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Potent Stent-Less Procedure Using Rotational Atherectomy and Drug-Coated Balloon to Right Coronary Ostial Lesion

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ABSTRACT

Background: Even in the drug-eluting stent era, ostial lesion of the right coronary artery (RCA) still remains therapeutic challenge for interventional cardiologists. **Case Series** Case 1 (76 y.o. male) with angina on effort underwent transradial stent-less percutaneous coronary intervention (PCI) using rotational atherectomy (RA) followed by drug-coated balloon (DCB) dilation alone (RA/DCB) against a calcified *de novo* RCA ostial lesion. Case 2 (86 y.o. female) with recurrent unstable angina and hemodialysis underwent transfemoral RA/DCB against a severe repeat in-stent restenosis probably due to calcified nodule in the RCA ostium. In the both patients, PCI was successfully completed under intravascular ultrasound imaging (IVUS) guidance without complications. Follow-up CAG performed 4–5 months after the procedure revealed no significant lumen narrowing in the both RCA ostial lesions.
Conclusions: The both cases suggest that stent-less PCI using RA/DCB under IVUS might be an alternative revascularization therapy of choice for calcified RCA ostial lesions.

Contents

1. Introduction

Drug-eluting stent (DES) implantation has been a mainstay of the interventional treatment of coronary artery disease; however, ostial lesion of right coronary artery (RCA) has a high risk for DES failure, such as in-stent restenosis (ISR) and target lesion revascularization (TLR) [1–3]. Even in the DES era, the RCA ostial lesion still therefore remains therapeutic challenge for interventional cardiologists. Nowadays stent-less percutaneous coronary intervention (PCI) using rotational atherectomy (RA) followed by drug-coated balloon (DCB) dilation (RA/DCB) under DES-unsuitable conditions has gained more interest [4–7]. In this mini-review, we have focused on utility of the stent-less PCI using RA/DCB for the RCA ostial lesion in a case series.

1.1. Case 1: de novo calcified RCA ostial lesion

A 76-year-old man with past smoking and hypertension was referred to our hospital due to angina on effort. Electrocardiography at admission was normal and transthoracic echocardiography showed no asynergy. Adenosine-stress myocardial perfusion scintigraphy with thallium depicted rest-redistribution of the inferoposterior-posterolateral wall perfusion. Right coronary angiography (CAG) indicated a severe stenosis in the distal segment in addition to an intermediate calcified stenosis at the ostium (Fig. 1A). Left CAG showed an

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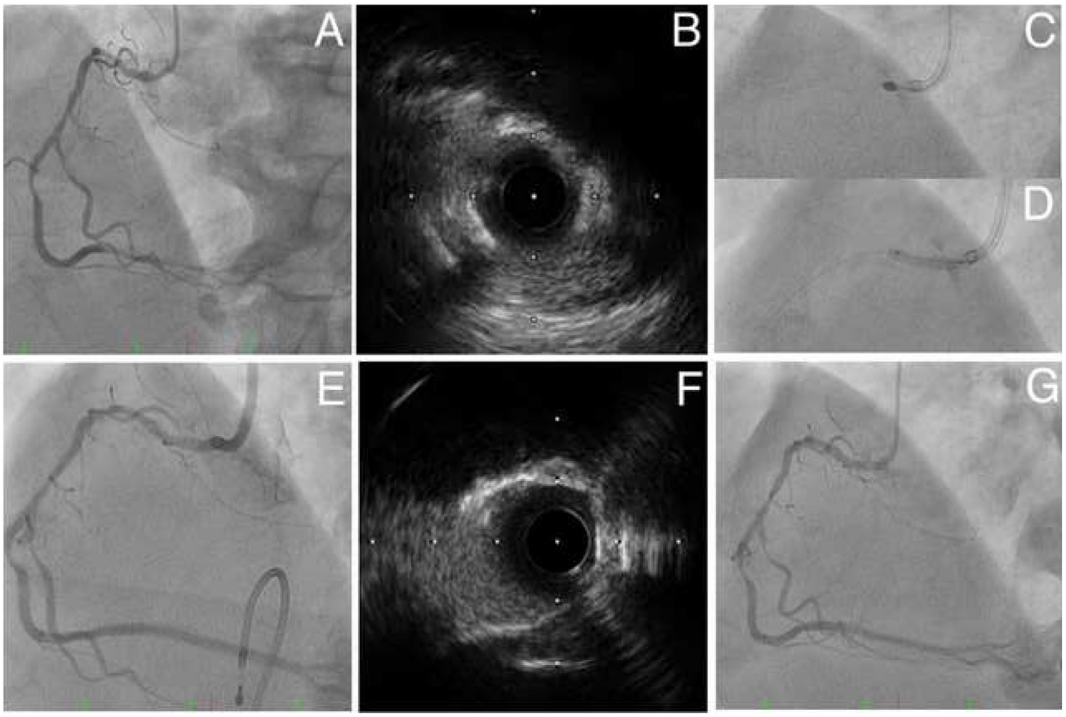

