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CONSENSUS STATEMENT

Global Consensus Recommendations on Improving the Safety of Chronic Total Occlusion Interventions

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Safety is of critical importance to chronic total occlusion (CTO) percutaneous coronary intervention (PCI). This global consensus statement provides guidance on how to optimise the safety of CTO) PCI, addressing the following 12 areas: 1. Set-up for safe CTO PCI; 2. Guide catheter–associated vessel injuries; 3. Hydraulic dissection, extraplaque haematoma expansion, and aortic dissections; 4. Haemodynamic collapse during CTO PCI; 5. Side branch occlusion; 6. Perforations; 7. Equipment entrapment; 8. Vascular access considerations; 9. Contrast-induced acute kidney injury; 10. Radiation injury; 11 When to stop; and, 12. Proctorship. This statement complements the global CTO crossing algorithm; by advising how to prevent and deal with complications, this statement aims to facilitate clinical practice, research, and education relating to CTO PCI.

Keywords Chronic total occlusion • Percutaneous coronary intervention • Consensus document • Global • Safety

Introduction

Chronic total occlusion (CTO) percutaneous coronary intervention (PCI) carries an increased risk of complications compared with non-CTO PCI [1–7]. Complications of CTO PCI can be classified into cardiac (coronary and non-coronary) and non-cardiac (Figure 1). Although a strict delineation between complications in CTO PCI vs non–CTO PCI is artificial, there is an increasing risk of complications from "bread and butter" PCI to complex high-risk indicated percutaneous coronary intervention (CHIPS) PCI to CTO PCI, making this paper relevant to all PCI. Although the indications for CTO PCI is beyond the scope of this paper, a balance—between how "indicated" a CTO PCI is, and the "risks" of complications for a CTO PCI—must be made, soberly and thoughtfully. Through global collaboration, consensus was recently reached on the definitions and terminology [8], guiding principles [9], and crossing algorithm for CTO PCI [10]. In the present manuscript, 147 co-authors from 139 centres in 52 countries developed 12 sets of recommendations for the prevention, early recognition, and treatment of CTO PCI complications.

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1. Set-Up for Safe CTO PCI

Cardiac catheterisation laboratories performing CTO PCI should have immediate access to echocardiography, covered stents, coils (ideally compatible with 0.014-inch microcatheters), an Activated Clotting Time (ACT) measurement device, and a pericardiocentesis kit. A trained team of operators, assistants, nurses, technicians, and radiographers with familiarity with CTO equipment, radiation dose minimisation, pericardiocentesis, and cardiopulmonary resuscitation is recommended. Mechanical circulatory support devices such as an intra-aortic balloon pump (IABP), Impella (Abiomed, Danvers, MA, USA) [11], and extracorporeal membrane oxygenation (ECMO) may be useful in selected patients. Modern X-ray machines provide lower radiation dose and should be preferentially used for CTO PCI, if available.

2. Guide Catheter–Associated Vessel Injury

A. Air and Thrombus Embolism

Although air and thrombus embolism can occur during any PCI, the risk is higher with CTO PCI due to routine use of the trapping technique, simultaneous use of multiple devices through the guide catheter, use of guide extension, and long procedure duration. Air embolism in the donor vessel during CTO PCI can be catastrophic, as it can lead to profound ischaemia and haemodynamic collapse. Dampening of the guide catheter pressure tracing may indicate presence of thrombus or air. Air embolism can be prevented by back bleeding and aspirating the guide catheter after every trapping manoeuvre. Prolonged blood stasis inside a guide catheter can lead to thrombus formation, despite adequate anticoagulation. The activated clotting time (ACT) should be checked 5–10 minutes after heparin administration, and then every 30 min, and maintained >300 seconds during antegrade and >350 seconds for retrograde CTO PCI [12]. Moreover, the guide catheters, especially the retrograde guide catheter, should be aspirated and flushed periodically throughout the case, especially if no injection is performed for prolonged periods of time. The retrograde guide catheter should be removed as soon as it is no longer needed.

Thrombosis of the donor vessel can be due to embolisation of catheter thrombus or thrombosis of the vessel itself. Retrograde CTO PCI is more prone to donor vessel thrombosis given the often- prolonged periods of microcatheter or anchoring balloon dwelling in the vessel. Periodic ACT monitoring and having a low threshold to treat donor vessel lesions before retrograde attempts may reduce the risk of donor vessel thrombosis [13].

