Tips and tricks in management of patients with Congenital Heart Diseases (CHD)

I-Approach patients with congenital heart disease

General examination:

©Features of famous syndromes:

1-Alagille syndrome:

- Branch Pulmonary stenosis
- Biliary obstruction (Look for jaundice)

2-Holt-Oram syndrome (Heart-Hand syndrome):

- ASD, VSD,
- Absent radius (Look for hand and thumb)

3-Turner syndrome:

- 45 XO
- Aortic aneurysm, dissection, coarctation
- Hypertension, DM
- Neck webbing
- Short stature
- Amenorrhea
- Broad chest

4-Klinefilter syndrome:

- 47 XXY
- ASD, Prolapse (mitral valve), PDA
- Tall stature
- Testicular atrophy

5-William syndrome:

- Supravalvular AS, Peripheral Pulmonary stenosis
- Systemic Hypertension
- Mental retardation
- Elfin facies:
  Depressed basal bridge
  Prominent forehead

6-DiGeorge syndrome:

It is considered part of CATCH syndrome

- Cono-truncal abnormalities:

  Truncus arteriosus

  TGA

  Double outlet right ventricle (DORV)

  Tetralogy of Fallot

  Interrupted aortic arch type B
- Parathyroid and thymus hypoplasia (immune-compromised)

7-Catch syndrome:

C: Cono-truncal abnormalities

A: Abnormal facies

T: Thymus gland hypoplasia

C: Cleft palate

H: Hypoparathyriodism

8-Leopard syndrome:

L: Lentiginosis

E: ECG abnormalities

O: Ocular abnormalities

P: Pulmonary stenosis

A: Abnormal genitalia

R: Retarted growth

D: Deafness

9-Marfan syndrome:

- Aortic aneurysm, aortic incompetence, mitral valve prolapse, Mitral incompetence
- Tall stature
- Long arm, Long leg, Arachnodactyly
- High arched palate
- Skeletal deformities
- Lens subluxation

10-Down syndrome:

- Trisomy 21
- VSD, Endocardial cushion defect, prolapse (mitral valve)

- Mental retardation
- Short stature, low seated ear, large tongue

11-Scimitar syndrome:

- Partial anomalous pulmonary venous drainage
- Right lung hypoplasia
- Systemic blood supply to right lung from the aorta
- Parenchymal lung abnormalities

12-Noonan syndrome:

- ASD, Pulmonary stenosis, Hypertrophic cardiomyopathy
- Morphological features as Turner but without chromosomal abnormalities

13-Others:

Alcohol intake during pregnancy:

- VSD
- Microcephaly, Microphthalmia, Micrognathia
- Growth and developmental abnormalities

Rubella infection during pregnancy:

- ASD, PDA, Pulmonary stenosis
- Microcephaly, cataract, deafness

Shone complex:

- Aortic coarctation, sub-aortic membrane
- Parachute mitral valve, supra-mitral ring

Lutmebacher syndrome

- Mitral stenosis, ASD

©Vital signs:

Blood pressure

1-Hypertension:

-Williams's syndrome (supravalvular AS)
-Turner syndrome
-Aortic coarctation (Upper limb hypertension)

NB: With supravalvular AS, the BP is higher on the Right arm compared to left arm due to shift of blood to innominate artery by coanda effect

Pulse

1-Bounding pulsation:

PDA, truncus arteriosus or systemic AV fistula

2-Weak lower limb pulsation with radio-femoral delay:

Aortic coarctation

3-Absent pulsations in in upper and lower limbs with intact superficial temporal artery pulsations:

Interrupted aortic arch

©Neck veins:

1-Left atrialization of neck veins (A wave equal to V wave):

- ASD

2-Prominent A wave:

- Pulmonary stenosis
- Tricuspid stenosis,
- Pulmonary hypertension (Eisenmenger)

©Cyanosis and clubbing:

1-Seen with congenital cyanotic heart disease

2-Differential cyanosis:

- Cyanosis in lower limb only is seen with PDA that is associated with shunt reversal (Right to left shunt) due to Eisenmenger syndrome or preductal Coarctation

3-Reversed Differential cyanosis:

- Cyanosis in upper limb only is seen in PDA with TGA

Precordial examination (Auscultation):

©Heart sounds

1-Single S1:

- Complete AV canal (Single AV valve)

2-Splitting of S1:

- Wide splitting of S1 and increase in tricuspid component (Sail sound) is seen with Ebstein anomaly

3-Single S2:

- Truncus arteriosus (Single valve)
- TGA (as Pulmonary artery became posterior so muffled pulmonary component)
- Total anomalous pulmonary venous drainage (if associated with pulmonary obstruction)
- Tetralogy of Fallot (if associated with pulmonary atresia)
- Fontan

4-**Wide Splitting of S2:**

- ASD
- Partial anomalous pulmonary venous drainage
- Total anomalous pulmonary venous drainage
- Pulmonary stenosis
- Ebstein anomaly

5-Narrow (close) Splitting of S2:

- Pulmonary hypertension (Eisenmenger)

6-**Ejection click:**

- Bicuspid aortic valve
- Pulmonary stenosis (decreases with inspiration)
- Pulmonary hypertension (Eisenmenger)

©Murmurs:

You have to comment on

I-Murmur of the original disease
II-Murmur of the associated defect

III-Functional (or flow) murmur

IV-Murmur of collaterals (if present)

-Examples:

1-Flow murmur

- In patients with ***ASD,*** there is there is flow murmur of ***pulmonary stenosis***, ***tricuspid stenosis*** due to increase in flow across Pulmonary and tricuspid valves
- In patients with ***VSD***, there is flow murmur of ***pulmonary stenosis***, ***mitral stenosis*** due to increase in flow through pulmonary and mitral valves

2-Murmur of collaterals:

- Heard over the back (with aortic coarctation)
- Murmur of Major Aorto-Pulmonary collaterals (MAPCas) is heard with tetralogy of Fallot with pulmonary atresia
- Collaterals with Truncus arteriosus (aorto-Pulmonary collaterals)

Investigations

© Chest x ray

1-ASD:

- Hilar dance is seen with fluoroscopy (Pulmonary plethora, prominent pulmonary pulsations)

2-Tetralogy of Fallot:

- Coeur en sabot or boot shaped heart, concave main Pulmonary artery and uplifted apex

3-D-TGA:

- Straight left cardiac border (formed of aorta)
- Egg on side

4-L-TGA:

- Straight left cardiac border
- Waterfall appearance (Right Pulmonary artery is more enlarged, prominent and elevated)

5-Ebstein:

- Box shaped heart or Water bottle appearance due to right atrial enlargement, right ventricular enlargement

6-Partial anomalous pulmonary venous connection or drainage:

- Scimitar syndrome (Right Pulmonary vein drain to IVC) appear as crescent vertical shadow in Right lung

7-Total anomalous pulmonary venous connection or drainage:

- Snow man appearance with supra-cardiac (non-obstructive type), common Pulmonary vein drain through innominate vein and left vertical veins drain to SVC and Right atrium

8-Aortic coarctation:

- Figure of 3 (pre and post-stenotic aortic dilatation) or rib notching (Intercostal collaterals)

©ECG

1-Tricuspid atresia:

- LVH with superior axis

2-ASD:

