INTRODUCTION

Acute coronary syndromes (ACS) with and without ST-segment elevation are most commonly caused by rupture of an atherosclerotic plaque, leading to thrombin generation, platelet activation, and thrombus formation.[1]

Although there have been improvements in outcome in recent years, these patients remain at high risk for ischemic events, both early during the initial hospitalization and long term.[2]

In patients with ACS, major bleeding is as common as recurrent myocardial infarction and occurs in about 5% of patients. A substantial proportion of the bleeding occurs at the vascular access site.[3,4]

More recently, there has been increasing awareness that bleeding is associated with an increased risk of adverse outcomes, including MI, stroke, and death.[5]

In 1948, Radner[6] first described transradial catheterisation using radial artery cut-down. In 1989, Campeau revisited Radner's idea and reported on percutaneous entry into the distal radial artery for selective coronary angiography in 100 patients.[7,8]

The main complications of femoral artery access are hematoma, arteriovenous fistula, arterial pseudoaneurysm, and

retroperitoneal hemorrhage. These complications are responsible for most of the bleeding that occurs in invasive procedures, especially in ACS & they are influenced by anatomic features, obesity, and puncture technique.[9]

The transfemoral approach (TFA) has been until presently the main-stay for arterial access PCI in the setting of acute STEMI, while the transradial approach (TRA) is gaining ground in elective as well as primary procedures.[10]

Aim of the Work

To assess the impact of transradial versus transfemoral approach for PCI on the outcome of patients presenting with acute coronary syndrome.

Chapter 1
ACUTE CORONARY SYNDROMES

Background & Pathogenesis:

Acute coronary syndromes are a group of conditions precipitated by a reduction or cessation of blood flow through the coronary arteries. An acute coronary syndrome (ACS) is a medical emergency requiring immediate intervention. ACS encompasses any narrowing or obstruction of the coronary arteries leading to acute symptoms.

Acute coronary syndromes include: [11]

- ST elevation myocardial infarction (STEMI)
- Non ST elevation myocardial infarction (NSTEMI)
- Unstable angina (UA)

ACS represents a life-threatening manifestation of atherosclerosis. It is usually precipitated by acute thrombosis induced by a ruptured or eroded atherosclerotic coronary plaque, with or without concomitant vasoconstriction, causing a sudden and critical reduction in blood flow. [12]

In the complex process of plaque disruption, inflammation was revealed as a key pathophysiological element. In rare cases, ACS may have a non-atherosclerotic etiology such as arteritis, trauma, dissection, thrombo-embolism, congenital anomalies, cocaine abuse, or complications of cardiac catheterization. The

key pathophysiological concepts such as vulnerable plaque, coronary thrombosis, vulnerable patient, endothelial dysfunction, accelerated atherothrombosis, secondary mechanisms of NSTE-ACS, and myocardial injury have to be understood for the correct use of the available therapeutic strategies. [12]

The lesions predicting ACS are usually angiographically mild, characterized by a thin-cap fibroatheroma, by a large plaque burden, or by a small luminal area, or some combination of these characteristics. [12]

Definitions:

According to the universal definition of myocardial infarction (MI), acute MI defines cardiomyocyte necrosis in a clinical setting consistent with acute myocardial ischaemia. Under these conditions, a combination of criteria is required to meet the diagnosis of acute MI, namely the detection of a rise and/or fall of a cardiac biomarker, preferably high-sensitivity cardiac troponin, with at least one value above the 99th percentile of the upper reference limit, and at least one of the following: [13]

a) Symptoms of ischaemia.

b) New or presumed new significant ST-T wave changes or left bundle branch block on 12-lead ECG.

c) Development of pathological Q waves on ECG.

d) Imaging evidence of new or presumed new loss of viable myocardium or regional wall motion abnormality.

e) Intracoronary thrombus detected on angiography or autopsy.

Two main forms of acute MI should be considered:

Type 1 MI: characterized by atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection with resulting intraluminal thrombus in one or more coronary arteries leading to decreased myocardial blood flow and/or distal embolization and subsequent myocardial necrosis. The patient may have underlying severe coronary artery disease (CAD) but, on occasion (i.e., 5% to 20% of cases), non-obstructive coronary atherosclerosis or no CAD, particularly in women. [13,14]

Type 2 MI: instances of myocardial necrosis in which a condition other than unstable coronary plaque contributes to an imbalance between myocardial oxygen supply and demand.[3] Mechanisms include coronary artery spasm, coronary embolism, coronary endothelial dysfunction, tachy-/bradyarrhythmias, anaemia, respiratory failure, hypotension, and severe hypertension. In addition, in critically ill patients and in patients undergoing major non-cardiac surgery, myocardial necrosis may be related to injurious effects of pharmacologic agents and toxins. [15]

