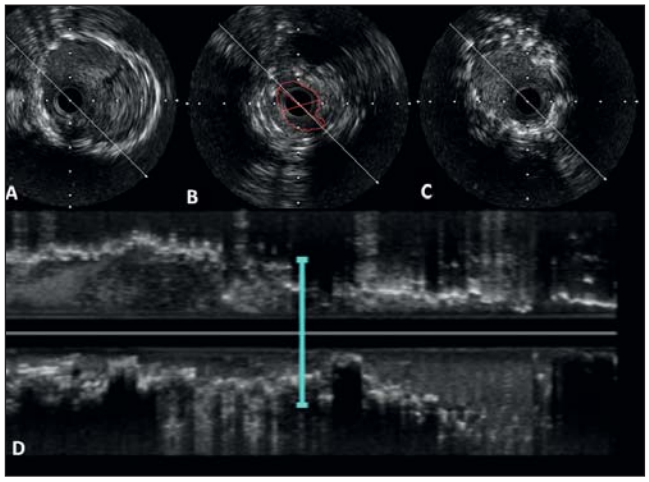

Fig. 4 – Cross sectional IVUS pictures show a distal intraaluminal thrombus (A), stent fracture in the middle part (B) and stent recoil or underdeployed stent in the proximal part (C). Cross sectional image shows the stent fracture in the middle part (blue line) (D).

showed critical in-stent restenosis. After the angiography, the RCA was dilated with a non-compliant balloon (NC Mercury 4×20 mm^2, Abbot, USA) and it was stented with a second generation DES (Promus 4×15 mm^2, Boston Scientific Scimed, Maple Grove, MN). Four months later the patient was admitted with inferior wall STEMI. ECG showed significant ST segment elevation in inferior leads. Coronary angiography revealed stent fracture grade V according the Nakazawa [9] classification and RCA occlusion in the proximal stent with TIMI 0 flow (Fig. 3A), while left coronary artery showed the same angiographic picture as before. After thrombus aspiration the flow was restored, but a critical stenosis was visible in the proximal stent (Fig. 3C). By the fluoroscopic image the possibility of a stent fracture emerged as an underlying mechanism of the stent thrombosis. Stent fracture (grade V) was proven by IVUS examination in the proximal stent (Figs. 3A, 3B, Figs. 4A–D). Two balloon dilatations were performed (2.5×15 and 3.5×20 Maverick, Boston Scientific Scimed, Maple Grove, MN) and finally the lesion was covered with a BMS (Liberte 4×24) (Fig. 3B). Postprocedural IVUS examination showed good stent expansion and apposition (Fig. 3B). One year after the procedure, the patient remained asymptomatic and at the mean time being treated with aspirin and clopidogrel.

Discussion

Complex coronary lesions (calcified, long) have better outcomes with RA and BMS implantation than with stenting without prior plaque modification [7], however, Clavijo et al. published the same results with RA as conventional DES implantation in the treatment of calcified coronary lesions [10]. Rathore et al. compared the restenosis, success rate and the in-hospital outcomes after RA with BMS compared with DES implantation and authors have found significantly better restenosis rate with DES implantation (11% vs. 28.1%, $p < 0.001$) [11]. With the introduction of DES the rate of restenosis has been significantly reduced but a new concern, the risk of ST has emerged. Previous studies reported a negative impact of severely calcified lesions on stent expansion [12]. IVUS has been shown to implant stents better so IVUS guided DES implantation is an attractive supposition. However, only retrospective studies exist of IVUS guided DES implantation. Roy et al. [13] have matched the IVUS and angiography guided DES implantations in 884 patients, and the authors have found that IVUS guided DES implantation underwent less direct stenting, more post-dilation, and had greater CBA and RA use. At 30 days and at 12 months, a higher rate of definite stent thrombosis was seen in the no IVUS group (0.5 vs. 1.4%; $p = 0.046$) and (0.7 vs. 2.0%; $p = 0.014$), respectively. Features on IVUS found to be associated with subacute ST included stent under-expansion, incomplete stent apposition (ISA), inflow/outflow disease, dissection, thrombus, and tissue prolapse [14]. Acute ISA is mostly technique dependant and may be due to suboptimal stent implantation or by severely calcified lesions not allowing for homogeneous stent expansion and resulting in localized stent underexpansion and ISA [15]. In these IVUS findings confirmed that the mechanisms underlying subacute stent thrombosis were mechanical and potentially treatable when identified. Recently optical coherence tomography (OCT) was introduced into clinical practice, which allows better visualization of the struts and clarifies better the apposition of the stents. With 3D reconstruction of the OCT image, SF can be also visualized [16]. Despite the better OCT images, there are no randomized studies confirming, that OCT guidance is better than IVUS guidance for DES implantation. The main goal of RA is to remove the superficial calcium in calcified lesions, which results in better stent expansion. Fujimoto et al. published lower restenosis and subacute ST rate (restenosis rate, 17.4% vs. 17.4%, $p = 0.061$; subacute thrombosis rate, 0% vs. 7.1%, $p = 0.31$) in hemodialized patients with RA than with traditional angioplasty with high-pressure

balloon inflation [17]. The potential mechanism of SF in patients without RA was the use of high pressures, which damaged the stent structure and detached the polymer coating from the struts. Debulking of calcification by RA should enable an adequate expansion of the stents with low pressure, and avoid the fracture of the struts. Our patient highlights this mechanism because first rotational atherectomy was performed in the middle part of the right coronary artery, but the proximal part was treated only with high pressure balloon inflation. The vessel size was too big for RA, but the plaque modification with CBA could be an option. Furuichi et al. [18] have compared the CBA with conventional high pressure balloon dilatation followed by RA and it was found, that in the ROTACUT group the final minimum stent cross-sectional area (CSA) was significantly larger compared to the control group (6.80 ± 1.27 mm² vs. 5.38 ± 1.89 mm²; $p = 0.048$). Other possible mechanisms of the SF include increased axial rigidity of the overlapping stent segments, higher radial forces in lengthy stents, and having the stent placed at a hinge point [17]. Sang-He Lee et al. reported SF as one of the leading roles of stent restenosis. The authors explain the possible mechanism of SF by the poor neointimal hyperplasia and the cardiac movement [19]. Compared to previous reports, our case was observed in proximal RCA location without severe angulation. The possible mechanism of the stent fracture in our case is the chronic wall stress and focal severe calcification causing a stent disruption, but even a relevant longitudinal shortening of the Pt–Cr may be considered. In our patient ST occurred in the nonatherectomized site in the RCA, but not in the lesion which had undergone RA. Therefore the case highlights the preventive effect of the RA on SF by reducing the amount of the superficial calcium.

Conclusion

Our case suggests that DES fracture should be treated with stent in stent implatation with greater radial force. This report emphasizes the role of massive intracoronary calcium as an inducer of stent fracture and the role of calcium debulking in SF prevention.

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