Percutaneous Coronary Intervention: An Overview

Najih Farooqi, Mobeen Farooqi, Mohamed K. Hussein, Ruqqaya Maham, and Anoosh Farooqui

ABSTRACT

PCI remains the mainstay treatment for acute cardiac ischemia. The indications for PCI include Stable Ischemic Heart Disease, STEMI and NSTEMI. The procedure involves the use of a guided catheter steered to the site of coronary arterial blockage, followed by dilation of the balloon at its end to remove the blockage and placement of a stent to maintain the patency. The choice of stent varies from case to case. Certain alternatives and adjuvant therapies are incorporated in order to adapt to specific scenarios. Complications can arise, which may require operative intervention. Mortality rate associated with the procedure is affected by the age and gender of the patient. The decision to opt for either CABG or PCI is based on the complexity of the vessel disease, and scales like the SYNTAX score are used to make this decision.

Keywords: Heart disease, NSTEMI, percutaneous coronary intervention (PCI), STEMI.

I. INTRODUCTION

Percutaneous coronary intervention (PCI), was first performed successfully by Andreas Gruntzig in 1977. It was not until 1982 that guidelines for balloon angioplasty were introduced. It is a well-established procedure used in Myocardial Infarction (MI) and to relieve angina symptoms in Coronary Artery Disease (CAD).

The coronary arteries are accessed by a peripherally-inserted catheter, usually through the femoral or radial artery and less commonly via the brachial or ulnar artery. After establishing peripheral access, the catheter is advanced to the heart under fluoroscopic guidance where the affected vessels are identified and re-vascularized [1]. The outcomes are affected by the size of the ischemia, the duration of the occlusion and the capacity of the collateral blood vessels to supply the affected area.

The physiology of the disease initiates with a critical narrowing, the blood supply of the affected organ is affected. This propagates the myocardial cells to turn towards anaerobic metabolism leading to build up of lactic acid. Moreover, the excess of intracellular H+ overloads the Na+/H+ channel in the cell wall. The cellular sodium influx activates the Ca2+/Na+ channel in the sarcoplasmatic reticulum. Cellular calcium overload results in disorganization of cytoskeleton and excess of reactive oxygen species, and along with mitochondrial collapse, proteolysis, edema, leukocyte infiltration and capillary rupture, ultimately leading to necrosis [2].

To cease this chain of events, timely revascularization is of essence in a patient with acute MI. PCI offers a feasible less invasive modality to achieve the goal of myocardial salvage.

II. INDICATIONS

A. Stable Ischemic Heart Disease (SIHD)

These are patients with previously diagnosed coronary artery disease (CAD), without any recent changes or complications of their symptoms. This includes patients with (1) Stable angina, and angina equivalent, (2) Post-ACS patients (3) Asymptomatic SIHD (Silent Ischemia) [3].

Stable angina refers to discomfort in chest, as well as in arm, jaw or shoulder, in CAD patients due to mismatched oxygen demand and supply. It occurs only on physical exertion or stress, is short lived and is relieved by rest or medication (such as nitroglycerin). Angina equivalent refers...
to having atypical symptoms including shortness of breath (SOB) on exertion as well as nausea, fatigue etc. [3]

American College of cardiology (ACC) [4] recommends Guideline Dependent Medical Therapy (GDMT) for all SIHD patients. This comprises antiplatelet therapy, beta-blockers, renin-angiotensin blockers, and vasodilators (calcium channel blockers, nitroglycerin etc).

SIHD patients are assessed for high-risk coronary lesions by stress testing.

An abnormal result is when the patient develops SOB, chest discomfort and cramping during testing. This ought to be followed by an angiographic evaluation.

PCIs are indicated in SIHD patients with:

- Non-complex narrowing of more than 50% in the left main coronary artery.
- FFR < 0.8 or non-LAD coronary stenosis of ≥70%, with anginal symptoms compromising the quality of life [4].

B. ST Elevation Myocardial Infarction (STEMI)

ST-elevation myocardial infarction (STEMI) refers to transmural myocardial ischemia resulting in myocardial necrosis [5]. It manifests with the typical chest pain and other ischemic symptoms, accompanied with the diagnostic ST segment elevation on echocardiography [0.2 mV (2 mm) in males > 40 years old and for men < 40 years it is greater than 0.25 (2.5 mm)], or greater than 0.15mV (1.5 mm) in Women [6].

