

Chapter 9

Intravascular Lithotripsy for Calcified Peripheral Arterial Disease



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Introduction

The presence of vascular calcification imparts specific difficulties for the cardiovascular interventionist to provide safe and effective therapies. Vascular calcification is often seen in patients with comorbidities, such as diabetes mellitus and chronic kidney disease, as well as in those with advanced age [1]. Moderate to severe vascular calcification is common, being present in up to one third of patients presenting with acute coronary syndromes and up to one half of patients undergoing peripheral artery revascularization [1, 2]. Extensive vascular calcification is associated with reduction in lesion crossing, device delivery, and adequate lesion preparation (including a decrease in the effect of antiproliferative therapies), which, in turn, is directly related to an increase in procedural failure [3, 4]. Recently, there has been increasing use of large-bore access for interventional therapies such as endovascular aneurysm repair (EVAR), thoracic endovascular aneurysm repair (TEVAR), and transcatheter aortic valve replacement (TAVR) with transfemoral access being the preferred site. Iliofemoral calcification is a predominant factor for the utilization of alternative access [5]. Current techniques in the management of noncompliant calcified lesions include high-pressure balloon angioplasty, specialty cutting and scoring balloons, and atherectomy devices, which are associated with increased risk of vessel dissection, acute closure, perforation, and no reflow phenomenon [6, 7].

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Intravascular lithotripsy (IVL) is a novel device therapy which modifies both intimal and medial calcified lesions using pulsatile sonic waves which are converted to mechanical energy. External shock wave lithotripsy has been used for decades in the treatment of renal calculi and gallstones [8, 9]. In a similar way, IVL utilizes electrohydraulically generated sonic waves which pass harmlessly through soft tissue and target high-density calcium in the intimal and medial walls of the artery. The pressures generated by the sonic waves fracture vascular calcium with effective dilating force of approximately 50 atmospheres (atm), thereby rendering the vessel more compliant and reducing elastic recoil [10].

IVL: Device Specifics

In 2016, Shockwave Medical, Inc. (Santa Clara, CA) received 510 k premarket approval for their peripheral IVL system for the treatment of calcified peripheral artery disease. This device, labelled the Shockwave M5, consisted of an over-the-wire system on an 0.014" wire platform (Fig. 9.1). The balloon sizes ranged from 3.5 to 7.0 mm, in 0.5 mm increments, in a single available 60 mm length mounted on a 110 cm shaft. The M5+ catheters became available in 135 cm shaft length with the additional availability of an 8.0 mm device in April 2022. The M5 catheter has a slightly increased profile when compared to comparable-sized noncompliant balloons (0.050–0.066 in), similar in crossing profile to contemporary cutting balloons. The M5 catheters up to 6.0 mm are compatible with a 6-Fr sheath with 6.5, 7.0 mm, 8 mm requiring a 7-Fr sheath. The balloon is semi-compliant with five emitters located between two radiopaque markers. Balloon preparation is done in the standard over-the-wire fashion with the central lumen being flushed prior to loading of the 0.014 in guidewire. A mixture of 50/50 saline/contrast is used to prepare the balloon. Balloon sizing is based on a 1.1:1 balloon-to-reference lumen ratio with the

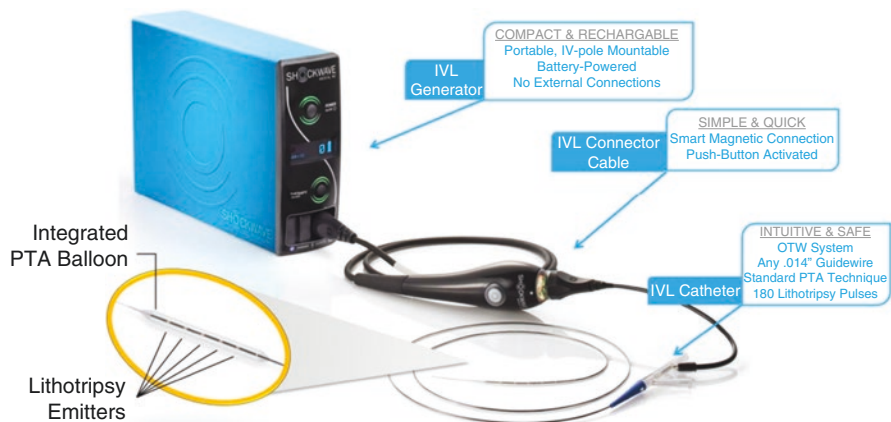


Fig. 9.1 Shockwave equipment and setup

balloon being inflated to subnominal pressures, typically starting at 4 atm and increasing to nominal pressure of 6 atm after several inflations. This ensures contact with the vessel wall and minimizes the risk of endothelial trauma. If contact with the vessel wall is not accomplished, the sonic waves will not reach the intimal or medial calcification as the energy does not traverse dead space. The M5 catheter is attached to the power generator which is programmed to deliver 30 pulses at a rate of 1 pulse per second. Each M5 catheter is capable of delivering a maximum of 300 pulses allowing for overlapping inflations of adjacent arterial segments. After each round of 30 pulses, it is imperative to deflate the balloon in order to remove microbubbles which are generated as a byproduct of the sonic waves. It is recommended that any single segment is treated with no more than 180 pulses. When overlapping inflations are done, it is important to ensure at least 1 cm of overlap in treated segments to avoid “geographic miss” of intentional treatment zones.

The Shockwave S4 is a smaller design catheter for below-the-knee angioplasty and was FDA approved for use in 2019. The balloon sizing is 2.5–4.0 mm in 0.5 mm increments with a single 40 mm length housing four emitters. The catheter comes in a 135 cm length and requires a minimum of 5 Fr sheath. Each catheter has the ability to provide 180 pulses in 20 pulse/cycle increments (Table 9.1). The coronary device, labelled Shockwave C2, is a shorter catheter specifically designed for intracoronary lithotripsy. This device obtained investigational device exemption in 2020 after several clinical trials provided data on safety and effective use [11, 12]. The C2 device is available in 2.5–4.0 mm balloons in 0.5 mm increments. The balloon length is 12 mm and available on a 138 cm catheter. The C2 requires a minimum of a 6 Fr guiding catheter. Each catheter is capable of delivering 80 pulses in 10 pulse/cycle increments to limit coronary artery occlusive time.

Table 9.1 Technical features of shockwave IVL catheters

Features	M5	S4	C2
Guide/Sheath size	6 Fr: 3.5 mm–6 mm 7 Fr: 6.5 mm, 7.0 mm	5 Fr	6 Fr guide
Balloon size (mm)	3.5, 4.0, 4.5, 5.0, 5.5, 6.0, 6.5, 7.0	2.5, 3.0, 3.5, 4.0	2.5, 3.0, 3.5, 4.0
Catheter length (cm)	135	135	138
Wire compatibility (in)	0.014	0.014	0.014
Balloon length (mm)	60	40	12
Number of emitters	5	4	2
Nominal pressure (atm)	6	6	6
Rated burst pressure (atm)	10	10	10
Pulses per catheter	300 pulses 30 pulses/cycle 1 pulse/s	180 pulses 20 pulses/cycle 1 pulse/s	80 pulses 10 pulses/cycle 1 pulse/s

atm atmosphere, *cm* centimeter, *Fr* French, *in* inch, *mm* millimeter

IVL in Peripheral Artery Disease

Although there is no standard definition for grading severity of calcification in peripheral arteries, several scoring systems have been proposed. The Society for Cardiovascular Angiography and Intervention (SCAI) in a 2018 consensus document defined severe calcification as $>180^\circ$ involving both sides of the vessel [13]. The Peripheral Arterial Calcification Scoring System (PACSS) takes into account the lesion length (greater than 5 cm) as well as location of calcification (intimal, medial, mixed, unilateral, bilateral) in the anteroposterior fluoroscopic projection. The PARC (Peripheral Academic Research Consortium) scoring system defines severe calcification as greater than 180° on both sides of the vessel and greater than one half of the total lesion length [14].

It is well described that in patients with peripheral artery disease, the presence of vascular calcification is associated with worse outcomes including higher Rutherford classification and higher rates of amputation [15]. The use of percutaneous transluminal angioplasty (PTA) with conventional angioplasty balloons in severely calcified peripheral artery disease is associated with low success rates due to acute recoil, suboptimal lesion expansion, and the potential of vessel injury, dissection, or even perforation [16]. Rotational and orbital atherectomy are able to achieve acute luminal gain by affecting superficial calcium, but medial calcium is unaffected [17]. IVL has been studied in an attempt to overcome the limitations of the previously available devices for calcium modification.

DISRUPT PAD I/II were multicenter, single-arm registries which enrolled a total of 95 patients with symptomatic peripheral artery disease (Rutherford 2–4), ankle-brachial index (ABI) <0.9 , and angiographic evidence of calcific femoropopliteal stenosis of $>70\%$ with at least 1 patent runoff vessel to the foot [18, 19] (Table 9.2). Using the PARC definition, severe calcification was seen in over half of participants, and procedural success, defined as residual stenosis of $<50\%$, was achieved in all patients. IVL showed a reduction in luminal stenosis from 76 to 23% with a mean acute luminal gain of 3.0 mm (1.2 ± 0.8 to 4.2 ± 0.6 mm) [18]. Clinically important outcomes from this showed that at 1 and 6 months, no target lesion revascularization (TLR) occurred and vessel patency rates were 100% and 82%, respectively. At 12 months, patency rates were 54% with TLR rates of only 21% [19]. In a subgroup analysis of DISRUPT PAD II, when optimal technique was performed, patency rates were elevated to 63%, there was an improvement of 15%, and TLR decreased to 8.6% at 12 months (Fig. 9.2). Optimal techniques include appropriate balloon sizing of 1.1:1 balloon-to-reference ratio and full lesion coverage of treatment zones with at least 1 cm of emitter overlap (Fig. 9.3).

