Facilitated percutaneous coronary intervention: current concepts, promises, and pitfalls

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The reperfusion era has dramatically and irrevocably transformed the management of ST-segment elevation myocardial infarction (STEMI). For the patient treated with fibrinolysis as the initial reperfusion strategy, subsequent options which are the subject of some controversy and the focus of this review are (i) facilitated percutaneous coronary intervention (PCI); (ii) pharmacoinvasive approach; (iii) 'Watchful waiting'. We performed a literature search of all available articles on facilitated PCI published in English language on Medline, including randomized controlled trials, meta-analyses, and review articles. This information provided the core for a critical appraisal of the current literature on this controversial topic. On the basis of current available data, facilitated PCI with a thrombolytic agent has no role in everyday clinical practice and facilitated PCI utilizing a glycoprotein IIbIIIa inhibitor agent has no considerable benefit on mortality in STEMI in contemporary practice. The evidence to date suggests that fibrinolytic-facilitated PCI is associated with higher mortality rates and adverse events compared with primary PCI.

Introduction

The reperfusion era has dramatically and irrevocably transformed the management of ST-segment elevation myocardial infarction (STEMI) over the last 30 years. Nonetheless, outside of the setting of randomized trials, a pervasive aspect of clinical practice is a marked variability in management, including the preferred modality of reperfusion therapy. In regard to the latter, there are two pivotal issues that are currently the subject of vigorous debate.

The first relates to improving door-to-balloon (DTB) times and the achievement of rapid reperfusion in hospitals with facilities for primary percutaneous coronary intervention (PPCI).1 The second relates to the majority of hospitals in the USA and around the world that is not PPCI-capable. It must be stated that the total ischaemic time, which is composed of symptom to first medical contact and first medical contact to reperfusion, remains the most critical interval in regard to time to treatment.2 Several randomized trials and a pooled meta-analysis have led to a general consensus that with 'all things being equal', PPCI is the optimal reperfusion strategy,3 with the caveat that outcomes may be institutional and operator-dependent, and in some centres, the time of day or the day of the week may be an important prognostic factor.4

At community facilities that lack 24 h PPCI capability and availability, the armamentarium of reperfusion strategies is more diverse and the issues quite complex and controversial (Figure 1). The clinician must balance patient, operator, institutional, and transport variables and do so without delay. The impact of incurred delays to transport a patient to a second facility for PPCI is magnified because the clinician may be withholding immediate fibrinolysis, a potentially life-saving treatment.

The risk of catastrophic bleeding from fibrinolysis as well as growing enthusiasm for PPCI has fuelled an understandable shift towards immediate transfer for an invasive therapy—a strategy that could potentially lead to adverse consequences, unless the total duration of ischaemia is taken into account. Recent analyses of the National Registry of Myocardial Infarction Registry (NRMI) demonstrated that achieving DTB times of <90 min is associated with lower in-hospital mortality.5 Total ischaemic time, which includes patient-related delays, transfer-related delays, and system-related delays, is a critical determinant of outcomes. Moreover, an approach to transfer all patients for PPCI has substantial implications for healthcare costs, the development of regional networks, transport systems, and rural critical access hospitals.

For the patient treated with fibrinolysis as the initial reperfusion strategy (Figure 1), subsequent options which are the subject of some controversy and the focus of this review are as follows:

(i) facilitated PCI (transfer for immediate angiography and PCI);
(ii) pharmacoinvasive approach (immediate transfer and rescue PCI for failed reperfusion or routine angiography within 24 h of successful reperfusion);
(iii) 'Watchful waiting' (transfer only in the event of recurrent ischaemia, either spontaneous or during a stress test).

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The current focus of interest is upon facilitated PCI vs. the pharmacoinvasive approach, as increasing evidence would suggest that early transfer to an invasive facility is the preferred approach even though these trials were rather small and the beneficial effects of early PCI related only to soft endpoints such as recurrent ischaemia. In this respect, trials comparing PPCI vs. fibrinolytic therapy need to be critically reviewed from the perspective of duration of ischaemia from symptom onset to reperfusion therapy in patients requiring transfer.