STEMI is classified depending on the part of myocardium involved:

- Inferior STEMI: Inferior heart wall infarct, mostly involving the Right coronary artery (RCA), whereas in some Left- dominant patients (15%) it is supplied by the left circumflex artery (LCX).
- Anterior STEMI: left anterior descending artery (LAD) occlusion leading to infarction of the anterior wall.
- Lateral STEMI: Lateral wall is supplied by both LAD and LCX.
- Posterior infarction: It is rarely ever an isolated infarction, and is mostly a complication of anterior and inferior infarctions.

Most patients with STEMI have multi vessel involvement, and so different PCI approaches are adopted [7]:

1) The artery with the most significant stenosis (culprit artery) is re-vascularized in the primary PCI. The remaining involved arteries (non-culprit) are addressed only if there is hypoperfusion or concerning outcomes on testing;  
2) All involved arteries (multi-vessel) can be revascularized in the primary PCI  
3) Primary PCI involving revascularization of the culprit artery followed by staged PCI of non-culprit arteries.  

Multi-vessel staged PCI is believed to bear better outcomes than the multi-vessel primary PCI [7]

C. Non ST Elevation Myocardial Infarction (NSTEMI)

Non-ST elevation myocardial infarction involves subendocardial ischemia [5]. Echocardiography in this case may show ST segment depression/elevation, T wave inversion or no significant abnormalities [8]. Its diagnosis is heavily based on clinical features and serum cardiac enzyme studies.

Patients with NSTEMI undergo extensive risk stratification, to decide correct management. The risk stratification involves assessing the patient’s prognosis and clinical outcomes by using a combination of clinical prediction (based on history, ECG findings and patients symptoms) and risk assessment scores [8] like: the TIMI (Thrombolysis In Myocardial Infarction) risk score, the PURSUIT (Platelet Glycoprotein IIb/IIa in Unstable Angina: Receptor Suppression Using Integrilin Therapy) risk score and the GRACE (Global Registry of Acute Coronary Events) risk score.

Following are the treatment options available based on predicted risk [8]:

- Low risk prediction: (GRACE: <109, TIMI<2): If hemodynamically stable are given intensive medication and are re-evaluated.
- High risk (GRACE: >140, TIMI >5) Angiography followed by revascularization within 12 h of symptoms onset.

III. TECHNIQUE

A. Access

The technique for performing PCI depends on its primary access. The usual access sites are via the femoral or radial artery. Less commonly the brachial or ulnar artery may be used. In the United States, the femoral artery is the most commonly used, although the use of radial route is increasing in both US and Europe, mainly due to its better outcomes in mortality and complication rates [9].

B. Procedure

For trans-radial approach: Radial styloid landmark is marked, an arteriotomy is made 2 cm proximal to it. Using the modified Seldinger technique, the radial artery is pierced with a micro puncture needle, and a hydrophilic sheath is installed [10]. After placing the sheath, a vasodilator is given intra-arterially (verapamil 5 mg or Nicardipine 500 µg), half of the dose given at the beginning and the rest after the procedure. Intravenous heparin (50 units/kg) is often used in combination [10].

For trans-femoral catheterization: The access site is common femoral artery, above its bifurcation and below the inferior epigastric artery. A micro puncture needle is used along with a sheath according to the modified Seldinger technique [10].

After placing the sheath, under fluoroscopic guidance, a guide wire is used to lead the catheters into the ascending aorta. After reaching the coronary openings in the sinuses and obtaining angiograms, IV anticoagulation is administered (based on weight) in order to reduce acute, subacute and long-term post-procedural ischemic events related to plaque rupture and coagulation system activation. All patients should take aspirin before the procedure [10].

The guide wire is advanced across the stenotic lesion in the selected coronary artery. All devices will be guided by this wire.

C. Pre-dilation

Balloon angioplasty is vessel dilation, it could be without stent placement.In which case the catheter is removed afterwards followed by closure of the access site.
The technique is also employed in pre-dilation before stent placement. Direct stenting without pre-dilation may be done in cases with easy access for stent placement. If there’s indication that the stenosis cannot be dilated, alternatives such as cutting balloon and atherectomy etc. may be used. Situations presenting a hindrance to dilation include moderate to severe coronary calcification, heavy plaque, or diffuse coronary artery disease.