IVL has also been shown effective in the treatment of below-the-knee disease in patients with critical limb ischemia (CLI) as well [20]. The DISRUPT BTK study reported 20 patients, Rutherford classes 3–5 (16 patients with CLI), with heavily calcified infrapopliteal lesions (angiographic stenosis 72.6%, mean lesion length

Table 9.2 Trials of IVL in peripheral artery disease

		DISRUPT PAD I/II (N = 95)	DISRUPT PAD III RCT (N = 306)		DISRUPT BTK (N = 20)
Design		Multicenter, single arm	Multicenter, prospective, randomized		Multicenter, single arm
Population			IVL	PTA	
	Rutherford 2	33.7%	17%	17%	–
	Rutherford 3	65.3%	77%	74%	20.0%
	Rutherford 4	1.1%	6%	8%	5.0%
	Rutherford 5	–	–	1%	75.0%
	Rutherford 6	–	–	–	–
Angiographic appearance			IVL	PTA	
	Severe calcification (PARC)	54.7%	82.9%	89.5%	47.6%
	RVD (mm)	5.3	5.3	5.4	3.2
	Lesion length (mm)	71.9	101	97	52.2
	CTO	18.9%	26%	31%	9.5%
Safety			IVL	PTA	
	Complications	1%	1.1%	15.1%	0.0%
	Grade > C dissections		0.0%	0.7%	
	Perforations Thromboembolic events		0.0%	0.7%	
Efficacy	Residual stenosis	24%	23.6%		26%
	Acute gain (mm)	3	3.4		1.5
Outcomes	30 days	Freedom from TLR: 100% Patency: 100%	Freedom from TLR at 12 months (IVL+DCB): 95.7% vs. 98.3% (PTA+DCB), P= .94		Freedom from TLR: 100% MAE: 0%
	6 months	Freedom from TLR: 96.8% Patency: 76.7%	Primary patency at 12 months (IVL+DCB): 80.5% vs. 68.0%, (PTA+DCB), P= .017		

CTO chronic total occlusion, MAE major adverse events [myocardial infarction, amputation, death], PARC peripheral academic research consortium, RVD reference vessel diameter, TLR target lesion revascularization

52.2 ± 35.8 mm). Procedural success was achieved in 95% of patients, with a residual percent stenosis of 26.2% and an acute lumen gain of 1.5 ± 0.5 mm. Two stents were implanted for residual stenosis, but none for flow-limiting arterial dissection, without major adverse events.

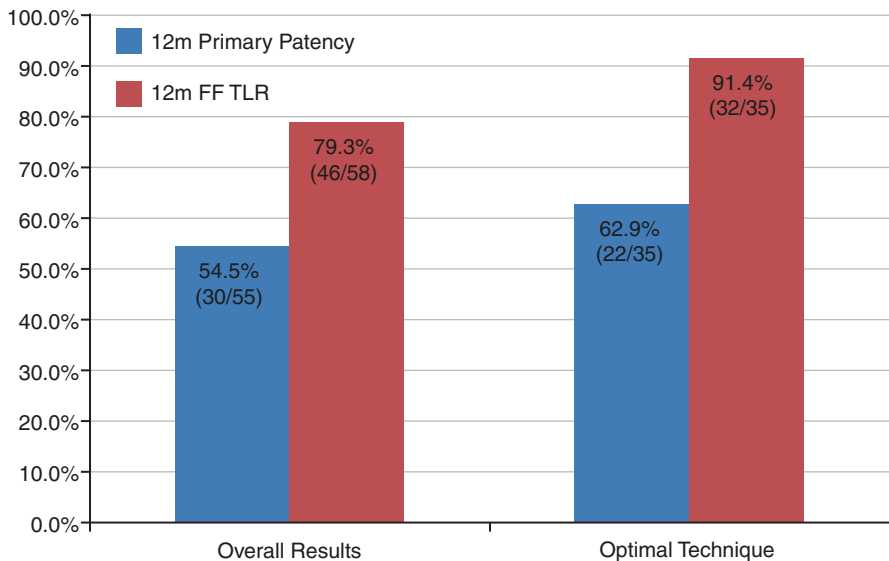


Fig. 9.2 12-month primary patency and freedom from target lesion revascularization (FF-TLR)

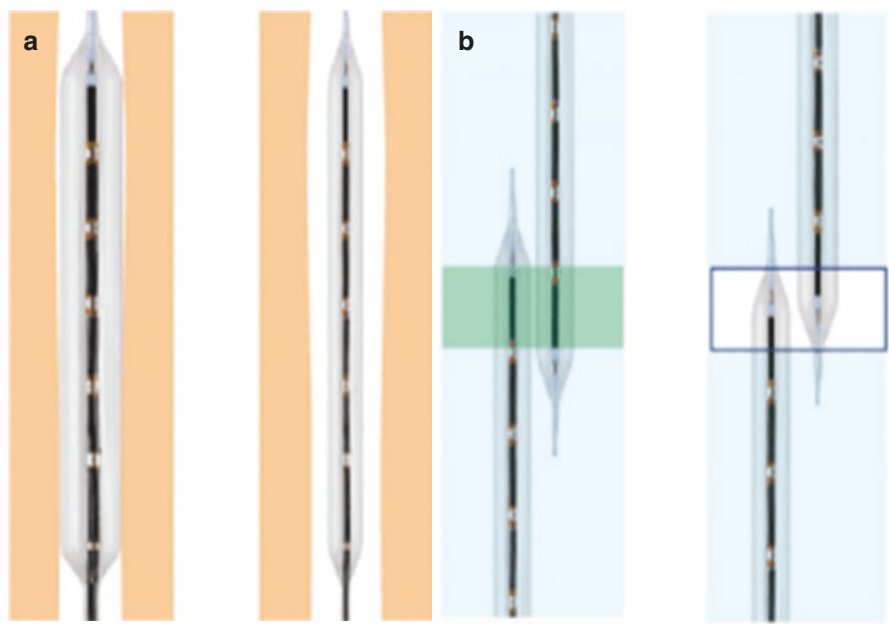


Fig. 9.3 Optimal technique. (a) Balloon sizing 1.1:1 balloon-to-reference vessel ratio. (b) At least 1 cm emitter overlap

Effect of Calcium on Drug Elution

The use of drug-coated balloons (DCB) as adjunctive therapy in peripheral angioplasty has been tested as a means to overcome the shortcomings of traditional angioplasty. Studies have shown excellent vessel patency and low rates of both target lesion revascularization (TLR) and complications [21–23]. Drug-eluting technologies have lower efficacy rates in severe calcific disease, likely a result of reduced drug penetration into the vessel wall [24]. As a result, combination therapy utilizing specialty balloons and atherectomy devices prior to DCB has shown promising results; however, long-term efficacy remains unproven.

The recently presented DISRUPT PAD III trial was designed to compare the use of combination IVL and DCB versus combination PTA and DCB [25]. DISRUPT PAD III provides the first level I evidence comparing the effect of calcific disease on drug elution. 306 patients were randomized in a 1:1 fashion to receiving IVL + DCB/stent versus PTA + DCB/stent with primary endpoint being procedural success, defined as residual stenosis of <30% without flow-limiting dissection. Powered secondary endpoints included primary patency and clinically driven target lesion revascularization (CD-TLR) at 12 months of 80.5% and 95.7% in the IVL + DCB group, vs. 68.0% and 98.3% in the PTA + IVL group, respectively; $P=0.94$ for FF TLR and $P=0.017$ for primary patency [26]. Lower maximum inflation pressures were seen in the IVL group (6.3 atm vs. 11.3 atm) which resulted in a 75% relative risk reduction in bailout stenting. DISRUPT PAD III also included 2% below-the-knee, 15% iliac, and 13% common femoral artery target lesions vs. only femoropopliteal lesions in DISRUPT PAD I/II. Moreover, DISRUPT PAD III lesion characteristics were more challenging- longer lesion lengths, greater calcification, higher percentages of CTO, and CLI patients. These encouraging results suggest that lesion preparation and calcium modification with IVL prior to drug-eluting devices are more effective, and safe, than PTA in the management of moderate to severe calcific peripheral artery disease. Although more evidence is needed, it can be surmised that calcium modification prior to DCB provides better milieu for drug elution.

IVL-Facilitated Large-Bore Vascular Access

With the increasing use of minimally invasive strategies for the management of aortic and cardiac valvular disease, large-bore vascular access is frequently required. Randomized trials in the investigation of TAVR included transfemoral as well as other alternative access sites in the approach to valve delivery [27]. Transfemoral access has become the access site of choice as studies have shown it to be the only superior access site when compared to traditional surgical aortic valve replacement [28]. Unfortunately, given concurrent peripheral artery disease with calcified aortoiliac bifurcations, up to 15–20% of TAVR candidates may be deemed ineligible for transfemoral access due to the inability to successfully and safely advance the

required large-bore delivery sheaths resulting in more invasive methods such as trans-axillary, trans-aortic, trans-apical, trans-carotid, trans-septal, and trans-caval [29]. Initial case reports suggested the feasibility of IVL technology to aid in the delivery of large-bore access [30, 31]. In a prospective registry of 40 patients with peripheral artery disease who were deemed ineligible for transfemoral access, IVL facilitated successful placement of the delivery sheaths in >90% with no iliofemoral perforations or dissections observed [32]. Numerous case reports have highlighted the efficacy of IVL in the facilitation of other large-bore vascular access including percutaneous LV assist devices, TEVAR, and EVAR [33, 34]. IVL therefore provides a useful tool in the management of this complex patient population.