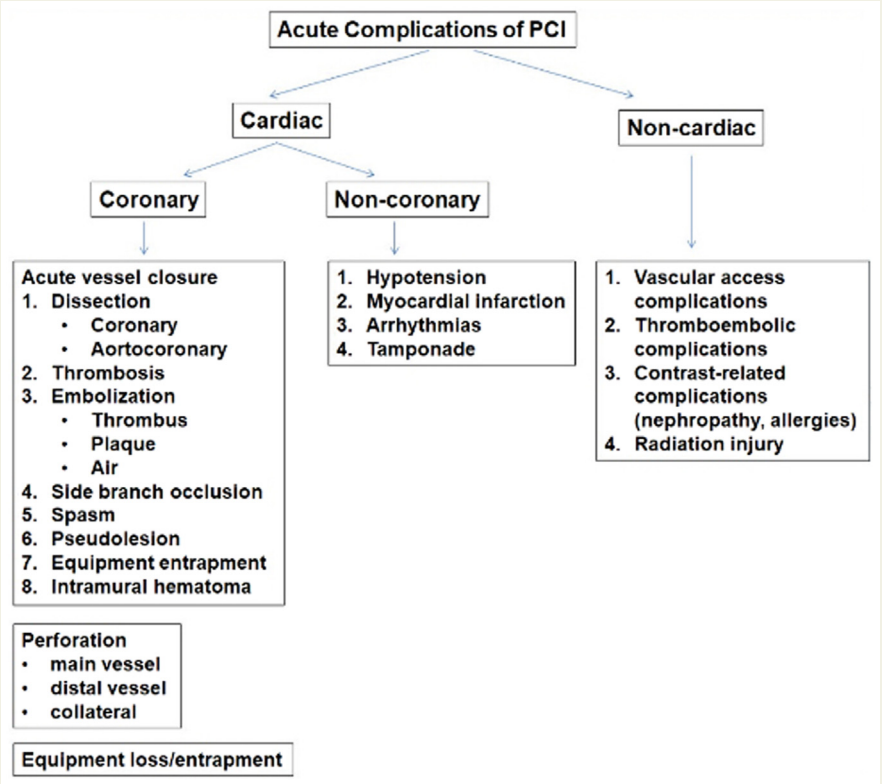

Figure 1 Classification of CTO PCI complications into cardiac (coronary and non-coronary) and non-cardiac.

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B. Guide-Induced Dissection

CTO PCI is particularly prone to guide-induced dissections as: i) aggressive guide catheters are often used to maximise support; ii) after CTO crossing, the loss of backward force may lead to deep engagement of the antegrade guide catheter increasing the risk of guide-induced dissection; and, iii) in retrograde CTO PCI, the donor guide catheter can be "sucked in" when the retrograde microcatheter or guidewire is pulled back, potentially leading to donor vessel injury. The donor guide catheter should be pulled back into the ascending aorta after wire externalisation, especially when backing out retrograde devices [13].

Prevention

Avoid oversized guide catheter and aggressive guide catheter manipulations. Pressure dampening can be avoided by slight withdrawal of the guide catheter, by placing a coronary wire in the aortic cusp that prevents deep guide catheter intubation, and in some cases by prophylactic stenting of diseased vessel ostia. Side hole catheters can reduce injection pressure and barotrauma while allowing some perfusion through the side holes, but it increases contrast use and caution must be taken against "false reassurance" of lack of pressure dampening. Inserting a coronary wire as soon as the guide catheter is engaged, before injections, can protect from coronary dissection but may also cause dissection with aggressive wire advancement. Gentle contrast injection, and the avoidance of automated injectors are recommended.

C. Donor Vessel Injury and Treatment

The donor vessel that provides collaterals to the CTO is, in most cases, engaged with a second guide catheter. The donor vessel can be injured during angiography or when instrumented during the retrograde approach. Donor vessel injury can be catastrophic since the donor artery supplies both its own territory and collateralises the CTO. The "ping-pong" (dual-guide catheter) technique [14] is recommended when using an ipsilateral collateral for retrograde CTO PCI. Single-guide catheter approach for ipsilateral retrograde increases the risks of guide-induced dissection during retrograde retrieval, may lead to cardiac strangulation by tension on the retrograde system, and hypotension, and carries risk of equipment entrapment.

In case of donor vessel injury, the haemodynamic status of the patient might quickly deteriorate, often necessitating inotropes, vasopressors, and mechanical circulatory support. Prompt treatment of the donor vessel should, hence, be the highest priority. Prophylactic insertion of a "safety" wire in the donor vessel prior to PCI can greatly facilitate treatment. In case of donor vessel dissection, the retrograde equipment should be removed (if possible) before stenting the donor vessel. CTO treatment attempts should, in most cases, stop unless the procedure is expected to be completed in a short period of time. With donor vessel thrombosis, aspiration thrombectomy and administration of a glycoprotein IIb/IIIa inhibitor might be needed. The ACTs should be monitored to ensure that adequate anticoagulation is achieved.