D. Direct Stenting

Indications for direct stenting without pre-dilation, include: 1) vessel diameter (>2.5 mm), 2) proximal lesion location, 3) absence of severe coronary calcification, 4) absence of significant angulation with a bend >45º, 5) absence of very severe lesions and bifurcation lesions, and 6) ST-elevation MI [11].

The standard strategy of starting with pre-dilation, followed by stent deployment, and ending with high-pressure post-dilation are believed to increase the time taken for the procedure, as well as increased radiation and higher costs.

E. Post-dilation

Balloon dilation performed after stent placement, to improve stent expansion. High-pressure balloon dilation is thought to be the most optimal, as studies show that it produces better stent expansion and apposition [12].

Aggressive mechanical expansion could lead to adverse effects such as distal embolization, microvascular injury and more intimal hyperplasia.

F. Imaging

Angiography is the current gold standard for imaging during PCI but has significant limitations such as inaccurate vessel diameter calculation, missing benign plaques [13].

New Catheter based intravascular imaging techniques such as intravascular ultrasound (IVUS) and optical coherence tomography (OCT) are being used nowadays. OCT utilizes infrared light, has better spatial resolution allowing better view of superficial surface of vessel wall, but the penetration isn’t as good as IVUS. IVUS on the other hand is ultrasound based, and has poorer resolution but can map the whole thickness of the vessel wall [13].

G. Post Procedure

In trans-radial approach the sheath is removed right after finishing the procedure, and a compression band is applied, which is left inflated for 90-120 minutes.

In trans-femoral catheterization, hemostasis is achieved by either manually compressing or by using vascular closure devices [10].

IV. CONTRAINDICATIONS

A. Absolute

Intolerance of long-term antiplatelet therapy, High risk for bleeding (thrombocytopenia, peptic ulcers, severe coagulopathy) and multiple restenosis post-PCI [1].

B. Relative

Vessel diameter less than 1.5 mm [1], diffuse disease of artery or vein [7], lack of onsite surgery backup, hypercoagulability, total occlusion of SVG, Kidney disease, critical left main blockage with no collateral flow and other hindrances in the coronary circuit [7].

V. ADJUVANT THERAPIES

A. Atherectomy

It is performed to de-bulk the atherosclerotic plaque from the affected vessel. Atherectomy has proved to be successful in managing disease of both coronary and peripheral vessels [14]:

• Directional atherectomy device

The device is a carbide cutting blade and a nose cone. A few devices are designed to cut through severe calcification. Due to high embolization potential, a distal embolic protection filter is most often used.

• Rotational and orbital atherectomy

The device uses an excimer laser to ablate soft plaque and thrombus, with minimal injury to the surrounding structures. It is not effective for calcified lesions [14].

B. Fibrinolytic Therapy

Results from clinical studies show a decrease in occurrence of stroke, recurrent ischemia, and repeat target vessel revascularization (TVR) and a lower absolute risk of death with coronary angioplasty as compared to fibrinolysis [15]. Fibrinolytic therapy is used in eligible patients if primary PCI is inaccessible. Thrombolysis may be followed by PCI:

• Facilitated PCI: Performing PCI right after medical therapy (fibrinolysis, platelet glycoprotein IIb/IIIa inhibitors, or both).

• Rescue PCI: PCI after failing of fibrinolytic therapy (defined as failure to achieve 50% ST-segment resolution within 60 minutes, following fibrinolytic therapy).

C. Thrombectomy

Catheters with a central cavity are used which are able to aspirate the thrombus, thus extracting it. Two types of mechanical devices are used in this: Manual and Rheolytic, manual ones aspirate the thrombus via a syringe, while Rheolytic devices break the thrombus into fragments before aspiration [16].

D. Antiplatelet Therapy

Aspirin and P2Y12 inhibitors combined, Dual antiplatelet therapy (DAPT), appears to reduce numbers of severe cardiac events in CAD patients [17]. 2016 ACC (American College of Cardiology) guidelines recommend long-term antiplatelet therapy CAD patients, regardless of them being treated with PCI, medical therapy alone, fibrinolytic therapy (FT) or coronary artery bypass graft (CABG).

About 1 month of treatment with a P2Y12 inhibitor is advised in patients with a bare metal stent (BMS) for Coronary Artery Diseases. Six and twelve months course for those with a drug-eluting stent (DES) for SIIHD and ACS, DOI: http://dx.doi.org/10.24018/ejmed.2022.4.4.1433
respectively. In patients at high risk for bleeding early discontinuation may be advised [17].