Indications for IVL in Specific Vascular Beds with Case Examples

Brachiocephalic Lesions in Patient with Symptomatic Arm Claudication or TIA

Endovascular treatment of brachiocephalic arteries is challenging due to their larger diameter, short length, and proximity to the intracranial vasculature [35]. The presence of calcific disease adds to procedural complexity and increases risks of complications due to perforation, dissection, embolization, and stent underexpansion. Case 9.1 demonstrates the use of IVL to treat symptomatic concomitant innominate and subclavian calcific disease.

Carotid In-Stent Restenosis (ISR) Due to Stent Underexpansion

Dense calcification of carotid bifurcation stenoses is a frequent exclusion for enrollment in studies of carotid artery stenting (CAS). In cases of CAS ISR due to stent underexpansion, IVL may be used to allow for further stent expansion (*off-label use*) as illustrated in Case 9.2. There are very limited options for treating underexpanded stents. High-pressure inflations may be ineffective or induce dissections or rupture. This is the first reported use of IVL for CAS ISR and was included in a first published review of IVL for use in calcified carotid lesions [36].

Mesenteric Ischemia Due to Calcific Stenosis of the Superior Mesenteric Artery (SMA)

The feasibility of IVL both for the treatment of symptomatic mesenteric ischemia for native de novo calcific stenosis and for the treatment of managing ISR due to stent underexpansion was recently reported by Khan et al. [37]. Case 9.3 illustrates

the ability to treat severe circumferential underlying calcified stenosis with IVL, allowing for full stent expansion.

Treatment of Aorto-Iliac Calcific Disease

IVL disrupts both intimal and deep wall calcification improving vessel compliance, allowing for the introduction of large-bore devices, and may offer an alternative for high-pressure dilation (with associated risk of dissection or rupture) or expandable sheaths and may obviate the need for open exposure with conduit placement (“pave and crack” technique). A recent report from a subset of the Disrupt PAD III study confirms the safety and efficacy of IVL for the treatment of calcified stenotic iliac arteries [38]. Case 9.4 demonstrates a case of severe aorto-iliac calcific occlusive disease in a patient deemed high risk for open repair treated with IVL and stenting, performed as an outpatient. Case 9.5 illustrates the off-label use of IVL for acute iliac stent suboptimal stent expansion.

Treatment of Calcific Common Femoral Artery (CFA) Stenosis

Endarterectomy has been an established treatment for CFA disease but is associated with extended length of stay and higher 15% composite rate of morbidity and mortality than endovascular techniques [39]. Brodmann evaluated 21 patients with calcified CFA stenoses treated with IVL with an acute lumen gain of 3.1 ± 1.3 mm, few non-flow-limiting dissections, and no perforations, distal embolization, thrombus, and no-reflow or abrupt closure [40]. IVL is an effective treatment for calcific common femoral disease as stand-alone therapy, or in combination with atherectomy and/or drug-coated balloon angioplasty, as seen in Case 9.6.

Calcified Femoropopliteal Lesions in Patients with Symptomatic Claudication or Critical Limb Ischemia

Calcified femoropopliteal lesions are often long occlusions, and traversal is often subintimal where use of atherectomy may result in a higher incidence of dissection or perforation. IVL, by virtue of its ability to penetrate transmural calcification, is uniquely suited to the treatment of subintimal calcification. As described earlier, in DISRUPT PAD III, IVL demonstrated a significant reduction in dissections and provisional stenting and less need for bailout stenting in the largest randomized clinical trial of severely calcified femoropopliteal lesions. Case 9.7 is an example of the utility of IVL to achieve an excellent result without the need for a stent in “no-stent zones” such as the CFA and popliteal arteries.

Calcified Below-the-Knee (BTK) Lesions in Patients with Symptomatic Claudication or Critical Limb Ischemia (CLI)

Medial calcification is more prevalent in BTK arteries and is a marker for amputation in patients with PAD [41]. This is particularly true for patients with diabetes mellitus, chronic kidney disease, and CLI, where diffuse calcific disease with infrapopliteal occlusions is common. Stents are limited to short lesions in proximal locations, and balloon angioplasty is burdened by a high rate of recoil and restenosis [42]. Atherectomy devices are problematic in these patients due to the risks of perforation with possible resultant compartment syndrome. Distal embolization after atherectomy may have catastrophic consequences and transform the CLI patient into an acute limb ischemia patient, with high risk of amputation. IVL has been shown to have a high procedural success and excellent safety profile.

Future Applications

As the population ages, calcified vascular disease is a growing challenge for the cardiovascular interventionist. Since its foray in the cardiovascular space, IVL has shown significant utility in the safe and effective treatment of moderate to severe calcified stenosis. Future applications will extend into other vascular beds. Already being evaluated is the use of IVL in the coronary arteries as has been adjudicated in the DISRUPT CAD studies, with recent FDA approval in the USA [11, 12, 43]. IVL has been used off-label in the treatment of aortic arch vessel angioplasty as well as to aid in the treatment of carotid artery revascularization, both transfemoral and trans-carotid [44, 45]. Increasing clinical experience supports the utility of combination atherectomy to create a pilot channel followed by IVL as the mechanism for enhanced luminal gain. Future improvements of IVL will include larger vessel diameters and longer balloon lengths. Currently, research and development are underway for the evaluation of IVL for the management of calcific cardiac valvular disease; however, no evidence currently exists to support its use. IVL has shown promising potential in many aspects of cardiovascular intervention, with its ceiling yet to be defined.

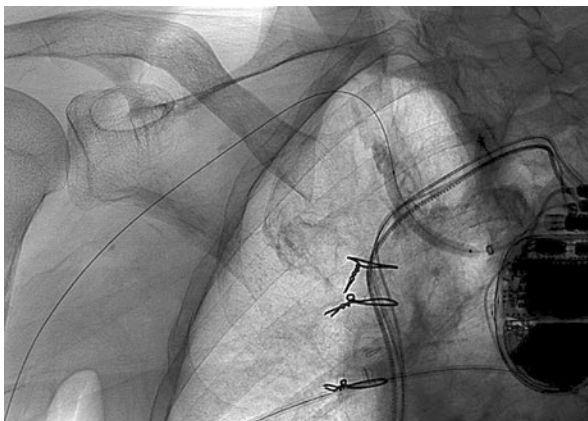
Case 9.1 Brachiocephalic



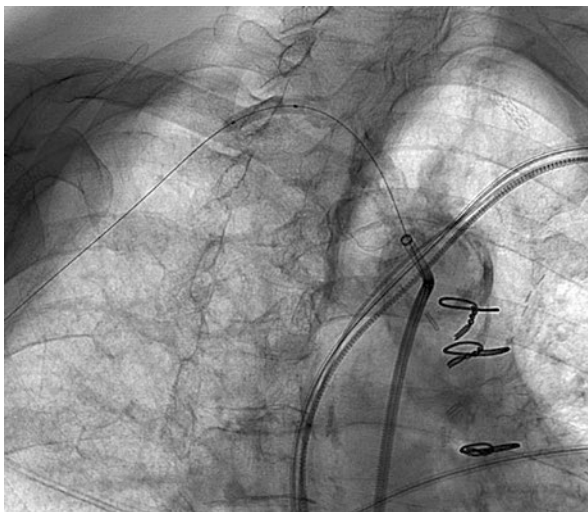
Baseline aortogram showing densely calcified innominate stenosis



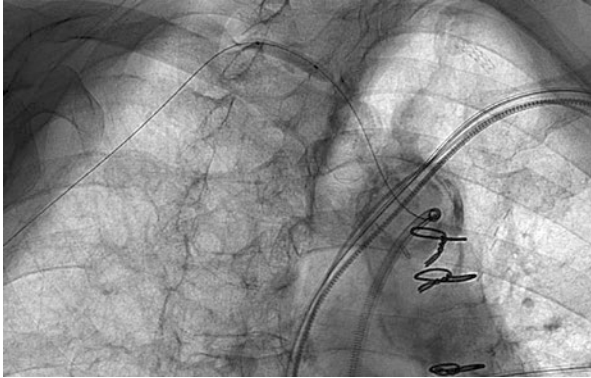
Selective innominate angiogram demonstrating eccentric densely calcified high-grade innominate artery stenosis and proximal right subclavian stenosis, left anterior oblique view (LAO)



PTA of the innominate artery stenosis with a 4 mm balloon after delivery of a 7F sheath from the left common femoral access