- rsr pattern in V1
- Right axis deviation with secundum ASD
- Left axis deviation with primum ASD (due to associated mitral cleft with LV overload and associated left anterior hemiblock)
- Inverted P wave in ECG with sinus venous ASD
- Crochetage sign: notch near R wave apex in Inferior leads with secundum ASD

3-Arrhythmias:

- **First degree AV block:**

  TGA, Ebstein, Endocardial cushion defect, ASD

- **Complete heart block:**

  AV canal, congenitally corrected TGA, Fontan

- **Atrial Arrhythmia:**
  ASD, Ebstein, Fontan, atrial switch operation for TGA

- **Ventricular Arrhythmia:**

  Post Fallot repair, ventricular dysfunction, Fontan

- **Pre-excitation:**

  Ebstein and congenitally corrected TGA

©Echocardiography:

1-TGA:

- In short axis view, Great vessels level, you will see 2 circles (representing aorta and Pulmonary arteries) instead of circle (aorta) and sausage shape (Pulmonary Artery)

2-AV canal (Endocardial cushion defect):

- AV valves (mitral and tricuspid valves) are at same level (Normally: tricuspid valve is more apical in position)

3-Anomalous Pulmonary venous drainage:

- Inability to see all (total anomalous) or some (partial anomalous) Pulmonary veins draining in the left atrium

©Angiography:

- **Goose neck deformity**

  Endocardial cushion defect (due to elongated narrow LVOT)

- **Hourglass appearance:**
  Supravalvular AS
- **Coronary anomalies:**

  1-Fallot:

  LAD arises from RCA, cross in front of RVOT

  2-CC-TGA:

  Mirror image coronary arteries:

  RCA supply LAD and LCX, left main resemble RCA

  3-Truncus arteriosus:
  Ostial coronary stenosis or anomalies

  4-Supravalvular AS:
  Ostial coronary stenosis

Spectrum of congenital heart diseases

	Left to right shunt	Right to left shunt	Obstructive lesion
Cyanosis/Clubbing	No cyanosis	Cyanosis /clubbing	No cyanosis
Chest infection	Frequent	Usually no	No recurrent chest infection
Precordium	Pulsatile precordium with or without precordial bulge	Silent precordium	Silent precordium but forcible LV/RV impulse
Cardiomegaly	Yes	No	No
Murmur	Shunt murmur and flow murmur if large shunt	No shunt murmur	Harsh ejection systolic murmur /thrill
Chest X ray	Increased pulmonary vascularity (plethoric lung)	Decreased pulmonary vascularity (oligemic lung)	Normal pulmonary vascularity
Congestive heart failure	Early in infancy	CHF: Late	CHF: Very late

Classifications of congenital heart disease

I-Acyanotic heart disease

Increased pulmonary blood flow (left to right shunt)		Normal pulmonary blood flow (obstructive lesions)	
RVH	**LVH or combined**	**RVH**	**LVH or combined**
-ASD -Partial anomalous pulmonary venous drainage	-VSD -PDA -AV canal	-Pulmonary stenosis -Mitral stenosis -Aortic coarctation in infants	-Aortic stenosis -Aortic incompetence -Mitral incompetence -Aortic coarctation -Interrupted aortic arch

II-Cyanotic heart disease

Increased pulmonary blood flow		Decreased pulmonary blood flow		
RVH	**LVH or combined**	**RVH**	**LVH**	**Combined**
-TGA -Total anomalous pulmonary venous drainage	-TGA with VSD -Single ventricle -Persistent truncus arteriosus	-Eisenmenger -Ebstein anomaly -Fallot tetralogy	-Pulmonary atresia -Tricuspid atresia	-Pulmonary stenosis with single ventricle -Pulmonary stenosis with TGA -Pulmonary atresia with persistent truncus arteriosus

Other cyanotic heart disease

1-Double outlet right ventricle (DORV)

2-Hypoplastic left heart syndrome (HLHS)

3-Heterotaxia(isomerism)

4-Persistent pulmonary hypertension of the newborn (PPHN)

Complications of cyanosis:

1-Neurological: Ischemic Stroke, cerebral hemorrhage, brain abscess

2-Hematological: iron deficiency anemia, polycythemia, bleeding tendency, coagulopathy

3-Hyperviscosity: visual disturbances, tinnitus, headache, poor concentration

4-Renal: proteinuria, urate nephropathy, acute renal failure

5-Rheumatological: osteoarthropathy, gout, scoliosis

Phlebotomy(venesection)

Indication: Symptomatic hypervelocity with hematocrit value above 65% provided that there is no dehydration and no iron deficiency

However, any patient with neurological symptoms, CT brain should be done first to exclude stroke or cerebral hemorrhage

Method: withdrawal of 250-500ml blood over 30-45 minutes plus volume replacement, it can be repeated every 24 hours until improvement of symptoms or Hemoglobin level 18-19 gm.

Target hemoglobin= 60-(saturation/2)

Timing of intervention in congenital heart disease

Disease	Timing
Truncus arteriosus	At the age of 2 month
Tetralogy of Fallot	At the age of 3 month(shunt), at the age of 3-24 month (total repair)
Total anomalous pulmonary venous drainage	At the age of 6 month
Complete AV canal	At the age of 6 month
VSD	At the age of 6 month
Partial AV canal	At the age of 1 year
ASD	At the age of 2-4 years
Partial anomalous pulmonary venous drainage	At the age of 2-4 years
Aortic coarctation	At the age of 2-4 years

How to approach adult patient presenting with cyanosis secondary to congenital heart disease?

We have to classify patients into 2 major categories:

1-Patients who have loud murmur, the DD Includes:

- Fallot tetralogy
- ASD with pulmonary stenosis
- Single ventricle
- Ebstein (included in this group but did not have loud murmur)

2-Patients without loud murmur but have loud P2 (accentuated pulmonary component of P2) the DD includes:

- Eisenmenger syndrome
- Single ventricle

PDA (Duct dependent circulation vs. indications of balloon atrial septostomy

Duct dependent circulation	Balloon atrial septostomy (Rashkind procedure)
1-To maintain pulmonary circulation: • Tricuspid atresia • Pulmonary atresia • Fallot tetralogy • Pulmonary stenosis (Severe)	• Tricuspid atresia • Pulmonary atresia • Failing Fontan • Pulmonary hypertension • RV dysfunction
2-To maintain systemic circulation: • Interrupted aortic arch • Hypoplastic left heart syndrome • Aortic coarctation • Critical aortic stenosis	• Hypoplastic left heart syndrome • Double inlet LV • LV dysfunction
3-To allow mixing of blood: TGA	• TGA • Total anomalous pulmonary venous connection

Systemic to Pulmonary Artery Shunting for Palliation

Advantages:

1-To improve oxygenation in patients with cyanosis

2-To increase pulmonary blood flow and allow pulmonary artery to grow

3-preserve LV function

4-Prepartion for Fontan (Ideal candidate for Fontan operation should have normal LV function and low pulmonary vascular resistance, both can be provided by palliative shunts

Disadvantages

1-Pulmonary artery distortion (kinking, thrombosis or occlusion)

2-LV volume overload (pulmonary hypertension, pulmonary vascular disease) if large shunt

Indications:

1-Fallot tetralogy

2-Tricuspid atresia

3-Pulmonary atresia either with or without VSD

4-Hypoplastic left heart syndrome

5-Single ventricle situation with pulmonary or aortic atresia

6-Ebstein anomaly with functional or anatomical pulmonary atresia

Types of shunt

1-Blalock Taussig shunt (BT shunt) either classic or modified

- **Classic:** Direct Anastomosis between subclavian artery or innominate artery to pulmonary artery
- **Modified:** Anastomosis between subclavian artery or innominate artery to pulmonary artery though interposition graft (Gore-Tex or polytetrafluoroethylene (PTFE)
- Result in increase in pulmonary blood flow thus decreased the cyanosis
- Should be done on opposite side of arch to avoid pulmonary artery kinking
- **Advantages:** Small shunt so overflow and no CHF and unlikely to increase pulmonary vascular resistance
- **Disadvantages:** subclavian steal (arm ischemia) and it is small shunt with risk of shunt obstruction due to thrombosis

2-Potts shunt (Obsolete now):

- Anastomosis between descending aorta and left pulmonary artery
- **Advantage: large** caliber to decrease risk of thrombosis
- **Disadvantage:** overflow can increase pulmonary vascular resistance and CHF

3-Waterston shunt (obsolete now):

- Extra pericardial anastomosis between ascending aorta and right pulmonary artery
- **Disadvantages:** The same as Potts

4-Cooley shunt:

- Intrapericardial anastomosis between ascending aorta and right pulmonary artery
- **Disadvantages**: As Waterston plus increased risk of adhesions

5-Central shunt (Mee shunt)

- Anastomosis between ascending aorta and main pulmonary artery
- Done in neonate below age of 3 month
- There must be other source for pulmo0nary blood flow such as PDA
- Useful if there are bilateral small pulmonary artery branches

6-Glenn shunt:

- **Classic:** Anastomosis between svc and divided right pulmonary artery (unidirectional)
- **Modified:** anastomosis between SVC to undivided right pulmonary artery (bidirectional Glenn, BDG), There is bidirectional flow from SVC to both right and left pulmonary arteries
- **Advantages:** No increase in flow (so no volume overload on ventricle)
- **Disadvantages:** Pulmonary vascular resistance must be low (so, child younger than 3-6 month are generally excluded)

7-Sano shunt

- RV to pulmonary artery shunt though a tube
- infants may suffer the catastrophic results of a stenosed or obstructed shunt because of their propensity to develop dehydration in the presence of an anatomically small shunt.
- In addition, a shunt provides a run-off from the systemic circulation in favor of the pulmonic circulation.
- A consequence of this run-off is the low diastolic pressures in the aorta that contribute to coronary insufficiency.
- Sano et al proposed a right ventricle–to–pulmonary artery shunt in an attempt to overcome the obstacles noted with a systemic-to-pulmonary artery shunt.

Pulmonary artery banding

Definition:

Palliative or staging procedure to protect the lung from high flow or high pressure acting as if surgically created pulmonary stenosis

Indications

I-Left to right shunt:

- Patients with left to right shunts have pulmonary overflow, so pulmonary artery banding may be considered as a staged approach for more definitive repair
- Patients with multiple muscular VSD (Swiss cheese) which is technically difficult for repair in neonates
- Patients with VSD associated with aortic coractation or interrupted aortic arch with contraindication for primary repair such as sepsis or multiple organ system failure

II-TGA who require training of LV as staged approach before arterial switch

III-Functionally single ventricular circulation with unrestrictive pulmonary blood flow

Important points to remember about transposition of Great vessels (TGA)

1-Normally:

Pulmonary artery is anterior and to the left and aorta is posterior and to the right

2-D-TGA:

- Aorta: is anterior and to the right
- RV is anterior and to the right
- Ventriculo-arterial discordance
- Aorta arises From RV and Pulmonary artery arises from LV

3- L-TGA or congenitally corrected TGA (CC-TGA):

- Aorta is anterior and to the left
- RV is posterior and to the left
- Ventriculo-arterial discordance and atrioventricular discordance
- Aorta arises from RV
- Pulmonary artery arises from LV
- LA is connected to RV and through a tricuspid valve
- RA is connected to LV through mitral valve

Takotsubo cardiomyopathy

(Brocken heart syndrome)

1-More common in post-menopausal female

2-Related to severe emotional stress

3-Pathogenesis is related to catecholamine toxicity

4-Moderate Troponin elevation despite extensive ECG change and Echo findings

5-Regional wall motion abnormalities (RWMAs) in echo involving multiple coronary territories

6-RWMAs most commonly involved apex and adjoining apical segments, less commonly mid myocardial segments, least commonly involve basal segments

7-Normal coronary angiography or discrepancy between RWMAs findings in echo and coronary angiography findings

8-No late gadolinium enhancement in cardiac MRI

9-Prolonged QT more than 500 msec

10-Usually reversible LV function

11-Most common finding in Left ventriculography is apical ballooning

12-ECG changes include ST elevation or depression

Reference: ESC guidelines for fourth universal definition of myocardial infarction (2018)

Cardiac amyloidosis

I-Types:

- **Primary (AL) amyloidosis:** due to deposition of Amyloid light chain most commonly due to multiple myeloma
- **Secondary (AA) amyloidosis**: due to deposition of Amyloid A protein, in Chronic Inflammatory state Such as rheumatoid arthritis
- **Familial Amyloidosis (ATTR):** due to deposition of mutated type of transthyretin
- **Senile systemic amyloidosis (SSA):** due to deposition of wild type of transthyretin

II-Clinical presentation:

Amyloidosis is one of the most common causes of restrictive cardiomyopathy and can present with dyspnea, lower limb edema, ascites, as well as syncope (due to autonomic neuropathy and orthostatic hypotension) and palpitation (AF)

III-ECG:

- Low voltage despite increased wall thickness in Echocardiography (Voltage mass mismatch)
- Pathological Q waves in absence of ischemia
- AF

IV-Echo:

2D findings

- Two Large atria with two small ventricles
- Increased LV thickening (Ground glass appearance)
- Thickening of AV valves (mitral and tricuspid valves)
- Thickened interatrial septum
- Pericardial effusion
- IVC plethora (Dilated, non-collapsed IVC)

Pulsed wave (PW) Doppler

- Restrictive pattern (grade III diastolic dysfunction)
- Marked LA dysfunction that can be manifested as very small A wave in PW Doppler(called atrial arrest)

Continuous wave (CW) Doppler:

- Estimated pulmonary artery systolic pressure more than 50mmHg

Tissue Doppler imaging (TDI):

- Medial e prime velocity less than 8cm/sec (very important sign)

- Elevated LV filling pressure) E/e prime ratio above 14

Color:

- Mitral and tricuspid incompetence with RCM

V-Diagnosis:

- Abdominal fat aspirate, Congo red stain
- Free light chain assay, protein electrophoresis, immune-fixation, bone marrow aspirate (to diagnose primary amyloidosis)
- Genetic study to diagnose Familial type
- Cardiac-MRI for tissue characterization
- Technetium scintigraphy can be used for diagnosis of TTR related amyloidosis

VI-Associated Features:

- Carpal tunnel syndrome
- Orthostatic hypotension and syncope (due to autonomic neuropathy secondary to transthyretin
- Hepatomegaly disproportional to degree of congestion

VII-Treatment:

1-Tafamidis

- Tafamidis is now approved for treatment of Familial type with autonomic neuropathy based on ATTR-ACT trial, Decrease all-cause mortality and HF hospitalization.
- There are 2 preparations: Vnydaqel and Vyndamax but both are contraindicated in Pregnancy

2-Anti-failure measures

- ACEI used cautiously as it can result in profound hypotension
- Beta blocker used cautiously as there is fixed stroke volume and COP depend mainly on HR
- Better to avoid digitalis due to high incidence of digitalis toxicity

3-Anticoagulation:

- For AF regardless CHADS-VASc score due to high incidence of LAA thrombus due to marked reduction of LA function

4-**Liver transplantation** :can be considered in familial type
5-**Chemotherapy and steroids:** for primary type
6-**Immunosuppression and anti-inflammatory:** in secondary type

Practical guidance on the use of angiotensin-converting enzyme inhibitors (or angiotensin II receptor blockers) in patients with heart failure with reduced ejection fraction

1-Check baseline renal function and electrolytes

2-Start with a low dose

3-Double the dose at not less than 2-week intervals

4-More rapid dose up-titration may be carried out in patients in hospital or who are otherwise closely monitored, tolerability permitting

5-Aim for target dose or the highest tolerated dose (remember: some ACE-I (or ARB) is better than no ACE-I).