The strategy of facilitated PCI is intuitively appealing, as this approach combines the benefits of early thrombolysis with those of immediate PCI. At the same time, it should be recognized that this combined strategy also entails the costs as well as the complications of thrombolysis and immediate PCI. Early fibrinolysis would aim to achieve partial or full reperfusion as a ‘bridge’ to mechanical stabilization of the ruptured plaque with subsequent PCI. Nonetheless, the facilitated approach does expose all treated patients to the bleeding risks of fibrinolytic therapy and to procedural-related complications in the milieu of a systemic fibrinolytic state. This strategy is particularly relevant for the USA, as although a hospital without PCI capability is the closest facility for 60% of the population, 80% live within 60 min of a PCI-capable hospital. With this important statistic in mind, it is crucial that we develop methods to establish a pre-hospital diagnosis of acute STEMI in our healthcare systems.

**Benefits and risks of reperfusion strategies**

**Fibrinolytic therapy**

The striking benefits of fibrinolytic therapy on mortality and infarct size when administered within 1–2 h of symptom onset should be emphasized. The Myocardial Infarction Triage and Intervention (MITI) Randomized Trial project investigators demonstrated that 2-year survival was 98% for patients treated with fibrinolysis within 70 min from symptom onset compared with 88% for those treated later (P = 0.12). In the ASSENT 3 trial, ~25% of patients treated with fibrinolytic therapy within the first hour did not have any enzymatic evidence of myocardial necrosis—what has been termed ‘aborted MI’. The PRAGUE 2 Study demonstrated a trend in favour of fibrinolytic therapy when administered within 3 h of symptoms vs. mechanical reperfusion; the study was however underpowered to demonstrate statistical significance.

Recent data from The Viennese Registry on Reperfusion Strategies in ST-Elevation Myocardial Infarction (Vienna STEMI Registry) showed that in-hospital mortality rates were lowest in patients treated within 2 h of symptom onset. There was a trend in favour of fibrinolytic therapy over PCI (5.1 vs. 7.8% P = 0.37). These results are comparable to those of Comparison of Angioplasty and Prehospital Thrombolysis in Acute Myocardial Infarction trial (CAPTIM), in which patients randomized <2 h after symptom onset had a trend towards lower 30-day mortality with prehospital fibrinolysis than those randomized to PCI (2.2 vs. 5.7%, P = 0.058). Boersma et al. presented a comprehensive meta-analysis of 25 trials comparing the efficacy of PCI vs. fibrinolysis in patients with acute STEMI. This study suggested a benefit for PCI during all time periods, but it must be stated that the authors utilized the time from randomization and not total ischaemic time to treatment, therefore not taking into account initial delays involved in patient transfer. A prospective multi-centre French registry demonstrated that among patients who were admitted to hospital within 3.5 h from symptom onset, after receiving pre-hospital fibrinolytic therapy (among whom 67% underwent angiography before...
discharge), the observed in-hospital mortality was 0% and 1 year mortality was 1%.19

However, fibrinolytic therapy has four main limitations.

(i) TIMI-3 flow at 90 min is achieved in only 60–70% of patients treated with full-dose fibrinolysis, and effective microvascular perfusion is only seen in 25–30% of patients.

(ii) The risk of bleeding, particularly in the elderly.

(iii) Many patients have absolute or relative contraindications.

(iv) Patients are at risk for re-occlusion after successful fibrinolysis.

Re-occlusion and recurrent MI after successful reperfusion with fibrinolysis portend a poor prognosis. Gibson et al.20 demonstrated that the frequency of symptomatic recurrent MI in four randomized trials of fibrinolytic therapy was 4.2%, with a mortality of 16.4 vs. 6.2% (P < 0.001) when compared with patients without recurrent infarction. It must be stated that in the GUSTO-V trial, the lower rate of re-infarction with combination therapy was not associated with a better prognosis at 1 year.21 Therefore, the findings from Gibson et al.20 from the Thrombolysis in Myocardial Infarction (TIMI) trials might overstate the impact of re-infarction on outcome.