IVL of right subclavian artery with a 7 mm x 60 mm Shockwave Medical balloon



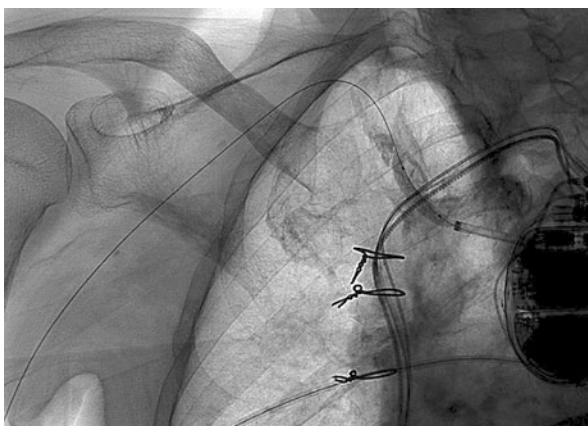
IVL of right subclavian artery with a 7 mm × 60 mm Shockwave Medical balloon



IVL of right subclavian artery with a 7 mm × 60 mm Shockwave Medical balloon



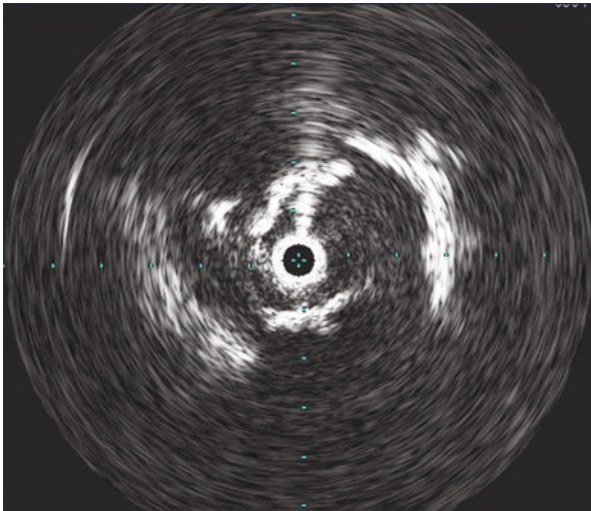
IVL of innominate artery with a 7 mm × 60 mm Shockwave Medical balloon



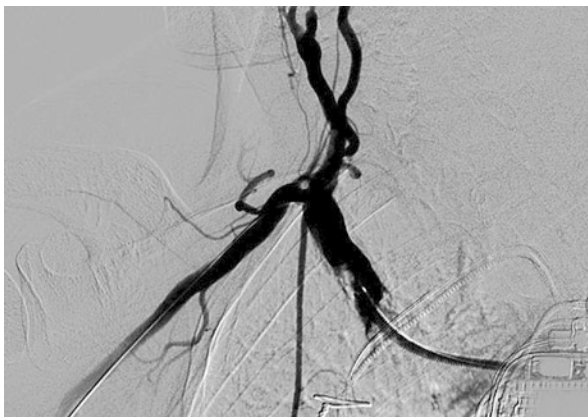
IVL of innominate artery with a 7 mm × 60 mm Shockwave Medical balloon



Post-IVL angiogram



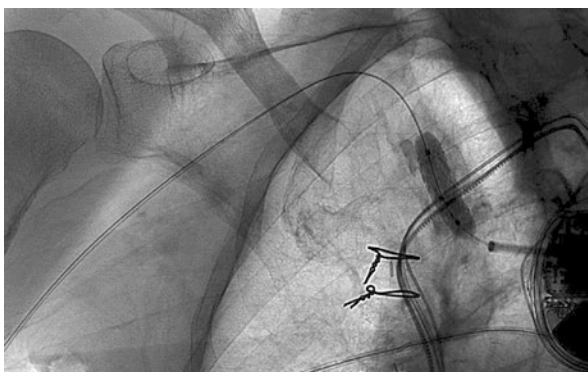
Intravascular ultrasound (IVUS) of the right subclavian post-IVL



Post-subclavian and innominate IVL, LAO



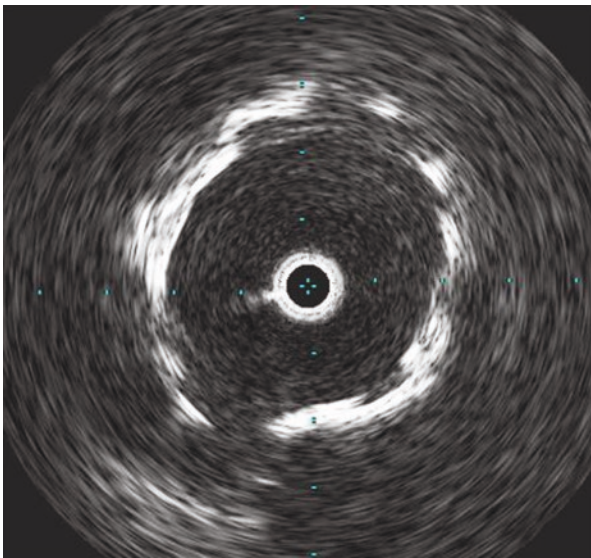
Post-subclavian and innominate IVL, RAO



Deployment of 8 mm × 24 mm Cordis Genesis balloon-expandable stent

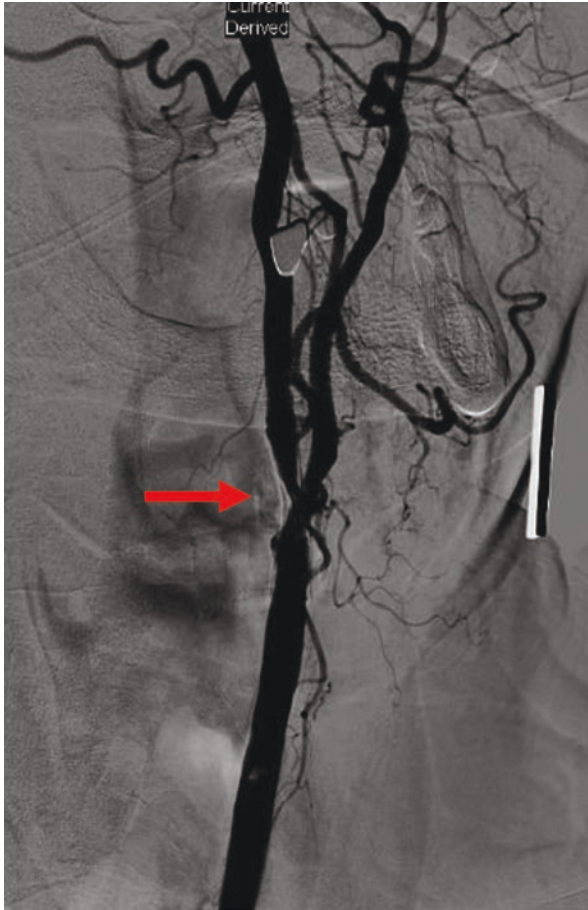


Final angiogram after post-dilation of stent with a 9 mm \times 20 mm balloon

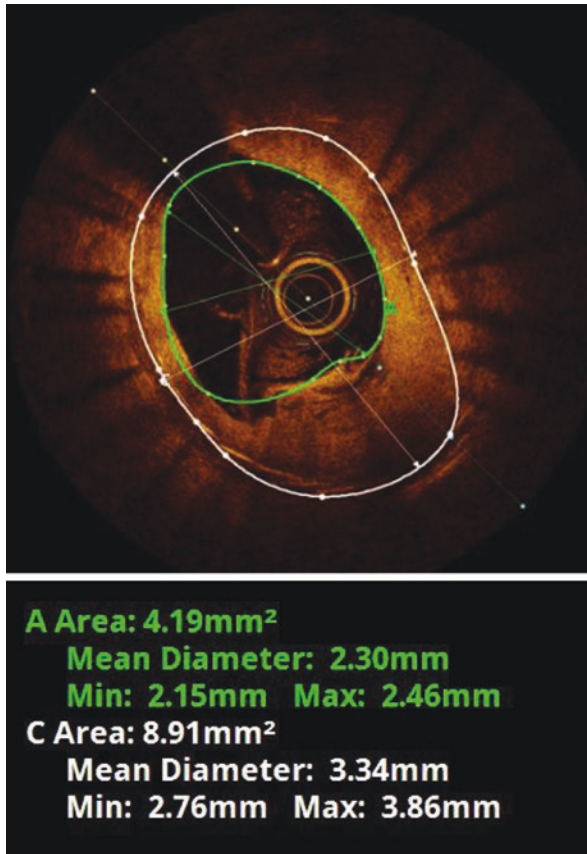


IVUS of innominate artery post-IVL/stenting

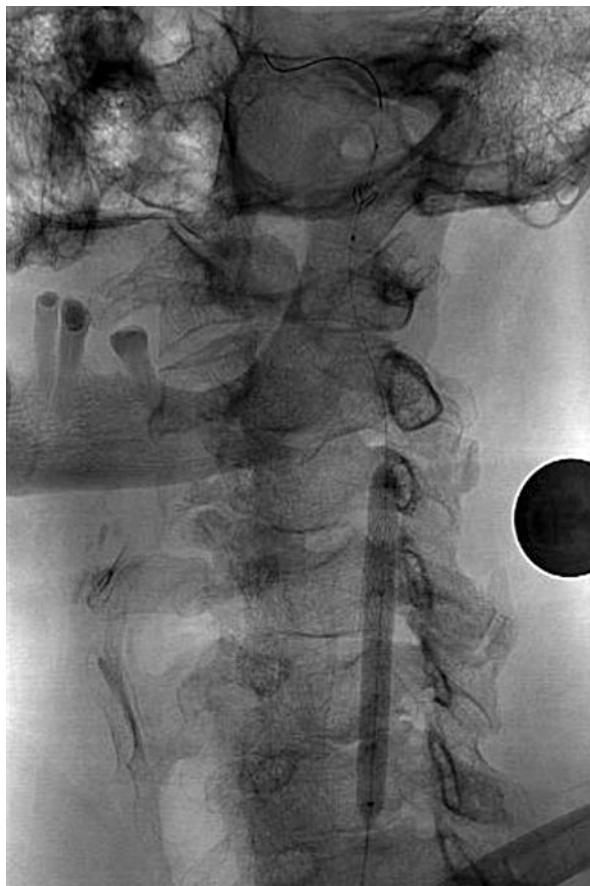
Case 9.2 Carotid In-Stent Restenosis (ISR): Off-Label Indication!