3. Hydraulic Dissection, Extraplaque Haematoma Expansion, and Aortic Dissection

A. Mechanism

Hydraulic dissection, extraplaque haematoma, and aortic dissection, represent a family of complications of CTO PCI sharing the following common mechanisms:

i). Creation of a connection between the true lumen and the extraplaque (formerly called "subintimal") space—this can be caused by guide-induced dissection (see section 2, ***Guide Catheter–Associated Vessel Injury***, above) [15], by aggressive wiring when the wire has entered the extraplaque space, or by ballooning during the reverse Controlled Antegrade Retrograde Tracking (CART) (Figure 2A).

ii). Hydraulic barotrauma. Pressure from contrast injection or systolic pressure can cause flow from the true lumen into the extraplaque space that can expand the extraplaque haematoma (Figure 2C). Wire advancement may also expand the extraplaque haematoma (Figure 2D).

iii). Exposure of the extraplaque space to arterial pressure, and constant inflow of blood may lead to distal true lumen collapse, aortic root haematoma, aortic dissection [16] and tamponade (Figure 2B).

B. Hydraulic Dissection

Guide-induced dissections are only noticed after contrast injection leading to distal or aortic root haematomas (Figure 2B). When a wire advances from the proximal true lumen into the extraplaque space, a potential pathway for hydraulic dissection is created. Inadvertent or deliberate (balloon-assisted microdissection, BAM) balloon rupture [17] can also lead to hydraulic dissections. Inflation of a balloon when performing reverse CART can also create a connection intraplaque and extraplaque space, possibly leading to hydraulic dissection.

Prevention

The risk of hydraulic dissection may be reduced by gentle injections and avoiding injection when the pressure waveform is dampened. Contrast injections should stop if a hydraulic dissection occurs, to minimise the risk of enlarging the dissection and intravascular ultrasound (IVUS) should be used for guiding PCI. Disconnecting the contrast syringe from the antegrade manifold can help prevent inadvertent contrast injection after proximal cap penetration.

C. Haematoma Expansion

Haematoma expansion is often the result of hydraulic dissection but may also be caused by wire manipulation (Figure 2D). Immediate cessation of contrast injection and inflow control are essential to prevent haematoma expansion. If the entrance site of the dissection can be identified,

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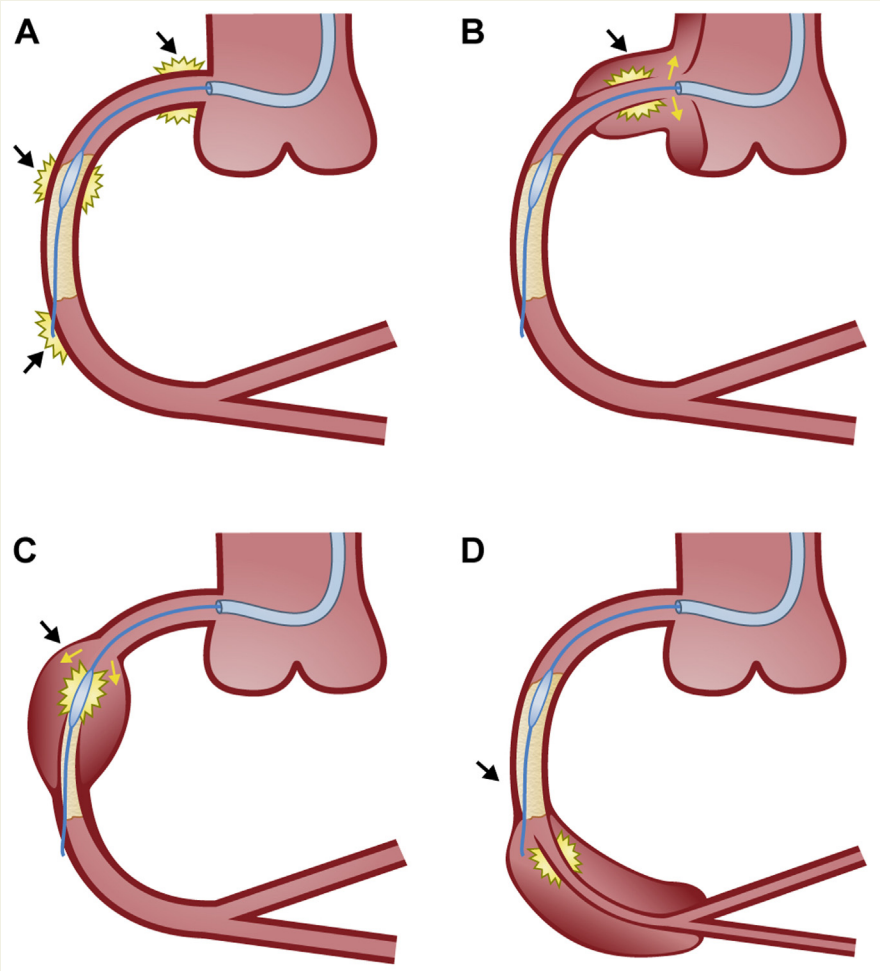