**Primary percutaneous coronary intervention**

Currently, there is a general consensus that PPCI is the preferred approach when delivered rapidly and in high-volume centres by experienced teams.2 However, PPCI is limited by intra-hospital and inter-hospital transport delays from first medical contact to balloon inflation or reperfusion in the catheterization laboratory. Transport times are significantly longer in the USA compared with those achieved in the DANish trial in Acute Myocardial Infarction-2 (DANAMI-2).22 The median DTB time in the DANAMI-2 trial was 112 min for transfer patients when compared with a median DTB of 180 min for transfer patients in the USA.23

An area of debate and controversy revolves around the optimal reperfusion strategy for STEMI patients who present to a community hospital without PCI capability, with symptoms of <2–3 h duration (Figure 2). Current guidelines recommend that if DTB <90 min is reliably attainable, then the preferred approach would be PPCI. If DTB, including the time for inter-facility transport, exceeds door-to-needle (DTN) time by >60 min, then fibrinolytic therapy should be considered (Figure 1).24,25 In some subsets of patients, longer delays may be acceptable.26

Time to reperfusion is a less critical determinant of mortality and myocardial salvage for the STEMI patient who presents with symptom duration >2–3 h (Figure 2).27 The benefits of fibrinolysis on myocardial salvage decrease over time, and studies have suggested that the superiority of PPCI becomes evident in the setting of a more prolonged duration of symptoms.28 Although only documented in case of streptokinase and in vitro, the prevailing hypothesis is that older clots become more resistant to fibrinolytic drugs, whereas their amenability to a mechanical approach remains constant.29

PPCI would be the preferred strategy after an ischaemic time of 3 h or longer, as during this phase time to treatment is less of a concern and the major priority is to obtain patency of the infarct-related artery (IRA). What the clinician needs to balance are the advantages of a mechanical approach vs. incurred transport-related delays, particularly when these delays exceed 120–180 min. Nonetheless, it should be emphasized that the earlier the presentation after the onset of symptoms, the greater the impact of transfer-related delays for PPCI and the stronger the case for prompt reperfusion therapy with fibrinolytic agents.30 It must be stated that practice patterns in the USA and Europe are indeed different, with pre-hospital ECGs and administration of pre-hospital fibrinolytics being less frequently utilized within the markedly diverse EMS systems in most US cities. As a result, approaches to patients with STEMI will likely have significant regional and national differences.

**Facilitated percutaneous coronary intervention**

**Definitions**

The term ‘facilitated PCI’ has been used to refer to multiple strategies of pharmacotherapies administered before immediate PCI, including full-dose or half-dose fibrinolytic agent or glycoprotein IIb/IIIa inhibitor, as well as combinations of these two agents. There is some debate as to whether the use of glycoprotein IIb/IIIa inhibitors alone without thrombolytic agents falls under the rubric of facilitated PCI. Some trials have suggested a benefit with glycoprotein IIb/IIIa inhibitors before PCI.31,32 A pooled analysis has shown that early administration of glycoprotein IIb/IIIa inhibitors before arrival at the catheterization laboratory leads to higher rates of TIMI grade 2 or 3 flow.33

**Rationale**

The rationale for facilitated PCI is based on the hypothesis that combining early pharmacologically mediated reperfusion with subsequent and immediate mechanical stabilization of the ruptured plaque will overcome delays to transfer the patient to a second facility. Several studies have suggested that an initially patent IRA with TIMI grade 2–3 flow may result in better outcomes during PCI when compared with an IRA with initial TIMI 0–1 flow.34–36 Facilitated PCI clearly achieves higher rates of TIMI grade 2–3 flow prior to catheterization, but this has not translated to improved clinical outcomes as measured by infarct size or mortality.

The outcomes associated with facilitated PCI, as well as any other reperfusion strategy, will directly depend upon the total duration of ischaemia prior to reperfusion therapy. The relationship between the duration of ischaemia, extent of myocardial salvage, and reduction in mortality is of paramount importance (Figure 2). There is a narrow but ‘golden’ window of opportunity within the first 2–3 h of an evolving STEMI, during which, time to treatment (rather than the method of reperfusion) is the most critical determinant. During this 2–3 h window, every 30 min delay is associated with an 8% increase in relative mortality at 1 year.4 A recent article by Pinto et al.,30 using 192 509 STEMI patients at 645 National Registry of Myocardial Infarction hospitals, has illustrated quite emphatically the impact of delays in time to reperfusion upon outcomes. After ~3 h, there remains a persistent benefit from reperfusion therapy but time to treatment is less critical. The overarching goal
during this period is to open the IRA,\textsuperscript{37} which is best achieved with PCI rather than fibrinolytic therapy.

