



Acute Coronary Syndrome

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mOh

ISCHEMIC HEART DISEASE (IHD)

IHD : imbalance between blood supply by the coronaries
.and oxygen requirements of myocardium

:CAUSES

Atherosclerosis : the most common cause (plaque .1
.formation started from birth)

.inflammation .2

.vasospasm .3

.thrombosis, and embolic .4

.congenital anomaly of the coronaries .5

CLINICAL PRESENTATION OF IHD

.SUDDEN CARDIAC DEATH .1

.EFFORT(STABLE ANGINA) .2

.UNSTABLE ANGINA.3

.NONSTEMI(NON ST ELEVATION MI) .4

.STEMI .5

.ISCHEMIC HF (ISCHEMIC CMP).6

.ISCHEMIC ARRHYTHMIA(AF ,VT ,VF).7

.Risk factors for IHD

:Modifiable

- Family history.
- Age (male>45
- Female >55)
- sex, and race

:Non modifiable

- Diabetes (IHD EQUIVALENT).
- HTN (causes endothelial dysfunction, increases collagen).
- SMOKING increasing plt activity, and catecholamine level, alters PG.s, and decrease HDL.
- DYSLIPIDEMIA.
- OBESITY.
- SEDENTARY LIFESTYLE
- NONTRADITIONAL RISK FACTORS: high lipoprotein A, high iron level,
- hyperhomocystenemia.

ACUTE CORONARY SYNDROME

- ACS– umbrella (temporary term) , including pts, presenting to RR with typical anginal pain with or without ECG changes.
- Spectrum of clinical presentation , ranging from unstable angina, STEMI, NONSTEMI, and sudden cardiac death.

● ACS includes :

- sudden cardiac death
- STEMI
- Non STEMI
- Unstable angina.

Pathophysiology

Mostly ,due to rupture of an atherosclerotic plaque,and partial or complete thrombosis of the infarct related artery, or increasing demand of myocardium

Vulnerable plaque(large lipid pool,rich with inflammatory cells, and thin fibrous cap)

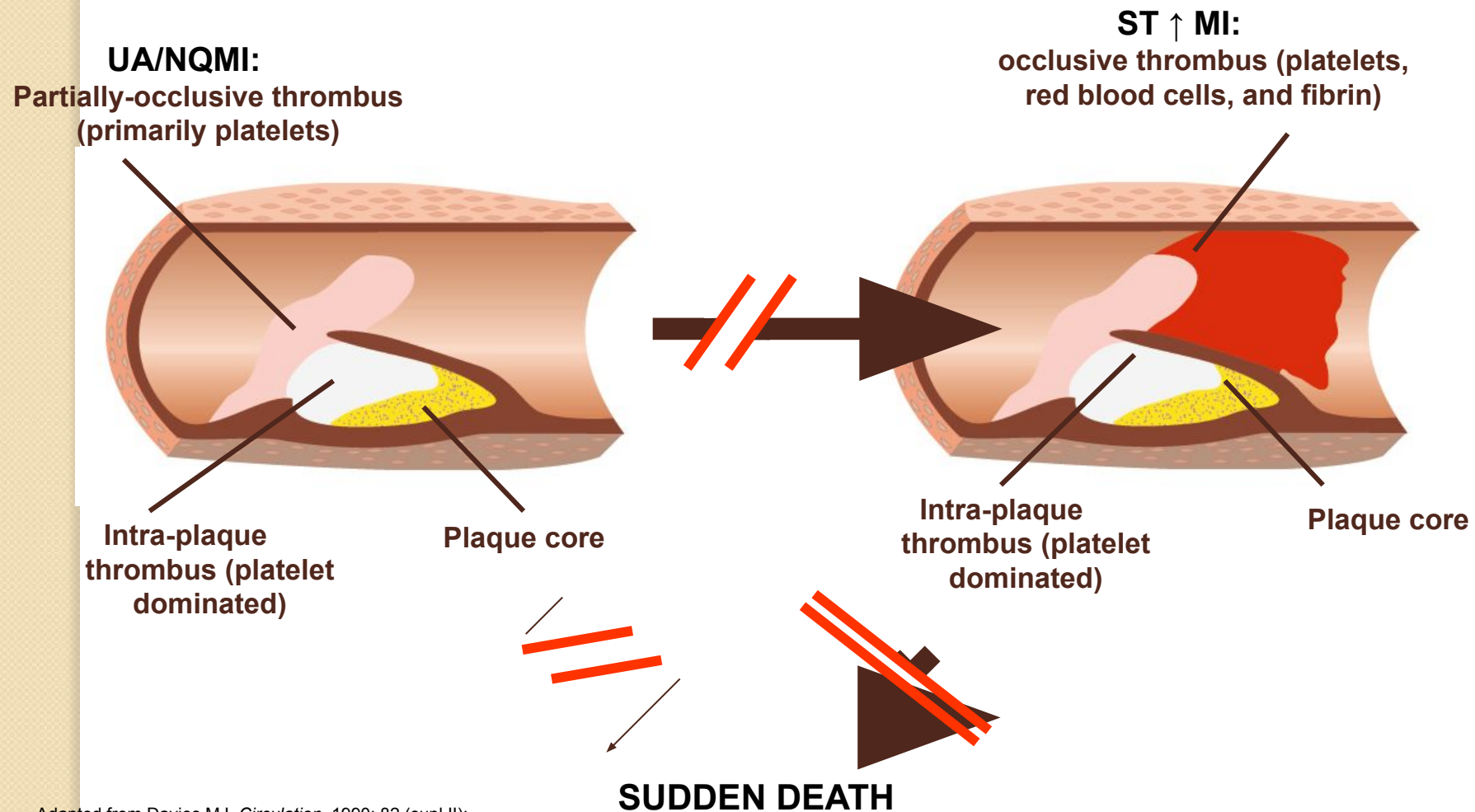
After any injury Inflammatory cells release metalloproteinase's(collagenases)wich lead to plaque rupture