6-Recheck blood chemistry (urea, creatinine, serum potassium) 1-2 weeks after initiation and 1-2weeks after final dose titration. Monitor blood chemistry 4 monthly thereafter.

7-Some rise in urea (BUN), creatinine, and potassium is to be expected after an ACE-I; if an increase is small and asymptomatic, no action is necessary

8-An increase in creatinine of up to 50% above baseline, or 266 μmol/L (3 mg/dL)/eGFR <25 mL/min/1.73 m2, whichever is the smaller, is acceptable

9-An increase in potassium to ≤5.5 mmol/L is acceptable

10- If urea, creatinine, or potassium does rise excessively, consider stopping concomitant nephrotoxic drugs (e.g. NSAID) and other potassium supplements or retaining agents (triamterene, amiloride) and, if no signs of congestion, reducing the dose of diuretic

11- If greater rises in creatinine or potassium than those outlined above persist despite adjustment of concomitant medications, the dose of the ACE-I (or ARB) should be halved and blood chemistry re-checked within 1–2 weeks; if there is still an unsatisfactory response, specialist advice should be sought

12- If potassium rises to >5.5 mmol/L or creatinine increases by >100% or to >310 μmol/L (3.5 mg/dL)/eGFR <20 mL/min/1.73 m2, the ACE-I (or ARB) should be stopped and specialist advice sought.

13-Blood chemistry should be monitored frequently and serially until potassium and creatinine have plateaued

Reference: ESC guidelines for management of HF, 2016(Web addenda)

LV Thrombus After Acute MI

The following are key points to remember from this review article about left ventricular (LV) thrombus after acute myocardial infarction (MI):

1-LV thrombus is not an uncommon complication of acute MI and is associated with systemic thromboembolism.

2-Contemporary epidemiologic data suggest the incidence of LV thrombus, detected using optimal imaging modalities, may be as high as 15% in patients with ST-segment elevation MI (STEMI) and up to 25% in patients with anterior MI.

3-Standard transthoracic echocardiography (TTE) is typically the screening modality of choice for LV thrombus detection and should be performed within 24 hours of admission in those at high risk for apical LV thrombus (e.g., those with large or anterior MI or those receiving delayed reperfusion).

4-If (1) the LV apex is poorly visualized, (2) anterior or apical wall motion abnormalities are present, or (3) high apical wall motion scores are calculated (≥5 on non-contrast TTE), contrast TTE or cardiac magnetic resonance should be considered based on local availability and resources.

5-The 2013 American College of Cardiology Foundation/American Heart Association (ACCF/AHA) STEMI guidelines advise that oral anticoagulation (OAC) may be considered in patients with STEMI with anterior apical akinesis or dyskinesis to prevent LV thrombus formation.

6- Similarly, the AHA/American Stroke Association 2014 guidelines on stroke prevention advise that anticoagulation may be considered for 3 months in patients with acute anterior STEMI and ischemic stroke or transient ischemic attack who have anterior apical akinesis or dyskinesis.

In patients with a diagnosed LV thrombus, OAC should be started immediately.

7-The 2013 ACCF/AHA STEMI guidelines advise that it is reasonable to add OAC to dual antiplatelet therapy among patients with STEMI and asymptomatic LV thrombus for 3 months, targeting a lower international normalized ratio (INR) goal of 2.0-2.5. The AHA/American Stroke Association 2014 stroke prevention guidelines recommend a similar duration, targeting a higher INR of 2.5.

8-The European Society of Cardiology 2017 STEMI guidelines advised that once an LV thrombus is diagnosed, OAC should be considered for up to 6 months, guided by repeated echocardiography and with consideration of bleeding risk and need for concomitant antiplatelet therapy.

The optimal duration of OAC in these patients is unclear, and decisions regarding continuation of OAC should be made on a case-by-case basis.

9-In 2014, the AHA/American Stroke Association guidelines on stroke prevention introduced a new recommendation advising that low molecular weight heparin, dabigatran, rivaroxaban, or apixaban may be considered as an alternative to vitamin K antagonists for post-MI LV thrombus or anterior or apical wall motion abnormalities with an LV ejection fraction <40%, who are intolerant of vitamin K antagonists because of non-hemorrhagic adverse events.

10-In addition to less potent antithrombotic regimens, adjunctive bleeding reduction or avoidance strategies should be considered. It is reasonable to treat all patients with proton-pump inhibitor therapy while receiving combination antithrombotic regimens.

11-Given a lack of clear randomized clinical trial data and great variability in the presentation and associated complications of LV thrombus, individualized approaches are indicated.

12- Ongoing studies from related therapeutic areas of varying antithrombotic regimens will continue to help delineate the optimal antithrombotic strategy for LV thrombus

Reference: ACC article by Dr. Debabrata Mukherjee, MD, FACC

Approach for patients with Coronary artery disease (CAD) and thrombocytopenia

1-This is one of the most challenging situations in cardiology practice that needs a lot of wisdom and precautions

2-To approach these patients we have to go through 2 parallel tracks:

-Thrombocytopenia track
-CAD track

I-Thrombocytopenia track:

1-What is the severity of thrombocytopenia?

Mild: Platelets count 150.000 to 100.000
Moderate: Platelets count 100.000 to 50.000
Severe: Platelets count less than 50.000

2-What is cause of thrombocytopenia?

If the etiology is malignancy, then what is the prognosis and life expectancy. (malignancy with expected survival less than 1 year, mostly we will adopt conservative strategy)

3-Is there treatment for thrombocytopenia?

Can we increase the platelet count by certain drugs (eg: increasing platelets by thrombopoietin agonist)
Or by treating the underlying condition (eg: Steroids in autoimmune disease)

II- Coronary artery disease track:

1-What is the clinical presentation?

