

# Recanalization of a chronic total occlusion in ST-segment elevation myocardial infarction patients: why and when?

Current guidelines recommend that primary percutaneous coronary intervention should be limited to the infarct-related artery in patients with acute myocardial infarction and multivessel disease. However, patients with acute myocardial infarction and concomitant multivessel coronary artery disease have a worse prognosis, mainly due to the presence of a chronic total occlusion (CTO) in a nonculprit lesion. Few studies have addressed the specific issue of CTO treatment in the setting of acute myocardial infarction. We review the currently available literature regarding coronary revascularization of the nonculprit CTO in ST-segment elevation myocardial infarction patients, starting with a clinical case of an infero–postero–lateral ST-segment elevation myocardial infarction in a multivessel coronary artery disease patient with concurrent left anterior descending CTO complicated by hemodynamic instability.

KEYWORDS: chronic total occlusion = multivessel disease = primary percutaneous coronary intervention = ST-elevation myocardial infarction

Primary percutaneous coronary intervention (PCI) in patients with ST-segment elevation myocardial infarction (STEMI) aims at early and sustained restoration of anterograde flow in the infarct-related artery (IRA) [1,2]. The strategy of the mechanical reopening of an acutely occluded coronary artery allows the reperfusion of ischemic myocardium, thus limiting infarct size and reducing hemodynamic and arrhythmic complications [3]. However, coronary angiography during primary PCI often shows that the atherosclerotic process can extend over the IRA. Of note, the finding of multivessel coronary artery disease (MVD) in the setting of STEMI is associated with higher morbidity and mortality, even after reperfusion therapy [4-6]. At present, multivessel PCI in the setting of acute myocardial infarction (AMI) is discouraged since it does not appear to confer a net beneficial effect on clinical end points [7]. Conversely, it has recently been shown that in patients with AMI and concomitant MVD, the higher mortality rate is mainly due to the presence of a chronic total occlusion (CTO) in a non-IRA rather than the mere presence of MVD [8]. However, current guidelines with regard to multivessel PCI in the setting of primary PCI are based on only a few clinical evidences and do not even address the specific issue of CTO treatment. Consequently, in this particular setting in which clear evidence from randomized controlled trials is still lacking, the usual strategy is mainly based on clinical judgment.

We review the currently available literature regarding coronary revascularization of nonculprit CTO in STEMI patients starting with a clinical case of an infero-postero-lateral STEMI in a MVD with concurrent CTO in a non-IRA complicated by hemodynamic instability. In particular, we aim to assess whether a revascularization of the nonculprit CTO has to be attempted and when it should be performed.

# **Clinical case**

A 64-year-old man was admitted to our emergency department owing to acute onset of chest pain and dyspnea in the last 2 h. He had a history of hypertension and smoking habit without previous cardiovascular events. Recent laboratory tests revealed normal renal function. On admission, physical examination revealed tachypnea and cyanosis with pulmonary rales in the absence of significant heart murmurs. Blood pressure was 100/60 mmHg and a 12-lead ECG showed a sinus tachycardia at 125 beats per min, an ST-segment elevation in inferior leads and poor anterior R-wave progression from V2 to V5 (FIGURE 1). The patient was treated with aspirin and clopidogrel and immediately transferred to our catheterization laboratory to undergo primary PCI. Upon arrival in the catheterization laboratory, the patient had persistent chest pain with Killip class III requiring mechanical ventilation. A femoral access (7 Fr) was chosen to eventually switch to intra-aortic balloon pump (IABP) counter-pulsation if clinically needed during the Antonio Maria Leone<sup>+1</sup>, Simona Giubilato<sup>1</sup> & Alberto Ranieri De Caterina<sup>1</sup>