Platelet activation and aggregation, activation of the coagulation system, decrease in NO release and vasoconstriction

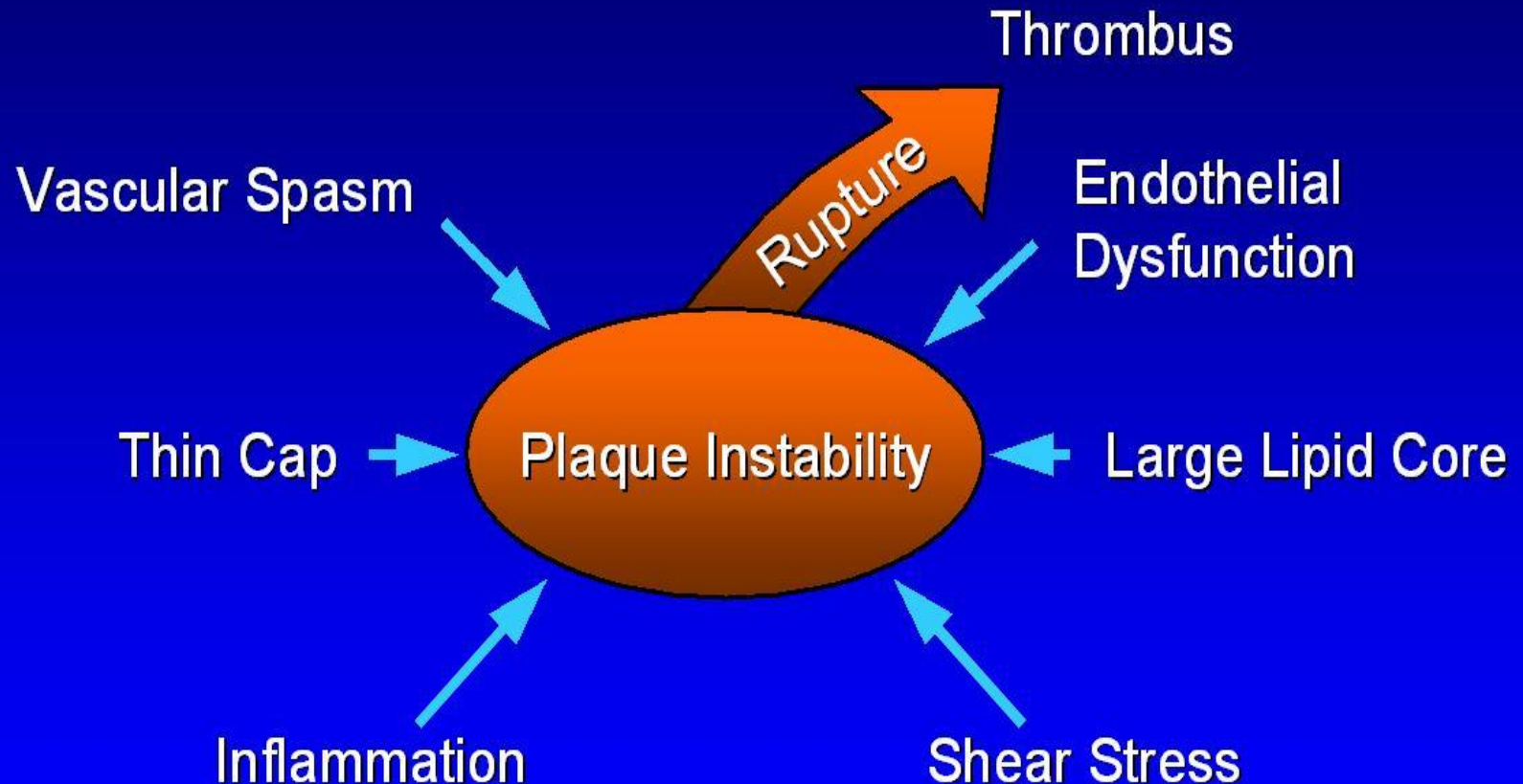
This lead to coronary intraluminal thrombosis and distal embolization