**Results**

Keeley et al.\textsuperscript{38} recently reported their second major pooled analysis comparing 17 randomized trials of facilitated vs. PPCI (Figure 3). Fibrinolytic-facilitated PCI was used in six trials, glycoprotein IIb/IIIa inhibitor-facilitated PCI in nine trials, and the combination of reduced dose thrombolytic- and glycoprotein IIb/IIIa-facilitated PCI in two trials. Despite achieving higher rates of pre-intervention TIMI 3 flow in the facilitated PCI group compared with PPCI (37 vs. 15\%, $P = 0.0001$), facilitated PCI was associated with higher mortality (5 vs. 3\%, $P = 0.04$). Furthermore, the higher mortality was confined to full-dose fibrinolytic therapy-alone-facilitated PCI regimens and not seen with glycoprotein IIb/IIIa inhibitors alone or with facilitated regimens of platelet glycoprotein IIb/IIIa inhibitors in combination with reduced-dose fibrinolitics. The authors concluded that a facilitated PCI strategy utilizing a fibrinolytic agent should be avoided unless under the auspices of a clinical research trial. Moreover, current guidelines do not support a strategy of PCI with full-dose fibrinolytic drugs. It should also be recognized that there is currently no evidence to support a strategy of half-dose lytics during transport prior to immediate PCI.

The largest study to date comparing facilitated PCI and PPCI is the Assessment of the Safety and Efficacy of a New Treatment Strategy with Percutaneous Coronary Intervention (ASSENT-4 PCI) investigation.\textsuperscript{39} This trial randomized patients with STEMI of <6 h duration to PPCI ($n = 838$) or to full-dose tenecteplase-facilitated PCI ($n = 829$). The study was terminated early by the Data and Safety Monitoring Board due to increased in-hospital mortality in the facilitated PCI arm compared with the PPCI group (6 vs. 3\%, $P = 0.0105$) (Table 1). The higher in-hospital mortality observed in the facilitated PCI was largely attributable to higher rates of total stroke [15/829 (1.8\%) vs. 0, $P < 0.0001$] and haemorrhagic stroke [8/829 (1.0\%) vs. 0, $P = 0.0037$].

Kastrati et al.\textsuperscript{40} randomized STEMI patients to half-dose fibrinolytic therapy combined with glycoprotein IIb/IIIa inhibitor vs. glycoprotein IIb/IIIa inhibitor alone prior to PPCI. Despite achieving higher rates of TIMI grade 3 flow with a combination of a half-dose fibrinolytic agent and a glycoprotein IIb/IIIa inhibitor compared with glycoprotein IIb/IIIa inhibitors alone, there was no difference in infarct size. The median time to treatment from onset of symptoms was 160 min for the former group and 164 min for the latter. These results support that moving along the ‘flat’ part of the

![Figure 3](image-url)

*Figure 3* Short-term death in patients treated with facilitated or primary percutaneous coronary intervention. This table summarizes the 17 trials analysed by Keeley et al. looking specifically at short-term mortality comparing facilitated with primary percutaneous coronary intervention. These trials randomly assigned 4504 patients (2237 to facilitated intervention, 2267 to primary intervention). As shown in the figure, the facilitation agent was platelet glycoprotein IIb/IIIa inhibitors in nine trials ($n = 1148$), thrombolytic therapy in six ($n = 2957$), and the combination of platelet glycoprotein IIb/IIIa inhibitors and reduced-dose thrombolytic therapy in two ($n = 399$). Reproduced from Keeley et al.\textsuperscript{38}
curve beyond 3 h after symptom onset (Figure 2) will not improve myocardial salvage or survival.

