Management of Nodular Coronary Calcium

A current understanding of coronary calcified nodules, including pathogenesis, imaging characteristics, and treatment considerations to support nuanced interventional decision-making.

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Despite marked advances in care, coronary heart disease remains a principal cause of morbidity and mortality in the United States.[1] Advanced calcified lesions, including calcified nodules (CNs), play an increasingly recognized role in both stable coronary disease and acute coronary syndrome (ACS).[2] Advanced coronary artery calcification has previously been shown to be an independent predictor of suboptimal percutaneous coronary intervention (PCI) outcomes, with higher rates of death, myocardial infarction (MI), and target vessel revascularization (TVR) when observed in the target lesion.[3-6] Although CNs are determined to be the cause of a minority of ACS presentations, they merit increased clinical attention due to unique considerations in their treatment and the strong evidence base demonstrating worse outcomes after intervention with contemporary treatment methods.[5]

CNs are associated with advanced age, diabetes mellitus, and advanced kidney disease.[5,7-9] The high prevalence of these risk factors underscores the importance of developing optimal strategies for these lesions. Increased utilization of optical coherence tomography (OCT) and intravascular ultrasound (IVUS) has added to our understanding of how these lesions affect both the coronary intervention itself and outcomes after the intervention.

These complex calcified lesions increase intervention complexity and worsen patient outcomes after PCI by several mechanisms, including altering stent expansion and deployment.[10] Thus, there remains a need for further elucidation of optimal treatment strategies for these lesions. This article provides a brief review on the pathogenesis of CNs, their associated clinical implications, available treatment modalities, and a suggested treatment strategy algorithm (Figure 1).

PATHOGENESIS

Previous studies have extensively detailed the development of the other two major etiologies of ACS—plaque rupture and plaque erosion.[2] The development of advanced calcified lesions, including CNs, and the understanding of which lesions are more likely to lead to symptomatic coronary ischemia or ACS has long remained an unanswered clinical question.[7]

CNs can be categorized as eruptive or noneruptive. An eruptive CN refers to the specific pathophysiologic situation in which an eccentric heavily calcified convex lesion with protruding calcium fragments eventually disrupts the fibrous cap of the coronary arterial intima and develops an associated occlusive or nonocclusive thrombus, resulting in ACS.[7,9] These calcified lesions may initially develop on the fringes of the necrotic core of an initially atheromatous coronary lesion. As microcalcification develops and jagged calcium fragments disrupt intravascular capillary vessels with subsequent microhemorrhage and healing, the lesion may continue to grow.[7] The CN continues to develop as calcific aggregates form, fragment, and layer upon themselves, eccentrically narrowing the vessel lumen prior to intimal disruption. Endothelial disruption with healed thrombosis or healed plaque rupture can also serve to enlarge an already heavily calcified lesion.[7]

This final step of endothelial disruption is in contrast to the entity of a noneruptive CN, which is also a heavily calcified lesion with convex eccentric luminal narrowing. However, this term is used when there is no disruption of the fibrous cap and thus no pathophysiologic substrate for ACS.[9] Therefore, active CNs associated with ACS are typically classified as eruptive CNs, whereas more stable nodular calcifications without fibrous cap disruption are referred to as noneruptive CNs (Figure 2).[11]

Imaging by IVUS may not be able to detect the difference between eruptive and noneruptive CNs due to the difficulty in visualizing thrombus or fibrous cap disruption.[9] Autopsy data from a large set of sudden coronary death patients revealed several predisposing characteristics for both patients and coronary vessels more likely to develop CNs.[7] These lesions are more likely to develop in vascular territories exposed to relatively more hinging motion and torsion during the cardiac cycle, resulting in added circumferential and axial stress, as well as shear stress from blood flow itself.[7] CNs were observed particularly frequently at the left main bifurcation and within the proximal to middle right coronary artery segment, especially in patients with tortuous coronary anatomy. Mechanical stress is thus suspected to be a primary contributor to CN development with eventual discontinuity of the fibrous cap.[12]

