Percutaneous coronary intervention

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Abstract

Percutaneous coronary intervention (PCI) is a cornerstone therapy in the management of coronary artery disease, a condition driven by atherosclerotic plaque formation and inflammation. Since its inception PCI has undergone major technological and procedural evolution, progressing from plain balloon angioplasty to contemporary drug-eluting stents and adjunctive intravascular imaging. PCI aims to restore coronary blood flow safely and effectively while minimizing re-stenosis, stent thrombosis and procedural complications. Indications encompass stable angina refractory to optimal medical therapy, non-ST elevation myocardial infarction where PCI improves symptoms and mortality, and ST elevation myocardial infarction, in which timely PCI is the preferred reperfusion strategy. Advances in pharmacotherapy, including potent $P2Y_{12}$ inhibitors and tailored durations of dual antiplatelet therapy, have further optimized outcomes. Pre-procedural computed tomography coronary angiography enables detailed anatomical and physiological assessment, enhancing procedural planning. Contemporary PCI emphasizes meticulous lesion preparation, appropriate calcium modification, and image-guided stent deployment to ensure optimal expansion. New technologies such as intravascular lithotripsy have improved the management of disease with heavily calcification. Continuing refinements are improving precision, safety and long-term clinical outcomes in people undergoing PCI.

Keywords Atherectomy; coronary angiography; coronary artery disease; drug-eluting stents; dual antiplatelet therapy; intravascular ultrasonography; myocardial infarction; optical coherence tomography; percutaneous coronary intervention; vascular calcification

Key points

- Treatment of coronary artery disease resulting from atherosclerosis is the leading indication for percutaneous coronary intervention (PCI)
- PCI has evolved from balloon angioplasty to contemporary thin-strut drug-eluting stents and image-guided precision techniques
- Indications for PCI include refractory stable angina, non-ST segment elevation myocardial infarction (NSTEMI) and ST segment elevation myocardial infarction (STEMI), where rapid reperfusion significantly improves outcomes
- CT coronary angiography provides detailed anatomical and physiological assessment that increasingly informs procedural planning
- Optimal outcomes rely on meticulous lesion preparation including calcium modification strategies, intravascular imaging and appropriate stent selection and optimization

Introduction

Coronary artery disease (CAD), caused by atherosclerosis, results in progressive chronic vessel luminal obstruction and downstream myocardial ischaemia or acute vessel occlusion as a result of plaque rupture.

Coronary atherosclerosis is caused by an accumulation of cholesterol-rich lipids within the arterial sub-intimal space and is driven by the well-established risk factors of male sex, a family history of CAD, hypertension, hypercholesterolaemia, diabetes mellitus, tobacco smoking, obesity and chronic inflammatory conditions.

This process is modulated by key immune cells, typically monocytes and T cells, which perpetuate the cycle of coronary inflammation and atherosclerosis development through a series of cytokine signalling pathways and subsequent DNA damage,[1] as outlined in Figure 1.

Percutaneous coronary intervention (PCI) has evolved significantly since its introduction in 1977, when Andreas Grüntzig performed the first successful coronary balloon angioplasty. The aim of PCI is to increase the coronary arterial luminal area to improve blood flow to the downstream myocardium. Early PCI was limited by high rates of acute vessel closure and re-stenosis, largely caused by elastic recoil, neo-intimal hyperplasia and acute arterial dissection that necessitated emergency coronary artery bypass grafting (CABG).

The advent of bare-metal stents in the late 1980s represented a major step forward, providing a mechanical scaffold that reduced acute vessel closure and improved procedural safety. However, in-stent re-stenosis remained a significant limitation, prompting the development of drug-eluting stents (DESs) in the early 2000s. By delivering anti-proliferative agents locally, first-generation DESs substantially reduced re-stenosis rates, although concerns regarding late stent thrombosis led to refinements in stent design and polymer technology.

Contemporary PCI incorporates thinner strut and second- and third-generation DESs with biocompatible or bioresorbable polymers improving long-term outcomes. Adjunctive PCI advances (e.g. intravascular imaging, physiological assessment with fractional flow reserve, improved antiplatelet regimens) have further enhanced precision and safety.