As trials that administered glycoprotein IIb/IIIa inhibitors without lytics in the ED before primary PCI tended to show improved IRA flow and trends for improved outcomes, a potential alternative approach that might warrant further evaluation includes the immediate administration of triple anti-platelet therapies (aspirin, clopidogrel, and glycoprotein IIb/IIIa inhibitors with an appropriate dose of heparin) at a community hospital before transfer for primary PCI. The recently, prematurely terminated Facilitated Intervention with Enhanced Reperfusion Speed to Stop Events (FINESSE) study compared the efficacy and safety of early administration of reduced-dose reteplase and abciximab combination or abciximab alone followed by routine, immediate PCI compared with PCI in approximately 2400 patients (clinicaltrials.gov number NCT00046228). The results will be presented late in 2007 (S. Ellis, personal communication). The CARESS in MI (Combined Abciximab REteplase Stent Study in acute myocardial infarction) trial has completed enrolment of approximately 600 patients, and the results will be available later this year.41 This trial utilized half-dose reteplase plus abciximab. Perhaps until the results of these trials become available, we should remember the words of Mark Twain who upon reading his obituary notice immediately sent a cable from London to the press in the USA stating ‘the reports of my death are greatly exaggerated’ (Mark Twain reference).42

Lack of benefit in trials of facilitated percutaneous coronary intervention—potential explanations

There are several potential explanations for the disappointing and somewhat surprising results of the trials of facilitated PCI. In the ASSENT-4 PCI patients presenting directly to a PCI hospital and randomized to the facilitated arm had a high mortality rate compared with all other groups. The initial hypothesis was that they had such a short interval from fibrinolytic bolus to first balloon inflation that they were ‘double burdened’ i.e. too short an interval for the lytic to work but heightened exposure to its bleeding propensity. Further inspection revealed that the lytic to balloon interval in the pre-hospital cohort was almost identically short, yet these patients had the lowest mortality in the trial. No cogent explanation has yet surfaced (A. Ross, personal communication).

The ASSENT-4 study has also been criticized for the absence of a heparin infusion after initial bolus, lack of up-front clopidogrel,43,44 and prohibition of glycoprotein IIb/IIIa use except in the setting of ‘bailout’ in the facilitated PCI arm. The open-label nature of the study may have also introduced bias.45 Patients treated with tenecteplase were less likely to undergo subsequent PCI at the time of the acute cardiac catheterization (86.7 vs. 90.9%), which could be because of higher rates of TIMI 3 flow at the time of angiography. Consequently, they were less likely to benefit from mechanical plaque stabilization with stenting. The median time from symptom onset to fibrinolytic administration was 153 min in the facilitated PCI group, but it should be emphasized that fibrinolytic-mediated reperfusion takes another 60 min, leading to a total ischaemia time of 213 min. At this stage, 3.6 h from symptom onset to reperfusion, the patient falls on to the flat part of the curve for myocardial salvage and mortality reduction (Figure 2).46

Lastly, PCI early (1–2 h) after fibrinolytic therapy can theoretically lead to a range of complications which can outweigh potential benefits of achieving TIMI grade 3 flow in the IRA prior to catheterization. These include fibrinolytic-induced platelet activation, intramural coronary haemorrhage, myocardial haemorrhage leading to ventricular free-wall rupture, haemorrhagic stroke, and other systemic bleeding. The unusual high mortality rate of >8% in the facilitated PCI arm of the ASSENT-4 trial is indeed surprising and unexplained. An electrocardiographic analysis of ST-segment resolution in the ASSENT-4 trial demonstrated better early reperfusion with facilitated PCI. Between 60–180 min, worsening ST-segment resolution was more frequent with facilitated PCI and associated with high rates of recurrent myocardial infarction consistent with a prothrombotic effect and re-occlusion.47 Further subset analyses of the ASSENT-4 trial will hopefully place the overall results into perspective.