Figure 2 Mechanism of hydraulic dissection, haematoma expansion, and aortic dissection. (A) Mechanism of creation of a connection between the true lumen and the extraplaque space: guide-induced dissection (arrow); ballooning during reverse CART (arrow); CTO wiring in the extraplaque space (arrow). (B) Hydraulic dissection via guide-induced dissection causing haematoma expansion into aortic cusp (yellow arrow) and ascending aortic dissection (yellow arrow). (C) Haematoma expansion around entry site from reverse CART or CTO proximal cap ballooning. (D) Extensive hydraulic dissection and haematoma expansion into distal RCA (yellow arrow) leading to distal vessel narrowing.
Abbreviations: CART, controlled antegrade retrograde tracking; CTO, chronic total occlusion; RCA, right coronary artery.

stenting over it can prevent haematoma expansion. Alternatively, an adequately-sized balloon can be inflated to occlude flow to the vessel, or a guide extension catheter can be advanced into the proximal cap of the CTO to limit blood inflow and prevent haematoma expansion.

Haematoma expansion can cause distal true lumen collapse. IVUS can provide important information about the size and extent of the haematoma. The Subintimal Transcatheter Withdrawal (STRAW) technique can help reduce the size of haematoma via aspiration through a microcatheter [18] or through the Stingray balloon. This is done by delivering the microcatheter tip to the extraplaque space and applying suction with an indeflator or syringe. Treatment options for distal true lumen collapse due to haematoma after successful CTO crossing include: cutting balloon to release the hematoma [19]; low pressure balloon inflation at the distal vessel; or, long stents to cover beyond the distal edge of the haematoma and expand the vessel.

Extraplaque haematomas can also track backwards towards the ostium and into the aortic root leading to an aortocoronary haematoma that can expand towards the ascending aorta, causing a type A aortic dissection (Figure 2B) [20], or towards the pericardium, potentially leading to acute aortic regurgitation and tamponade [21]. Treatment is with stenting, with the stent protruding 1–2 mm into the aorta [22]. A large enough stent should be used to prevent further blood entry into the aortic root. IVUS is often helpful for stent size selection. If there is persistent contrast inflow after stenting, a covered stent can be used. Serial non-invasive imaging (with computed tomography or

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transoesophageal echocardiography) could confirm that the dissection is not progressing and should be considered. Emergency surgery is rarely needed [23].

4. Haemodynamic Collapse During CTO PCI

Haemodynamic collapse during CTO PCI may occur for several reasons [13], as outlined below.

A. Access Site Bleeding /Retroperitoneal Haematoma

Femoral access should be obtained using ultrasound- and fluoroscopy-guided techniques. Vascular access complications can cause hypotension during or after CTO PCI. Use of ultrasound can decrease the risk of vascular access complications, primarily driven by a reduction in local haematomas, especially in complex CTO-PCI [24]. A high arterial puncture location increases the risk of retroperitoneal bleed [25]. Non–access site-related bleeding complications have a similar prevalence to access site complications, but are associated with a significantly worse prognosis, partly related to the severity of the bleed [25].

B. Aortic Insufficiency

Patients can have a fall in systolic pressure, tachycardia, and a low pulse pressure due to acute aortic insufficiency that is often induced by an Amplatz left (AL-1) guide catheter [26], but can also occur with extra-back up (XB/EBU) type guide catheters. Correcting the guide catheter position (withdrawing it, until it is not pushing against the valve) promptly resolves aortic insufficiency and normalises the blood pressure.

C. Ischaemia

Ischaemia during CTO PCI can lead to hypotension, especially during retrograde CTO PCI. Occlusion of a large dominant collateral might create severe ischaemia in target vessel territory. In patients with low ejection fraction, preferential use of the antegrade approach is recommended as the retrograde approach carries increased risk of haemodynamic compromise. Ventricular support device use, limiting contrast volume and limiting ischaemic time are important in this patient cohort.

D. Donor Vessel Lesions

Donor vessel lesions, even if moderate, can lead to ischaemia and haemodynamic compromise, if crossed during retrograde CTO PCI. Diseased donor vessels should be treated prior to proceeding with retrograde CTO PCI.

E. Donor Vessel Ischaemia

Donor vessel spasm and the accordioning effect could also lead to compromised flow. Intracoronary administration of vasodilators, such as nitroglycerin, often helps resolve the spasm. Meticulous care is needed when utilising the left internal mammary artery for retrograde access; use of highly tortuous internal mammary artery grafts should be avoided.

F. Perforations

Perforations might also lead to tamponade and hypotension.

G. Anaphylaxis

Allergic reaction to contrast or other substances can cause hypotension and should be considered in the differential diagnosis.

5. Side Branch Occlusion

A. Mechanism

Occlusion of side branches that originate within the CTO body is usually inconsequential as they usually fill by collaterals. In contrast, occlusion of a side branch at the distal or proximal cap can lead to periprocedural myocardial infarction (MI) (Figure 3).