Indications for PCI

The indications for PCI broadly fall into two categories. First is patients who have stable angina with symptoms caused by

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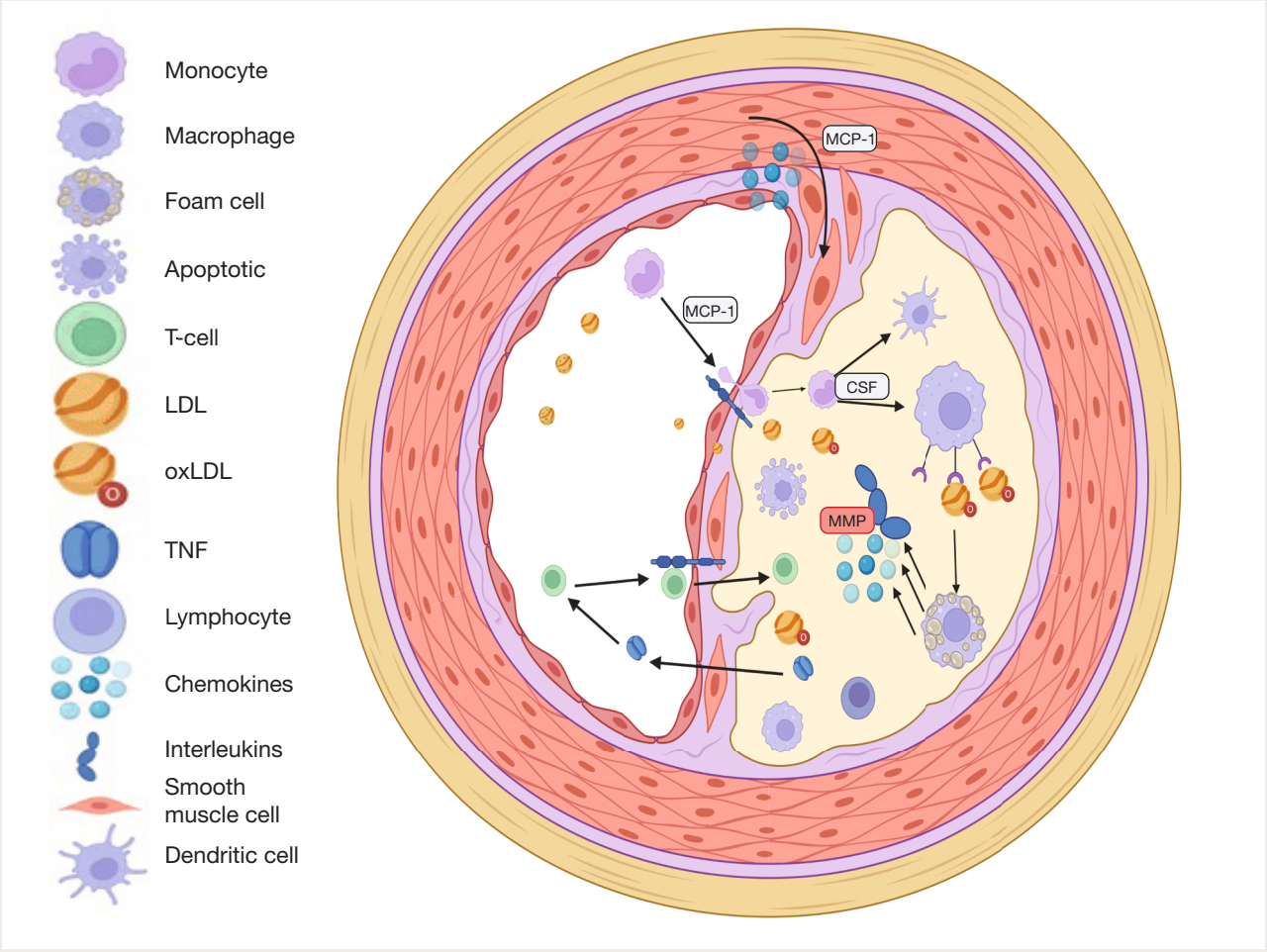
Schematic representation of the pathogenesis of atherosclerosis.