Fig. 1. Case 1. Rotational atherectomy (RA) followed by drug-coated balloon (DCB) dilation (RA/DCB) for calcified RCA ostial lesion (Panels A, C, D, E, G, right anterior view). Panel A initial CAG showed a moderate stenosis in the RCA ostium in addition to a severe stenosis in the distal RCA. Panel B during the PCI for distal RCA lesion, IVUS depicted an almost circumferential calcification at the RCA ostium. Panel C ablation with a 2.0 mm burr. Panel D adjunctive 3.0-mm DCB dilation. Panel E post-procedural CAG. Panel F post-procedural IVUS showed a RCA ostial lumen enlargement. Panel G five-month follow-up CAG revealed no significant vessel narrowing in the RCA ostial lesion.

intermediate calcified stenosis in the proximal segment of left anterior descending coronary artery (LAD), and a severe stenosis in the distal segment of left circumflex coronary artery (LCx). Subsequent fractional flow reserve (FFR) measurement revealed 0.75 at the distal LAD. Taking the myocardial scintigraphic, CAG, and FFR findings into consideration, we planned to perform transradial PCI against the distal RCA lesion and the proximal LAD lesion. Firstly, we implanted an everolimus-eluting stent (EES) (Synergy, 2.25/32 mm, Boston Scientific) in the distal RCA lesion under intravascular ultrasound imaging (IVUS) guidance. During the procedure, we confirmed a significant vessel narrowing with almost circumferential calcification at the RCA ostium by IVUS (Fig. 1B). Secondly, 20 days later, we performed RA (Rotalink Plus, 1.5/2.0 mm burrs, Boston Scientific), inflated a cutting balloon (3.0/10 mm), and implanted an EES (3.0/24 mm) in the proximal LAD lesion.

Thirdly, 4 months later, taking the IVUS findings and risk of stent under-expansion/ISR into consideration, we planned to avoid ostial stenting and perform stent-less PCI using RA/DCB for the calcified RCA ostial lesion. Using a 7-Fr guiding catheter (Launcher, SAL 0.75 SH, Medtronic) *via* left radial artery and a rotawire extrasupport, we performed RA with a 2.0 mm burr (initial 180,000 rpm) under IVUS guidance (Fig. 1C) and started a temporary pacing because of intermittent bradycardia due to atrio-ventricular block. We continued RA, modified burr speed gradually down to 140,000 rpm by 20,000 rpm, and then inflated a DCB (SeQuent Please, 3.0/15 mm, B. Braun) at 7 atm for 60 s (Fig. 1D). Final CAG and IVUS showed an acceptable result without flow delay (Fig. 1E) and a relatively smooth luminal surface without dissections (Fig. 1F), respectively. Five-month follow-up CAG revealed no significant vessel narrowing in the RCA ostial lesion (Fig. 1G).