Side branches can be occluded by dissection, plaque shift, or subintimal shift, when the wire has gone subintimal beyond the bifurcation at the proximal or distal cap [27]. For

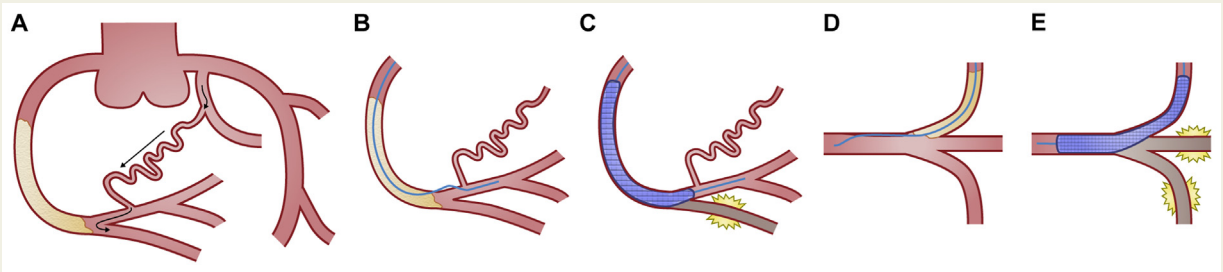

Figure 3 Side branch short cut. (A) The blood supply to the posterior descending artery (PDA) is from the atrioventricular branch (AV) (black arrows). (B) CTO wire is advanced through the extraplaque space in the posterolateral vessel (PLV) distal to the PDA origin. (C) After stenting the PDA ostium is occluded (black arrows). (D) Ostial LAD CTO with retrograde wire travelling extraplaque in the left main and entering the aorta at the left main ostium. (E) Stenting causes occlusion of the ramus intermedius and the circumflex.
Abbreviations: CTO, chronic total occlusions; LAD, left anterior descending.

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example, in a RCA CTO with the distal cap at the posterior left ventricular (PLV) posterior descending artery (PDA) bifurcation, the blood supply to the PDA can be from the circumflex atrioventricular (AV) groove that perfuses the PLV and then, via the bifurcation, the PDA (Figure 3A). If an antegrade CTO wire enters the true lumen in the PLV and not at the bifurcation, the PDA may occlude after stenting into the PLV (Figure 3B). The PDA will lose the original collateral supply from the AV groove resulting in periprocedural MI (Figure 3C). In a similar manner, in an ostial left anterior descending (LAD) CTO, if one attempts retrograde wire crossing, there is a risk of the retrograde wire travelling extraplaque into the left main and entering the aorta at the left main ostium (Figure 3D and E). Stenting over this wire may lead to circumflex occlusion and MI. This same principle can be applied to any significant size side branch near either cap of the CTO.

B. Prevention

Recanalising a side branch that occluded after stenting can be very challenging, therefore prevention is critical.

Bifurcation at the proximal cap

Major side branches at the proximal cap should be wired prior to CTO crossing attempts. Sometimes placement of an IVUS in a proximal side branch can facilitate antegrade crossing. In cases of retrograde crossing, the extended reverse CART technique (i.e., re-entry proximal to the proximal cap) should be avoided [28]. Instead, antegrade wiring into the CTO body beyond the proximal side branch should be done first, to allow reverse CART within the occluded segment. Using guide extension reverse CART beyond the proximal side branch can minimise the risk of side branch occlusion.

Bifurcation at the distal cap

The global CTO crossing algorithm [10] recommends the use of retrograde approach, if there is a bifurcation at the distal cap. It also recommends use of IVUS before stenting CTOs with bifurcation at the distal cap. Major side branches should be wired (often using a dual lumen microcatheter) before stenting, to reduce the risk of occlusion.

C. Treatment

If despite preventive measures, the side branch becomes occluded, a dual lumen microcatheter is usually used for rewiring (the sidecar technique can be an alternative, if dual lumen not available). IVUS can help identify the origin of the occluded side branch: plaque shifts that cause occlusion can often be stented and wired with a low penetration force wire, followed by kissing balloon inflation, while an extraplaque subintimal shift usually requires a high penetration force wire and a two-stent strategy [29]. If this fails, retrograde crossing or antegrade dissection and re-entry [30] can be used, followed by 2-stent bifurcation PCI. As a last resort, the subintimal tracking and re-entry (STAR) technique [31], with ballooning into the side branch (investment procedure) [10] can restore flow into the side branch, minimise the risk of periprocedural MI, and facilitate a staged procedure.