E. Statin Therapy

Statin therapy is known for better outcomes when used in medical treatment for stable and unstable coronary disease. Statins before PCI has demonstrated a lower incidence of peri-procedural MI. The outcomes could be attributed to the plaque stabilizing and even regressing ability of the statins [18].

VI. PERIPROCEDURAL COMPLICATIONS:

Mostly complications in PCI are involving the coronary circuit catheterization and angiography. The main complications are as follows:

- **Perforation**
  Coronary artery perforations are common [19] and are graded on the basis of Ellis grading. The use of athero ablative devices and IVUS have been linked to an increased risk of perforation.

- **Peri-Procedural MI**
  Accompanied elevated enzymes and significant ECG findings [19]. Could cause significant.

- **Retroperitoneal bleeding**
  It is a rare occurrence after cardiac catheterization, mostly in cases of arterial puncture above the inguinal ligament, when femoral access is used for the procedure. It can be prevented in most cases by carefully tracing landmarks.

- **Intramural hematoma**
  Collection of blood in the tunica media of the vessel. Higher incidence in PCI involving balloon angioplasty, compared to stenting.

- **Distal embolization**
  Embolization of plaques leading to microvascular obstructions and end organ ischemia.

- **Side branch Occlusion**
  Stent placements across a major side branch may cause its occlusion.

- **Arrhythmias**
  It may occur during the catheterization and range from Ventricular and atrial fibrillation to bradycardia and conduction abnormalities. The type may be affected by site of catheterization and predisposing factors like spesis, MI, pre-existing cardiac abnormalities and guide wire. Arrhythmias causing hemodynamic instability require immediate intervention [20].

- **Allergic reactions**
  Hypersensitivity reactions can occur to the radiocontrast, the local anesthetic and/or the anticoagulant (heparin). Precautions such as preservative free anesthetic, non-ionic contrast and alternative anticoagulants may benefit.

- **Acute renal failure (ARF)**
  Renal failure during PCI can occur due to multiple etiologies [21].

- **Radiocontrast induced ARF**
  It is mostly seen with iodinated radiocontrast use and in diabetic and chronic renal disease patients. Patients' serum creatinine should be monitored to keep a check on their renal status. Iso-osmolar nonionic agents are preferred for at risk patients.

  - **Renal atheroembolism induced ARF**
    Atheroembolism is a very important cause of renal insufficiency in PCI patients, as kidneys are one of the major organs at risk of embolism leading to ischemic injury.

  - **Hemodynamic instability induced ARF**
    Due to hypo-perfusion of the kidneys leading to renal ischemia and acute tubular injury. [21]

  - **Infections**
    Infections in PCI patients is a rare occurrence nowadays, owing to better hygiene precautions, but is still quite a serious one. Infections can effect procedure outcome ranging from longer hospital stay to major adverse cardiovascular events (MACE).

  - **Radiation effects**
    PCI exposes the patients to a significant amount of radiation, which could result in adverse effects like cataract formation or malignant transformation. Measures have been taken to limit this exposure by decreasing frame rate and improving fluoroscopic image storage, both of which have shown to significantly reduce the radiation dose [22].

  - **Access site bleed**
    More common with femoral approach than the radial approach. But major adverse events are more or less the same [9].

Stenting has its own share problems:

- **Failure of deployment**
  higher incidence initially with first generation stents, newer generation stents show better placement success.

- **Stent jail**
  It refers to situations where a side branch is compromised due to the positioning of the stent across a major coronary vessel [23].

- **Stent Thrombosis**
  This could be acute, subacute or late, but is a major complication and could lead to MI and death [23]. BMS showed high rates of restenosis and so Drug Eluting Stents (DES) were made.

- **Stent infections**
  Mostly involves staphylococcus and pseudomonas infections, and more so with DES stents. Infections can prove to be very much fatal and thus are a very important complication.

Many of these complications may eventually lead to MI. Most of these can be addressed within the catheterization lab, but some may progress enough to necessitate CABG and open heart surgery. Access to onsite cardiac surgery is thought to be one of the factors contributing to a decrease in mortality in PCI cases with complications, but this is still a theory under debate.

VII. DEVICES AND AIDS

A. Stents

There are 2 types of stents: Bare metal stents (BMS) or Drug-eluting stents (DES).