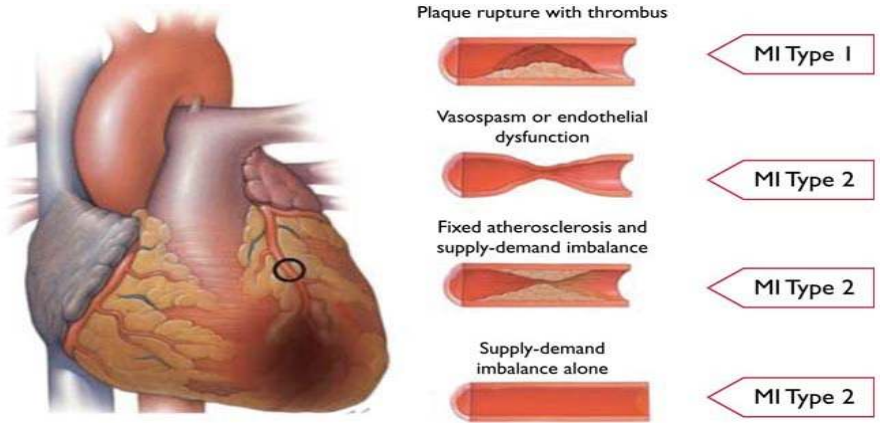

Figure (1): The differentiation between MI types 1 and 2 according to the condition of the coronary arteries. [13]

The leading symptom that initiates the diagnostic and therapeutic cascade is chest pain, but the classification of patients is based on the electrocardiogram (ECG).

Two categories of patients may be encountered: [16]

1. **Patients with acute chest pain and persistent (>20 min) ST-segment elevation:** This is termed ST-elevation ACS (STE-ACS) and generally reflects an acute total coronary occlusion. Most of these patients will ultimately develop an ST-elevation MI (STEMI). The therapeutic objective is to achieve rapid, complete, and sustained reperfusion by primary angioplasty or fibrinolytic therapy.
2. **Patients with acute chest pain but without persistent ST-segment elevation:** These patients have rather persistent or transient ST-segment depression or T-wave inversion, flat T waves, pseudo-normalization of T waves, or no ECG

changes at presentation. The initial strategy in these patients is to alleviate ischaemia and symptoms, to monitor the patient with serial ECGs, and to repeat measurements of markers of myocardial necrosis. At presentation, the working diagnosis of non-ST-elevation ACS (NSTE-ACS), based on the measurement of troponins, will be further qualified as non-ST-elevation MI (NSTEMI) or unstable angina (UA). In a certain number of patients, coronary heart disease will subsequently be excluded as the cause of symptoms.

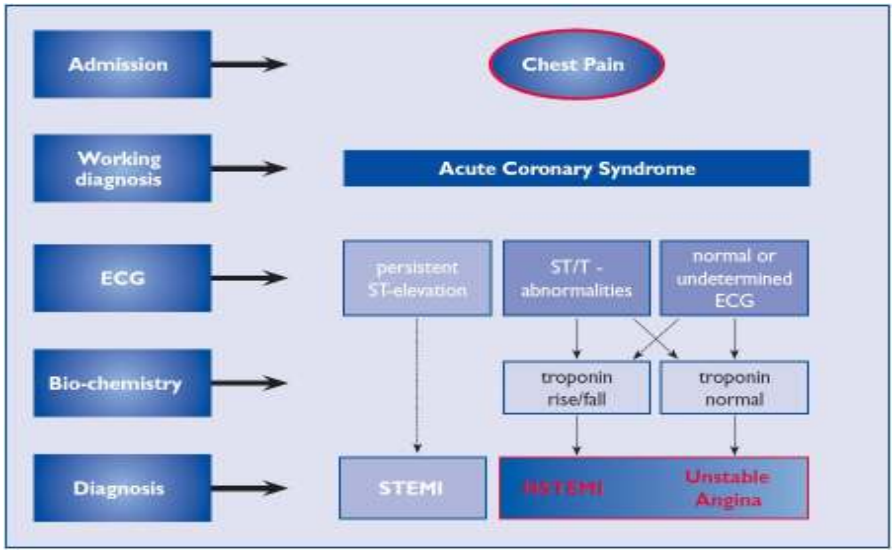

Figure (2): The spectrum of ACS. [16]

Initial diagnosis of STEMI:

A working diagnosis of myocardial infarction must first be made. This is usually based on a history of chest pain lasting

for 20 min or more, not responding to nitroglycerine. Important clues are a history of CAD and radiation of the pain to the neck, lower jaw or left arm. The pain may not be severe. Some patients present with less-typical symptoms, such as nausea/vomiting, shortness of breath, fatigue, palpitations or syncope. These patients tend to present later, are more likely to be women, diabetic or elderly patients. [17]