6. Perforation

CTO PCI carries increased risk of perforation especially in older patients and women, in very calcified vessels, and long occlusions, and with use of rotational atherectomy, antegrade dissection/re-entry and the retrograde approach [32]. Clinically relevant perforations associated with major adverse effects are larger in size, more proximal or at collateral location, and have a high-risk shape ('cloud-like' or 'floating') [33]. Perforations are classified by 2 descriptors: the first descriptor denotes the location of the perforation (large vessel, distal branch, septal collateral and epicardial collateral); and, the second descriptor denotes its severity. Both descriptors have important implications regarding management [8].

A. Large Vessel Perforation

Beware of perforations after stenting in the extraplaque space after knuckle wiring, especially in heavily calcified lesions. In long extraplaque space stenting with large haematoma, the perforation may continue despite covered stent implantation, unless the covered stent covers the entrance to the subintimal space all the way to the distal true lumen.

Large vessel perforations during CTO PCI can occur during CTO crossing attempts or during lesion preparation and stenting. Wire exits from the vessel architecture during CTO crossing are typically benign (except in epicardial collaterals), as long as the wires are not followed by devices. Therefore, microcatheter and balloon advancement should be avoided until the operator is certain that the wire is within the vessel architecture. Knuckle wire advancement is preferred if the course of the vessel is unclear, as it is less likely to cause a perforation compared with stiff tip wires [34]. Care should be taken during knuckle wiring to ensure that the knuckle does not enter a side branch and cause perforation. Dilation of the vessel during reverse CART needs to be done after assessment of the vessel size by fluoroscopy (using the size of the knuckled wire) or by IVUS (preferred).

The first step in every perforation is to inflate a balloon to stop bleeding into the pericardium. Large vessel perforations are usually treated with covered stents [35,36] that could be delivered through the same or through a second guide catheter ("ping-pong" technique [37]) as the blocking balloon [28,38,39]. Another way to seal a large vessel perforation is to create a dissection flap at the site of perforation and stent over the flap [39].

B. Distal Vessel Perforation

Distal vessel perforations are usually caused by too distal advancement of a coronary wire or another device, such as the CrossBoss catheter (Boston Scientific, Natick, MA, USA). Polymer-jacketed and stiff wires are more likely to cause

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perforation, hence they should be exchanged for a workhorse wire as soon as possible after CTO crossing.

Distal vessel perforations may lead to slow bleeding and tamponade even several hours after the procedure [32]. Treatment is usually with coil embolisation, using the block and deliver technique; another technique is to deploy a covered stent covering the origin of the perforated branch. Other embolisation materials (such as fat, suture, glue, particles, thrombin, clotted blood, etc.) can be used depending on local availability and operator familiarity and preference.

C. Collateral Perforations

Septal collateral perforations usually do not result in adverse consequences, although septal haematomas may cause outflow obstruction leading to dry tamponade, and ventricular arrhythmias [40,41]. If a large septal haematoma develops, fenestration into the right ventricle (RV) could be performed by directing a high penetration force wire via the haematoma into the RV and passing a microcatheter or balloon [42]. Epicardial collateral perforations can lead to rapid tamponade or loculated collection of fluid especially in post-coronary artery bypass grafting patients, that might lead to cardiac chamber compression [33,43].

Some CTOs have intra-coronary antegrade bridging collaterals. Inadvertent crossing through these channels may cause rupture, especially during microcatheter or balloon crossing [44].

D. Collateral Wire Injury

During retrograde CTO PCI, when force is applied to a bare wire across a retrograde channel or bypass graft, the wire may cut into the channel causing perforation [45]. Therefore, during retrograde CTO PCI, the entire retrograde channel should always be protected by a microcatheter.

E. Persistent Staining

Despite the concerning visual appearance, persistent contrast stain usually suggests bleeding in a contained space. Contrast that disappears is usually into a cardiac chamber, or pericardium.

F. Intact Pericardium

Presence of intact versus non-intact pericardium is a crucial determinant of the consequences of a perforation. Perforations in prior CABG patients are more likely to cause loculated effusions leading to chamber compression that may require surgery or computed-tomography guided drainage.

G. Prevention and Management

Coronary wire manipulation through collaterals should be performed with caution and the position of the wire should always be confirmed prior to microcatheter advancement. 'Back bleeding' through the microcatheter should be confirmed prior to contrast injection. If there is no back bleeding, the microcatheter should be withdrawn. Epicardial collaterals should not be crossed using the surfing technique.

Suction through the microcatheter might occasionally be sufficient to seal a collateral perforation. Coiling [46] from both sides that feed the collateral might be required to seal perforations. Occasionally, additional treatment, such as injection of autologous clots, macerated fat, thrombin, or fibrin glue [47], may be required to achieve haemostasis [48].