.Infectious theory

Clinical manifestations of arterial thrombosis



Acute Coronary Syndromes: Summary



Fuster et al. *N Engl J Med*. 1992;326:242-250.

Thérroux et al. *Circulation*. 1998;97:1195-1206.

CLINICAL PRESENTATION

:Symptoms include

angina:burning chest pain, pressure or tightness,.1

.lasts several minutes

.Pain in the arm or jaw.2

.nausea, and vomiting.3

.dyspnea.4

.diaphoresis.5

Atypical presentation:abd pain, unexplained

.fatigue, dizziness and PAINLESS ISCHEMIA

Management

- in RR- short precise history- 1/2 min
 - Door needle time
 - Time means muscle
- Perform ECG- Normal ECG doesn't mean Normal heart.

- Basic investigations: CBC, KFT, SE, RBS
- **Cardiac enzymes** onset duration
- Myoglobin 1-2 hour 1 day
- TroponinT,I 3 hours 10 days
- CPK(MB) 4 hours 3 days
- Ast 6h 7 days
- LDH 12 h 14 days

- 
- First Aid: **MONA**
 - -Morphin 1ml/10ml
 - -Oxygen 5 l/min
 - -Nitroglycerin **except** in hypotension or RV infarction
 - -Aspirin 300 mg chewable.

- **In CCU**

- Repeat ECG :1- resolved ST↑
2-New ST ↑
- Repeat morphine tell relief of chest pain.



● STEMI

Indications for SK

1-ST elevation in limb or chest leads

Reciprocal changes- ischemia at distance

2-New LBBB

3-True dorsal MI

Contraindications for SK

Absolute

Relative

- Active non comprisable bleeding
 - Brain Hge or tumor
 - Pregnancy
 - Acute Aortic dissection
1. ↑ BP
 2. prolonged CPR
 3. active PUD
 4. ischemic infarction
 5. major surgery
 6. active hepatitis
 7. previous administration of SK

Signs of successful SK therapy

- Relief of chest pain
- Regression of ST segment $>50\%$ within 2 hours
- Reperfusion arrhythmia
- Early peak of cardiac enzymes

Continued STEMI

- B. blockers – small dose IV(bisoprolol, Metoprolol)
- ACEI
- Statins – 80 mg atorvastatin for 1 month
- Clopidogrel – 300 mg ,then 75 mg – 1 month.
- Nitrate- angina or LVF.
- ASA 75 -325 mg.
- Low molecular weight heparin.
- Treat complications.

Unstable angina and NONSTEMI

Unstable angina

- angina Denovo –new onset < one month.
- Crescendo (progressive) angina.
- post MI angina .
- rest angina.