A-Stable CAD: conservative

B- Low risk non-ST elevation ACS: conservative

C-STEMI or high-risk Non-ST elevation ACS: invasive strategy

2-Antithrombotics:

You can use antiplatelet therapy if platelets count above 50 000

3-Strategy:
If platelets count above 50 000 and there is strong indication for invasive strategy (STEMI or high-risk non-ST elevation ACS) follow the following steps:

A-Radial access

B- Use Biofreedom stent to give DAPT only for 1 month then P2Y12 inhibitor monotherapy

C-Clopidogrel is the P2Y12 inhibitor of the choice

D-Use PPI

E- in Cath lab, Heparin dose can be reduced to 50 unit /kg

F-Avoid use of Glycoprotein IIb III a

Reference: Position paper by ESC /Position paper by SCAI

Evidence for Use of Inotropes and Vasopressors in Cardiovascular Disease (position paper from Circulation)

1-Cardiogenic Shock Complicating Acute Myocardial Infarction

- The American College of Cardiology/American Heart Association guidelines for management of hypotension complicating AMI suggest the use of dobutamine as a first-line agent if systolic blood pressure ranges between 70- and 100-mm Hg in the absence of signs and symptoms of shock.
- Dopamine is suggested in patients who have the same systolic blood pressure in the presence of symptoms of shock.
- However, definitive evidence supporting use of specific agents in this setting is lacking.
- Moderate doses of these agents maximize inotropy and avoid excessive α1-adrenergic stimulation that can result in end-organ ischemia.
- The deliberate combination of dopamine and dobutamine at a dose of 7.5 μg/kg/min− each was shown to improve hemodynamics and limit important side effects compared with either individual agent administered at 15 μg/ kg/min.
- Moderate doses of combinations of medications may potentially be more effective than maximal doses of any individual agent.
- When response to a medium dose of dopamine or dopamine/dobutamine in combination is inadequate, or the patient's presenting systolic blood pressure is <70 mm Hg, the use of norepinephrine has been recommended.
- With an antithrombotic effect in addition to its pressor qualities, norepinephrine may be the optimal choice under these conditions compared with epinephrine, which can exacerbate lactic acidosis and promote thrombosis in coronary vasculature.

2- Congestive HF

- Current American College of Cardiology/American Heart Association guidelines for diagnosis and management of chronic HF in the adult do not recommend the routine use of intravenous inotropic agents for patients with refractory end-stage HF (class III recommendation) but do state that they may be considered for palliation of symptoms in these patients (class IIb recommendation).
- The European Society of Cardiology acute HF guidelines also stress that few controlled trials with intravenous inotropic agents have been performed.
- However, these guidelines do point out that in an appropriate clinical setting of hypotension and peripheral hypoperfusion, particular agents may be indicated

with slightly different levels of recommendation (dobutamine and levosimendan, class IIa; PDIs and dopamine, class IIb).

- The most commonly recommended initial inotropic therapies for refractory HF (dobutamine, dopamine, and milrinone) are used to improve CO and enhance diuresis by improving renal blood flow and decreasing SVR without exacerbating systemic hypotension.

3- Cardiopulmonary Arrest

- Epinephrine, with its potent vasopresso r and inotropic properties, can rapidly increase diastolic blood pressure to facilitate coronary perfusion and help restore organized myocardial contractility.
- However, it is not clear whether epinephrine actually facilitates cardioversion to normal rhythm, and its use has been associated with increased oxygen consumption, ventricular arrhythmias, and myocardial dysfunction after successful resuscitation. Repeated high-bolus doses (5 mg) appear no more effective than repeated standard doses (1 mg) at restoring circulation.

4-Postoperative Cardiac Surgery

- Milrinone and dobutamine were both found to be effective in improving general hemodynamic parameters compared with placebo in a European multicenter, randomized, open-label trial.

5- Right Ventricular Infarction

- Significant right ventricular free-wall ischemia leads to immediate dilation of the right ventricle within a constrained pericardium.
- A rapid increase in intrapericardial pressure and intraventricular septal shift alters LV geometry, impairing LV filling and contractile performance.
- These combined effects result in a drop in CO that may exacerbate shock.
- Excessive intravenous fluid beyond a right atrial pressure >15 mm Hg to improve a “preload-dependent” right ventricle can result in deterioration of LV performance.
- Dobutamine improves myocardial performance in this setting.

Tips and tricks in management of cryptogenic stroke and Patent Foramen oval (PFO)

1-This topic is considered of the controversial topics in cardiology

2-To be able to select the appropriate patient who will benefit from PFO closure, we should go through 2 parallel tracks:

-Neurologist track
-Cardiologist track

I-Neurologist track:

1-Every effort should be made to **confirm** that this stroke is really **cryptogenic stroke** (so all work up should be done to exclude other types of stroke either large vessel or Small vessel or embolic or other determined etiology)

2- **History:** ask about Valsalva or straining or cough or prolonged travel or DVT before the event

3-Assessment of **ROPE** score (risk of paradoxical embolization):

-In general, if the patient is young with cortical Infarction and no CV risk factors then It is mostly cryptogenic
-If patient presenting with multiple cortical or subcortical infarction mostly it is embolic

4-**Recuritment window**: PFO device closure is done within 180 days from the onset of stroke

5-**Recruitment age:** 18-60 years

II-Cardiologist track:

1-Every effort should be made to exclude hidden AF or embolic source

2-It is recommended to ask for rhythm monitoring (External loop) for 4 weeks to exclude silent or paroxysmal AF. This duration can be reduced to 1-2 weeks if patient below age of 40, no Risk factors for AF (such as Valvular heart disease or hypertension, hyperthyroidism or alcohol intake).

3-Predictors of paroxysmal AF such as CHADS-VASc score and in ECG predictors such as frequent PACs, P wave dispersion.

4-Echo assessment should include assessment of LA size (including LA volumes), LA function (including emptying fraction) as well as LAA function and emptying velocity

5-Full Assessment of PFO including the bubble study (appearance of bubbles in the LA within 3 cardiac cycles). To be candidate for PFO closure, right to Left shunt must be demonstrated with or without Valsalva.

6-Presence of Interatrial septal aneurysm increases the risk and the possibility that this PFO is incriminated in the cryptogenic stroke

7-Patients maintained on long term oral anticoagulant are not candidate for device closure of PFO

8-After device closure of PFO, patients are maintained on aspirin, then if there is recurrent cryptogenic stroke on top of device and antiplatelet, shifting to oral anticoagulant instead of aspirin may be considered

8-Trials that showed that PFO closure device is superior to antithrombotic include:
CLOSE
REDUCE
RESPECT trials

Tips and tricks in Brugada syndrome

1-Brugada syndrome is a familial cardiac disease leading to ventricular arrhythmias and SCD.

2-It is characterized by a typical ECG morphology and an increased susceptibility to present ventricular arrhythmias and sudden death in the absence of structural heart disease.

3-The characteristic ECG pattern, known as coved-type or type 1, consists of a persistent ST-segment elevation in right precordial leads followed by negative T-waves, and must be distinguished from other conditions that also present with right ST-segment elevation.

4-To date, 19 genes have been associated with the disease, being SCN5A the most common gene.

5-The ICD is the only proven effective therapy for patients at high risk so far, despite several pharmacological approaches that are also currently being used.

6-Brugada pattern is only ECG Brugada pattern, if it was associated with symptoms suggestive of Brugada like syncope or ventricular arrhythmias or survivors of sudden cardiac death or Family history, then we can call it Bragada syndrome

7-Brugada Pattern is seen with many causes like electrolytes imbalance or fever or drugs

8-No need for provocative test with spontaneous type I Brugada

9-To confirm Brugada pattern, you can change ECG leads by placing V3 above V1 and V5 above V2

10-Male gender is predominant, and it is autosomal dominant

Expert Consensus Recommendations on Brugada Syndrome Therapeutic Interventions

Class I

1. The following lifestyle changes are recommended in all patients with diagnosis of BrS:

a) Avoidance of drugs that may induce or aggravate ST-segment elevation in right precordial leads (for example, visit Brugadadrugs.org),

b) Avoidance of excessive alcohol intake.

c) Immediate treatment of fever with antipyretic drugs.