The retrograde channel integrity should be checked before removal of the retrograde equipment [13]. The steps for safely removing the retrograde gear must include:

i) Protection of the retrograde channel with microcatheter during pulling back of retrograde wire to avoid a cheese-cutting effect;
ii) Backing out of retrograde guide catheter during retrograde gear pull back to avoid guide catheter sucking in and retrograde vessel injury; and,
iii) Maintaining access to both sides of the retrograde channel until contrast angiography can demonstrate its integrity. Guide catheter injections to check channel integrity are preferable to selective injections, especially in septal channels, as selective injections may cause septal haematoma enlargement. If there is collateral channel perforation but the CTO PCI is unsuccessful, multiple collaterals may have to be coiled, as coiling from CTO side is not possible. Bilateral angiography should always be done to ensure there is no further contrast extravasation.

If there is uncertainty about active pericardial bleeding, echocardiographic contrast administration can help confirm or exclude active bleeding into the pericardial space [49]. Pericardiocentesis should be performed in case of tamponade.

Echocardiography in the recovery area can detect pericardial effusion earlier. Protamine should only be administered after all equipment has been removed from the patient, and the pericardial effusion has been drained, as protamine may cause clotting of the pericardial blood. If surgery may be needed, protamine should not be used [50].

7. Equipment Entrapment

Equipment delivery can be challenging in CTO PCI, given high prevalence of extensive disease, calcification, and tortuosity, and may lead to stent loss and equipment entrapment, such as wire fracture, microcatheter damage and entrapment, and atherectomy burr entrapment [51–53].

Careful lesion preparation minimises the risk of stent loss during delivery attempts. In many cases, stent deployment or crushing is preferable to stent retrieval.

Knuckled coronary wires should be advanced without rotation to reduce the risk of wire fracture and entrapment. Fractured wire tips may be best left in place, but wire unravelling [52] should be ruled out, as it can lead to coronary or aortic thrombosis.

Microcatheter over-torquing can lead to the tip fracture or wire entrapment inside the microcatheter [53]. In case of

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difficulty advancing a microcatheter or rotating the wire, it is often better to change the microcatheter.

The risk of equipment entrapment is higher during retrograde CTO PCI. Short coronary wires and stiff tip wires should never be snared during retrograde procedures; snaring of dedicated externalisation wires is preferred. If an externalization wire cannot be passed, snaring of a polymer-jacketed wire on the radiopaque part (e.g., Sion Black, Asahi Intecc) with a releasable snare (e.g., sumi2G snare) is recommended [54,55]. The tips of the antegrade and retrograde equipment should not get in contact over the externalised wire.

Equipment entrapment can be treated percutaneously in most cases; emergency surgery is rarely required. Advancing another wire and performing balloon angioplasty or excimer laser [53] around the entrapped equipment can often facilitate retrieval, as can use of guide catheter extensions. The hub of the trapped equipment has to be cut before inserting a guide extension over it, and often the outer sheath of the device (e.g., rotablator, IVUS, or optimal coherence tomography [OCT] catheter) has to be removed after cutting, in order to deliver a balloon alongside it in the guide catheter. If equipment fragments are left in the coronary circulation, it is best to stent over them to prevent migration, unless they are located distally.

8. Vascular Access Considerations

In most cases, CTO PCI requires two (and sometimes three) arterial access points, usually of large calibre (7 or 8 French), increasing the risk of vascular access complications. The risk could be reduced by the following measures.

i). Use of radial access
Radial access – both proximal and distal [56,57]—is associated with lower risk of bleeding and has been increasingly used for one (for example, femoral–radial) or both (bi-radial) guide catheters during CTO PCI [58–63]. Complex PCI via radial access was associated with similar success compared with femoral access in the Complex Large-bore Radial Percutaneous Coronary Intervention (COLOR) randomised-controlled trial [64].

ii). Optimal technique when obtaining femoral access
Use of fluoroscopy and ultrasound for puncturing the femoral artery, micro-puncture technique [65], performing femoral angiography at the beginning of the case, and possibly using arterial closure devices may minimise the risk of femoral access complications [66]. Use of long sheaths can improve guide catheter support, especially in patients with severe iliac tortuosity and prevent sheath movement [24,67].
iii). Use of two access points. In CTO PCI, two access points are used in most cases, allowing use of the contralateral access to check for vessel integrity and for balloon tamponade if needed.

9. Contrast-Induced Acute Kidney Injury

CTO PCI often requires administration of a large volume of contrast, increasing the risk of contrast-induced acute kidney injury. The risk can be reduced as follows.

i). Reducing contrast toxicity
Contrast induced kidney injury can be reduced by hydration, statin administration, and discontinuation of nephrotoxic medications.

ii). Reducing contrast volume
The global CTO crossing algorithm recommends keeping the contrast volume to less than 3 times the estimated glomerular filtration rate or even lower if the patient has comorbidities [10,68]. Contrast volume can be reduced by: a) careful analysis of previous angiograms; b) optimal timing of the antegrade contrast injection to coincide with maximal retrograde filling; c) microcatheter tip injections both antegrade (instead of injections through the guide catheter) and retrograde (through the most dominant collateral); d) use of the retrograde approach, especially if selective collateral channel injection is used instead of guide catheter injection to visualise the collateral channel anatomy along with use of the reverse controlled antegrade and retrograde tracking (CART) for proximal true lumen entry; e) use of biplane; and, f) use of IVUS [69].