The potential disadvantages of performing a mechanical intervention in a fibrinolytic landscape were suggested by the results of earlier trials performed between 1987 and 1991 (the era of the learning curve of PCI), which compared early angiography with routine PCI with a policy of ‘watchful waiting’ after fibrinolytic therapy (Figure 4). Results were discordant with a reduction in recurrent ischaemic events in both, little change in ventricular function, and a trend towards harm in others.48–52 More recently, several pilot studies which reflect the contemporary era of mechanical intervention demonstrated safety and a signal pointing towards efficacy.6,7,53 A recent meta-analysis suggested that after the administration of fibrinolytic therapy, early angiography and PCI, if appropriate, are superior to a strategy of ‘watchful waiting’.54

Pharmacoinvasive strategy

Dauerman and Sobel45 highlighted the concept of ‘pharmacoinvasive recanalization’, which is defined as pharmacological reperfusion followed by routine, delayed coronary angiography and PCI. The Which Early ST-elevation
myocardial infarction Therapy (WEST) pilot study supports the efficacy and safety of combining fibrinolytic therapy with routine, delayed PCI. This study enrolled 304 STEMI patients: Group A received fibrinolytic therapy (weight-adjusted TNK) \((n = 100)\) followed by usual care; Group B received fibrinolytic therapy followed by invasive management within 24 h, including rescue PCI \((n = 104)\); Group C patients underwent PPCI \((n = 100)\). Mortality and re-infarction at 30 days were higher in Group A when compared with Group C \((13 \text{ vs. } 4\%, \ p = 0.021)\). There was no difference in outcomes between the pharmacoinvasive Group B and Group C \((6.7 \text{ vs. } 4.0\%, \ p = 0.378)\). The incidence of major systemic bleeding was similar in all three groups.

These results are indeed interesting, but significant differences exist between the ASSENT-4 trial and the WEST trials. First, patients in the WEST trial presented relatively ‘early’ with a median time from symptom onset to fibrinolytic therapy of 130 min in the pharmacoinvasive group compared with 153 min from symptom onset to fibrinolytic therapy in the ASSENT-4 trial. The median time from symptom onset to balloon inflation was 926 min for the pharmacoinvasive group compared with 263 min for the facilitated PCI group in ASSENT-4. The performance of routine, delayed PCI –13 h after fibrinolytic therapy in the WEST trial may explain the lower rate of complications such as haemorrhagic stroke when compared with the ASSENT-4 trial in which PCI was performed –2 h after fibrinolytic therapy. The timing of mechanical intervention may be a ‘critical issue’ as there may be an optimal window after fibrinolysis or a pharmacoinvasive approach after successful fibrinolysis. The optimal window or timing for facilitated PCI is a potentially attractive option. In this respect, the nuances of results of the FINESSE and CARESS trials are clinically important for the future or lack thereof of this strategy. Whether the numbers of patients enrolled in these trials will provide sufficient statistical power remains to be determined. Nonetheless, the prevailing impression is that the momentum has shifted away from the strategy of facilitated PCI towards a pharmacoinvasive approach.

**Future directions**

On the basis of the accumulated evidence, it is critical for PCI-capable hospitals to implement protocols and critical care pathways to achieve a DTB <90 min on all eligible STEMI patients. The term PCI-capable is not synonymous with PCI available during off-hours and rapid reperfusion. In community hospitals that are not PCI-capable, it is equally important to implement protocols and critical care pathways to choose a reperfusion approach, fibrinolysis or PPCI, rapidly and to immediately transfer the patient to a PCI-capable hospital for either rescue PCI after failed fibrinolysis or a pharmacoinvasive approach after successful fibrinolysis. The optimal window or timing for cardiac catheterization for the pharmacoinvasive approach remains undefined (currently within 6–24 h).

Future strategies should also focus on decreasing total ischaemic time beyond what may be achievable by improving DTN and DTB. The duration from symptom onset to first hospital presentation remains an opportunity as patients continue to delay –2 h prior to first medical contact. In addition, less than one-third of STEMI patients currently use emergency medical services or 911, with the majority transported by family members or friends to the emergency department. A recent editorial has pointed out that this component of delay virtually ensures that for the majority of patients undergoing fibrinolytic-facilitated PCI, infarct artery reperfusion will occur on the ‘flat’ part of the curve (Figure 2). Specific interventions should be targeted at patients at greatest risk for an STEMI, including, but not limited to, those with prior MI, prior CHF, prior PCI, and prior CABG.