2. ICD implantation is recommended in patients with a diagnosis of BrS who:

a) Are survivors of a cardiac arrest and/or

b) Have documented spontaneous sustained VT with or without syncope.

Class IIa

3. ICD implantation can be useful in patients with a spontaneous diagnostic type I ECG who have a history of syncope judged to be likely caused by ventricular arrhythmias.

4. Quinidine can be useful in patients with a diagnosis of BrS and history of arrhythmic storms defined as more than two episodes of VT/VF in 24 hours.

5. Quinidine can be useful in patients with a diagnosis of BrS:

a) Who qualify for an ICD but present a contraindication to the ICD or refuse it and/or

b) Have a history of documented supraventricular arrhythmias that require treatment.

6. Isoproterenol infusion can be useful in suppressing arrhythmic storms in BrS patients.

Class IIb

7. ICD implantation may be considered in patients with a diagnosis of BrS who develop VF during programmed electrical stimulation (inducible patients).

8. Quinidine may be considered in asymptomatic patients with a diagnosis of BrS with a spontaneous type I ECG.

9. Catheter ablation may be considered in patients with a diagnosis of BrS and history of arrhythmic storms or repeated appropriate ICD shocks.

Class III

10. ICD implantation is not indicated in asymptomatic BrS patients with a drug-induced type I ECG and on the basis of a family history of SCD alone.

Reference: Consensus recommendations for ICD placement in patients with Brugada Syndrome. Priori et al. (2013).

Cardiovascular manifestations of systemic diseases

©Systemic lupus erythematosis (SLE):

I-Cardiovascular manifestation of SLE

- Pericardium: Constrictive pericarditis, pericardial effusion
- Myocardium: Myocarditis
- Endocardium:Endocarditis (Non-bacterial thrombotic endocarditis, Libman sacks endocarditis)
- Pulmonary hypertension
- Coronary artery disease: Premature atherosclerosis

II-Diagnostic criteria for SLE

SOAP BRAIN MD

S: Serositis (pleurisy, pericarditis)
O: Oral ulcers
A: Arthritis(non-erosive)
P: Photosenstivity

B: Blood; hemolytic anemia, leucopenia, lymphopenia, thrombocytopenia
R: Renal (Proteinuria, red cell cast, granular casts)
A: ANA
I: Immune, anti-DNA, anti-smith, antiphospholipid syndrome
N: Neuro:Psychosis, seizures

M: Malar rash
D: Discoid rash

You need at least 4 with at least 1 Clinical and 1 Immune criteria

©Rheumatoid Arthritis (RA)

I-Cardiovascular manifestation of RA

Similar to the cardiovascular manifestation of SLE

II-Diagnostic criteria

- Inflammatory Arthritis involving 3 points or more
- Positive Rheumatoid factor plus or minus anti-CCP (cyclic citrulinated peptide)
- Increase in ESR and CRP
- Duration of symptoms more than 6 weeks
- Similar disease excluded especially psoriatic arthritis, acute viral polyarthritis, poly-articular gout or SLE
- Other features and differential diagnosis:
    - Morning stiffness (30 minutes)
    - More common in females

      (Seronegative arthritis is more common in males)
    - Affection of hand, wrist and knee joints but spare distal interphalangeal (DIP) joints and thoraco-lumbar joints

      (Seronegative Arthritis involve spine and sacroiliac joints with Inflammation of tendon and ligament and their insertion points in bones, enthesitis)
    - Symmetrical Arthritis

      (Rheumatic fever and seronegative arthritis are asymmetrical)
    - Destructive affection of the joints (can result in joint deformity)

      (Rheumatic fever and SLE are non-destructive Arthritis)

©Scleroderma

I-Cardiovascular manifestations:

- Group I pulmonary arterial hypertension (associated conditions; connective tissue disorders)
- Myocardial infarction (coronary spasm)
- Pericardial effusion
- Cardiomyopathy
- Raynaud’s phenomenon

II-Types

1-Limited type: CREST syndrome

- Calcinosis
- Raynaud’s
- Esophageal dysmotility
- Sclerodactyly
- Telangiectasia

2-Diffuse type:

- Renal crisis
- Acute Pulmonary edema
- Acute severe hypertension
- Acute Renal failure
- Thrombocytopenia
- Microangiopathy

III-Diagnosis:

- Anti-RNP
- Anticentromere
- Antiscleroderma 70

©Sarcoidosis

Non caseating granuloma affecting mainly lung and heart

I-Cardiovascular manifestations:

- Pulmonary hypertension (Group 5 PH)
- Restrictive cardiomyopathy
- Ventricular aneurysm
- Ventricular Arrhythmia
- AV block
- Thinning of basal septum
- Regional wall motion abnormalities in non-coronary territory
- Vasculitis that can involve small to large size vessel including aorta

II-Diagnosis:

- Serum ACE
- Urinary calcium
- CXR or CT chest(Hilar lymphadenopathy)
- Cardiac MRI
- PET scan

Cardiovascular manifestations of Vasculitis

I-Classifications of vasculitis according to size:

©Small vessel vasculitis
©Medium size vasculitis
©Large vessel vasculitis
©Variable size vasculitis

©Small vessel vasculitis:

1-ANCA positive vasculitis:

- Wegner
- Churg Strauss
- Microscopic polyangiitis

2-Immune complex associated:

"HCG"

- Henoch Schoenlein purpura
- Increased level of cryoglobulin
- Increased level of complement
- Good pasture syndrome

©Medium size vasculitis:

- Polyarteritis nodosa
- Kawasaki disease

©Large vessel vasculitis:

- Takayasu vasculitis
- Giant cell (temporal arteritis)

©Variable size vasculitis:

- Bechet
- Cogan

II- Vasculitis associated with possible etiology:

"DRIM"

- D: Drugs: cocaine or hydralazine
- R: Radiotherapy
- I: Infection

  Bacterial: TB, syphilis

Viral: HBV, HCV, Herpes

Fungal: Aspergillus

- M: Malignancy

III-Vasculitis associated with Systemic disease:

- Antiphospholipid syndrome
- SLE
- Rheumatoid arthritis
- Scleroderma

IV: Characteristic features of each type of vasculitis:

© Small vessel vasculitis:

1-Wegenr Vasculitis:

- Epistaxis
- Nasal crust
- Recurrent sinusitis
- Hematuria (Glomerulonephritis)

2-Good pasture syndrome:

- Hemoptysis
- Hematuria (Glomerulonephritis)

3-Churg Strauss:

- Myocarditis
- Coronary vasculitis
- Cutaneous manifestation
- Eosinophilia
- Kidney affection
- Lung affection: Bronchial asthma

4-Henoch Schoenlein purpura:

- Purpuric eruption
- Arthralgia
- Hematuria (Glomerulonephritis)

©Medium size Vasculitis:

1-Polyarteritis nodosa

- Systemic hypertension
- Cutaneous manifestation similar to Erythema nodosum

- Neuropathy especially Mononeuritis multiplex
- Glomerulonephritis and Renal infarction
- Lung affection (alveolitis and lung infarction)
- Coronary artery vasculitis (beaded appearance in coronary angiography)