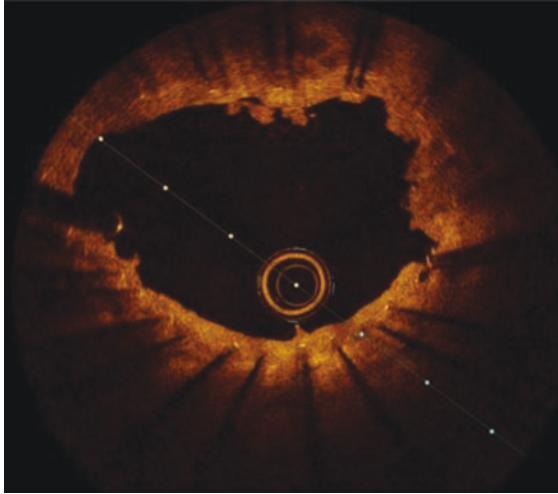
Baseline angiogram demonstrating calcific stenosis of CCA bifurcation with 80% stenosis within the stent, arrow



Baseline optical coherence tomography (OCT) image showing circumferential vessel wall calcification with reduced stent diameters and cross-sectional area



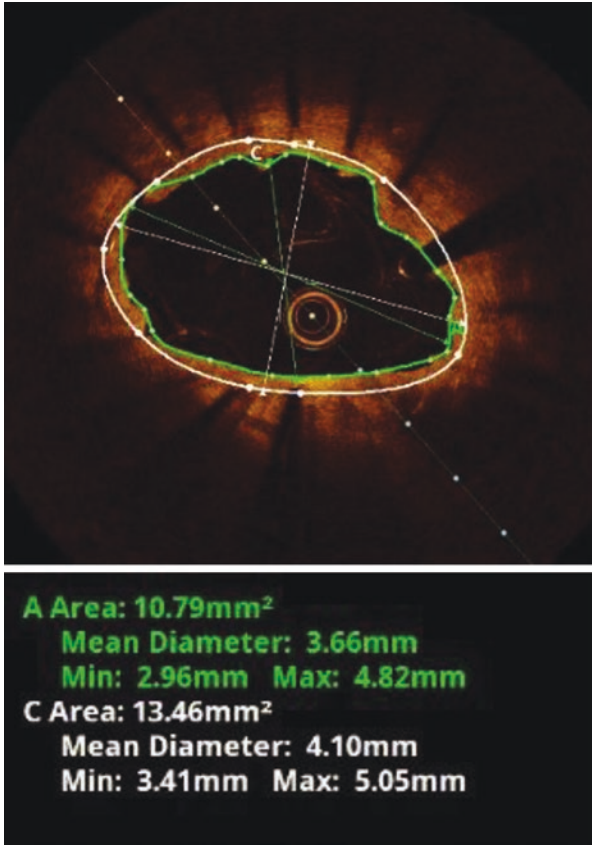
IVL of the carotid artery with 6 mm x 60 mm Shockwave balloon



OCT post-IVL showing doubling of stent lumen cross-sectional area



Post-IVL dilation with a 6 mm × 40 mm Bard Lutonix drug-coated balloon (DCB)



OCT post-IVL/DCB showing minimal additional lumen gain and stent expansion post-DCB—most gain from IVL



Final angiogram post-IVL/DCB demonstrating excellent stent expansion after IVL/DCB

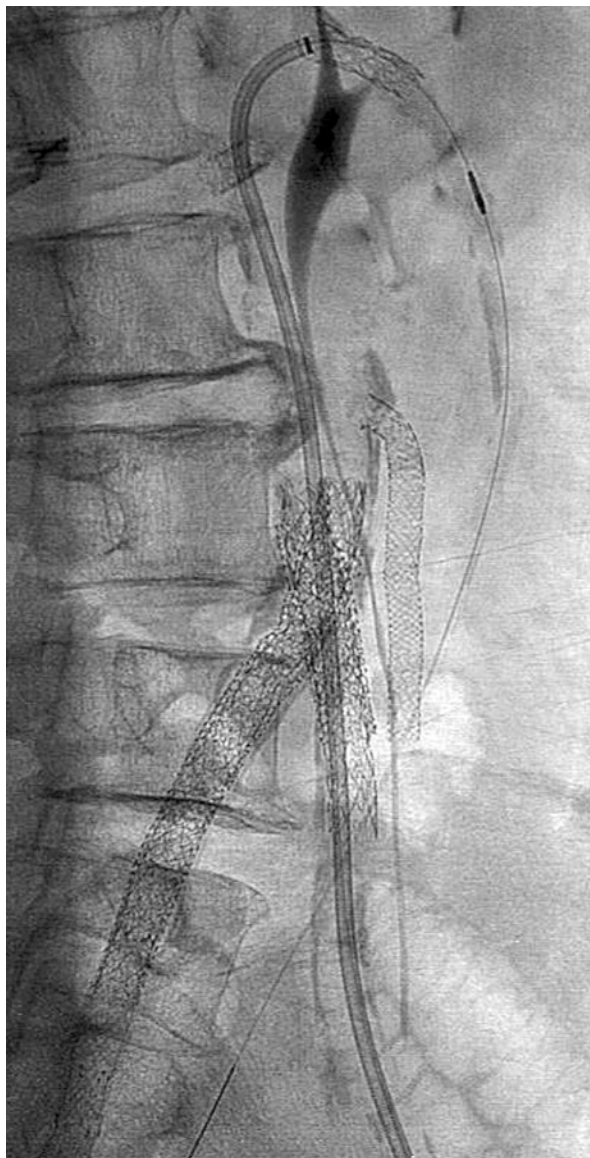
Case 9.3 Calcified Superior Mesenteric Artery Stenosis



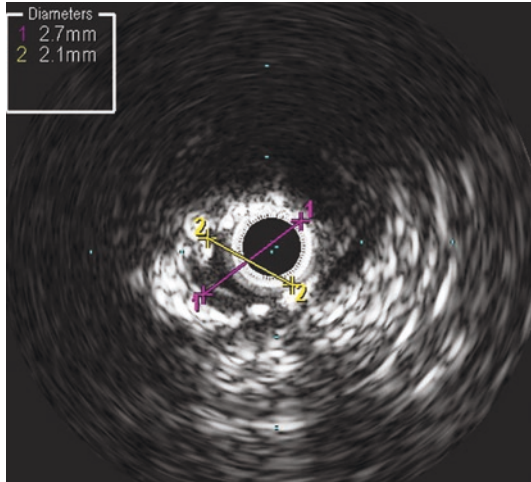
Baseline angiogram showing celiac occlusion, patent iliac, IMA stents, and patent ostial SMA stent with proximal SMA calcified stenosis



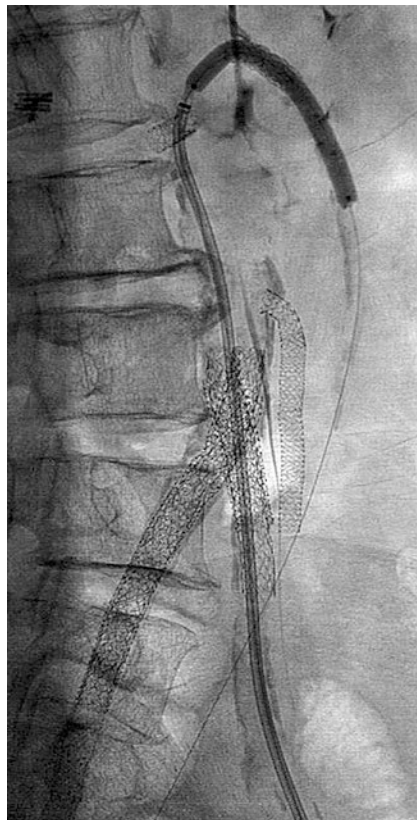
Selective SMA angiogram demonstrating patent ostial SMA stent with calcified proximal stenosis



Intravascular ultrasound of the SMA



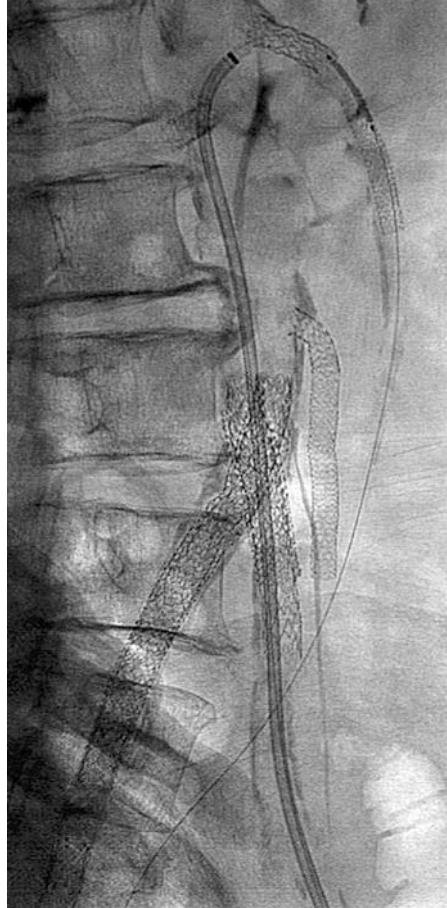
IVUS image showing circumferential severe calcification with high grade cross sectional stenosis



