Silent ischemia

Angina with normal coronaries

Cardiac risk of non-cardiac surgery

PAD

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Silent ischemia:

- Occurring in up to 20% to 40% of patients with coronary syndromes.
- 21% of MIs are silent.
- Silent ischemia may be documented by a variety of diagnostic modalities, including resting ECG, ambulatory ECG (AECG), nuclear scintigraphy, and echocardiography.

Clinical presentation:

Type I:

- Have asymptomatic ischemia with no history of CAD with asymptomatic MI patterns.
- Patients may also present with arrhythmias or sudden death from subsequent scar. These patients are considered to have an *ineffective "anginal warning system"*
- * Silent ischemia is often discovered by TMT.

Type II: have symptomatic MI but subsequent asymptomatic ischemia due to *abnormal pain threshold.*

Ischemia is often missed because of a lack of symptoms. Patients in this category have a positive TMT or the rarely ordered AECG.

Type III: the largest patient population with silent ischemia. These patients with known CAD have both symptomatic and asymptomatic ischemia. About 75% of ischemic episodes are silent and only 25% are symptomatic.

Diagnostic testing:

1. TMT (without imaging) in asymptomatic patients with possible myocardial ischemia on AECG or severe coronary calcification on EBCT.
2. Use of exercise plus imaging stress testing (echo and nuclear) in asymptomatic patients with positive TMT.
3. Periodic stress test is indicated in asymptomatic and has prior PCI.

Mechanism of silent ischemia:

** The exact explanation for a lack of symptoms remains unknown. It likely represents abnormal modulation of cardiac pain perception.

** The association between diabetes, silent ischemia and painless infarction has been attributed to autonomic neuropathy.

** A higher threshold for pain has been related to increased baseline plasma B-endorphin levels and increased age.

** A potential connection exists between baroreceptor function and pain perception.

** Suggestion, that the afferent signals at the thalamic level may be a potential mechanism for silent ischemia.

Management of silent ischemia:

- Medications effective in preventing symptomatic ischemia (i.e., nitrates, CCB and BB) and in decreasing myocardial O_2 demand are also effective in reducing or eliminating episodes of silent ischemia.

- Metoprolol better than diltiazem in reducing the number and duration of ischemic episodes. However, the combination of CCB and BB was more effective than either agent alone.

- Use of aspirin, BB, ACEI, and statins are highly indicated in asymptomatic patients with evidence of previous MI.

- The combination of anti-anginal drug therapy and aspirin appeared to significantly reduce cardiac events.

Prognosis:

** Myocardial ischemia, whether symptomatic or asymptomatic, is associated with poorer outcomes.

** Patients with frequent and accelerating episodes of ST-segment depression on AECG monitoring are at higher risk for subsequent cardiac events.

** Patients with silent ischemia had a three fold higher risk of subsequent cardiac events.

** Circadian effects of asymptomatic ST depression on AECG have been noted with changes being more common in the morning hours; however, nocturnal ST-segment changes have been associated with multi-vessel CAD or left main narrowing.

Angina with normal coronary arteries

1. ***Syndrome X***

** Effort-induced angina like discomfort in the setting of normal coronary arteries by angiography (without inducible spasm on ergonovine provocation testing).

** This chest pain is usually indistinguishable from ischemic angina caused by obstructive coronary disease and therefore considered a diagnosis of exclusion.

Presentation:

** Syndrome X is a diagnosis given to patients with persistent anginal symptom, often with abnormal stress testing despite a normal angiogram and negative workup for non-cardiac chest pain.

- ✔ Syndrome X has an increased occurrence in female over male (3:1).
- ✔ Abnormal cardiac physical examination findings and LV dysfunction on stress testing are both uncommon in syndrome X
- ✔ Approximately **10%** of patients who report CSA on effort will have angiographically normal coronary arteries.

Etiology and pathophysiology:

1) endothelial dysfunction
2) microvascular ischemia
3) abnormal pain perception

* Behavioral and psychiatric conditions often coexist in syndrome X.

Diagnostic testing:

CTA may play an important role in avoiding invasive angiography.

Treatment:

- ✔ Aggressive modification of cardiac risk factors including lifestyle modification and lipid treatment.
- ✔ BB have been shown to be very effective in controlling anginal symptoms and considered superior to CCB and nitrates.
- ✔ Other treatments that include tricyclic antidepressants (imipramine), oral aminophylline, and estrogen in postmenopausal women (with caution).

Prognosis of syndrome X

** The prognosis for patients is generally favorable with good long-term outcomes.

** Patients with persistent anginal synmtoms and/or evidence of significant myocardial ischemia on stress testing seem to be at higher risk and have significantly higher event rates, including premature death, MI, and stroke (should be treated aggressively with risk factor and lifestyle modification).

2. Coronary artery spasm

** Vasospasm in coronary arteries, rare, may coexist with atheroma, especially in UA and triggered by exertion, emotional stress, cold exposure, cocain use, and drugs.