BMS are the original stents, which revolutionized coronary intervention procedures in the late 1990s, these are made of metals such as stainless steel, cobalt chromium or platinum.
chromium. BMS decreased the rates of restenosis and abrupt closure compared to balloon angioplasty, and thus decreased the rate of target lesion revascularization (TLR). Endothelial stent coverage of bare metal stents completes by 12 weeks and decreases stent thrombosis risk significantly [24].

The more recently developed DES have led to better outcomes following PCI, such as less numbers of TLRs and restenosis [1]. DES comprises a polymer coated framework which elutes an anti-proliferative drug into the coronary wall, thus reducing rates of restenosis and stent thrombosis. First-generation DES are sirolimus-eluting and paclitaxel-eluting stents. The second-generation DES contain everolimus or zotarolimus, and these are associated with a reduced restenosis and stent thrombosis compared to their predecessors [10].

DES are preferred over BMS in most PCI settings in light of their more favorable outcomes. Chronic total occlusion, bifurcation stenosis, diabetes, Post heart transplant vasculopathy, calcified lesions and saphenous vein graft are some of the indications for DES usage. However, BMS are still used, especially in patients with high bleeding risk, have large arteries, and those who can’t bare DAPT for a whole year or require a surgery in the next 12 months [10].

B. Balloon Catheters

It’s a flexible tube with a small balloon attached to its one end. The balloon is inflated once the catheter reaches the site of stenosis. It is actually the inflation of the balloon that results in stretching of the vessel wall, including the media and the adventitia, and disruption of the intimal plaque resulting in enlargement of the lumen of the vessel. There are many different balloon catheters (fixed wire, monorail etc.), and use different balloon materials with varying compliance, effecting degrees of expansion with increasing pressure. The latest catheters have reduced diameter of the deflated balloon which allows easier passage through the stenosed vessels [1].

C. Closure Devices

To control the little bleeding at the access point at the end of the procedure Manual compression for 15-20 minutes followed by staying still for 4-6 h, has been the gold standard. But it is time consuming and uncomfortable. Vascular closure devices are an alternate approach to manual compression. The devices available fall into two categories, passive closure devices and active closure devices [25].

- Passive devices

Stop the bleed by causing clot formation or mechanical pressure. However, these aren’t very time efficient and the patient may still have to stay immobile for a while, as well as with mechanical compression.

- Active devices

They use different techniques like collagen plugs, suture-based products and clips to directly close the entry site. This closes the access site but because it has a part of the device remaining in the artery, complications like hematoma, pseudoaneurysms, infections and arteriovenous fistulas can occur.

Vascular closure devices cause less pain and discomfort, compared to manual compression. They are time saving meaning early rehabilitation and shorter hospital stay, thus being cost efficient. They prove to be the better option for older and unhealthy individuals who find it difficult to lie flat on their backs. Complications are rare but do occur, like device failure leading to uncontrolled bleeding making one revert to manual compression. Risk of infection is always present, although a small one, as well as a risk of hematoma or aneurysm formation [25].

D. Distal Embolic Protection Devices

Manipulating an atherosclerotic lesion causes displacement of thrombotic debris and could lead to distal embolism. Embolic protection devices (EPDs) prevent or reduce plaque debris reaching the distal capillary beds and potentially reduce adverse clinical events i.e., Cerebral stroke. [26].

VIII. ALTERNATIVES TO PCI

A. Coronary Artery Bypass Graft (CABG)

ACC guidelines for choosing CABG over PCI are as follows [27]:

- More than 50% blockage of left main coronary artery.
- LAD and LCX arteries with >70% stenosis.
- Mild or stable angina in individuals with 3 vessel disease.
- Proximal LAD stenosis with three vessel disease accompanied with poor left ventricular function.
- Stable angina patient with 1 or 2 vessel disease and significant viable myocardium in high risk area, Syntax score > 33.
- LAD blockage of >70% accompanied by an ejection fraction (EF) of <50%.
- Vessel anatomy and its diameter are also a limit factor for stent usage. Tortuous and calcified arterial segments also present a challenging situation [10].

Syntax score is an angiographic risk assessment tool, which helps determine the complexity of coronary artery disease. It is used to decide the revascularization approach between PCI or CABG. The scale takes into account lesion characteristics like bifurcations, chronic total occlusions, thrombus, calcification, and small diffuse disease and allocates a score ranges from 0 to greater than 60 in very complex coronary anatomy lesions. PCI is recommended if there’s a significant stenosis in unprotected CAD with anatomic conditions associated with low risk of complications and high likelihood of favorable outcomes: Low Syntax score (22 or less) with clinical features that increase risk of operative complications i.e. COPD, myocardial. A syntax score >33 is taken as an indication for CABG [27].