1.2. Case 2: in-stent calcified RCA ostial lesion

An 86-year-old woman with hemodialysis was transferred to our hospital due to recurrent unstable angina. She had undergone an axillo-bifemoral bypass surgery for calcified stenosis of bilateral just proximal common iliac arteries 4 years ago, and repeat transbrachial PCIs twice for RCA ostial lesion 90 days and 50 days before (Fig. 2). In spite of presence of almost circumferential calcification on IVUS, we did not perform RA during the both prior PCI procedures (Fig. 2), because the residual bilateral iliac artery disease did not allow support device usage when needed. In the first PCI, we pre-dilated with a scoring balloon catheter at 16 atm, implanted an EES (2.75/24 mm) at 18 atm from the RCA ostium to the proximal segment, and post-dilated with a 3.0 mm noncompliant balloon catheter at 22 atm (Fig. 2A, B, E, F, I, J). In the second PCI for the RCA ostial ISR, we dilated with a 3.0 mm noncompliant balloon catheter at 20 atm, and then inflated a DCB (3.5/15 mm) at 9 atm for 60 s; however, final IVUS showed a residual calcified nodule inside the stent (Fig. 2C, D, G, H, K, L).

In the current procedure, in order to employ a support device when needed during RA-based PCI, we planned to perform endovascular intervention for left iliac artery before CAG/PCI. After the endovascular intervention, we performed CAG *via* left femoral artery. Right CAG confirmed a recurrent severe ISR at the RCA ostium and diffuse stenosis in the proximal-mid segment with flow competition (Fig. 3A, white arrows) in the distal segment, while left CAG showed no interval change, but delineated collaterals to the distal RCA. Pre-procedural IVUS depicted concentric superficial high-echoic mass with significant shadowing inside the stent, suggesting the RCA ostial ISR due to calcified nodule (Fig. 3B, C). Using a 7-Fr guiding catheter (Launcher, SAL 0.75 SH, Medtronic) and a rotawire extrasupport, we performed RA from the RCA ostial ISR to the mid portion with a 1.5 mm burr (initial 200,000 rpm) under IVUS guidance (Fig. 3D, Supplementary Fig. 1). Then, we added RA for the RCA ostial ISR with a 2.0 mm burr (initial 160,000 rpm) and started a temporary pacing because of intermittent bradycardia (Fig. 3E, Supplementary Fig. 1). Unexpectedly, the IVUS catheter entrapment around the distal edge of the old EES occurred just after ablation with 2.0 mm burr. After successful retrieval of the IVUS catheter, we did not attempt repeat IVUS examinations thereafter. After dilation at the ostial ISR with a 3.5 mm cutting balloon catheter (Fig. 3F), we advanced a guide extension catheter, and dilated with a 2.25 mm scoring balloon catheter at 14–20 atm in the proximal-mid

