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Editorial Note: Abrupt Vessel Closure in Percutaneous Coronary Intervention - Lessons from the Cath Lab

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Introduction

The past thirty years have seen a shift in percutaneous coronary intervention (PCI) from simple balloon angioplasty to modern day practices characterized by drug eluting stents (DES) along with powerful antiplatelet medications and sophisticated intra-vascular imaging. With this progress, complication rates have steadily declined. Yet, the specter of abrupt vessel closure (AVC) continues to haunt the catheterization laboratory. Though the incidence of AVC has fallen from approximately 3% in the balloon angioplasty era to 0.3% in the DES era [1], its occurrence remains a life-threatening challenge.

AVC is defined as the sudden cessation of antegrade coronary blood flow during or immediately after PCI. It is not only catastrophic in its clinical consequences but also a litmus test of the operator's preparedness, judgment, and technical skill. In this editorial note, we revisit AVC from a contemporary lens, distilling mechanistic insights, risk factors, management strategies, and pearls of wisdom that every interventional cardiologist must carry into the lab.

The Historical Perspective

Early PCI with plain balloon angioplasty was marred by high rates of AVC, mostly due to elastic recoil, flow-limiting dissections, and acute thrombosis. From the adoption of bare metal stents, recoil and dissection related AVC were reduced, and later DES with dual antiplatelet therapy reduced thrombotic complications. AVC is modern, still occurring much more with advanced complexity and in patients with more advanced comorbidities. This is still a relative anomaly despite these contradictions in progress accomplished [2].

The Multifactorial Mechanisms

The most common mechanism of AVC remains dissection with or without thrombus formation [3]. Other culprits include intramural hematoma, acute stent thrombosis, intracoronary thrombus, no-reflow phenomenon, vasospasm, and iatrogenic embolization of air or plaque. Recognizing the mechanism rapidly is crucial, as management differs substantially.

Clinical and Angiographic Risk Factors

Not all lesions or patients carry equal risk. Predictors include:

- ***Clinical Factors***: acute MI presentation, unstable angina, renal impairment, female sex.
- ***Angiographic Factors***: thrombus-laden lesions, degenerated saphenous veins grafts, diffuse or long lesions, heavy calcification, and marked angulation [4].

Operators need to plan interdisciplinary risk and prepared bailout strategies in these scenarios.

Dissection - The Classic Culprit

Dissection still remains the number one cause of AVCs. The NHLBI classification (Types A through F) subdivides dissections by angiographic form with increasing risk of AVC: about 3% in Type B to greater than 60% in Type F [5].

Guide catheter-induced dissections, stent-edge dissections, and spiral dissections are the most frequent iatrogenic varieties today. Intravascular ultrasound (IVUS) or optical coherence tomography (OCT) can help distinguish true lumen from false lumen and guide treatment.

Managing Dissection-Induced AVC

The cardinal principle is: "Never lose the wire in the true lumen." Even when hemodynamic support is required, arterial access should be preserved around the wire. Once true lumen position is confirmed, sealing the dissection with balloon inflation or stenting is the mainstay.

Extensive dissections may need a distal-to-proximal stenting strategy to prevent propagation. If wire position is uncertain, IVUS-guided parallel wire techniques or CTO approaches may be applied [6].

Intramural Hematoma - The Silent Threat

Intramural hematoma is under-recognized because angiography often shows only new stenosis at stent edges without dissection flap. IVUS/OCT is invaluable for diagnosis.

Management differs from dissection: plain ballooning is ineffective and may worsen the hematoma. Options include stenting beyond the hematoma edge, cutting balloons for fenestration, or emerging methods like subintimal aspiration [7]. Recognizing this entity early prevents futile or harmful interventions.

Intracoronary Thrombosis

Despite potent dual antiplatelet therapy and meticulous anticoagulation, acute thrombosis still contributes to AVC, especially in thrombus-rich lesions or CTO PCI [8].

Treatment options:

- ***Procedural approaches***: such as manual or mechanical thrombectomy.
- ***Pharmacologic options***: including glycoprotein IIb/IIIa inhibitors, escalation of anticoagulation, or—in refractory cases—intracoronary thrombolysis.
- ***Monitoring***: maintaining strict control of activated clotting time (ACT), with reassessment every 30 minutes, is essential to minimize the risks of both under- and over-anticoagulation.