Flanking regions proximal and distal to the CN were also noted to have significant sheet and/or circumferential calcification, with a relatively low proportion of collagen at the culprit lesion site. The lack of tensile strength due to an absence of collagen, contrasted to the relatively high proportion of sheet calcium and collagen content both proximal and distal to these lesions, thus appears to further predispose to intimal disruption. The mechanical stress that these sites undergo induces fragmentation of existing calcium deposits, resulting in further aggregation of calcium in a nodular eccentric formation that can impinge on the vessel lumen.[7,13,14]

Several studies have demonstrated that both eruptive and noneruptive CNs are more often found in patients of advanced age, patients with diabetes mellitus, and patients with advanced kidney disease or end-stage renal disease.[5,7] The latter is most likely due to chronically elevated serum calcium and phosphate levels in the setting of bone mineral disease, which may also independently stimulate osteogenic transformation of smooth muscle cells.[5]

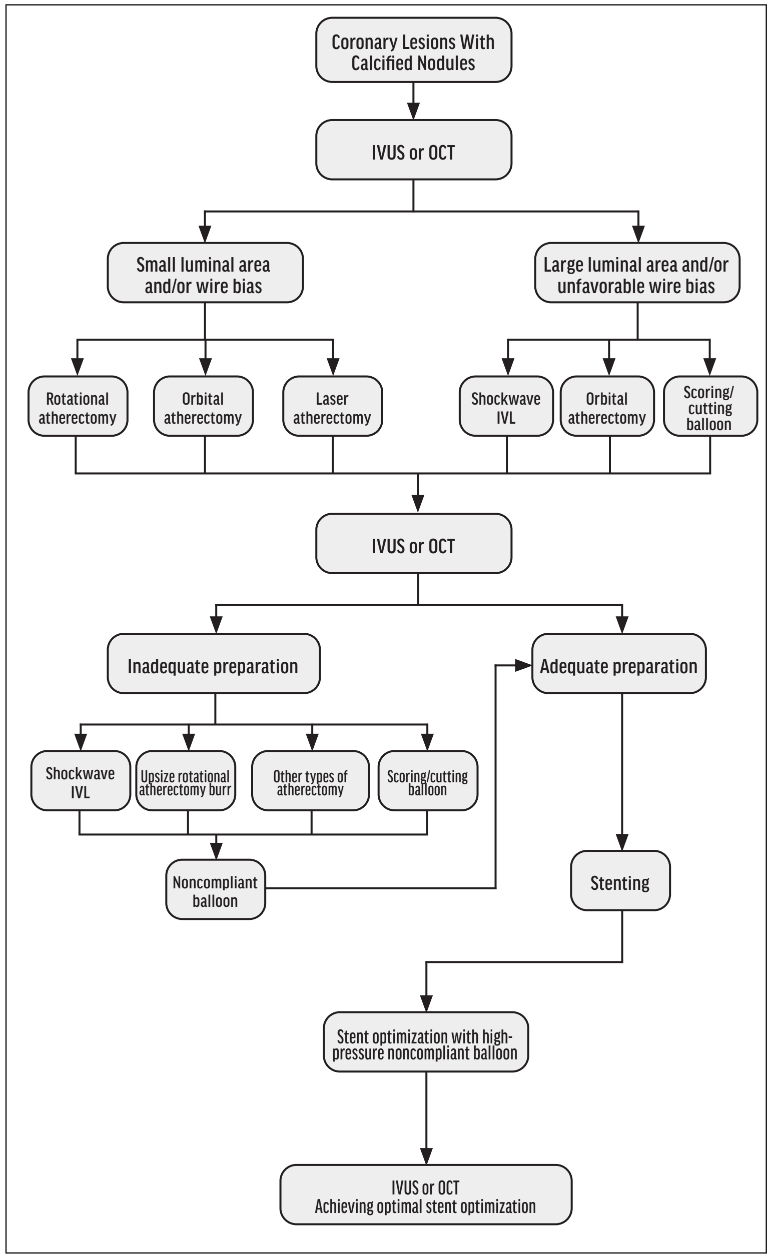

Figure 1. Our treatment algorithm for coronary lesions with CNs.