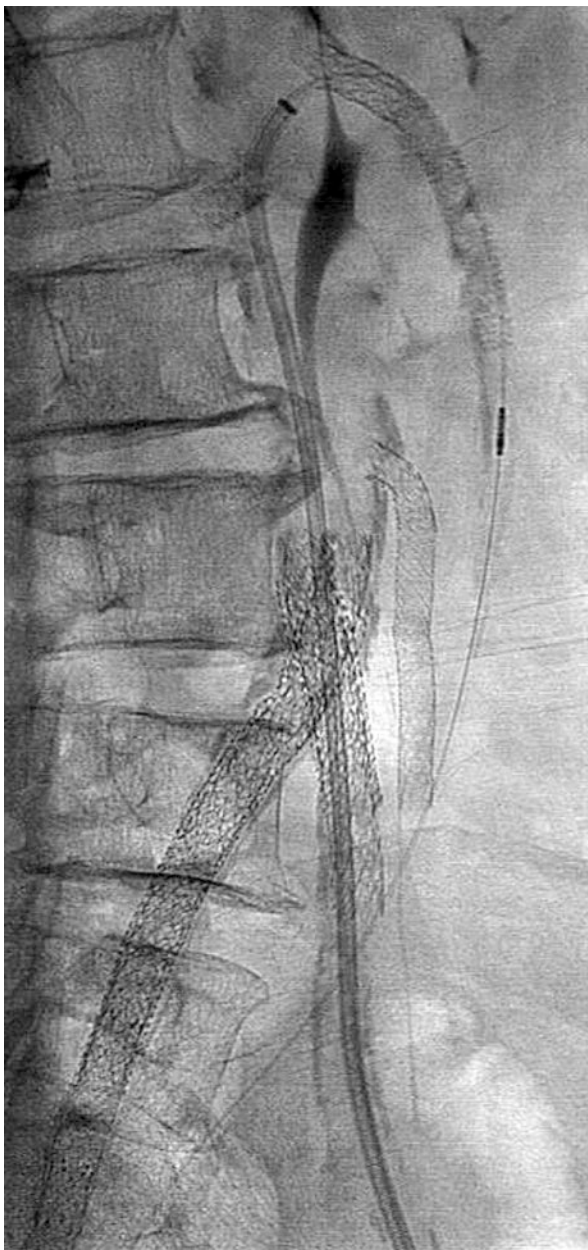
IVL SMA 5.0 mm x 60 mm balloon



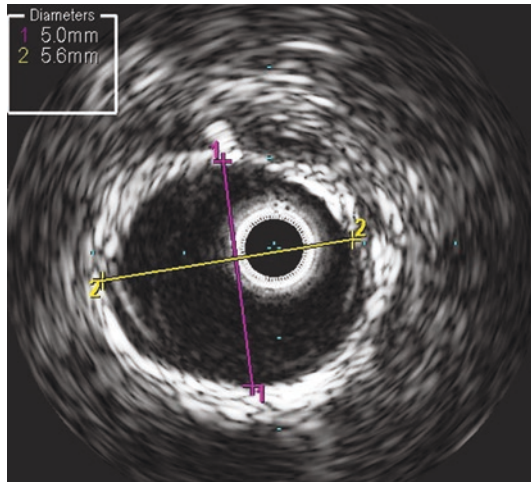
Angiogram post-IVL showing good lumen expansion



4.5 mm × 30, 5.0 mm × 15 mm Onyx drug-eluting stents after IVL



IVUS post-stenting

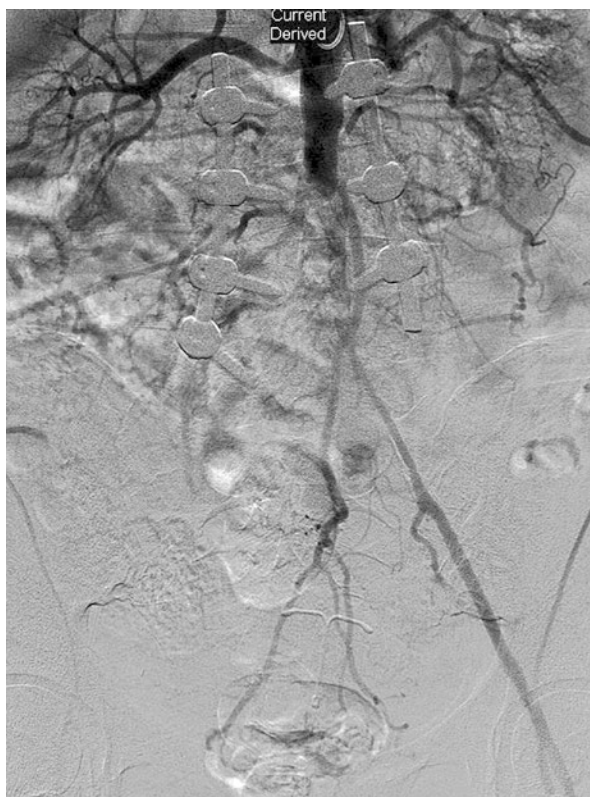


IVUS image post-IVL/stenting showing full stent apposition and expansion

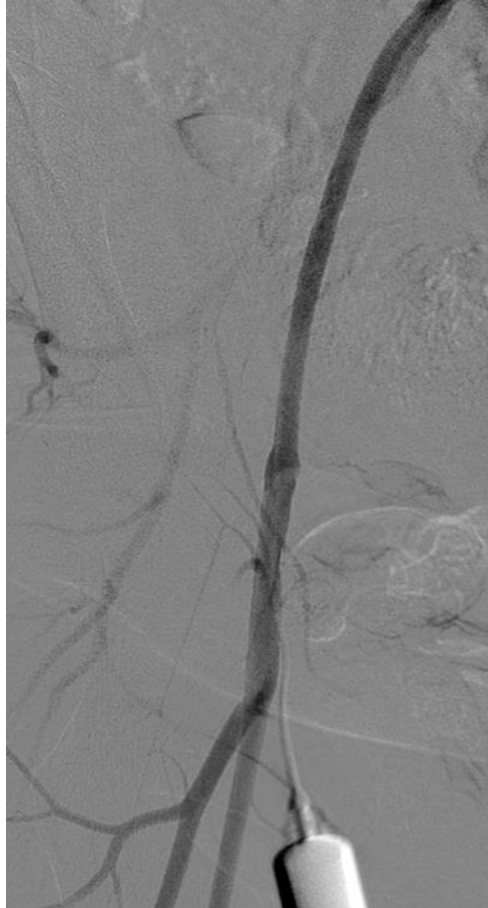


Final angiogram post-IVL/stenting

Case 9.4 Calcified Aorto-Iliac Occlusions



Baseline angiogram showing distal aortic occlusion with dense calcified common iliac artery occlusions



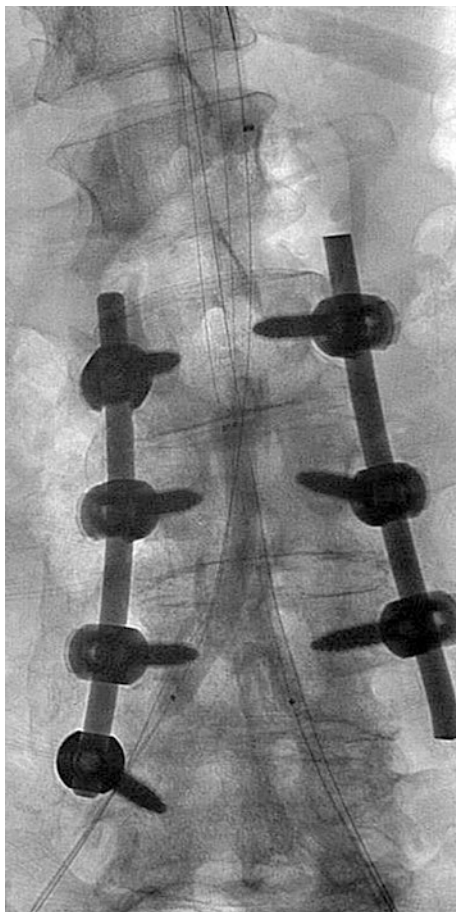
Angiography after obtaining retrograde right CFA access