Cholesterol-rich lipoproteins cross the tunica intimal by transcytosis and become oxidized (oxLDL). Chemoattractant protein-1 (MCP-1) is released, and monocytes are recruited and enter the intimal space. Under the influence of specific colony-stimulating factors, monocytes differentiate into macrophages and take up the deleterious oxLDL via specific scavenger receptors. After intracellular oxLDL metabolism, lipid droplets are formed within the cytoplasm, forming foam cells. Smooth muscle cell migration results in the formation of a fibrous cap protecting the circulating blood from the lipid-rich pool beneath. LDL, low-density lipoprotein. TNF, tumour necrosis factor.

Figure 1

epicardial CAD despite 'optimal medical therapy'. Current UK National Institute for Health and Care Excellence guidelines suggest that patients should have uncontrolled symptoms despite two antianginal medications or prognostically important CAD (left main stem disease, left anterior descending stenosis, a large area of ischaemia) before PCI is offered.

However, recent randomized data from the ORBITA II trial suggest that PCI may be as good as medical therapy for reducing angina. Importantly, contemporary randomized data show that PCI for individuals with stable angina does not infer a mortality benefit.

The complexity of individual CAD influences outcomes and modality for revascularization; discussion in a multidisciplinary heart team is essential in identifying the most appropriate form of revascularization when deciding between CABG and PCI in complex cases. Patient factors including co-morbidities and anatomical risk scoring should be undertaken using, for instance the SYNTAX score, a well-validated tool to define the complexity of CAD.[2]

The second category is patients with acute coronary syndrome (ACS), for whom PCI confers both a symptomatic and, importantly, a mortality benefit.

CABG can also be considered after a multidisciplinary team discussion for patients with non-ST segment elevation myocardial infarction (NSTEMI) after coronary angiography if the CAD anatomy is highly complex, whether or not the patient has a past medical history of diabetes mellitus. Important caveats are continuing ischaemic chest pain, recurrent ventricular arrhythmias or haemodynamic instability in which urgent revascularization with PCI is often mandated.

With regard to ST segment elevation myocardial infarction (STEMI), PCI is the preferred reperfusion strategy over fibrinolysis provided it can be feasibly undertaken within 120 minutes

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of first presentation to the medical services (paramedics or emergency department). Important considerations include inter-hospital transfer time to a PCI centre; adjunctive fibrinolysis can be considered if the reperfusion time is expected to be >120 min. Importantly, PCI for STEMI has a strong evidence base for improved mortality, reinfarction and stroke.

Pharmacotherapy for PCI

Dual antiplatelet therapy (DAPT), consisting of aspirin and a $P2Y_{12}$ receptor inhibitor, is essential to reduce the risk of acute and subacute stent thrombosis, myocardial infarction and death. Aspirin is universally administered before PCI as a loading dose of 300 mg orally. Its mechanism — irreversible cyclooxygenase-1 inhibition — provides rapid and sustained suppression of thromboxane A_2-mediated platelet aggregation. Aspirin is continued indefinitely after PCI unless contraindicated.

The choice of $P2Y_{12}$ inhibitor depends on the clinical setting. Clopidogrel, a pro-drug requiring hepatic activation, has historically been the most widely used agent. A loading dose of 300–600 mg is followed by 75 mg daily. Although effective, clopidogrel exhibits variable laboratory inter-individual responses because of genetic polymorphisms and drug–drug interactions. Consequently, more potent agents have become preferred in ACS.

Prasugrel and ticagrelor offer faster onset, greater platelet inhibition and improved clinical outcomes in ACS patients undergoing PCI. Prasugrel is administered as a 60 mg loading dose followed by 10 mg daily. However, it is contraindicated in individuals with previous stroke or transient ischaemic attack, and should be used cautiously in patients aged ≥75 years or with low body weight.

Ticagrelor, given as a 180 mg loading dose followed by 90 mg twice daily, is suitable for a broader range of patients, although dyspnoea and bradyarrhythmias can limit its use because of production of adenosine as an intermediate metabolite.

In stable CAD, clopidogrel remains acceptable, as the incremental benefit of prasugrel or ticagrelor over clopidogrel is less clear in lower risk populations.

The optimal duration of DAPT is determined by the balance between ischaemic and bleeding risks. Traditionally, 12 months of DAPT has been recommended after PCI for ACS, with shorter durations of 3–6 months considered for stable patients with contemporary DESs.