2-Kawasaki disease:

Acute phase (during childhood):

- Fever, skin rash, lymphadenopathy, strawberry tongue

Cardiac manifestations:

- Myocarditis
- Aortic incompetence, mitral incompetence
- Aorta and Pulmonary artery Vasculitis
- Coronary vasculitis: result in coronary aneurysms

©Large vessel vasculitis:

1-Takayasu syndrome:

- More common in females
- Upper limb systemic hypertension
- Lower limb claudication
- Unequal brachial pulse volume in both upper limbs
- Unequal blood pressure (usually left subclavian vasculitis)
- Aortic obstruction more common (aortic aneurysm less common)

Diagnostic criteria for Takayasu vasculitis According to American college of rheumatology (ACR), you need 3 of the following 6 criteria:

1-Age at disease onset below 40 years

2-Claudication of the extremities
3-Decrease pulsation of 1 or both brachial arteries

4-Difference of at least 10mmHg in SBP between both arms

5-Bruit over one or both subclavian arteries or abdominal aorta

6-Arteriographic narrowing or occlusion of entire aorta, its primary branches or large arteries in proximal upper or lower extremities not due to atherosclerosis or Fibromuscular dysplasia or other causes

2-Giant cell vasculitis (temporal arteritis):

- Above Age of 50 years
- Affect extracranial portions of aortic arch branches
- Scalp tenderness
- Weak superficial temporal artery pulsation

- Stroke
- Blindness
- Jaw claudication
- Aortic aneurysm

©Variable size Vasculitis: Behcet disease

*Diagnostic criteria include **recurrent oral ulcers** (at least 3 times per year) plus 2 of the following:*

- **Recurrent genital ulcers**
- **Eye manifestations:**
  1-Hypopyon
  2-Uveitis
  3-Retinal vasculitis
- **Skin manifestations:**

  1-Erythema nodosum
  2-Pseduofolliculitis
  3-Acneform nodules

  4-Pathergy test (papule/pustule at the site of needle prick within 24-48 hours)

Cardiovascular manifestations of Behcet:

- Aortic aneurysm
- Pulmonary aneurysm
- Venous Thromboembolism
- Superficial thrombophlebitis

©Vasculitis associated with Systemic disease:

Antiphospholipid syndrome (APS)

Criteria for diagnosis include clinical and lab criteria; you need one clinical criterion and one lab criterion)

Clinical criteria:

- Recurrent arterial or venous thromboembolism
- Recurrent abortion (after first trimester)

Lab criteria:

- Lupus anticoagulant
- Anticardiolipin antibodies
- Anti-beta two glycoprotein

  (2 positive results 12 weeks apart)

Sleep apnea and cardiovascular diseases

I-Definition, etiology and mechanism of cardiovascular risk

©Obstructive Sleep apnea:

Definition:

- Defined as more than 5 episodes of apnea or hypopnea per hour sleep
- Higher incidence in obese patients

Etiology:

- Secondary to inspiratory collapse of airway during sleep

Mechanism of cardiovascular risk

During nighttime:

- Nocturnal hypoxia and hypercapnia stimulate chemoreceptors which result in sympathetic activation

During daytime:

(MIECOS)

- **M**etabolic dysregulation (insulin and leptin resistance)
- **I**nflammatory state (increase in CRP, amyloid A)
- **E**ndothelial dysfunction
- Hyper**c**oagulable state
- Oxidative stress
- Sympathetic over activity (vasoconstriction of blood vessels and increase in HR)

©Central Sleep apnea

Etiology

- In patients with heart failure with increase in LV filling pressure and subsequent pulmonary congestion
- Pulmonary congestion simulates lung vagal irritant receptors
- Stimulations of lung vagal irritant receptors results in abrupt hyperventilation
- Hyperventilation results in decrease in PCo2 below threshold for ventilation
- Thus, Trigger sleep apnea

©Mechanism of cardiovascular risk

- Increase in PCo2 and decrease in Po2 stimulate chemoreceptors which leads to arousal and sympathetic stimulation

II-Cardiovascular manifestation of Sleep apnea

© Obstructive Sleep apnea (OSA)

1-Hypertension:

- Resistant hypertension
- Nocturnal, non-dipping Hypertension

2-Heart failure:

- OSA can results in heart failure (mainly be sympathetic over activity)
- Heart failure can results in OSA by soft tissue edema of upper airway (upper airway obstruction)

3-Arrhythmias

- AF
- Bradycardia and heart block
- Brady arrhythmia is caused by Diving reflex (hypoxia, stimulate sympathetic system which results 8n Vasoconstriction in all vessels Except brain and heart, as it stimulates vagal supply of the heart resulting in AV block and sinus arrest
- 50% of sudden cardiac death occur at night (10pm-6 am)

4-Ischemic heart disease:

- OSA is independent predictor of CAD due to MEICOS (See mechanism of CV risk)

5-Stroke:

- Due to AF or MEICOS (see mechanism of CV risk)

©Central Sleep apnea:

- Independent risk factor for Mortality or heart transplantation on patients with HF

III-Diagnosis of Sleep apnea:

1-Clinical suspicion:

- **Type of patient:** Usually Elderly male obese (but not necessarily to be obese) patient with snoring, daytime somnolence and apnea presenting with resistant HTN, resistant HF, recurrent AF or recurrent strokes

2-Investigations:

- Sleep study or polysomnography to calculate Apnea hypopnea index (AHI) which represent number of apnea or hypopnea per Sleep hours
- **To diagnose OSA:**
    - More than 5 attacks with symptoms

      or More than 15 attacks without symptoms
    - More than 30 attacks are associated with increase in CV risk and all-cause Mortality
- **Other tests:**
    - Epworth score
    - Pulse oximetry showing night desaturation

IV-Treatment

© Obstructive Sleep apnea:

1-Life style modification:

Weight loss

Stop alcohol
Avoid sedative drugs

Avoid sleeping over the back

2-CPAP

3-Surgical options in refractory cases:

- Dental appliances to decrease backward displacment of mandible
- Uvuloplasty to decrease airway obstruction
- Tracheostomy in life threatening conditions

©Central Sleep apnea:

- The treatment is mainly the treatment of heart failure

Depression in cardiac patients

I-Why it is important for the Cardiologist to know about depression?

1-Depression affects about 25% of cardiac patients

2-Approximately 25% of chest pain in ER is due to panic attack

3-About 75% of patients with depression presenting with somatic complaints

4-Depression triple all-cause mortality and quadrable the mortality from MI or stroke

5-Patients with depression are more prone to have heart disease

6-Depression is considered one of major cardiovascular risk factors (at least as the same cardiovascular risk as DM and HTN) as shown in INTERHEART study and Framingham study

II-What is the mechanism of cardiovascular risk of depression?

1-Disturbance in hypothalamic-pituitary adrenal axis, which results in increase in cortisol level which enhance atherosclerosis

2-Trigger inflammatory response (as evidenced by increase in CRP level)

3-Enhanced sympathetic activity due to autonomic disturbance

4-Defect in limbic system

5-Platelets hyperactivity with hypercoagulable state

6-Endothelial dysfunction

III-How to diagnose Depression?

You need 5 of these points, (but must include one of first two points) that are present on daily basis for 2 successive weeks. It should be present most of the day and impair function.