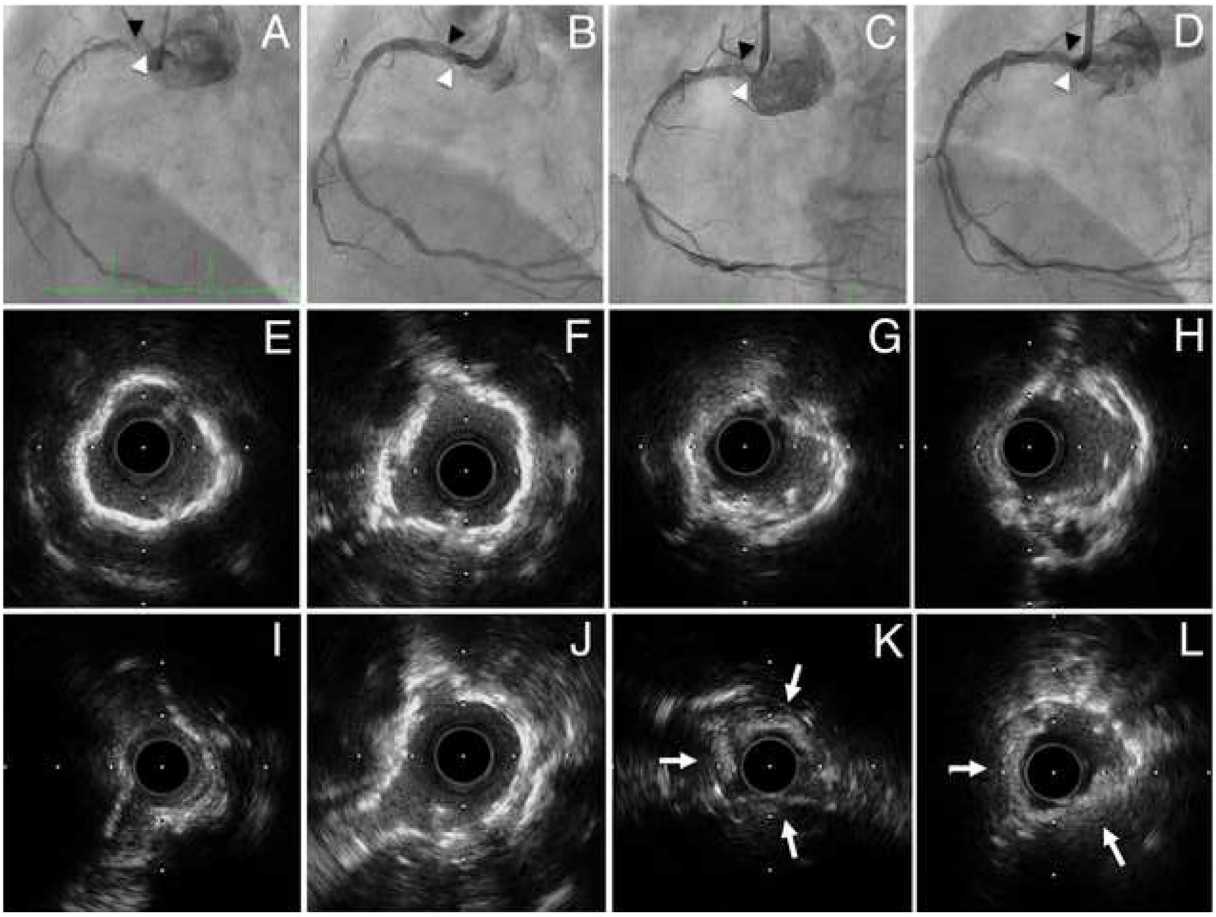

Fig. 2. Case 2. First percutaneous coronary intervention (PCI) using DES for calcified RCA ostial lesion (Panels A, B, E, F, I, J) and second PCI using DCB for calcified RCA ostial ISR (Panels C, D, G, H, K, L). Pre-procedural (Panels A, C) and post-procedural CAG (Panel B, D). White arrowheads and black arrowheads indicated RCA ostium and just distal to ostium, respectively (Panels A–D, left anterior view). Pre-procedural (Panels E, I corresponding to white arrowhead, black arrowhead, respectively in Panel A; Panels G, K corresponding to white arrowhead, black arrowhead, respectively in Panel C), and post-procedural IVUS images (Panels F, J corresponding to white arrowhead, black arrowhead, respectively in Panel B; Panels H, L corresponding to white arrowhead, black arrowhead, respectively in Panel D). IVUS images at the second PCI showed calcified nodule (white arrows) inside the stent just distal to ostium (Panels K, L).

segment (Fig. 3G). Subsequently we inflated a DCB (SeQuent Please, 2.5/30 mm, B. Braun) at 10 atm for 60 s in the mid segment (Fig. 3H), and another DCB (SeQuent Please, 2.5/20 mm, B. Braun) at 14 atm for 60 s in the proximal segment (Fig. 3I). Then we performed a DCB (SeQuent Please, 4.0/20 mm, B. Braun) dilation at 12 atm for 60 s against the ostial ISR lesion (Fig. 3J), and final CAG showed an acceptable result (Fig. 3K). Four-months follow-up CAG revealed no significant in-stent lumen narrowing in the RCA ostium (Fig. 3L). During the follow-up CAG, we could not advance a pressure wire to the distal segment of the RCA, and did not exam a FFR. Instead, we performed an instantaneous wave-free ratio (iFR) pullback study from the mid RCA (bifurcation of right ventricular branch), and iFR values at bifurcation of right ventricular branch, just distal to the RCA ostium, and aorta were 0.94, 0.97, and 0.98, respectively. Thus, we could not rule out the possibility of residual myocardial ischemia in the RCA territory; however, taking the small drop (0.01) in iFR at the RCA ostium into consideration, we judged that the RCA ostial in-stent lesion itself was not severe, and did not perform further repeat PCI for the RCA ostium.