Recent evidence has supported individualized strategies, including shorter DAPT followed by $P2Y_{12}$ inhibitor monotherapy in selected patients with an increased bleeding risk. Conversely, prolonged DAPT can be appropriate in patients with high ischaemic risk features such as previous stent thrombosis, diffuse disease or complex PCI.[3]

Adjunctive intra-procedural agents can be used in specific scenarios. Glycoprotein IIb/IIIa inhibitors are reserved mainly for therapy in cases of extensive thrombus or a no-reflow situation. Cangrelor, an intravenous $P2Y_{12}$ inhibitor with rapid onset and offset, may be used when oral loading is not possible.

Computed tomography coronary angiography (CTCA) for procedural planning

CTCA has emerged as an invaluable non-invasive imaging modality for evaluating CAD and increasingly serves an important role in procedural planning for PCI. With continuous improvements in scanner technology, spatial resolution and post-processing capabilities, CTCA can provide detailed anatomical information that complements invasive angiography and influence both indications for PCI and the technical strategy required to achieve optimal results.

Furthermore, CTCA can assess coronary physiology by computational fluid dynamics to simulate blood flow and calculate the pressure drop across lesions approximating to invasive fractional flow reserve; this allows both vessel-specific and lesion-specific ischaemia assessment. This can refine decision-making about whether PCI is truly indicated and prevent unnecessary invasive assessment in borderline or ambiguous cases.[4]

One of the principal advantages of CTCA is its ability to offer comprehensive, three-dimensional visualization of the coronary tree. This allows accurate delineation of lesion length, vessel tortuosity, bifurcation anatomy and plaque distribution before catheterization. In patients with diffuse or multivessel disease, CTCA can identify lesions unsuitable for PCI or highlight areas where intervention might offer limited benefit, thereby refining patient selection and reducing unnecessary procedures.

CTCA is particularly valuable for characterizing plaque morphology. High-resolution imaging enables the detection of calcification, lipid-rich plaque, positive remodelling and features associated with plaque vulnerability. Quantification of calcific burden helps operators decide whether calcium modification techniques might be required (Figure 2).

Basic procedural techniques

Vascular access and initial set-up

The procedure begins with vascular access. The transradial approach is now considered the preferred route for most patients because of its lower risk of bleeding and vascular complications, alongside improved patient comfort and faster post-procedural mobilization. When radial access is not feasible (e.g. small-calibre arteries, previous radial occlusion, need to use large-bore catheters) the femoral approach remains a crucial alternative.

Guiding catheter engagement and coronary wiring

A guiding catheter is advanced to the coronary ostium under fluoroscopy. Selection depends on the coronary anatomy, operator preference and support required for device delivery. Coronary wiring is in most cases performed with a workhorse guidewire; hydrophilic or specialized chronic total occlusion wires can be selected for more complex lesions. Wire manipulation techniques (e.g. torque control, shaping, microcatheter use) facilitate safe navigation through tortuous or obstructed segments.

Lesion preparation and adjunctive technologies

Lesion preparation is a fundamental component of contemporary PCI. Simple lesions require only balloon pre-dilatation; however, calcified, fibrotic or eccentric plaques often necessitate advanced plaque modification strategies. Intravascular imaging is important in understanding the properties of the plaque, allowing appropriate preparation to be undertaken.

Cutting and scoring balloons provide controlled plaque incisions, reducing vessel recoil and facilitating optimal stent