Registries show that up to 30% of patients with STEMI present with atypical symptoms. Awareness of these atypical presentations and a liberal access to acute angiography for early diagnosis might improve outcomes in this high-risk group. [18]

Timely diagnosis of STEMI is key to successful management. ECG monitoring should be initiated as soon as possible in all patients with suspected STEMI to detect life-threatening arrhythmias and allow prompt defibrillation if indicated. A 12-lead ECG should be obtained and interpreted as soon as possible at the point of FMC (first medical contact). [18]

Typically, ST-segment elevation in acute myocardial infarction, measured at the J point, should be found in two contiguous leads and be ≥0.25 mV in men below the age of 40 years, ≥0.2 mV in men over the age of 40 years, or ≥0.15 mV in women in leads V2–V3 and/or ≥0.1 mV in other leads (in the absence of left ventricular (LV) hypertrophy or left bundle branch block (LBBB). [13,19]

In patients with inferior myocardial infarction, it is advisable to record right precordial leads (V3R and V4R) seeking ST elevation, in order to identify concomitant right ventricular infarction. Likewise, ST-segment depression in leads V1–V3 suggests myocardial ischemia, especially when the terminal T-wave is positive (ST-elevation equivalent), and may be confirmed by concomitant ST elevation ≥0.1 mV recorded in leads V7–V9. [19]

The presence of ST-depression >0.1 mV in 8 or more surface leads, coupled with ST elevation in aVR and/or V1 but an otherwise unremarkable ECG, suggests ischemia due to multivessel or left main coronary artery obstruction, particularly if the patient presents with haemodynamic compromise. [20]

Blood sampling for serum markers is routinely carried out in the acute phase but one should not wait for the results before initiating reperfusion treatment. Troponin (T or I) is the biomarker of choice, given its high sensitivity and specificity for myocardial necrosis. [21]

Table (1): Recommendations for initial diagnosis of STEMI. [21]

Recommendations	Class [a]	Level [b]
A 12-lead ECG must be obtained as soon as possible at the point of FMC, with a target delay of ≤10 min.	I	B
ECG monitoring must be initiated as soon as possible in all patients with suspected STEMI.	I	B
Blood sampling for serum markers is recommended routinely in the acute phase but one should not wait for the results before initiating reperfusion treatment.	I	C
The use of additional posterior chest wall leads (V_7–V_9 ≥0.05 mV) in patients with high suspicion of infero-basal myocardial infarction (circumflex occlusion) should be considered.	IIa	C

Logistics of care for patients with STEMI:

Prevention of delays is critical in STEMI as the most critical time of an acute myocardial infarction is the very early phase, during which the patient is often in severe pain and liable to cardiac arrest. [22]

In addition, early provision of therapy, particularly reperfusion therapy is critical to its benefit. Thus, minimizing delays is associated with improved outcomes. [22]

Revascularization in STEMI:

There are several components of delay in STEMI: [23]

- **Patient delay:** that is, the delay between symptom onset and FMC. To minimize patient delay, the public should be made aware of how to recognize common symptoms of acute myocardial infarction and to call the emergency services.
- **Delay between FMC and diagnosis:** a good index of the quality of care is the time taken to record the first ECG. the goal should be to reduce this delay to 10 min or less.
- **Delay between FMC and reperfusion therapy:** This is the 'system delay'. It is more readily modifiable by organizational measures than patient delay. It is an indicator of quality of care and a predictor of outcomes.

Reperfusion strategies:

If the reperfusion therapy is primary PCI, the goal should be a delay (FMC to wire passage into the culprit artery) of ≤90 min and in high-risk cases with large anterior infarcts and early presenters within 2 h, it should be ≤60 min. If the reperfusion therapy is fibrinolysis, the goal is to reduce this delay (FMC to needle) to ≤30 min. [24,25]

In PCI-capable hospitals, the goal should be to achieve a 'door-to-balloon' delay ≤60 min between presentation in the hospital and primary PCI (defined as wire passage into the culprit artery). This delay reflects the organization and performance of the PCI-capable hospital. [24,25]

From the patient's perspective, the delay between symptom onset and provision of reperfusion therapy (either starting fibrinolysis or passing a wire through the culprit vessel) is possibly the most important, since it reflects total ischemic time. It should be reduced as much as possible. [24,25]