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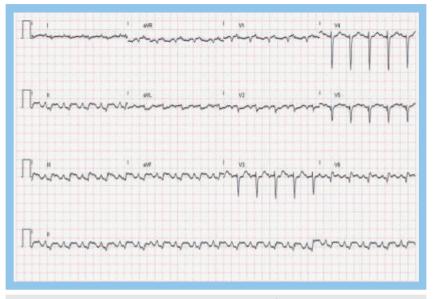
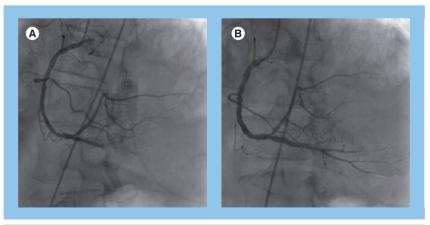


Figure 1. EKG shows ST-segment elevation on inferior–lateral leads and poor anterior R-wave progression.

procedure. Coronary angiography revealed a diffuse coronary artery disease (CAD) with acute thrombotic occlusion of the posterior descending artery (PDA) of the right coronary artery (RCA) and a CTO of the proximal left anterior descending (LAD) previously supported by septal collaterals from the PDA. Left ventricular (LV) function appeared significantly impaired. Anterograde flow in the IRA was restored by multiple manual thrombus aspirations with Invatec (Italy) Diver CE Max. Next, the culprit lesion was stented with a Chrono Carbostent 3 × 16 mm at 18 atm, obtaining a final thrombolysis in myocardial infarction (TIMI)-3 flow and myocardial blush grade 2 (FIGURE 2). Nevertheless, the patient continued to be hemodynamically unstable. Thus, considering the low dose of contrast used and the short duration of the recanalization of the



**Figure 2. Right coronary artery angiography. (A)** Infarct-related lesion: thrombotic occlusion of large posterior descendent artery. **(B)** Final result after primary percutaneous coronary intervention with stent implantation.

culprit vessel, LAD was engaged with a Cordis Extra Back Up guiding catheter and LAD CTO was crossed with an intermediate wire (ACS, Abott Vascular) supported by an over-the-wire  $2.0 \times 20$  mm balloon in a relatively easy manner. The procedure was completed by implanting a carbostent & implantable device: chrono Carbostent  $3.5 \times 25$  mm postdilated with a noncompliant balloon  $3.5 \times 20$  mm at 26 atm with an optimal angiographic result (FIGURE 3). The patient was sent to a coronary care unit without the need for an intra-aortic balloon pump and discharged after 6 days with a moderately impaired LV function. No major adverse coronary events (MACEs) were recorded at the 6-month clinical follow-up.

The present case represents a situation occurring with an increasing frequency in interventional cardiology, consisting of a patient with an AMI and MVD further complicated by the concurrent presence of a CTO in the non-IRA. Nevertheless, despite the relatively high frequency of this scenario, clear evidence from randomized clinical trials that can guide the strategy of the interventional cardiologist is lacking. Consequently, the correct clinical management of similar cases can be inferred only indirectly from the literature.

# Negative prognostic role of multivessel coronary artery disease & noninfarct-related artery CTO in the setting of acute myocardial infarction Multivessel CAD in the setting of primary PCI represents a frequent finding, ranging from 40 to

65% of STEMI patients undergoing urgent coronary angiography [4,5]. The presence of MVD is associated with a significant increase in the rate of early recurrence of MACEs, especially reinfarction and need for revascularization [4,9,10]. More importantly, the presence of MVD has been shown to be an independent predictor of 1-year mortality after mechanical reperfusion of the culprit vessel [10].

The presence of MVD can adversely affect survival through several mechanisms. Notably, patients with MVD have a greater incidence of comorbidities such as older age and a higher rate of cardiovascular risk factors, possibly contributing to an adverse prognosis. However, MVD has been shown to be an independent predictor of mortality even after adjustment for all these risk factors [10]. A reduced reperfusion success may play a relevant role. Indeed, Sorajja *et al.* have demonstrated that in patients with single-, double- and triple-vessel disease, despite similar rates of epicardial TIMI-3 flow, successful myocardial reperfusion – as assessed by ST-segment resolution – is impaired in patients with significant disease remote from the infarct artery, proportional to the extent of CAD [11]. Accordingly, a lower successful myocardial perfusion, as assessed by myocardial blush grade or ST-segment resolution, results in a lower survival rate despite the restoration of normal epicardial blood flow in the culprit vessel [12,13].