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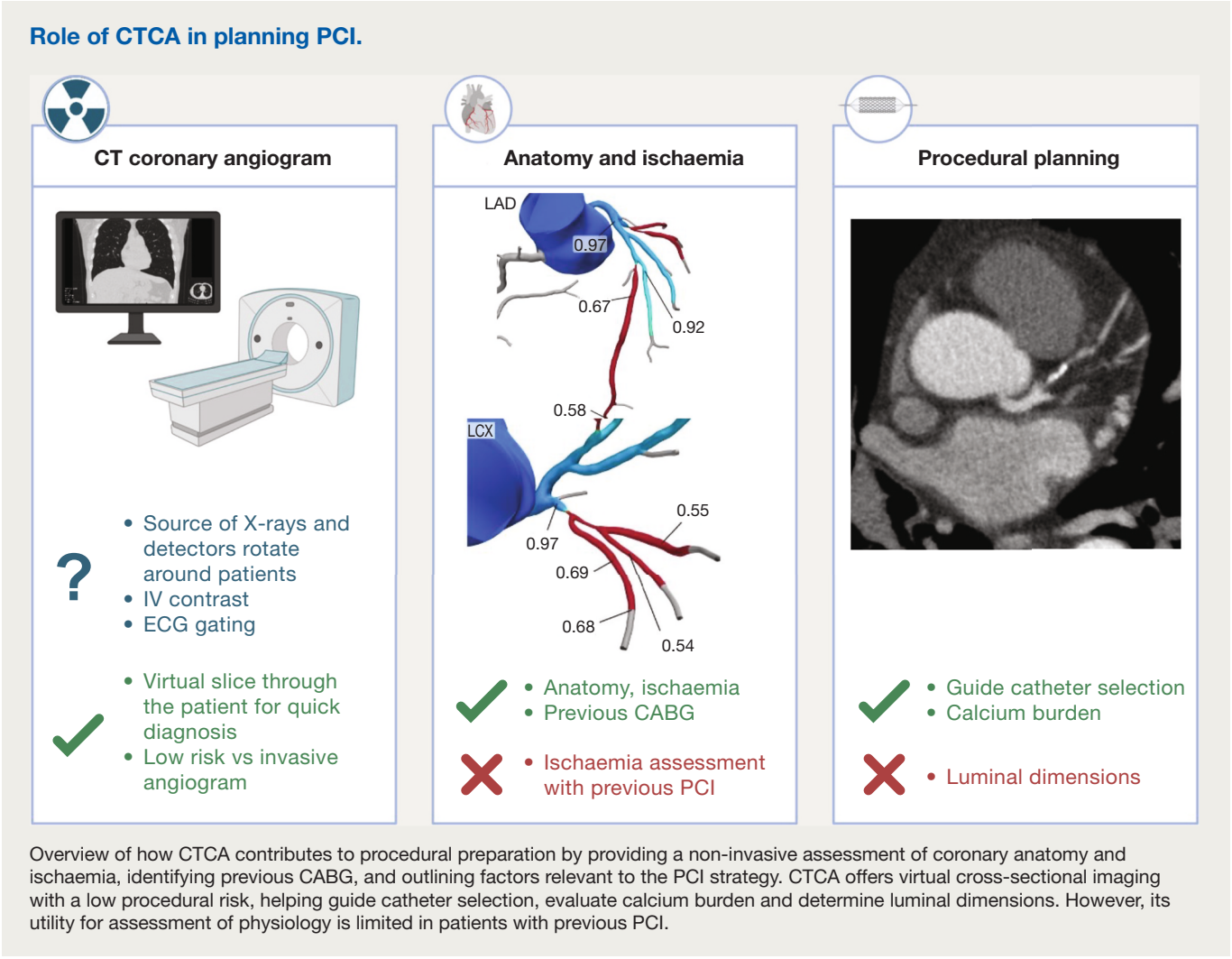

Figure 2

expansion. Proper lesion preparation is particularly important in left main stem disease, diffuse atherosclerosis where stent underexpansion predisposes to re-stenosis or stent thrombosis. Heavily calcified vessels may require further preparation with intravascular lithotripsy (IVL) and rotational or orbital atherectomy.

Intravascular imaging and physiological assessment

The integration of intravascular imaging — principally intravascular ultrasonography (IVUS) and optical coherence tomography (OCT) — has transformed PCI practice. Imaging allows a detailed assessment of plaque composition, vessel dimensions and landing zones, enabling precise stent sizing. Post-deployment imaging is equally important as it detects malapposition, underexpansion, edge dissections and thrombus. Multiple studies have demonstrated improved outcomes when PCI is guided by IVUS or OCT, particularly in complex anatomy such as left main bifurcations.

Physiological tools, such as fractional flow reserve and non-hyperaemic pressure ratios (e.g. instantaneous wave-free ratio), support appropriate lesion selection. They help avoid unnecessary stenting, reduce procedural time and improve long-term outcomes by ensuring functional and not merely angiographic lesion significance.

Stent deployment and optimization

DESs are deployed across the target lesion after careful sizing. High-pressure balloon inflation ensures adequate stent expansion, while post-dilatation with non-compliant balloons is often necessary to optimize the stent geometry. Bifurcation lesions may require advanced techniques using >1 stent.