1-Depressed mood

2-Anhedonia which means loss of interest in all pleasurable activities

3-Sleep disturbance (insomnia or hypersomnia)

4-Suicidal attempts

5-Pyschomotor agitation or retardation

6-Weight disturbance (un-intentional weight loss or weight gain)

7-Inability to concentrate
8-Guilt sensation

9-Fatigue and Loss of energy

IV- How to treat depression?

1-Group therapy and psychotherapy

2-Behavioral therapy

3-Pharmacological therapy (Selective serotonin reuptake inhibitors, SSRI) which include:

- Escitalopram (Cipralex): Starting dose 10mg
- Sertraline (Lustral): Starting dose 50mg

  The Sad-Heart study showed that sertraline Decreases mortality and non-fatal MI in patients with CAD
- Citalopram (Celexa): Starting dose 20mg
- Fluoxetine (Prozac): Starting dose 20mg
- Paroxetine (Seroxat): Starting dose 20mg

The Reference for this topic is a lecture by our Great Professor Dr. Mohsen Ibrahim, professor of cardiology, Cairo university

Analgesics in cardiac patients

I-How NSAIDs work?

Arachidonic acid is present in 3 organs
1-Stomach
2-Kidney
3-Blood vessels

1-Stomach:

- COX-1 transform Arachidonic acid into PGE2
- Role of PGE2: Gastric protection against peptic ulcer

2-Kidney:

- COX-1 transform Arachidonic acid into PGE2
- COX-2 transform Arachidonic acid into Prostacyclin (Prostaglandin I2)
- Role of PGE2 and Prostacyclin is to increase renal blood flow and increase GFR

3-Blood vessels

- COX-1 transform Arachidonic acid into Thromboxane A2
- Role of Thromboxane A2: VC of blood vessels and increases platelets aggregations
- COX-2 transform Arachidonic acid into prostacyclin
- Role of Prostacyclin: VD of blood vessels and Decrease platelet aggregations

II- Drugs

1-Selective COX-1 inhibitor: Aspirin

- Decreases PGE2 in stomach (risk of peptic ulcer)
- Decreases PGE2 in kidney (risk of nephrotoxicity)
- Decreases Thromboxane A2 in blood vessels (cardioprotective)

2-Selective COX-2 inhibitors: Celecoxib and rofecoxib

- No gastric side effects
- Nephrotoxicity
- Cardiotoxicity by decreasing Prostacyclin (so increase in VC of blood vessels and increase of platelet aggregations due to un-balanced increase in Thromboxane A2 from Arachidonic acid by COX-1)

3-Non-selective COX-1 and COX-2 inhibitors: NSAIDs

- Mild to Moderate anti-inflammatory action:

Such as Naproxen and Ibuprofen

- Marked anti-inflammatory action:
  Such as Diclofenac and indomethacin as well as piroxicam and meloxicam

III-What are the safest analgesics in cardiac patients?

You have to follow the following order when you prescribe analgesics for cardiac patients

1-First line:

Start with

"PNAT"

Paracetamol
Narcotic analgesics
Acetyl salicylic acid
Tramadol

Then
Non-acetylated salicylates (eg: salsalate)

2-Second line:
Naproxen and ibuprofen

3-Third line:
Diclofenac and indomethacin

4-Fourth line:

Piroxicam and meloxicam

Important rules

- ❖ In cardiac patients, avoid selective COX-2 inhibitors (such as celecoxib and rofecoxib)
- ❖ The second, third and fourth lines are NSAIDs.
- ❖ The rule is to avoid the use of NSAIDs (second, third and fourth lines)
- ❖ If you have to give NSAIDs, use the lowest effective doses and for the shortest duration (3-5days)
- ❖ Use Proton pump inhibitors whenever possible
- ❖ Ibuprofen decreases the antiplatelet effects of aspirin if given together
- ❖ So, you have to give aspirin with ibuprofen, give both at different times; Either to give aspirin first, then give Ibuprofen after 2 hours Or to give Ibuprofen first, then give aspirin after 4 hours
- ❖ Aspirin for post MI pericarditis: Up to 1500mg per day, the antiplatelet function of Aspirin is maintained.

Sexual Activity in cardiac patients:

General Recommendations

1-Women with CVD should be counseled regarding the safety and advisability of contraceptive methods and pregnancy when appropriate (Class I; Level of Evidence C).

2-It is reasonable that patients with CVD wishing to initiate or resume sexual activity be evaluated with a thorough medical history and physical examination (Class IIa; Level of Evidence C).

3-Sexual activity is reasonable for patients with CVD who, on clinical evaluation, are determined to be at low risk of cardiovascular complications (Class IIa; Level of Evidence B).

4-Exercise stress testing is reasonable for patients who are not at low cardiovascular risk or have unknown cardiovascular risk to assess exercise capacity and development of symptoms, ischemia, or arrhythmias (Class IIa; Level of Evidence C).

5-Sexual activity is reasonable for patients who can exercise ≥3 to 5 METS without angina, excessive dyspnea, ischemic ST-segment changes, cyanosis, hypotension, or arrhythmia (Class IIa; Level of Evidence C)

6-Cardiac rehabilitation and regular exercise can be useful to reduce the risk of cardiovascular complications with sexual activity for patients with CVD (Class IIa; Level of Evidence B)

7-Patients with unstable, decompensated, and/or severe symptomatic CVD should defer sexual activity until their condition is stabilized and optimally managed (Class III; Level of Evidence C).

8-Patients with CVD who experience cardiovascular symptoms precipitated by sexual activity should defer sexual activity until their condition is stabilized and optimally managed (Class III; Level of Evidence C).

9-Men and women with stable CVD who have no or minimal symptoms during routine activities can engage in sexual activity. This includes patients with:

- Canadian Classification System class I or II angina;
- New York Heart Association (NYHA) class I or II heart failure;
- Mild to moderate valvular disease;
- No symptoms after MI
- Successful coronary revascularization
- Most types of congenital heart disease (CHD)

- Ability to achieve ≥3 to 5 METS during exercise stress testing without angina, ischemic electrocardiographic changes, hypotension, cyanosis, arrhythmia, or excessive dyspnea.

10-In patients with unstable or decompensated heart disease, Sexual activity should be deferred until the patient is stabilized and optimally managed. This include patients with any of the following conditions:

- Unstable angina,
- Decompensated heart failure
- Uncontrolled arrhythmia
- Significantly symptomatic and/or severe valvular disease,

11-In patients whose exercise capacity or cardiovascular risk is unknown, exercise stress testing can be useful to assess exercise capacity and development of symptoms, ischemia, cyanosis, hypotension, or arrhythmias

Reference: Circulation

Cardiovascular contraindications to commercial airline flight (UK guidelines)

1-Uncomclicated MI within 7 days

2-Complicated MI within 4-6 weeks

3-Unstable angina

4-Decompesnated congestive heart failure

5-CABG within 10 days

6-Cerebrovascular accident within 3 days

7-Uncontrolled arrhythmia

8-Severe symptomatic valvular heart disease

UK Driving guidelines for cardiac cases:

1- Private car/Motorcycle

- Treated ACS with normal EF >>No driving for 1 week
- Untreated ACS >> no driving for 1 month.

2- Bus/Lorry?

- No driving for 6 weeks. His license should not be renewed except after informing the Authorities (DVLA) & he should pass Stage 3 on ETT without medications & without ischemic symptoms