Importantly, several studies have recently shown that the higher mortality rate observed in patients with AMI and MVD is mainly determined by the presence of a CTO in a non-IRA and not by the mere presence of MVD. The finding of a CTO in non-IRA in the setting of primary PCI is not so uncommon, as randomized trials and registries indicate that it represents almost a third of all cases with MVD and 12–13% of all patients with STEMI [8,9].

Moreno et al., while assessing the event-free survival rate among 630 patients with AMI treated with PCI within 12 h after symptom onset according to the presence of single vessel disease, MVD without CTO and MVD with CTO, demonstrated that the group of patients with MVD and CTO had the worst outcome [9]. In accordance with these results, in a larger population, Van der Schaaf et al. demonstrated that STEMI patients with MVD had a higher mortality at 1-year follow-up compared with patients with single vessel disease and that this increased risk profile in MVD patients is mainly due to CTO of non-IRA (odds ratio [OR]: 3.8; 95% CI: 2.5-5.8) [8]. In a recently published paper, the same group, while confirming that CTO rather than the mere presence of MVD is a strong predictor of mortality during the first 30 days after PCI (OR: 3.6; 95% CI: 2.6-4.7; p < 0.01), have also shown that the negative prognostic value of CTO is maintained up to a 5-year follow-up, even excluding patients who died within 30 days of the acute event (OR: 1.9; 95% CI: 0.8-1.6; p < 0.01) [14]. Interestingly, the presence of a CTO, but not of MVD alone, was strongly associated with a decrease in LV ejection fraction (OR: 3.5; 95% CI: 1.6–7.8; p < 0.01) suggesting that a possible explanation for this worse outcome might reside in the fact that STEMI patients with a non-IRA CTO undergo a more pronounced postinfarction worsening of LV function and an unfavorable LV remodeling process.

# **Importance of CTO revascularization in mutivessel coronary artery disease** While the aforementioned findings highlight the prognostic relevance of a CTO in MVD in the setting of AMI, the long-term benefit of a



**Figure 3. Left coronary artery angiography. (A)** Chronic total occlusion of nonculprit lesion: proximal occlusion of left anterior descending artery. **(B)** Final result after successful chronic total occlusion treatment.

CTO recanalization is limited to patients with MVD only. In fact, Valenti *et al.*, testing the efficacy of a modern percutaneous approach to CTO treatment using drug-eluting stents in a series of 486 consecutive patients with at least one CTO, found that PCI success (in almost three out of four patients) conferred a long-term survival benefit compared with a failed recanalization only in patients with MVD [15]. Of note, this survival benefit was restricted to MVD patients receiving a complete revascularization, while patients with incomplete revascularization showed a worse prognosis.

The site of the occlusion influences the prognostic impact of a CTO, as well as the clinical relevance of its recanalization. In a large cohort of stable patients, Safley et al. demonstrated that the benefit of reopening a CTO was evident in the recanalization of a chronically occluded LAD since its success was associated with reduced long-term mortality (hazard ratio [HR]: 0.61; 95% CI: 0.42-0.80), while this was not the case for the left circumflex artery or for RCA (HR: 0.84; 95% CI: 0.53-1.32 and HR: 0.95; 95% CI: 0.63-1.44, respectively) [16]. Nevertheless, a clinical benefit in the reopening of non-LAD CTO can be predicted when a large area of myocardial vitality is subtended by a CTO. Consequently, in order to decide whether to obtain a revascularization of a CTO lesion, either percutaneous or surgical, a careful evaluation of myocardial viability in the territory of the occluded vessel, using nuclear imaging, MRI or stress echo, is generally warranted. However, when these techniques are not available, the simple 12-leads resting ECG has a strong prognostic relevance since, while

in the presence of Q-waves myocardial viability has been demonstrated in more than half of cases [17], their absence represents an absolutely reliable marker of myocardial viability and of functional recovery in the territory of the occluded vessel [18].