PCI in heavily calcified vessels

Coronary calcification poses a significant challenge in PCI, impairing stent deliverability, restricting optimal stent expansion and increasing the risk of complications such as perforation and stent thrombosis. Heavily calcified lesions are becoming increasingly common because of the ageing population, higher prevalence of diabetes mellitus and metabolic syndrome along with increased statin use. Several specialized techniques are available, each suited to different patterns and severities of calcification.

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Balloon-based strategies remain the initial approach for many lesions. Non-compliant balloons can modify superficial calcium when inflated at high pressures; however, their efficacy is limited in deep or circumferential calcification. Cutting and scoring balloons provide more controlled plaque modification by using microblades or scoring elements to create focal incisions, facilitating subsequent balloon expansion and stent deployment. These devices are particularly useful in eccentric or moderately calcified lesions but their effectiveness diminishes in the presence of substantial or concentric calcium.

Atherectomy devices offer more robust calcium debulking. Rotational atherectomy uses a diamond-coated burr rotating at high speed to ablate superficial calcium and facilitate device passage. It is especially beneficial for lesions that are uncrossable or undilatable by a balloon. Orbital atherectomy employs an eccentrically mounted crown that sands calcium as it orbits, allowing gradual lumen enlargement. Both techniques carry risks, including slow flow, perforation and burr entrapment, necessitating careful patient selection and operator expertise.

IVL has emerged as a transformative technology for the management of deep or circumferential calcification. IVL delivers localized ultrasonic pressure waves that fracture both intimal and medial calcium while minimizing damage to the surrounding soft tissue. It is deployed using a low-pressure balloon, reducing the risk of vessel perforation.

IVL has shown high procedural success with favourable safety profiles in a range of complex lesions, including left main interventions and bifurcations. Its predictable calcium-modifying effect makes it an increasingly preferred option for diffuse or heavily calcified arteries.[5] Figure 3 summarizes the most commonly used calcium modification techniques.

Commonly occurring complications and their management

PCI is a generally safe procedure. The overall risk of major adverse cardiac events is <1% in stable patients, although the risk is marginally higher in individuals with NSTEMI and STEMI. Understanding the mechanisms underlying these complications is essential for prompt recognition and effective management.

Coronary dissection and perforation

Coronary dissection results from disruption of the intimal layer, often from aggressive guidewire manipulation, balloon inflation or stent deployment, particularly in calcified or tortuous vessels. Dissection can impair distal flow and precipitate myocardial ischaemia. Management depends on severity: minor dissections often seal spontaneously after stenting, whereas extensive dissections require immediate stent implantation to restore luminal integrity.

Coronary perforation, although less common, arises from high-pressure inflations, oversized balloons or the use of atherectomy devices. It can lead to pericardial tamponade, necessitating urgent pericardiocentesis, prolonged balloon inflation, implantation of covered stents or, rarely, emergency CABG.

No-reflow phenomenon

The no-reflow phenomenon is characterized by impaired microvascular perfusion despite successful epicardial reperfusion. It is most frequently observed in PCI for STEMI or NSTEMI, where distal embolization of thrombus, endothelial injury and microvascular spasm contribute to impaired flow.

Management includes intra-coronary vasodilators such as adenosine or verapamil, alongside aspiration thrombectomy in select cases. Adequate anticoagulation and minimization of device-induced trauma are key preventive strategies.

Acute or subacute stent thrombosis

Stent thrombosis is a rare but potentially fatal complication, typically presenting as acute myocardial infarction. It is often related to stents under expansion, malapposition, procedural complications such as edge dissection or inadequate antiplatelet therapy.

Prevention relies on appropriate lesion preparation, intravascular imaging to optimize stent deployment, and ensuring correct antiplatelet loading. Management includes urgent repeat PCI, aspiration of thrombus, high-pressure balloon dilatation and escalation of antithrombotic therapy.

Access-site complications

Vascular complications, including haematoma, pseudoaneurysm, arteriovenous fistula or retroperitoneal haemorrhage, remain important considerations, particularly with femoral access. They arise from inadvertent high or low arterial puncture, inadequate haemostasis or anticoagulation.

Management ranges from manual compression and ultrasound-guided thrombin injection for pseudoaneurysm, to blood transfusion or surgical repair in severe bleeding. Early recognition through vigilance for hypotension, flank pain or falling haemoglobin concentration is essential.