2. Discussion

Interventional treatment for calcified RCA ostial lesions still has a therapeutic limitation even in the modern DES era. Aorto-ostial lesions are histopathologically reported to be heavily calcified, fibrotic, and sclerotic [8,9]. In addition, the distribution of surrounding muscle bundles differs between the RCA ostium and left main (LM) ostium [10]. Muscle bundles of the RCA originate independently of the elastin-muscle fibers of the aorta and a large circumferential sphincter-like muscle mass frequently exists at the RCA ostium, while the LM ostium has elastin-muscle fibers similar to and continuous with those of the aorta [10]. Moreover, the adventitia of the RCA ostium is thicker than that of the LM ostium [10]. In addition to heavily calcified plaque itself and hinge motion around the ostium, these histopathological characteristics of surrounding muscle fibers described above might lead to a stent under-expansion, a stent recoil, and a stent fracture, resulting in refractory ISR and repeat TLR in RCA ostial lesions. Indeed, stenting in RCA ostial lesions had a 10 times higher risk of TLR than that in LM ostial lesions [2]. Moreover, the under-expanded stent becomes a just residual small metal cage, and tends to interfere with future repeat PCI.

A previous report regarding first-generation sirolimus-eluting stent implantation at RCA ostial lesions indicated 13.5% of TLR rate at 8 months [1], while a recent report from Japanese expert PCI operators has demonstrated that TLR rate of RCA ostial lesions was 5.4% at 1 year after second-generation biolimus-eluting stent implantation [11]. In the latter report, all the TLR cases had shorter length between RCA ostium and preprocedural minimum lumen diameter site (mean 2.3 mm) [11]. According to another report from j-Cypher registry, overall patients undergoing first-generation sirolimus-eluting stent implantation at RCA ostial lesions had 17.5% and 28.2% of TLR rate at 1 year and 5 years, respectively, and end-stage renal disease on hemodialysis as well as ISR lesion were independent risk factors for TLR within 1 year in those patients [3]. Thus, 4–5 months interval between initial PCI