### When to attempt a complete revascularization in STEMI patients with multivessel coronary artery disease: evidence from literature

Taken together, all these data suggest that a patient with an AMI and MVD presenting a concurrent CTO of the LAD and with evidence of myocardial viability in the territory of CTO should undergo CTO recanalization. However, as previously mentioned, current guidelines recommend a staged ischemia-driven strategy of treating nonculprit lesion in patients with AMI and MVD. This suggested approach is referred to a single study by Corpus et al. demonstrating not only a lack of benefit of treatment of nonculprit lesions, but also a higher rates of reinfarction, target vessel revascularization and MACEs among patients undergoing multivessel PCI [19]. Moreover, in this study, the proportion of patients undergoing multivessel PCI within the same procedure was only 5% (26 out of 506) of all patients showing MVD. Finally, the higher rate of mortality among patients with multivessel PCI during the same procedure occurred within the hospitalization and it was mainly driven by the presence of cardiogenic shock at admission, a situation in which the same guidelines recommend a complete revascularization, as discussed below.

More recently, other studies have demonstrated that simultaneous nonculprit vessel PCI in the setting of AMI is feasible and safe [20-24], while the benefit of this more aggressive approach remained unclear. In the Hepacoat for Culprit or Multivessel Stenting for Acute Myocardial Infarction (HELP AMI) trial, Di Mario et al. demonstrated that the 1-year incidence of repeat revascularization was similar among patients randomized to IRA treatment only or to complete multivessel treatment [22]. By contrast, in a retrospective analysis of more than 3500 patients treated with primary PCI, Hannan et al. demonstrated that PCI limited to IRA was associated with significantly lower inhospital mortality than multivessel PCI, while patients undergoing staged PCI of nonculprit vessels within 60 days had a significantly lower 12-month mortality rate than patients undergoing culprit vessel PCI only [23]. Moreover, Ochala *et al.*, testing the hypothesis that an aggressive multivessel revascularization strategy would have beneficial effects on LV function and on clinical outcomes, found that despite a trend towards an improvement in LV ejection fraction at 6 months, no significant difference in the incidence of MACEs during follow-up was present [24].

Several reasons might explain the uncertain benefit of multivessel PCI in the setting of AMI. First, it is well known that myocardial infarction is associated with an enhanced thrombotic and inflammatory state. Thus, in the acute setting and in the immediate short term after AMI, an unfavorable thrombotic and inflammatory milieu compounded by multiple areas of vascular injury induced by multivessel intervention might create a high-risk situation for recurrent ischemic events and restenosis. Moreover, the prolonging of the procedure and the consequent increase in contrast media utilization exposes the patient to a higher risk for contrast-induced nephropathy, a complication known to adversely impact the prognosis of STEMI patients [25]. Finally, it has been shown that degree of stenosis in moderately obstructive nonculprit lesions may be overestimated during primary PCI owing to the widespread vasoconstriction of the entire coronary tree and to the change in reference segment observed at angiographic follow-up [26]. This observation has important implications for decision-making on complete revascularization strategy in patients with MVD, possibly leading to the unnecessary and eventually harmful treatment of nonsignificant coronary stenosis.

However, all the studies that have tested the potential benefit of the aggressive strategy of multivessel revascularization during primary PCI have important limitations. First, the sample size is always small. Second, most of the evidence is based on retrospective observations. Last, and more importantly, the prognostic relevance of non-IRA CTO in the setting of AMI is not addressed at all.

# Hemodynamic instability: the relevant role of a non-IRA CTO

An exception to the general rule of limiting PCI to the IRA in patients with AMI and MVD is represented by cardiogenic shock. In fact, given the notion that the restoration of blood flow is the major predictor of survival in these patients and based on the results of the Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock? (SHOCK) trial [27], current guidelines also express a consensus of attempting multivessel PCI in patients with AMI and cardiogenic shock [7]. Again, no mention of treatment of a non-IRA CTO is given in the guidelines for PCI. This is a relevant issue, since the presence of CTO mainly affects mortality in the first days after AMI, as evident by the early separation of the Kaplan-Meyer survival curves in the studies by van der Schaaf et al. and Claessen et al. owing to higher rate of acute decompensation or cardiogenic shock [8,14]. In fact, as previously suggested by Conde-Vela et al., among patients with AMI and MVD, CTO of other coronary arteries is the strongest predictor of cardiogenic shock at admission [28]. This observation is further supported by the fact that, in the study by Claessen et al., the independent long-term prognostic value of CTO among 30-day survivors, although still significant, was lower than that observed for 30-day mortality (ORs: 1.9 vs 3.6, respectively) [14].