IMPLICATIONS IN PCI

CNs worsen patient outcomes both in the setting of ACS and in the treatment of symptomatic stable lesions. Heavily calcified lesions complicated by CNs often have severe eccentricity due to protrusion of calcium fragments on one side of the vessel and relatively less disease on the opposing side of the vessel. These features make PCI more complex due to difficulty in dilating the lesion, resulting in failure to deliver equipment, including stents, and stent underexpansion.[11]

Large studies examining OCT-guided intervention have demonstrated a higher likelihood of cardiac death or MI related to treated culprit lesions with CNs, with a higher incidence of major adverse cardiac events (MACE) noted even compared to other morphologies

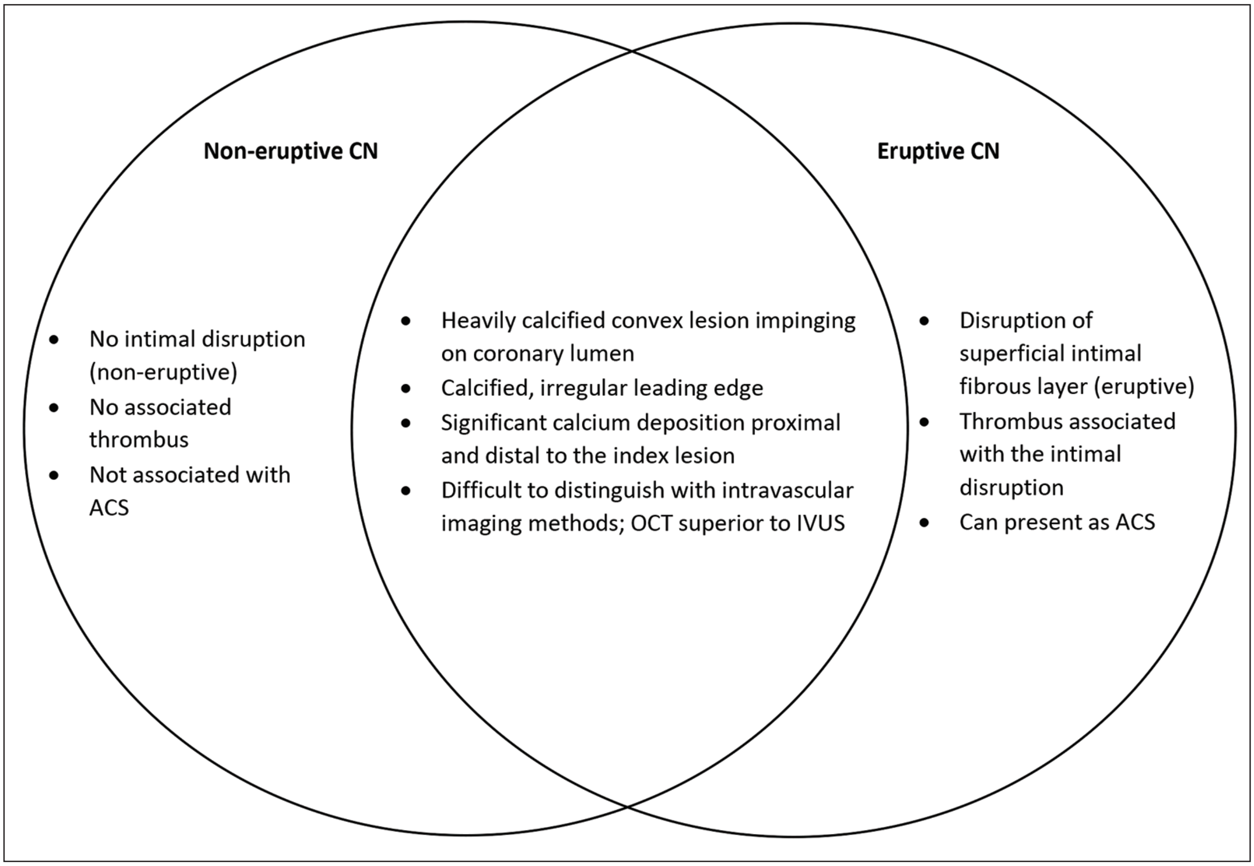

Figure 2. Characteristics of eruptive versus noneruptive CNs.

of advanced calcification (eg, calcified sheets, nonnodular severe calcification).[15,16]

Several purported mechanisms have been suggested for this disparity, including malapposition of deployed stents within the vessel lumen, asymmetric or incomplete stent deployment, and disruption of stent struts or stent polymer. Stent underexpansion is associated with restenosis and stent thrombosis and is strongly associated with calcified coronary lesions, including CNs.[10,17-19] Stent underexpansion and asymmetric deployment persist and are common despite high-pressure balloon inflation.[19-21] Furthermore, the higher pressures used to attempt full, symmetric stent expansion may lead to stent fracture or edge dissection, with nodular calcium acting as the nidus of dissection.[20-22] ACS attributable to eruptive CN has a higher risk of recurrence and need for TVR, and it is an independent predictor of MACE.[8,10,20]

CNs can also protrude and reappear within a stented vessel, causing neointimal disruption while also acting as a nidus for neointimal hyperplasia.[8] This protrusion can cause catastrophic stent complications, including sudden coronary death.[9,23] Eruptive CN development has also been observed de novo within the neointima of a stented vessel years after implantation.[24]

Irregularity in the surface of advanced calcified lesions appears to increase the likelihood of need for revascularization.[23] The likelihood of CN recurrence within stents underscores the need for calcium modification techniques and intravascular imaging before and after stent deployment given the propensity for CNs, especially eruptive CNs, to protrude through stent struts.[23]