.Unstable angina ,Non STEMI

.NonSTEMI,associated with elevation of cardiacenzymes
UAP and non STEMI. Recurrence more than
.STEMI

The same management as STEMI, with some
:exceptions

.No role for SK

Use of LMWH , or fondaparinux(synthetic
pentasaccharides,2.5 mg s/c daily)

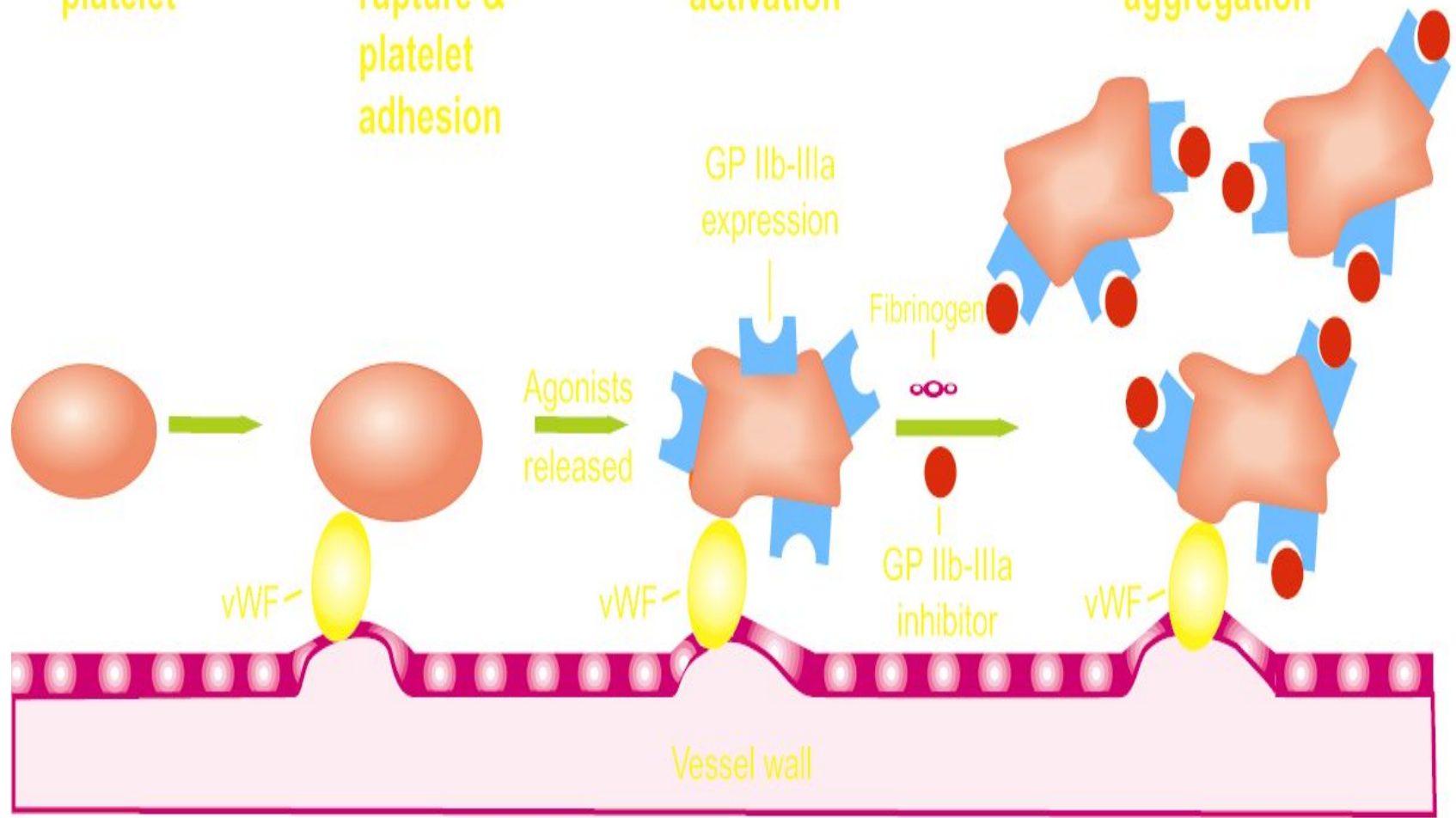
GP IIb IIIa receptors inhibitors (abciximab,
.eptifibatide)