The high incidence of cardiogenic shock in patients with STEMI and a CTO in a non-IRA appears to be related to the pathophysiological concept that a CTO per se always determines a severe impairment of myocardial perfusion. Consequently, even though in the presence of a CTO collateral branches are sufficient to maintain full systolic contractile function in some patients, in most cases they just provide a minimum nutritional supply to hibernating myocardium. Accordingly, Werner et al., invasively measured collateral function by intracoronary pressure and flow recordings in the collateralized arterial segment in patients with stable CAD and a CTO and demonstrated that angiographically well-developed collaterals do not provide a sufficient functional supply to the occluded arterial segment [29]. Moreover, even in patients with normal regional LV function, collaterals provide a normal coronary flow reserve in less than 10%. The high prevalence of coronary steal in CTO indicates that even patients with well collateralized CTO may benefit from a revascularization. If this is true in a stable situation, it is easy to understand the dramatic consequence of the severe and abrupt impairment of myocardial perfusion that follows the acute occlusion of an arterial segment that provides collaterals to the territory supplied by a pre-existing CTO.

This was exactly what happened in the case of the patients presented here, where the residual perfusion of the occluded LAD territory, previously provided by septal collaterals, was acutely lost owing to the thrombotic occlusion of the PDA branch of the right coronary artery. Moreover, the successful recanalization of the CTO promptly allowed the patient to hemodynamically stabilize.

# Conclusion

The available data indicate that MVD has a tremendous deleterious effect on prognosis of STEMI patients and that this mainly depends on the presence of a CTO in a non-IRA. While the treatment of MVD in patients with STEMI represents a controversial issue in contemporary interventional cardiology, the prognostic impact of a non-IRA CTO and its management are not adequately taken into account by current guidelines. This is not a secondary issue, especially considering the relatively high incidence of a CTO in the context of an urgent coronary angiography in patients with STEMI and its strong predictivity of cardiogenic shock. In general, in the absence of acute decompensation of shock, an ischemia-driven staged procedure might be of first choice in case of non-IRA CTO, since it might have more risks than advantages. However, starting from a paradigmatic clinical case and analyzing the available literature, we believe that in particular conditions, such as in case of hemodynamic or electrical instability, in the presence of ECG findings suggesting myocardial viability in the territory of occluded vessel, in a patient with preserved renal function and when the procedure can be completed in a short time with a reasonable amount of contrast medium, PCI of the CTO might be attempted within the same procedure.

## **Future perspective**

Definitive data should come from specifically designed randomized clinical trials. However, these are unlikely to be performed for practical reasons. Consequently, clinical judgment, based on the available data and on the specific characteristics of the patient, has to be applied in this particular situation.

## Financial & competing interests disclosure

The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

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### Executive summary

- The finding of a chronic total occlusion (CTO) in noninfarct-related arteryin the setting of primary percutaneous coronary intervention represents almost a third of all cases with multivessel coronary artery disease and 12–13% of all patients with ST-segment elevation myocardial infarction.
- The presence of a concurrent CTO in a ST-segment elevation myocardial infarction patient is associated with an increased risk of cardiogenic shock and with worse long-term prognosis.
- In case of hemodynamic instability, percutaneous coronary intervention of a noninfarct-related artery CTO might be attempted within the same procedure.