INTRAVASCULAR IMAGING

IVUS

Coronary angiography alone may be insufficient to distinguish advanced calcified lesions from other noncalcified lesions. Previous work has helped elucidate the features of eruptive and noneruptive CNs on IVUS, with subsequent histopathologic comparison to verify these findings.[25] These features include an irregular leading edge of hyperechoic material consistent with calcification and a nearly uniformly convex shape impinging on the coronary lumen. This is in contrast to either dense fibrotic lesions or fibrocalcific lesions that, although they may share some IVUS features with CNs, will demonstrate a concave border and not appear as a bright and protruding mass from the vessel wall.[26] Additionally, high-definition IVUS has the ability to differentiate between eruptive and noneruptive CNs, based on the continuity of the fibrous cap, which remains intact in noneruptive CNs.[11]

OCT

When examined with OCT, calcified lesions appear as a heterogeneous area of interest with sharply delineated borders and high backscattering.[22] OCT allows for enhanced quantification of calcified lesion thickness, area, and volume compared to IVUS. With this imaging modality, CNs appear as bright, protruding masses with an irregular surface, and similar to other calcified lesions, they have sharply delineated borders with high backscatter.[27] Most relevant to the preceding discussion, the higher resolution of OCT allows for improved detection of erosion or interruption of the intimal surface and fibrous cap for a given lesion, as well as detection of mural thrombus, which may aid in the distinction between eruptive and noneruptive CNs.[14] Additionally, OCT allows the differentiation between deformable and nondeformable calcified lesions. Most CNs are deformable, with reduced luminal protrusion after PCI, achieving asymmetry and eccentricity indices > 0.7. In contrast, approximately one-third of noneruptive CNs are nondeformable because of their rigid structure.[11]

CORONARY CALCIUM MODIFICATION MODALITIES

There are some commonly used modalities for the treatment of advanced calcification that may also have a role in the treatment of CNs.