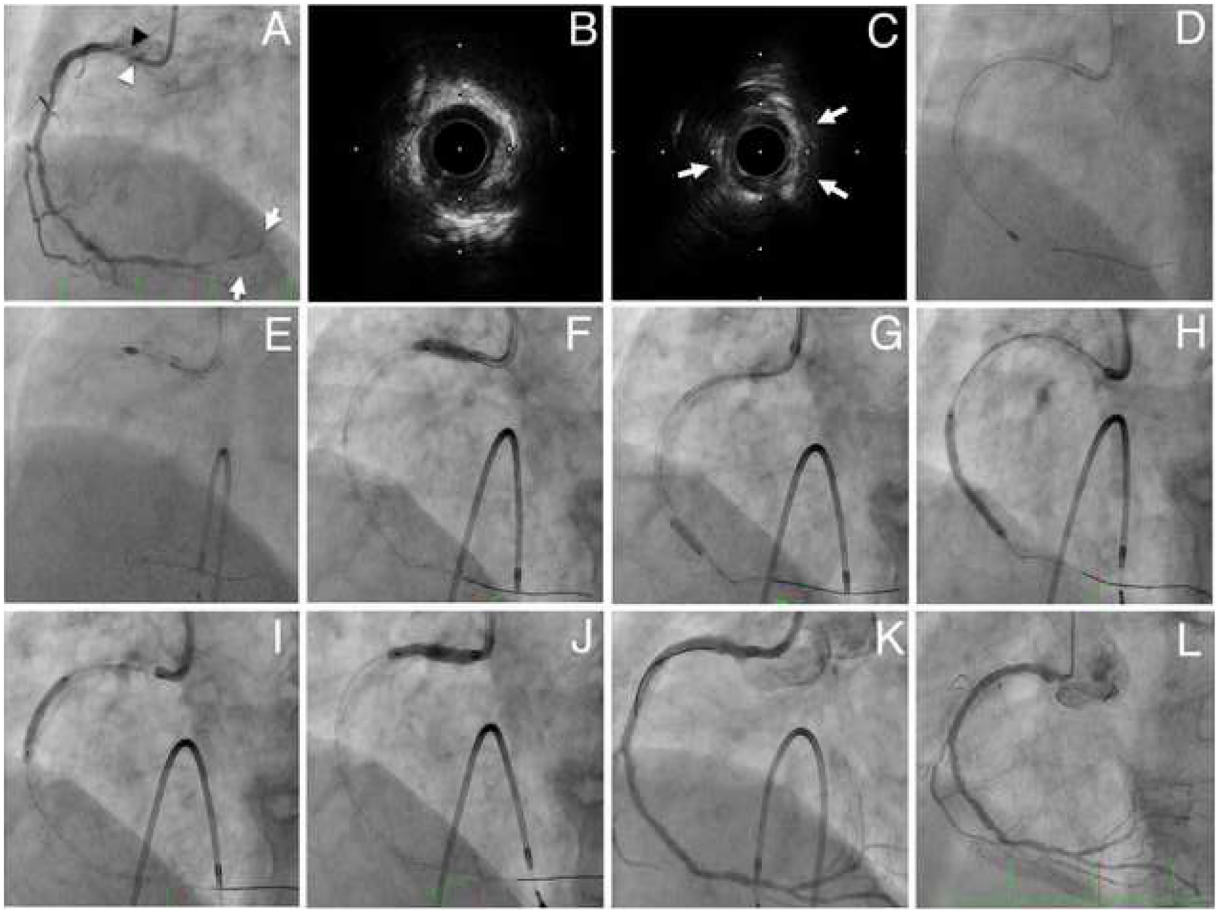

Fig. 3. Case 2. Third PCI using RA/DCB for the recurrent ISR at calcified RCA ostium. Panel A pre-procedural CAG revealed a recurrent severe ISR at the RCA ostium and diffuse stenosis in the proximal-mid portion. White arrowhead and black arrowhead indicated RCA ostium and just distal to ostium, respectively (Panel A, left anterior view). Panels B, C pre-procedural IVUS images (Panels B, C corresponding to white arrowhead, black arrowhead, respectively in Panel A) depicted a lumen narrowing due to surrounding calcified nodule (white arrows) inside the stent. Panel D ablation with a 1.5 mm burr from the RCA ostium to the mid segment. Panel E ablation with a 2.0 mm burr for the RCA ostium. Panel F a 3.5 mm cutting balloon dilation for the RCA ostium. Panel G guide extension catheter (GEC)-supported dilation with a 2.25 mm scoring balloon in the proximal-mid segment of RCA. Panels H, I GEC-supported dilation with 2.5 mm DCBs in the proximal-mid segment of RCA. Panel J dilation with a 4.0 mm DCB in the RCA ostium. Panel K post-procedural CAG showed an acceptable result. Panel L four-months follow-up CAG revealed no significant vessel renarrowing in the in-stent RCA ostial lesion.

and follow-up CAG might be short for the present cases, and longer follow-up period (6–9 months) would be more appropriate particularly in case 2. Even in the new-generation DES era, stent-less PCI might be an option of interventional treatments for the calcified RCA "just" ostial lesion.