## **Bibliography**

Papers of special note have been highlighted as: • of interest

== of considerable interest

- White HD, Norris RM, Brown MA *et al.*: Effect of intravenous streptokinase on left ventricular function and early survival after acute myocardial infarction. *N. Engl. J. Med.* 317, 850–855 (1987).
- 2 Keeley EC, Boura JA, Grines CL *et al.*: Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. *Lancet* 361, 13–20 (2003).
- 3 de Boer MJ, Suryapranata H, Hoorntje JC *et al.*: Limitation of infarct size and preservation of left ventricular function after primary coronary angioplasty compared with intravenous streptokinase in acute myocardial infarction. *Circulation* 90, 753–761 (1994).
- 4 Muller DW, Topol EJ, Ellis SG et al.: Multivessel coronary artery disease: a key predictor of short-term prognosis after reperfusion therapy for acute myocardial infarction. Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) Study Group. Am. Heart J. 121(4 Pt 1), 1042–1049 (1991).
- 5 Kahn JK, Rutherford BD, McConahay DR, Johnson WL, Giorgi LV, Hartzler GO: Results of primary angioplasty for acute myocardial infarction in patients with multivessel coronary artery disease. J. Am. Coll. Cardiol. 16, 1089–1096 (1990).
- 6 van der Schaaf RJ, Timmer JR, Ottervanger JP *et al.*: Long-term impact of multivessel disease on cause-specific mortality after ST elevation myocardial infarction treated with reperfusion therapy. *Heart* 92, 1760–1763 (2006).
- Impact of multivessel disease on long-term mortality in ST elevation myocardial infarction (STEMI) patients.
- 7 Silber S, Albertsson P, Avilés FF et al.: ESC Guidelines for percutaneous coronary intervention. The task force for Percutaneous Coronary intervention of the European Society of Cardiology. Eur. Heart J. 26, 804–807 (2005).

- van der Schaaf RJ, Vis MM, Sjauw KD et al.: Impact of multivessel coronary disease on long-term mortality in patients with ST-elevation myocardial infarction is due to the presence of a chronic total occlusion. Am. J. Cardiol. 98, 1165–1169 (2006).
- Demonstrates that negative long-term prognosis of patient with acute myocardial infarction and multivessel disease is related to the presence of a non-infarct-related artery (IRA) chronic total occlusion (CTO).
- 9 Moreno R, Conde C, Perez-Vizcayno MJ et al.: Prognostic impact of a chronic occlusion in a noninfarct vessel in patients with acute myocardial infarction and multivessel disease undergoing primary percutaneous coronary intervention. J. Invasive Cardiol. 18, 16–19 (2006).
- 10 Shihara M, Tsutsui H, Tsuchihashi M et al.: In-hospital and one-year outcomes for patients undergoing percutaneous coronary intervention for acute myocardial infarction. Am. J. Cardiol. 90, 932–936 (2002).
- Sorajja P, Gersh BJ, Cox DA *et al.*: Impact of multivessel disease on reperfusion success and clinical outcomes in patients undergoing primary percutaneous coronary intervention for acute myocardial infarction. *Eur. Heart J.* 28, 1709–1716 (2007).
- 12 Costantini CO, Stone GW, Mehran R et al.: Frequency, correlates, and clinical implications of myocardial perfusion after primary angioplasty and stenting, with and without glycoprotein IIb/IIIa inhibition, in acute myocardial infarction. J. Am. Coll. Cardiol. 44, 305–312 (2004).
- 13 McLaughlin MG, Stone GW, Aymong E et al.: Prognostic utility of comparative methods for assessment of ST-segment resolution after primary angioplasty for acute myocardial infarction. J. Am. Coll. Cardiol. 44, 1215–1223 (2004).
- 14 Claessen BEPM, van der Schaaf RJ, Verouden BJ *et al.*: Evaluation of the effect of a concurrent chronic total occlusion on long-term mortality and left ventricular function in patients after primary percutaneous coronary intervention. *JACC Cardiovasc. Intverv.* 2, 1128–1134 (2009).