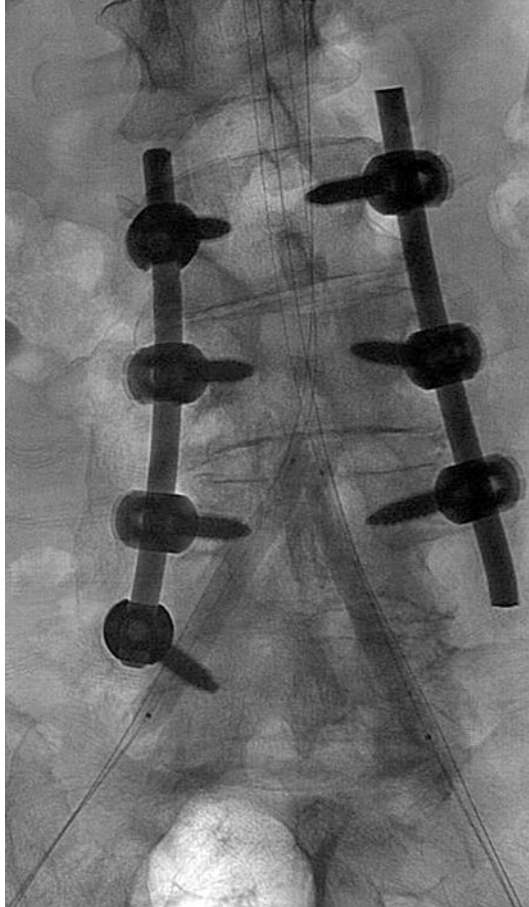
Angiography after obtaining retrograde left CFA access



Right iliac occlusion crossed antegrade, wire then exteriorized



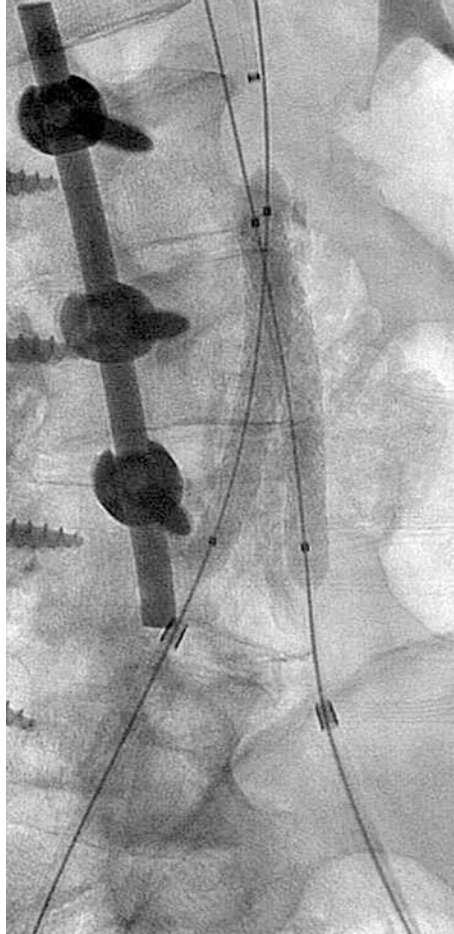
Upsized to 7F sheaths, pre-dilated with 5 mm balloons



Kissing inflations of distal aorta with two 7 mm × 60 mm Shockwave balloons



Kissing inflations of proximal common iliac arteries with two 7 mm × 60 mm Shockwave balloons



Angiogram post-IVL showing restoration of antegrade flow



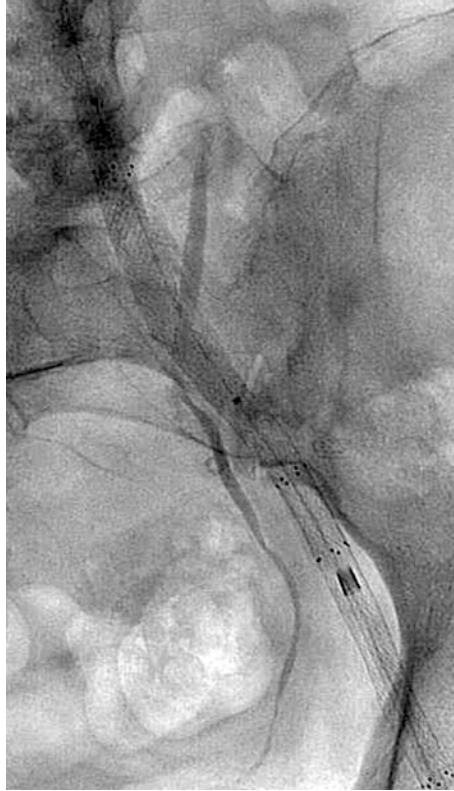
Final angiogram after post-dilation with 8 mm balloons

Case 9.5 IVL to Treat Stent Underexpansion

Baseline angiogram showing eccentric calcified iliac stenosis



Significant residual stenosis post-stenting



IVL of iliac ISR with 7 mm × 60 mm Shockwave balloon



Successful stent expansion post-IVL

Case 9.6 IVL to Treat Stent Underexpansion

Baseline angiogram showing high-grade calcified CFA stenosis



IVL CFA with 7 mm × 60 mm Shockwave Medical balloon



Angiogram after IVL shows full vessel expansion

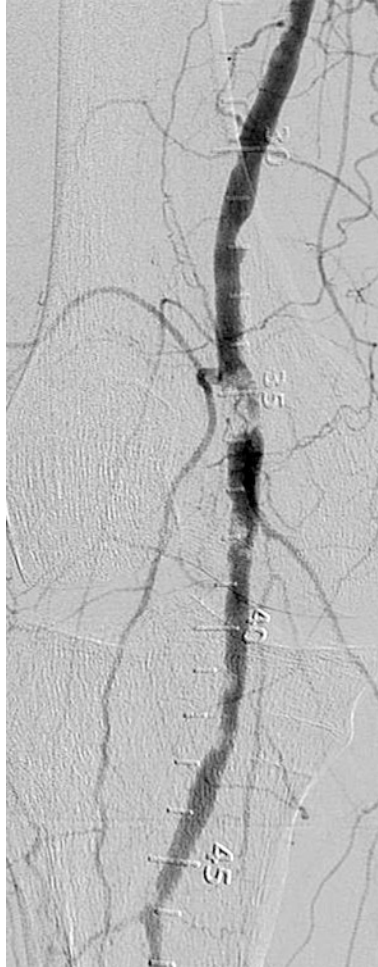


DCB CFA with 7 mm × 60 mm

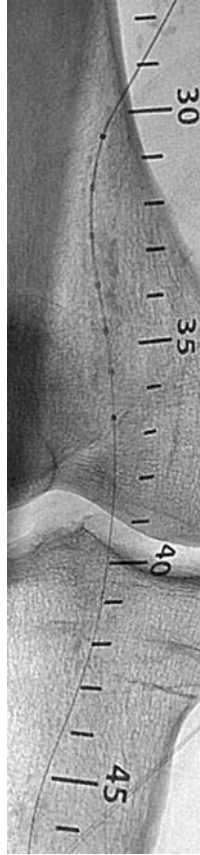


Final angiogram post-IVL/DCB

Case 9.7 IVL to Treat a Heavily Calcified Popliteal Artery “No-Stent Zone” Stenosis



Baseline angiogram showing heavily calcified popliteal artery stenosis



Fluoroscopy demonstrating severe calcification pre-inflation of IVL balloon



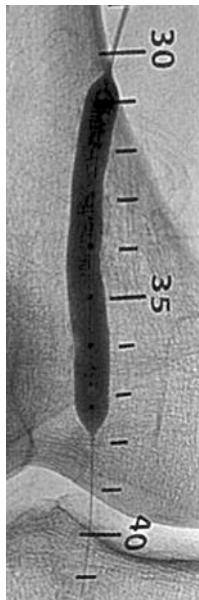
IVL of proximal popliteal stenosis with 6 mm x 60 mm Shockwave balloon



IVL of behind knee popliteal with same balloon



Angiogram post-IVL



IVL popliteal artery with 6.5 mm x 60 mm Shockwave balloon



Angiogram post-IVL showing excellent vessel expansion



DCB of the popliteal artery with 7 mm x 80 mm IN.PACT balloon



Final angiogram showing excellent expansion without dissection

References

1. Généreux P, Madhavan MV, Mintz GS, et al. Ischemic outcomes after coronary intervention of calcified vessels in acute coronary syndromes. Pooled analysis from the HORIZONS-AMI (harmonizing outcomes with revascularization and stents in acute myocardial infarction) and ACUITY (acute catheterization and urgent intervention triage strategy) TRIALS. *J Am Coll Cardiol.* 2014;63:1845–54.
2. Soor GS, Vukin I, Leong SW, Oreopoulos G, Butany J. Peripheral vascular disease: who gets it and why? A histomorphological analysis of 261 arterial segments from 58 cases. *Pathology.* 2008;40:385–91.
3. Frink RJ, Achor RW, Brown AL Jr, Kincaid OW, Brandenburg RO. Significance of calcification of the coronary arteries. *Am J Cardiol.* 1970;26:241–7.
4. Tan K, Sulke N, Taub N, Sowton E. Clinical and lesion morphologic determinants of coronary angioplasty success and complications: current experience. *J Am Coll Cardiol.* 1995;25:855–65.
5. Noble S, Roffi M. Overcoming the challenges of the transfemoral approach in transcatheter aortic valve implantation. *Interv Cardiol.* 2013;8(2):131–4.
6. Walker KL, Nolan BW, Columbo JA, et al. Lesion complexity drives the cost of superficial femoral artery endovascular interventions. *J Vasc Surg.* 2015;62:998–1002.
7. Fitzgerald PJ, Ports TA, Yock PG. Contribution of localized calcium deposits to dissection after angioplasty. An observational study using intravascular ultrasound. *Circulation.* 1992;86:64–70.
8. McAteer JA, Bailey MR, Williams JC Jr, et al. Strategies for improved shock wave lithotripsy. *Minerva Urol Nefrol.* 2005;57(4):271–87.
9. Davros WJ, Garra BS, Zeman RK. Gallstone lithotripsy: relevant physical principles and technical issues. *Radiology.* 1991;178:397–408.