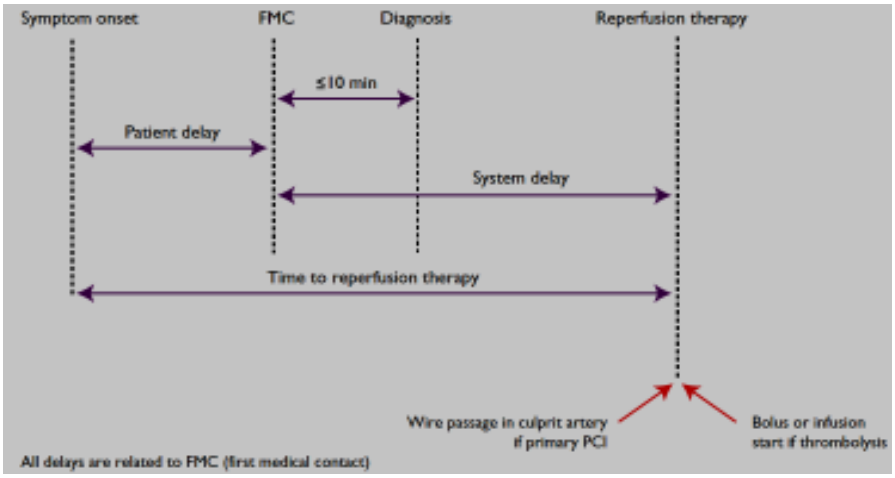

Figure (3): Components of delay in STEMI & ideal time intervals for intervention. [21]

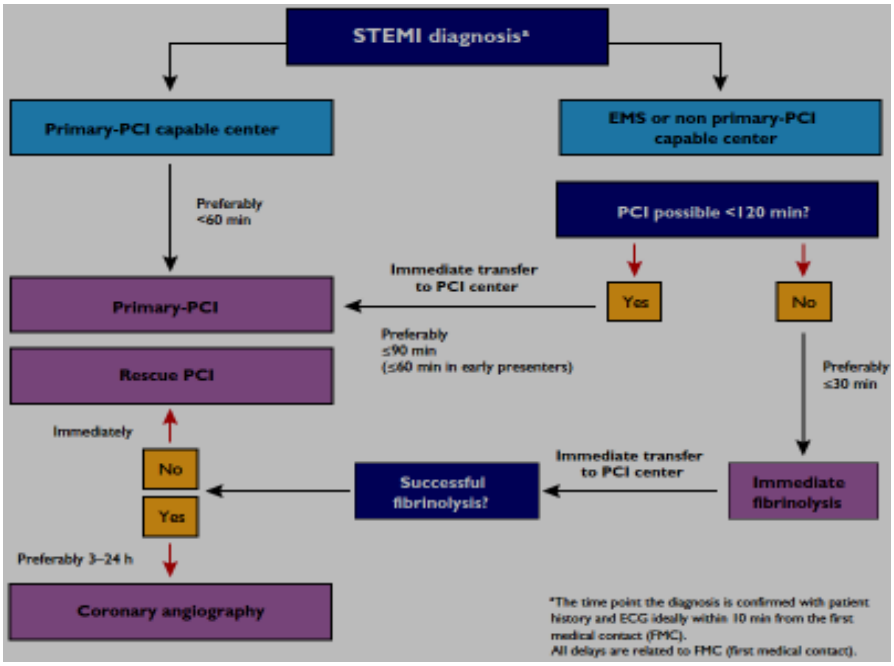

Figure (4): Prehospital, in-hospital management, & reperfusion strategies within 24 h of FMC. [26]

Indications of Primary PCI in STEMI:

1- Reperfusion therapy is indicated in all patients with time from symptom onset <12 hours duration and persistent ST segment elevation or (presumed) new LBBB (Class I A). [27,28]

2- Primary PCI is the recommended reperfusion therapy over fibrinolysis if performed by an experienced team in a timely fashion (Class I A). [29,30]

3- In patients with time from symptom onset >12 hours, primary PCI is indicated in the presence of continuing ischaemia, life-threatening arrhythmias or if pain and ECG changes have been stuttering (Class I C). [31]

4- Primary PCI is indicated for patients with severe acute heart failure or cardiogenic shock due to STEMI independent from time delay of symptom onset (Class I B). [32]

5- Reperfusion therapy with primary PCI should be considered in patients presenting late (12–48 hours) after symptom onset (Class IIa B). [33,34]

In settings where primary PCI cannot be performed in a timely fashion, fibrinolysis should be considered, particularly if it can be administered pre-hospital (e.g. in the ambulance) [35] and within the first 120 minutes after symptom onset. It should be followed by transfer to PCI-capable centres for routine coronary angiography in all patients (preferably within 3-24 hrs) and for rescue PCI in case of unsuccessful fibrinolysis (as soon as possible). [36,37]

Important points in optimizing & guiding primary PCI (strategies & techniques):

The infarct-related artery (culprit vessel) should be treated during the initial intervention. Evidence supporting immediate (preventive) intervention in non-infarct-related lesions is a matter of debate and may be considered in selected patients (Class IIb B). [38,39]

Multivessel PCI during STEMI should be considered in patients with cardiogenic shock in the presence of multiple, critical stenoses or highly unstable lesions (angiographic signs