Rotational Atherectomy and Orbital Atherectomy

The Rotapro rotational atherectomy (RA) system (Boston Scientific Corporation) uses a concentrically spinning, diamond-coated burr with a front cutting mechanism on a proprietary support wire spinning 140,000 to 200,000 revolutions per minute, aiding in advanced lesion modification for eventual delivery of stent delivery and expansion. The burr allows for differential cutting and ablation of noncompliant materials, such as atherosclerotic plaque and calcium, rather than vascular tissues.[28] In contemporary practice, it is frequently used in the setting of advanced calcification, including eruptive and noneruptive CNs, when the guidewire may be passed but the delivery of other equipment remains challenging.

The 1.5-mm burr size is most commonly used to modify the lesion before further preparation with other modalities—rather than using a larger burr for a stand-alone debulking strategy that previously was more commonplace.

RA is thought to be effective for modifying concentric calcified lesions, as the burr can directly contact and ablate the calcium sheet, potentially causing calcium fracture and creating a larger lumen for subsequent treatment with high-pressure, noncompliant (NC) balloon dilation or, more recently, Shockwave intravascular lithotripsy (IVL [Shockwave Medical], referred to as a "rotatripsy" strategy). However, CNs are severely eccentric, with protruding calcium debris on one side of the vessel opposed to normal vascular tissue. They may also have a larger luminal area compared to a severe concentric calcified lesion. These two characteristics reduce the effectiveness of RA, as the most commonly used burr size (1.5 mm) may make contact with the nodular lesion, bypassing the lesion without any modification.[28]

The Diamondback 360 coronary orbital atherectomy (OA) system (Abbott) uses an eccentrically mounted, diamond-coated crown between the device nose and driveshaft that rotates to elliptically ablate coronary lesions. Similar to RA, the burr is meant to selectively sand down inelastic material of atherosclerotic and calcified plaques while sparing vascular tissues. The device uses a proprietary guidewire to pass the nose beyond the lesion, and the ablation crown can then be used to ablate advanced calcific lesions. The eccentric motion of the ablation crown and driveshaft allows for debulking of higher-volume lesions within larger vessels compared to RA, especially for straight vessel segments; however, RA is still preferred for aorto-ostial lesions.[28]

Both OA and RA are subject to the phenomenon of wire bias, in which the guidewire is no longer centrally oriented after accessing a lesion with proximal or distal vessel tortuosity or angulation, resulting in cutting that is not parallel with the vessel path.[29] Wire bias may help bring the burr preferentially closer to the desired nodular lesion, but the effect is difficult to predict and unreliably seen on intracoronary imaging. If the burr more frequently contacts the normal side of the vessel, aggressive ablation may predispose to complications. Thus, vessel dissection and perforation are feared complications for both methods. Burr entrapment is also possible and thought to be less common with OA due to the bidirectional cutting mechanism.[29]

A small number of studies specifically review interventions involving RA on CNs and show that outcomes are unfavorable when these lesions are present, with an elevated risk of cardiovascular death, MI, and clinically driven target lesion revascularization compared to lesions without this morphology.[30-32] However, there is likely a bias toward more advanced lesions in general among these data, given the associated risk factors and advanced nature of these calcified lesions when such a comparison is drawn.