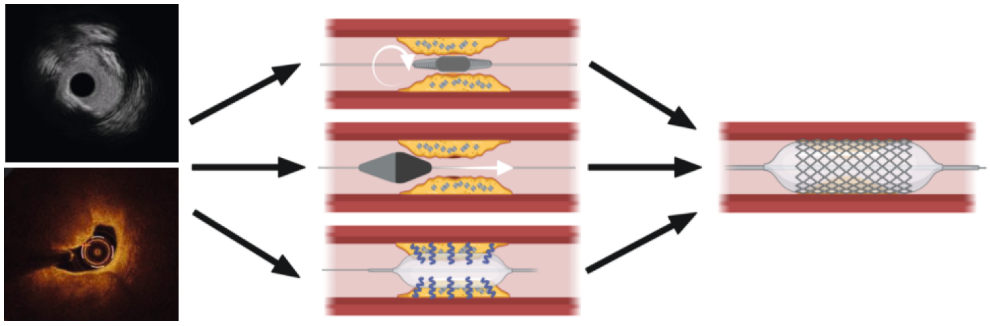

Figure 3 Calcium modification workflow guided by intravascular imaging. Schematic representation showing how IVUS and OCT imaging inform the selection of calcium modification strategies – rotational atherectomy, orbital atherectomy or IVL. Each modality provides lesion-specific insights that guide the choice of plaque-modifying technique before coronary stent implantation.

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Contrast-associated acute kidney injury

This occurs from renal vasoconstriction, oxidative stress or direct tubular toxicity. The risk is highest in patients with pre-existing renal impairment, diabetes or haemodynamic instability. Preventive strategies include pre-procedure hydration, minimizing contrast volume, using iso-osmolar agents and avoiding nephrotoxic medications.

Management is largely supportive, with careful monitoring of renal function and fluid balance; dialysis can be required in severe cases.

Arrhythmias

PCI can provoke arrhythmias through ischaemia, reperfusion injury or mechanical irritation of the coronary arteries. Ventricular tachycardia can occur during balloon inflation or after reperfusion in STEMI. Bradyarrhythmias are common in right coronary interventions for atrioventricular nodal ischaemia.

Management includes correction of haemodynamic instability, administration of anti-arrhythmic drugs, temporary pacing when required and defibrillation for life-threatening rhythms. ◆

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TEST YOURSELF

To test your knowledge based on the article you have just read, please complete the questions below. The answers can be found at the end of the issue or online here.

Question 1

A 62-year-old man presented with exertional chest pain classic for angina pectoris despite treatment with bisoprolol and amlodipine. CT coronary angiography has shown a 70% stenosis over an 8 mm length of the mid-left anterior descending artery (LAD), CT fractional flow reserve is 0.67. There is no coronary artery calcification. The remaining coronary arteries have mild atheromatous plaque with no significant stenosis or reduction in CT fractional flow reserve (CT FFR).

According to current guidance, which of the above factors best justifies percutaneous coronary intervention in this patient?

A. Continuing angina despite the use of two antianginal medications with demonstration of ischaemia.
B. 70% stenosis of the mid-LAD
C. Absence of coronary artery calcification
D. Expectation of improved mortality
E. CT FFR of 0.67 in the mid-LAD

Question 2

A 72-year-old female presented with acute coronary syndrome. Invasive angiography demonstrates severe mid-right coronary artery disease as the culprit for the presentation.

Investigation

- Intravascular ultrasound imaging (IVUS) of the lesion demonstrates circumferential deep calcification at the point of tightest stenosis.

Which calcium modification strategy is most effective for treating this presentation.

A. High-pressure non-compliant balloon
B. Cutting balloon
C. Rotational atherectomy
D. Intravascular lithotripsy
E. Scoring balloon

Question 3

A 57-year-old woman underwent percutaneous coronary intervention for a non-ST segment elevation myocardial infarction. Despite successful stent deployment there was thrombolysis in myocardial infarction flow 1–2 (TIMI 1–2) in the epicardial vessel, and the patient developed hypotension and ECG changes consistent with impaired microvascular perfusion.

What is the most likely diagnosis?

A. Acute stent thrombosis
B. Coronary perforation
C. No-reflow phenomenon
D. Retroperitoneal haemorrhage
E. Contrast-associated acute kidney injury

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