Air Embolism

Though uncommon (0.2-0.8%), air embolism can trigger catastrophic AVC. It is usually iatrogenic, caused by improper flushing of catheters or ruptured balloons [9].

Mild cases may be asymptomatic; severe embolism can lead to cardiogenic shock. Management includes:

- 100% oxygen (to hasten absorption).
- Forceful saline injection or aspiration via guiding/microcatheter.
- Balloon disruption of large bubbles.

Prevention through meticulous catheter preparation is paramount.

Vasospasm - The Masquerader

Catheter or wire manipulation can induce focal or diffuse coronary spasm, occasionally misinterpreted as dissection or thrombus. The diagnostic hallmark is resolution after intracoronary vasodilators (nitroglycerin, verapamil, nicorandil).

Recognizing vasospasm avoids unnecessary stenting, which could otherwise worsen outcomes [10].

The No-Reflow Phenomenon

No-reflow is characterized by reduced TIMI flow despite no mechanical obstruction, due to microvascular dysfunction, distal embolization, or reperfusion injury [10].

Risk factors include: small vessels, long stents, cardiogenic shock, PCI of left main or grafts, and delayed reperfusion. Diagnosis requires excluding other causes (dissection, hematoma, thrombus, spasm).

Management: distal delivery of intracoronary vasodilators (adenosine, nicorandil, nitroprusside, verapamil). Refractory cases may respond to low-dose epinephrine.

The General Algorithm - "60-second rule"

In all AVC cases, calm, structured response is essential. Doll et al. proposed the "60-second rule": pause for one minute, if hemodynamically safe, to review angiograms, consider differentials, and form a plan [12].

Immediate intervention is required in unstable patients (shock, arrest), but when stable, taking this pause improves diagnostic clarity and avoids unnecessary or harmful maneuvers.

Imaging - Seeing Beyond the Angiogram

Conventional angiography often misses subtle dissections, hematomas, or small thrombi. IVUS and OCT provide critical insights into mechanism and guidewire position. Their use in AVC cannot be overemphasized [13].

Hemodynamic & Supportive Care

Every case of AVC demands parallel resuscitation:

- Atropine and fluids for bradycardia/hypotension.
- Vasopressors/inotropes and intra-aortic balloon pump if unstable.
- Antiarrhythmic drugs or cardioversion for malignant arrhythmias.

Mechanical circulatory support (Impella, ECMO) may be required in refractory collapse.

Lessons for Prevention

Preventive wisdom is as critical as bailout skill:

- Avoid deep catheter engagement.
- Flush lines meticulously to prevent air.
- Gentle balloon inflations and cautious stent deployment, especially in calcified or lipid-rich plaques.
- Ensure effective anticoagulation and DAPT.

As the aphorism goes, "*An ounce of prevention is worth a pound of cure*."

The Educational Imperative

Because AVC is infrequent in the DES era, direct experience is limited. Hence, education must rely on case reviews, simulation, and morbidity-mortality meetings. Sharing knowledge—whether in conferences, digital platforms, or peer discussions—ensures that operators remain prepared for these rare but deadly events. As one expert aptly noted, "*You may see AVC once a year, but you must be ready every day*."

Conclusion

Abrupt vessel closure in PCI is a rare but formidable adversary. Its mechanisms are diverse, ranging from dissection and thrombosis to hematoma, vasospasm, and no-reflow. Recognition of risk factors, calm adherence to structured algorithms, and judicious use of intravascular imaging are central to successful management.

Ultimately, AVC tests not only technical skill but also composure, teamwork, and judgment in the cath lab. By preparing for the worst while striving for the best, interventional cardiologists can continue to transform PCI into a safer, more predictable therapy for patients worldwide.

Author Contributions

The sole author of the Editorial note is Dr Rohit Mody. He is sole responsible for data curation, formal analysis, resources, software, validation, visualization, writing - original draft, Writing, review & editing.

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Availability of Data and Materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

Ethical approval was not required since it is an accepted procedure.

Consent for Publication

Written consent has been obtained to publish the review article from the guardian. The consent copy is available with the authors and ready to be submitted if required.

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