10. Ali ZA, Brinton TJ, Hill JM, et al. Optical coherence tomography characterization of coronary lithoplasty for treatment of calcified lesions: first description. *JACC Cardiovasc Imaging*. 2017;10(8):897–906.
11. Brinton TJ, Ali ZA, et al. Feasibility of shockwave coronary intravascular lithotripsy for the treatment of calcified coronary stenoses. *Circulation*. 2019;139:834–6.
12. Ali ZA, Nef H, et al. Safety and effectiveness of coronary intravascular lithotripsy for treatment of severely calcified coronary stenoses; the disrupt CAD II study. *Circ Cardiovasc Interv*. 2019;12:e008434.
13. Feldman DN, Armstrong EJ, Aronow HD, et al. SCAI consensus guidelines for device selection in femoral-popliteal arterial interventions. *Catheter Cardiovasc Interv*. 2018;92(1):124–40.
14. Patel MR, Conte MS, Cutlip DE, et al. Evaluation and treatment of patients with lower extremity peripheral artery disease: consensus definitions from peripheral academic research consortium (PARC). *J Am Coll Cardiol*. 2015;65(9):931–41.
15. Rocha-Singh KJ, Zeller T, Jaff MR. Peripheral arterial calcification: prevalence, mechanism, detection, and clinical implications. *Catheter Cardiovasc Interv*. 2014;83(6):E212–20.
16. Thukkani AK, Kinlay S. Endovascular intervention for peripheral artery disease. *Circ Res*. 2015;116(9):1599–613.
17. Dini CS, Tomberli B, Mattesini A, et al. Intravascular lithotripsy for calcific coronary and peripheral artery stenoses. *EuroIntervention*. 2019;15:714–21.
18. Brodmann M, Werner M, Brinton TJ, et al. Safety and performance of lithoplasty for treatment of calcified peripheral artery lesions. *J Am Coll Cardiol*. 2017;70(7):908–10.
19. Brodmann M, Werner M, Holden A, et al. Primary outcomes and mechanism of action of intravascular lithotripsy in calcified, femoropopliteal lesions: result of disrupt PAD II. *Catheter Cardiovasc Interv*. 2019;93(2):335–42.
20. Brodmann M, Holden A, Zeller T. Safety and feasibility of intravascular lithotripsy for treatment of below-the-knee arterial stenoses. *J Endovasc Ther*. 2018;25(4):499–503.
21. Schneider PA, Laird JR, Tepe G, et al. Treatment effect of drug-coated balloons is durable to 3 years in the femoro-popliteal arteries: long-term results of the IN.PACT SFA randomized trial. *Circ Cardiovasc Interv*. 2018;11(1):e005891.
22. Lugenbiel I, Grebner M, Zhou Q, et al. Treatment of femoropopliteal lesions with the AngioSculpt scoring balloon—results from the Heidelberg PANTHER registry. *Vasa*. 2018;47(1):49–55.
23. Zeller T, Langhoff R, Rocha-Singh KJ, et al. Directional atherectomy followed by a paclitaxel-coated balloon to inhibit restenosis and maintain vessel patency: twelve-month results of the DEFINITIVE AR study. *Circ Cardiovasc Interv*. 2017;10(9):e004848.
24. Fanelli F, Cannavale A, Gazzetti M, et al. Calcium burden assessment and impact on drug-eluting balloons in peripheral arterial disease. *Cardiovasc Intervent Radiol*. 2014;37(4):898–907.
25. Adams G, Shammam N, Mangalmurti S, et al. Intravascular lithotripsy for treatment of calcified lower extremity arterial stenosis: initial analysis of the disrupt PAD III study. *J Endovasc Ther*. 2020;27(3):473–80.
26. Tepe G, Brodmann M, Bachincky W, Holden A, Zeller T, Mangalmurti S, Nolte-Ernsting C, Virmani R, Parikh S, Gray W, for the Disrupt PAD III Investigators. Intravascular lithotripsy for peripheral artery calcification: mid-term outcomes from the randomized disrupt PAD III trial. *JSCAI*. 2022. <https://doi.org/10.1016/j.jscai.2022.100341>.
27. Bavaria JE, Tommaso CL, Brindis RG, et al. 2018 AATS/ACC/SCAI/STS expert consensus systems of care document: operator and institutional recommendations and requirements for transcatheter aortic valve replacement: a joint report of the American Association for Thoracic Surgery, the American College of Cardiology, the Society for Cardiovascular Angiography and Interventions, and the Society of Thoracic Surgeons. *J Am Coll Cardiol*. 2019;73:340–74.
28. Holmes DR, Nishimura RA, Grover FL, et al. Annual outcomes with transcatheter valve therapy: from the STS/ACC TVT registry. *J Am Coll Cardiol*. 2015;66:2813–23.
29. Biasco L, Ferrari E, et al. Access sites for TAVI: patient selection criteria, technical aspects, and outcomes. *Front Cardiovasc Med*. 2018;5:88.

30. Di Mario C, Chiriatti N, et al. Lithoplasty-assisted transfemoral aortic valve implantation. *Eur Heart J*. 2018;41(8):942.
31. Gorla R, Cannone GS, et al. Transfemoral aortic valve implantation following lithoplasty of iliac artery in a patient with poor vascular access. *Catheter Cardiovasc Interv*. 2019;93:E140–2.
32. Di Mario C, Goodwin M, et al. A prospective registry of intravascular lithotripsy-enabled vascular access for transfemoral transcatheter aortic valve replacement. *JACC Cardiovasc Interv*. 2019;12(5):502–4.
33. Rosseel L, De Backer O, Søndergaard L, Bieliauskas G. Intravascular iliac artery lithotripsy to enable transfemoral thoracic endovascular aortic repair. *Catheter Cardiovasc Interv*. 2011;95:E96–9.
34. Riley RF, Corl JD, Kereiakes DJ. Intravascular lithotripsy-assisted Impella insertion: a case report. *Catheter Cardiovasc Interv*. 2019;93(7):1317–9. <https://doi.org/10.1002/ccd.28168>.
35. Mordasini P, Gralla J, Do DD, Schmidli, Keseru B, Arnold M, Fischer U, Schmidli G, Brekenfeld C. Percutaneous and open retrograde endovascular stenting of symptomatic high-grade innominate artery stenosis: technique and follow-up. *AJNR Am J Neuroradiol*. 2011;32(9):1726–31.
36. Giannopoulos S, Speziale F, Vadal G, Soukas PA, Kuhn BA, Stolz CL, Foteh MI, Mena-Hurtado C, Armstrong EJ. Intravascular lithotripsy for treatment of calcified lesions during carotid artery stenting. *J Endovasc Ther*. 2021;28(1):93–9. <https://doi.org/10.1177/1526602820954244>. Epub 2020 Sep 1. PMID: 32869718.
37. Khan MS, Baig M, Hyder ON, Aronow HD, Soukas PA. Intravascular lithotripsy for the treatment of severely calcified mesenteric stenosis. *JACC Case Rep*. 2020;2(6):956–60.
38. Armstrong EJ, Soukas PA, Shammas N, Chamberlain J, Pop A, Adams G, de Freitas D, Valle J, Woo E, Bernardo NL. Intravascular lithotripsy for the treatment of calcified, stenotic iliac arteries: a cohort analysis from the disrupt PAD III study. *Cardiovasc Revasc Med*. 2020;21(10):1262–8. <https://doi.org/10.1016/j.carrev.2020.02.026>. Epub 2020 Mar 2. PMID: 32147133.
39. Nguyen BN, Amdur RL, Abugideiri M, et al. Postoperative complications after common femoral endarterectomy. *J Vasc Surg*. 2015;61(6):1489–1494.e1.
40. Brodmann M, Schwindt A, Argyrios A, Gammon R. Safety and feasibility of intravascular lithotripsy for treatment of common femoral artery stenoses. *J Endovasc Ther*. 2019;26(3):283–7.
41. Guzman RJ, Brinkley DM, Schumacher PM, et al. Tibial artery calcification as a marker of amputation risk in patients with peripheral artery disease. *J Am Coll Cardiol*. 2008;51(2):1967–74.
42. Bauman F, Fust J, Engelberger RP, et al. Early recoil after balloon angioplasty of tibial artery obstructions in patients with critical limb ischemia. *J Endovasc Ther*. 2014;21(1):44–51.
43. Hill JM, Kereiakes DJ, et al. Intravascular lithotripsy for treatment of severely calcified coronary artery disease. *J Am Coll Cardiol*. 2020;76(22):2635–46.
44. Case B, Yurasi C, Waxman R, et al. Intravascular lithotripsy facilitated percutaneous endovascular intervention of the aortic arch: a single-center experience. *Cardiovasc Revasc Med*. 2020;21(8):1006–15.
45. Henry CL, Hansen SK, et al. Intravascular lithotripsy during trans-carotid arterial revascularization for highly calcified lesions in high-risk patients. *J Vasc Surg Cases Innov Tech*. 2020;7(1):68–73. <https://doi.org/10.1016/j.jvscit.2020.10.018>.