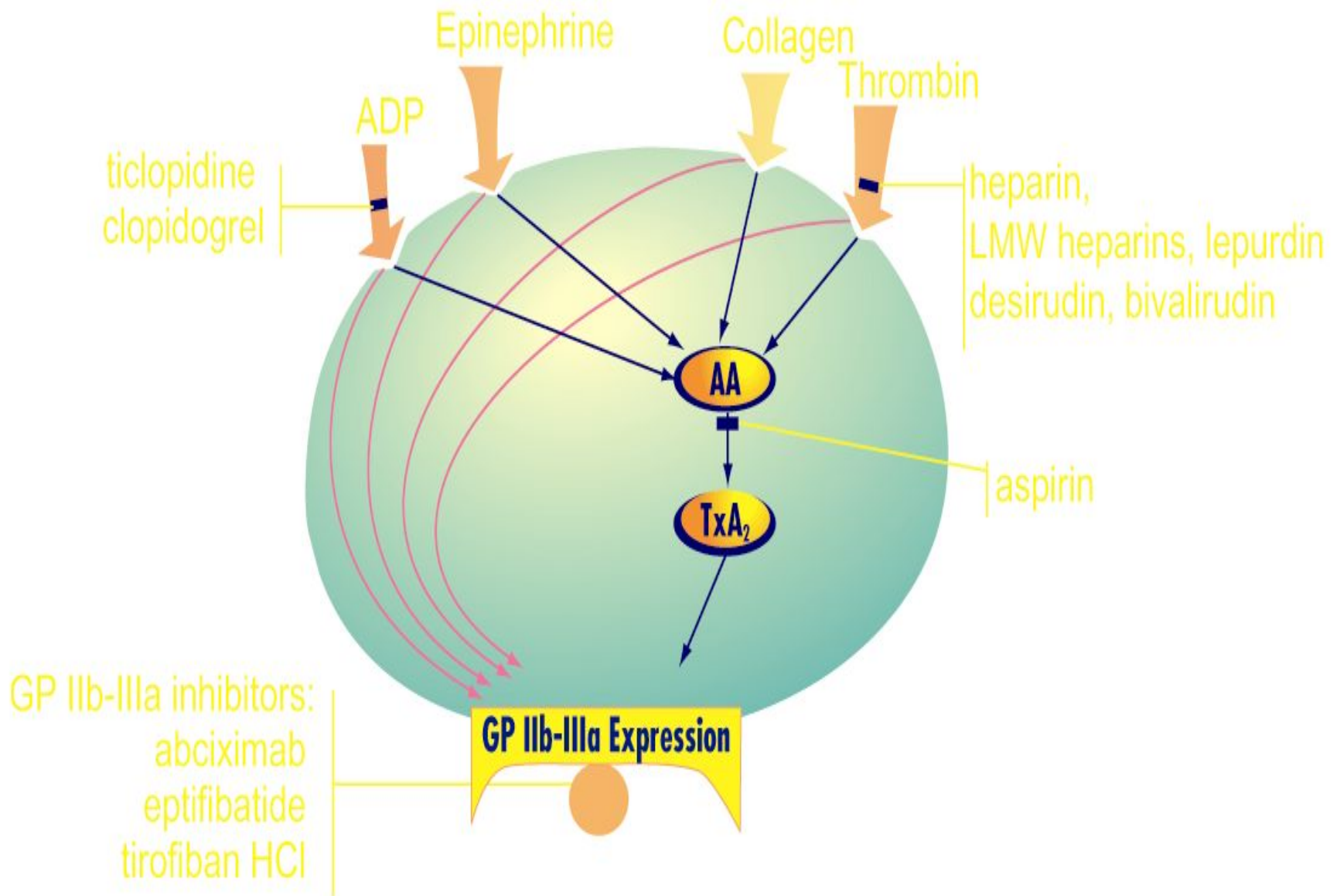
Resting
platelet

Plaque
rupture &
platelet
adhesion

Platelet
activation

Prevention of platelet
aggregation





RISK STRATIFICATION

:TIMI SCORE

age >65 .1

three or more risk factors for CAD.2

prior coronary stenosis ,>50%.3

ST deviation on presentation.4

two or more anginal episodes /24h.5

use of aspirin in last 7 days.6

.elevated serum markers.7

Coronary angiography in ACS

- Primary angiography- circumstances:
 - Highly qualified center and team for 24 h.
 - close to ICCU..DBT-DNT<2 h.
- Rescue angiography (within 72 h).
- late presentation >12 h.
- failed SK or previous administration of SK.
- cardiogenic shock.