Among consecutive CNs in a propensity-matched analysis of RA versus no RA, use of RA was not associated with a significant improvements in IVUS-derived acute lumen area gain, stent malapposition, or clinically driven TVR.[33] Conversely, another large retrospective cohort of CN lesions demonstrated a strong association between nonuse of RA and restenosis.[34]

Super-High–Pressure Balloons

The OPN NC balloon (SIS Medical AG) makes use of an angioplasty balloon that can uniformly inflate to and tolerate higher than typically used pressure to modify coronary lesions. It fills a niche within calcium modification with its ability to modify and potentially fracture larger sheets of calcium while allowing for fuller stent expansion, particularly when used to modify sheet calcification with concentric luminal narrowing.[35] The OPN NC balloon is double layered and designed to withstand high pressures (> 35 atm) while preventing the relative proximal and distal dilation—commonly referred to as "dog-boning"—observed in other NC balloons. However, there is concern that in the setting of an eccentric lesion like a CN, the high pressures with this balloon might increase the risk of vessel trauma or perforation rather than producing the desired fracture of calcific plaque.[36]

Specialty Balloons

Specialty balloons include cutting and scoring balloons and are designed to fracture large calcific plaques during deployment, thereby improving compliance and increasing minimal stent area at the lesion site. These balloons use wires or microsurgical blades arranged around an angioplasty balloon. The AngioSculpt scoring balloon catheter (Philips) is a semicompliant nylon balloon surrounded by three external nitinol spiral scoring wires for treatment of fibrocalcific and calcified lesions and in-stent restenosis. The Wolverine cutting balloon (Boston Scientific Corporation) is frequently used in contemporary practice and features three microsurgical blades triangularly arranged around an angioplasty balloon, with a narrower profile compared to earlier cutting balloons.

The efficacy of scoring and cutting balloons is affected by the eccentricity and resistance of CNs. Scoring wires and cutting blades may only modify the compliant, normal side of the vessel, thus resulting in asymmetric balloon expansion without fracturing the nodule. There are minimal safety and efficacy data regarding use of modified balloons in the setting of CN.[28]

Data comparing RA, specialty balloons, and super-high–pressure balloons for OCT-guided treatment of severely calcified lesions demonstrated similar stent expansion and in-hospital MACE rates among all three groups, with the super-high–pressure balloon group demonstrating less stent eccentricity and the RA group demonstrating the highest percentage of strategy success.[37] However, these data did not distinguish CNs from other advanced calcification.

IVL

Shockwave IVL uses pressure generated by an electrical charge that vaporizes fluid in an angioplasty balloon to fracture lesions, including fibrocalcific plaque and advanced calcified lesions. Shockwave IVL increases vessel compliance and allows for stent deployment as it targets deeper calcium than would necessarily be contacted by other advanced calcium treatment methods.[28] In comparison to RA, the risk profile is thought to be low, with a lower risk of atheromatous embolization and comparable data in terms of strategy success rate, lumen gain, and residual stenosis.[38]

A subanalysis of Disrupt CAD demonstrated favorable periprocedural and long-term outcomes with IVL for managing CNs compared to calcified lesions without CN.[39] In our experience, the circumferential acoustic energy transmitted by the Shockwave balloon may be the best modality for overcoming some of these discussed challenges in the treatment of CNs.[40] The acoustic energy is thought to only modify the calcification rather than the compliant vessel. In a large luminal lesion with a CN, the optimal size of the Shockwave balloon is selected via intracoronary imaging to ensure full contact with the lesion when the balloon is inflated to a low pressure (3-5 atm), while delivering as high as 50 atm of acoustic energy to the lesion.

If treating a critically stenosed calcified lesion with CN, the lesion may be initially modified with RA followed by an adequately sized IVL device to ensure adequate lesion preparation. Anecdotally, we do still find some CN cases that are not adequately modified despite the combined use of each of these modalities, with the unfortunate result of stent underexpansion.

Specifically for CNs, patient-level pooled data from the regulatory approval studies for coronary IVL demonstrated improvement in luminal gain, minimal stent area, and expansion after treatment, with no major complications and no major difference in these parameters between CN and non-CN lesions.[41,42]

CONCLUSION

This article describes the pathophysiology, clinical characteristics, and treatment methods for advanced calcified lesions, with the intent of summarizing some of the available evidence base to support nuanced decision-making regarding interventions on these complicated lesions. ■

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