- Demonstrates that the concurrent presence of a non-IRA CTO in STEMI patients is associated with an unfavorable left ventricular remodeling.
- 15 Valenti R Migliorini A, Signorini U *et al.*: Impact of complete revascularization with percutaneous coronary intervention on survival in patients with at least one chronic total occlusion. *Eur. Heart J.* 29, 2336–2342 (2008).
- 16 Safley DM, House JA, Rutherford BD, Marso SP: Success rates of percutaneous coronary intervention of chronic total occlusions and long-term survival in patients with diabetes mellitus. *Diab. Vasc. Dis. Res.* 3, 45–51 (2006).
- Schinkel AFL, Bax JJ, Boersma E *et al.*:
  Assessment of residual myocardial viability in regions with chronic electrocardiographic Q-wave infarction. *Am. Heart J.* 144, 865–869 (2002).
- 18 Schwarz G, Figulla HR, Werner GS: Resting 12-lead electrocardiogram as a reliable predictor of functional recovery after recanalization of chronic total coronary occlusions. *Clin. Cardiol.* 28, 293–297 (2005).
- 19 Corpus RA, House JA, Marso SP *et al.*: Multivessel percutaneous coronary intervention in patients with multivessel disease and acute myocardial infarction. *Am. Heart J.* 148, 493–500 (2004).
- 20 Kong JA, Chou ET, Minutello RM, Wong SC, Hong MK: Safety of single versus multi-vessel angioplasty for patients with acute myocardial infarction and multi-vessel coronary artery disease: report from the New York State Angioplasty Registry. *Coron. Artery Dis.* 17, 71–75 (2006).
- 21 Roe MT, Cura FA, Joski PS *et al.*: Initial experience with multivessel coronary intervention during mechanical reperfusion for acute myocardial infarction. *Am. J. Cardiol.* 88, 170–3 (2001).
- 22 di Mario C, Sansa M, Airoldi F *et al.*: Single vs multivessel treatment during primary angioplasty: results of the multicentre randomised Hepacoat for Culprit or Multivessel Senting for Acute Myocardial Infarction (HELP AMI) Study. *Int. J. Cardiovasc. Intervent.* 6, 128–133 (2004).

- 23 Hannan EL, Samadashvili Z, Walford G et al.: Culprit vessel percutaneous coronary intervention versus multivessel and staged percutaneous coronary intervention for ST-segment elevation myocardial infarction patients with multivessel disease. JACC Cardiovasc. Interv. 3, 22–31 (2010).
- 24 Ochala A, Smolka GA, Wojakowski W *et al.*: The function of the left ventricle after complete multivessel one-stage percutaneous coronary intervention in patients with acute myocardial infarction. *J. Invasive Cardiol.* 16, 699–702 (2004).
- 25 Marenzi G, Lauri G, Assanelli E et al.: Contrast-induced nephropathy in patients undergoing primary angioplasty for acute myocardial infarction. J. Am. Coll. Cardiol. 44, 1780–1785 (2004).
- 26 Hanratty CG, Koyama Y, Rasmussen HH et al.: Exaggeration of nonculprit stenosis severity during acute myocardial infarction: implications for immediate multivessel revascularization. J. Am. Coll. Cardiol. 40, 911–916 (2002).
- 27 Hochman JS, Sleeper LA, Webb JG et al.: Early revascularization in acute myocardial infarction complicated by cardiogenic shock. SHOCK Investigators. Should we emergently revascularize occluded coronaries for cardiogenic shock. N. Engl. J. Med. 341, 625–634 (1999).
- Highlights the importance of immediate and complete revascularization in patients with STEMI and cardiogenic shock.
- 28 Conde-Vela C, Moreno R, Hernández R et al.: Cardiogenic shock at admission in patients with multivessel disease and acute myocardial infarction treated with percutaneous coronary intervention: related factors. Int. J. Cardiol. 123, 29–33 (2007).
- 29 Werner GS, Surber R, Ferrari M, Fritzenwanger M, Figulla HR: The functional reserve of collaterals supplying long-term chronic total coronary occlusions in patients without prior myocardial infarction. *Eur. Heart J.* 27, 2406–2412 (2006).
- Demonstrates that in the presence of a CTO, collateral branches do not provide a sufficient functional supply to the occluded vessel.