Preparing pts for primary or rescue angiography

- clopidogrel 300 mg
- clexane 1 mg /kg
- 300 mg ASA.
- 80 mg atorvastatin.
- integrilin infusion (GPII B III A) INHIBITOR.

Elective angiography

- All pts with UAP or NON STEMI.
- STEMI :
 - Post MI angina
 - +ve ETT one week after MI
 - Young patients.

Other types of angina

.ANGINA DE CUBITUS .1

.POSTPRANDIAL ANGINA .2

.FIRST WIND ANGINA .3

.LINKED ANGINA.4

.ANGINAL EQUIVALENT : DYSPNEA, DIZZINESS, DISCOMFORT .5

:PRINSMATAL(VARIANT,VASOSPASTIC ANGINA) .6

NIGHT AFFECT MIDDLE AGED FEMALES, SMOKERS, OCCURING AT MID
OR EARLY MORNING, ASSOCIATED WITH TRANSIENT ST
.ELEVATION

SPECIFIC PRESENTATION

X SYNDROME: TYPICAL EFFORT ANGINA, WITH POSITIVE .1
EXERCISE TOLERANCE TEST, BUT NORMAL CORONARY
ANGIOGRAPHY (ARTERIOLES AND CAPILLARIES
.AFFECTED)

PAINLESS ISCHEMIA: DUE TO INCREASED ENDOGENOUS .2
ENDORPHINS, HIGH PAIN THRESHOLD (MORE
.DANGEROUS)

.AFFECT ELDERLY , DIABETICS , POST SURGERY , POST CABG

COMPLICATIONS OF ACUTE MI

Early complications: arrhythmia and HB, LVF, early pericarditis and .tromboembolic complications

LATE COMPLICATIONS: post MI angina, late pericarditis, CHF , LV aneurysm, frozen shoulder , frozen .chest, complications of treatment

Arrhythmia

VT: sync DC SHOCK, AMIODARONE OR
.LIDOCAINE

.VF: synch DC SHOCK

.AF

.Atrial and ventricular extra systole

.HB with all its grades

LVF

:KILLIP classification.(4 grades)

.Gr 1. gallop rhythm , mortality about 5%

.Gr 2 basal crepitation, mortality 25%

.Gr 3 pulmonary oedema,mortality 40%

Gr 4 cardiogenic shock. Mort 56% in best
.centers

.LVF MANAGEMENT

GR 1,2,3:TREATMENT OF HEART FAILURE(oxygen ,diuretics,morfine,ACE inhibitors.)

Gr 4 (cardiogenic shock) NA, DOPAMINE DOBUTAMINE, INTRAAORTIC (.BALLOON,RESCUE ANGIOGRAPHY

PERICARDITIS

Early pericarditis: 2-3 days – chest pain when lying , relieved with sitting,
.pericardial rub.- treatment : NSAIDS

Late pericarditis (Dresslers syndrome):
fever, pleurisy,pericarditis ,possible
.effusion

.Treatment: NSAIDS,AND STEROIDS

ECG in pericarditis

PERICARDITIS

Concave ST elevation.1

Diffuse changes.2

PR depression,(specific).3

Late dynamic changes .4

MI

convex ST

localized change

.rapid changes

MECHANICAL COMPLICATIONS

RUPTURE PAPILLARY MUSCLE: MR, .1
,PULMONARY OEDEMA

dyspnea rupture IVS : harsh pansystolic murmur,.2
.biventricular failure

rupture of free wall (tamponade) :TRIADE: .3
congested neck veins, muffled HS, hypotension,
.kussmaules and fredrech sign,paradoxic pulse
pseudo aneurysm .4

.TREATMENT : URGENT SURGERY

.LV ANEURYSM

Localized plugging of myocardium , in both
.systole and diastole

arrhythmia .1

.HF .2

.THROMBOEMBOLIC manifestation .3

.rupture .4

TREATMENT: ICD, aneurysmorrhaphy,
.aneurysmectomy

INFERIOR MI COMPLICATIONS

,Anatomy of coronaries, dominance

RCA BRANCHES: 1. SA BRANCH, 2. AV
.BRANCH, 3. RV BRANCH, 4. PDA, PLB

RV INFARCTION: hypovolemic

.hypotension

.CHB, PAPILLARY MUSCLE RUPTURE

Coronary anatomy