As a primary interventional treatment for ISR, usefulness of DCB has already been established [12]. In addition, even for *de novo* coronary artery lesions, several registries and small-sized randomized controlled trials comparing with DES have indicated acceptable results regarding safety and efficacy of DCB particularly among small vessel *de novo* lesions [13–16]. During the DCB-alone procedure, predilation before DCB is essential, and RA, which tends not to cause acute recoil or major dissection but to debulk thickened intima and make a relatively smooth luminal surface, might be one of suitable pre-treatments before DCB. Potent candidates undergoing RA/DCB procedures had lesion/clinical characteristics unsuitable for DES implantation, such as undilatable/diffuse calcified lesion, bifurcation lesion, coronary sequelae of Kawasaki disease, and bleeding tendency [4–7]. A recent report from Finnish investigators regarding RA/DCB procedure in patients with calcified *de novo* coronary artery lesions has shown no acute closures of the target vessels as well as 1.5% and 3.0% of ischemia-driven TLR at 1 and 2 years, respectively [5]. Stent-less PCI using RA/DCB could therefore be a revascularization therapy of choice as a first attempt for the *de novo* calcified RCA ostial lesion.

On the technical point of view, there are no established data to demonstrate that low-speed RA is superior to high-speed RA. Low-speed RA might have higher frequency of complications, such as burr stuck or coronary perforation, than high-speed RA. However, as to incidence of slow flow and other complications, there were no significant difference between low-speed (140,000 rpm) RA and high-speed (190,000 rpm) RA [17]. Since there is a possibility that low-speed RA might yield larger lumen than high-speed RA, we therefore endeavored to obtain larger lumen as much as possible by "gradual" low-speed RA under IVUS in case 1. Taking possible risk related to low-speed RA into consideration, we carefully advanced burrs little by little using a slow-bumping motion, and modified burr speed gradually from 180,000 rpm down to 140,000 rpm by 20,000 rpm during each ablation in case 1. In contrast, in ISR lesions like case 2, we should avoid low-speed RA procedure, because of high risk of burr stuck.

Calcified nodule-associated ISR has also been becoming a significant problem even in the new-generation DES era [18,19]. This type of ISR tends to be resistant to DCB (DCB failure), and frequently requires repeat PCI, as shown in case 2. A recent report has shown that calcified nodule was observed more frequently at the ostial and mid RCA, compared with other coronary locations [20]. In addition, hemodialysis, local hinge motion, and maximum calcium arc by optical coherence tomography have been associated with the presence of calcified nodule [20]. Although effective interventional treatment for ISR originating from calcified nodule has never been established; however, debulking

by RA prior to DCB dilation might be an appropriate revascularization strategy for those ISR.

3. Conclusion

Stent-less PCI using RA followed by DCB dilation under IVUS could be a candidate of revascularization therapy for calcified RCA ostial *de novo*/ISR lesions.

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Disclosures

The authors have no conflicts of interest regarding the content of the manuscript.