IX. FACTORS AFFECTING OUTCOMES

A. Age-based Outcomes

Elderly patients (age > 65) - more frequently present with atypical features of acute coronary syndrome and may be overlooked as appropriate candidates for PCI. Additionally, they tend to report late to emergency services, which may place these patients beyond the indicated window to treat with PCI [28] One year survival after PCI only decreases after 65 years of age, and declining more drastically in patients over 85 years old [28]. Risk factors common to patients above the age of 65 years included hypertension, diabetes, dyslipidemia
and previous myocardial infarction, indicating an increasing need for preventive measures aimed for this population.

Young patients (Age < 65) have shown good prognosis following PCI, with low mortality and adverse cerebral as well as cardiovascular events. Younger patients were more likely to experience nonfatal re-infarction [29].

Work by Turk et al. shows that the incidence of in-hospital mortality significantly increased with increasing age [29]:

<table>
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<tr>
<th>Table I: In-Hospital Mortality According to Age</th>
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<tr>
<td>Age group</td>
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<tr>
<td>&lt;60 years old</td>
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<tr>
<td>&gt;60 to &lt;70 years old</td>
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<td>&gt;70 to &lt;80 years old</td>
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<td>&gt;85 years old</td>
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Mortality rates within 12 hours of first medical contact were higher with advancing age (P < 0.001), as well as older patients are prone to stay longer in hospital.

B. Gender-based Outcomes

Gender is listed as one of the factors influencing the prognosis post-PCI. Literature indicates that women tend to have worse prognosis after PCI [30]. However, it is not an independent predictor of death post-procedure.

In a 30 year cohort study conducted by [31] found no difference between men and women mortality post-PCI in general. However, in-hospital mortality was higher in women than men.

Similarly, a study by [30] states that procedural success rates, MI and death rates were comparable by sex in young and older age groups during in-hospital stay. However, at 1 year follow-up, young women had higher incidence of MACE compared with young men. The higher MACE rate in women was attributable to higher rates of CABG [8.9% versus 3.9%; P<0.001] and repeat PCI [19.0% versus 13.0%; P=0.005], therefore, young women were at a higher risk of repeat revascularizations compared with men of similar age [30].

In the study conducted by [32] the first differences were noted on the baseline characteristics. Women were more likely to be older, have more cardiovascular risks such as hypertension, obesity and diabetes, and have a history of heart failure. Men were more likely to be smokers, have received thrombolysis for recent MI, have a family history of coronary disease and have more vessels with obstructive coronary artery disease.

Complications related to PCI, in-hospital mortality and all-cause mortality at 30 days and one year were significantly lower in men.

Female sex and patient’s age at the time of the intervention were separately significant predictors of mortality when using multiple regression analysis, however there was no statistically significant interaction between age and gender for all-cause mortality at 30 days and at one year [32].

C. Smoking

Smoking has been linked to a higher incidence of CAD. But studies have observed recent smokers to have a better post STEMI outcome than the nonsmokers, this has been termed as the ‘smoker’s paradox’ [33].

The truth is that as smoking accelerates atherosclerosis, so smokers with CAD present earlier in life and thus have better outcomes compared to Non-smoker CAD patients, who are ipso facto older. Once we count these factors out, the smokers are at a significantly higher risk for re-infarction, all-cause mortality and hospitalization [33].

X. EMERGENCY CABG IN PCI PATIENTS

In some cases PCI may fail and the practitioner may have to opt for CABG surgery. Indications for choosing CABG over PCI include the following:

- Hemodynamic compromise, ongoing ischemia or threatened occlusion with significant myocardium at risk, coronary rupture and failed stent deployment [34].
- ACC’s guidelines classify indications for emergency CABG after failed PCI as following [34]:
  - Failed PCI with continuous ischemia and significant myocardium at risk due to high risk occlusion,
  - Patients with or without impaired coagulation and sternotomy, presenting with hemodynamic compromise.
  - Foreign body (broken guidewires or stents) retrieval from a risky location.
- With widespread stent use and effective antiplatelet therapies, emergency CABG after failed PCI is a rare occurring.

REFERENCES