10. Radiation Injury

A. Radiation Injury in CTO PCI

Radiation injury is especially important in CTO PCI, due to longer procedural time and often high radiation dose [70]. Skin injury from radiation presents late and is often missed [71]. The air kerma radiation dose should be continuously monitored during the procedure, as it correlates well with radiation skin injury. The global CTO crossing algorithm recommends termination of the procedure "if the air kerma radiation dose is >5 Gray, unless the procedure is well advanced" [10].

B. Prevention

i). Reduce total radiation dose
Multiple strategies can reduce the radiation dose [71], such as low magnification and collimation, using ≤7.5 frame per second fluoroscopy, avoiding steep angles, using the fluoroscopy store function instead of cine angiography whenever possible, for example, for documenting the position of balloons and stents, and avoidance of panning.

Further radiation dose reduction can be achieved by using the trapping technique for device exchanges, using a marker torquer on wires (locking the torquer on the wire, when the wire tip is just inside the microcatheter tip before wire removal, and keeping it there when reusing the wire into the microcatheter, to reduce X-ray use to detect when the wire

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reaches the tip of the guide catheter) and detailed pre-procedural angiogram review, avoiding ad hoc CTO PCI. Some X-ray systems [72] stop imaging when the operator's eyes are not looking at the screen, reducing unnecessary fluoroscopy radiation.

ii) Reduce radiation concentration
Routine preprocedural examination of the patient's skin, if they have undergone a previous long procedure, can help with planning fluoroscopic views to avoid further radiation skin injury. Calculation of the cumulative dose prior to repeat CTO PCI cases can reduce the risk of radiation skin injury. Some angiography machines (Infinix^{TM}-i cardiovascular X-ray, Toshiba Corporation, Tokyo, Japan; Dose-map, General Electric, Boston, MA, USA) have built-in software to display skin dosage information, allowing adjustment of the Xray tube position to scatter radiation dose reducing skin injury risk. Frequent change of views during CTO PCI can also have a similar effect. Disposable radiation shields (such as the Radpad, Kansas City, MO, USA) can be applied to previously injured skin areas. In patients requiring repeat CTO PCI, an adequate period of recovery (at least 6 months) is advised, as dermatitis can occur late [73].

C. Patient Follow-Up

Patient who receives high radiation dose (>5 Gy) should receive patient information sheets, followed by clinical examination and photography after 30 days. This can help avoid unnecessary and potential harmful skin biopsy, that can result in non-healing wounds [74].

11. When to Stop

Four parameters should be constantly evaluated during the procedure to determine the need for stopping: radiation dose, contrast volume, procedure time, and risk of the remaining treatment options. The global CTO crossing algorithm recommends contrast, radiation, and procedural time limits for stopping a procedure [10]. In addition, lack of needed experience in ADR/retrograde, occurrence of a serious complication, and operator or patient fatigue favour stopping CTO PCI [10]. In the event of a complication such as donor vessel injury, the complication should be addressed prior to finishing the procedure. Any complication must be addressed, prior to resuming the CTO PCI. CTO PCI is an elective procedure, and "success" in a procedure should never be at the cost of safety.

Before stopping a CTO crossing attempt, determine if anything can be done to improve the chances of success during the next attempt (investment procedure) [10]. Investment can be considered using subintimal dissection and re-entry (STAR) and subintimal plaque modification (SPM) [75,76]. CTO PCI can be reattempted in 6–8 weeks, unless there is concern for radiation skin injury, in which case a longer delay to reattempt is suggested, as above.

12. Proctorship

The global CTO crossing algorithm recommends proctorship, if advanced crossing techniques such as use of the Stingray system or the retrograde approach are needed but the operator is unfamiliar with these techniques: "Proctoring may not only improve the operator CTO PCI skills but can also improve the safety of the CTO procedure, especially for highly complex lesions and patients" [10]. Therefore, proctorship is an important part of CTO PCI safety [77]. Having well established referral and request systems for proctors for complex CTO cases is part of the setup for a safe CTO program.

Conclusion

In CTO PCI, success should not be achieved at the cost of safety. A thorough understanding of potential complications, continuous monitoring of the patient for early complication detection, and prompt treatment can help prevent complications and/or minimise their adverse impact. Addressing the 12 areas outlined in this document could improve the safety of CTO PCI.

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For the Disclosures of all the CTO Global Consensus Group contributors (included those listed below), please see Appendix A.

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Appendix A

Disclosures of the CTO Global Consensus Group

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