References

[1] Sakamoto H, Ishikawa T, Mutoh M, Imai K, Mochizuki S. Angiographic and clinical outcomes after sirolimus-eluting stent implantation to de novo ostial lesion of the right coronary artery: a retrospective study. Circ J 2008;72:880–5.
[2] Luz A, Hughes C, Magalhães R, Bisceglia T, Descoutures F, Tamamm K, et al. Stent implantation in aorto-ostial lesions: long-term follow-up and predictors of outcome. EuroIntervention 2012;7:1069–76.
[3] Ko E, Natsuaki M, Toyofuku M, Morimoto T, Matsumura Y, Oi M, et al. Sirolimus-eluting stent implantation for ostial right coronary artery lesions: five-year outcomes from the j-Cypher registry. Cardiovasc Interv Ther 2014;29:200–8.
[4] Shiraishi J, Matsubara Y, Yanagiuchi T, Shikuma A, Shoji K, Nishikawa M, et al. Rotational atherectomy followed by drug-coated balloon dilation in possible coronary sequelae of Kawasaki disease. Int Heart J 2016;57:367–71.
[5] Rissanen TT, Uskela S, Siljander A, Kärkkäinen JM, Mäntylä P, Mustonen J, et al. Percutaneous coronary intervention of complex calcified lesions with drug-coated balloon after rotational atherectomy. J Interv Cardiol 2017;30:139–46.
[6] Shiraishi J, Ohshiro M, Matsubara Y, Hyogo M, Uchiyama H, Sawada T. Optical frequency domain imaging –guided rotational atherectomy followed by drug-coated balloon dilation to the non-calcified lesion in a patient with severe thrombocytopenia. Cardiovasc Interv Ther 2018;33:395–7.
[7] Shiraishi J, Koshi N, Matsubara Y, Nishimura T, Shikuma A, Shoji K, et al. Stent-less percutaneous coronary intervention using rotational atherectomy and drug-coated balloon: a case series and a mini review. Cardiovasc Revasc Med 2018;19:705–11.
[8] Stewart JT, Ward DE, Davies MJ, Pepper JR. Isolated coronary ostial stenosis: observations on the pathology. Eur Heart J 1987;8:917–20.
[9] Popma JJ, Dick RJ, Haudenschild CC, Topol EJ, Ellis SG. Atherectomy of right coronary ostial stenoses: initial and long-term results, technical features and histologic findings. Am J Cardiol 1991;67:431–3.
[10] Boucek RJ, Takeshita R, Brady AH. Microanatomy and intramural physical forces within the coronary arteries (man). Anat Rec 1965;153:233–41.
[11] Nasu K, Oikawa Y, Habara M, Shirai S, Abe H, Kadotani M, et al, NO-RECOIL Registries Investigators. Efficacy of biolimus A9-eluting stent for treatment of right coronary ostial lesion with intravascular ultrasound guidance: a multi-center registry. Cardiovasc Interv Ther 2018;33:321–7.
[12] Byrne RA, Joner M, Alfonso F, Kastrati A. Drug-coated balloon therapy in coronary and peripheral artery disease. Nat Rev Cardiol 2014;11:13–23.
[13] Waksman R, Serra A, Loh JP, Malik FT, Torguson R, Stahnke S, et al. Drug-coated balloons for de novo coronary lesions: results from the Valentines II trial. EuroIntervention 2013;9:613–9.
[14] Wöhrle J, Zadura M, Möbius-Winkler S, Leschke M, Opitz C, Ahmed W, et al. SeQuentPlease World Wide Registry: clinical results of SeQuent please paclitaxel-coated balloon angioplasty in a large-scale, prospective registry study. J Am Coll Cardiol 2012;60:1733–8.
[15] Latib A, Colombo A, Castriota F, Micari A, Cremonesi A, De Felice F, et al. A randomized multicenter study comparing a paclitaxel drug-eluting balloon with a paclitaxel-eluting stent in small coronary vessels: the BELLO (Balloon Elution and Late Loss Optimization) study. J Am Coll Cardiol 2012;60:2473–80.
[16] Nishiyama N, Komatsu T, Kuroyanagi T, Fujikake A, Komatsu S, Nakamura H, et al. Clinical value of drug-coated balloon angioplasty for de novo lesions in patients with coronary artery disease. Int J Cardiol 2016;222:113–8.
[17] Sakakura K, Funayama H, Taniguchi Y, Tsurumaki Y, Yamamoto K, Matsumoto M, et al. The incidence of slow flow after rotational atherectomy of calcified coronary arteries: a randomized study of low speed versus high speed. Catheter Cardiovasc Interv 2017;89:832–40.
[18] Alfonso F, Cuesta J, Bastante T, Aguilera MC, Benedicto A, Rivero F. In-stent restenosis caused by a calcified nodule: a novel pattern of neoatherosclerosis. Can J Cardiol 2016;32 (830.e1-3).
[19] Yumoto K, Kanaya T, Tanaka S, Fukuzawa T, Watanabe T, Aoki H. Calcified nodule as an unsolved cause of coronary stent thrombosis in the new-generation drug-eluting stent era. Cardiovasc Interv Ther 2018;33:169–70.
[20] Lee T, Mintz GS, Matsumura M, Zhang W, Cao Y, Usui E, et al. Prevalence, predictors, and clinical presentation of a calcified nodule as assessed by optical coherence tomography. JACC Cardiovasc Imaging 2017;10:883–91.