Other strategies to shorten total ischaemic time include an earlier diagnosis utilizing a pre-hospital electrocardiogram and developing systems and networks for ambulance triage to bypass community hospitals without PCI capability, as well as to bypass the emergency department of PCI.
Facilitated PCI capable hospitals and transport the 'definite STEMl' patient directly to the catheterization laboratory. These are complex issues with major regional and national differences in the manner in which they will be addressed.64

The importance of pre-hospital fibrinolysis has been emphatically demonstrated especially in Europe, and the presence of emergency physicians on ambulances or the electronic integration of pre-hospital electrocardiograms into systems of care will further reduce times to the early diagnosis and treatment of STEMI patients. This approach requires collaboration and integration across a continuum from emergency medical services, emergency departments in a geographic region, and cardiac catheterization laboratories. This strategy must be sensitive to critical care access hospitals in rural communities as well as competitive scenarios between urban PCI-capable hospitals, with the overriding goal being the most expedient care for the STEMI patient. The triage or transport of every chest pain, unstable angina, or non-STEMI patient immediately to a cardiac catheterization laboratory is neither realistic nor desirable.

A final strategy to increase access and expedite care for STEMI patients would be to allow community hospitals to perform PCI without on-site surgery.65,66 This model of care for STEMI patients would be to allow community hospitals to immediately transport patients to a cardiac catheterization laboratory is expedient care for the STEMI patient. The triage or transport of every chest pain, unstable angina, or non-STEMI patient immediately to a cardiac catheterization laboratory is neither realistic nor desirable.

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Supplementary material

Supplementary material is available at European Heart Journal online.

Conflict of interest: none declared.

References


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Bilateral coronary artery occlusion after aortic valve replacement in a patient with porcelain ascending aorta

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A 75-year-old woman with diabetes and chronic renal failure underwent aortic valve replacement (St. Jude Medical Biocor 21 mm, St Paul, MN, USA) for severe aortic stenosis. Pre-operative cardiac catheterization showed normal coronary arteries and a severe calcification of the aortic valve and the ascending aorta (Panel A). The patient suddenly had chest pain with hypotension on day 12 after surgery. The ECG suggested an inferior acute myocardial infarction. Emergency coronary angiography revealed total ostial occlusion of the right coronary artery (Panel B, black arrow) and a mobile intraluminal filling defect in the left main coronary artery (Panel B, white arrow). The patient developed electromechanical dissociation and died during catheterization. Necropsy showed severe atherosclerotic disease of the ascending aorta and accumulation of calcium-like material between the prosthesis and the aortic root wall, which completely occluded the right coronary ostium (Panel C). The left main coronary artery was patent. Histochemical analysis confirmed the presence of calcified fragments of atheromatous plaque located around the aortic prosthesis (Panel D).

It can be speculated that the manipulation of an atherosclerotic ascending aorta during cardiac surgery and the erosion produced by the prosthesis stent in the wall of a small-sized calcified ascending aorta could have precipitated the accumulation of calcium-like material around the aortic prosthesis. This material might as well be responsible for the mobile left coronary occlusion. This case emphasizes the risk of cardiac surgery in patients with severely diseased ascending aorta.

Panel A. Pre-operative cardiac catheterization. Fluoroscopic image showing an extensive calcification of the mitral annulus, and severe calcification of the ascending aorta and the aortic valve. Aortography shows moderate aortic regurgitation and coronary angiograms are normal. RCA, right coronary artery; LCA, left coronary artery.

Panel B. Emergency aortography performed during inferior myocardial infarction shows a total ostial occlusion of the right coronary artery (black arrows) and a mobile intraluminal filling defect in the left main coronary artery which moves during both phases of the cardiac cycle (white arrows).

Panel C. Macroscopic examination shows an atherosclerotic aortic root with a transverse competent suture and a biological prosthesis without thrombotic material in its valves. An accumulation of calcium-like material is identified between the aortic prosthesis and the aortic root, which is more abundant in the right side (black arrows). The white arrows point to the left coronary ostium.

Panel D. Microscopic analysis demonstrates fragments of atheromatous plaque with macrophages, lymphocytes, cholesterol crystals, and important deposits of calcium (purple crystals) located around the aortic prosthesis (HE 100×).