** Less than 1% of cases, vasospasm may occur without detectable atheroma on angiography.

** This is sometimes known as *vaspspastic*, *variant angina*, and may be accompanied by spontaneous and transient ST elevation on the ECG (*Prinzmetal's angina*), *or angina inversa*.

** Diagnosis made by inducible spasm on ergonovine provocation testing on angiography

** CCB, nitrates and other coronary vasodilators are the most useful therapeutic agents but may be ineffective

***BB is contraindicated in variant angina (block some B2 receptors and leave unopposed alpha-mediated receptors effect, vasoconstrictive)*

3. Takotsubo syndrome

- ✔ (LV apical ballooning syndrome, stress-induced cardiomyopathy or broken heart).

- ✔ Clinical features include sudden onset of chest pain, ECG changes (often ST elevation) mimicking an AMI, usually in the setting of severe emotional distress and catecholamine surge.

- ✔ The angiogram shows normal coronary arteries, and diagnosis is made on the typical appearance seen on LV ventriculogram or echocardiogram with basal hyperkinesia and severe apical systolic wall motion abnormality.
- ✔ Most patients recover LV function and require only hemodynamic and pharmacologic support.

Cardiac risk of non-cardiac surgery

- Non-cardiac surgery, particularly major vascular, abdominal or thoracic surgery can precipitate serious peri-operative cardiac complications, such as MI, VF, pulmonary embolism, cardiac arrest and death, in patients with CAD and other forms of heart disease.

- A hypercoagulable state is part of the normal physiological response to surgery, and may promote coronary thrombosis leading to an ACS in early post-operative period.

- Patients with a history of recent PCI or ACS are at greatest risk and, whenever possible, elective non-cardiac surgery should be avoided for 3 months after such an event.

** Antiplatelet agents, statins and BB reduce the risk of peri-operative MI in patients with CAD and should be prescribed throughout the peri-operative period.

** Careful attention to fluid balance during and after surgery is particularly important in patients with impaired left ventricular function and valvular heart disease because ADH is released as part of the normal physiological response to surgery.

** Patients with severe valvular heart disease, particularly AS and MS, are also at increased risk because they may not be able to increase their cardiac output in response to the stress of surgery.

** AF may be triggered by hypoxia, myocardial ischaemia or heart failure, and is a common postoperative complication in patients with pre-existing heart disease.

Many risk index used to assess the cardiovascular risk in non-cardiac surgery including:

1. Original Goldmans modified cardiac risk index
2. Detsky`s modified cardiac risk index
3. Revised cardiac risk index (Lee criteria): which is the most widely used now, with 6 independent predictors of major cardiac complication:

1. High risk surgery (intrathoracic, intraperitoneal, supra-inguinal procedure)
2. Hx of IHD (history of MI or positive TMT, current complain of chest pain though to be due to MI or ECG with pathological Q-wave, use of nitrate therapy. Prior CABG, PTCA , PCI included if they have only ischemic chest pain
3. Hx of HF (CHF, PND, pulmonary odema, physical examination showing bilateral rales or S3 gallop or chest radiography showing pulmonary vascular redistribution)

4. Hx of cerebrovascular disease Hx of TIA or stroke
5. DM require insulin therapy
6. Serum creatinine more than 2 mg/dl

0= very low risk of (MI, pulmonary embolism, VF, complete heat block, cardiac arrest), 0.4%.
1= low risk, 0.95%
2= moderat risk, 6.6%
3= high risk, > 11%

Major risk factors for cardiac complications of non-cardiac surgery

- ❑ Recent (< 6 mo) MI or unstable angina
- ❑ Severe CAD: left main stem or three vessel disease
- ❑ Severe stable angina on effort
- ❑ Severe left ventricular dysfunction (EF ≤ 35 %)
- ❑ Severe valvular heart disease (especially AS)

Peripheral arterial disease (PAD)

- In developed countries, almost all PAD is due to atherosclerosis and so shares common risk factors with CAD.
- As with CAD, plaque rupture is responsible for the most serious manifestations of PAD.
- Approximately 20% of middle-aged people in the UK have PAD but only one-quarter of them will have symptoms.
- The clinical manifestations depend upon the anatomical site, the presence or absence of a collateral supply, the speed of onset and the mechanism of injury.

Factors influence the clinical manifestation of PAD:

1. Anatomical site:
   - cerebral circulation (TIA, amaurosis fugax, vertebrobasillar insufficiency),
   - renal arteries (RF, HT),
   - mesenteric arteries (mesenteric angina, acute intestinal ischemia),
   - limb (leg more than arm) causing intermittent claudication, CLI and acute limb ischemia.
   - Aorta; aortic dissection, aneurysm

2. Collateral supply

3. Speed of onset: sudden occlusion of normal artery cause severe distal limb ischemia, while slow development induce collateralization.

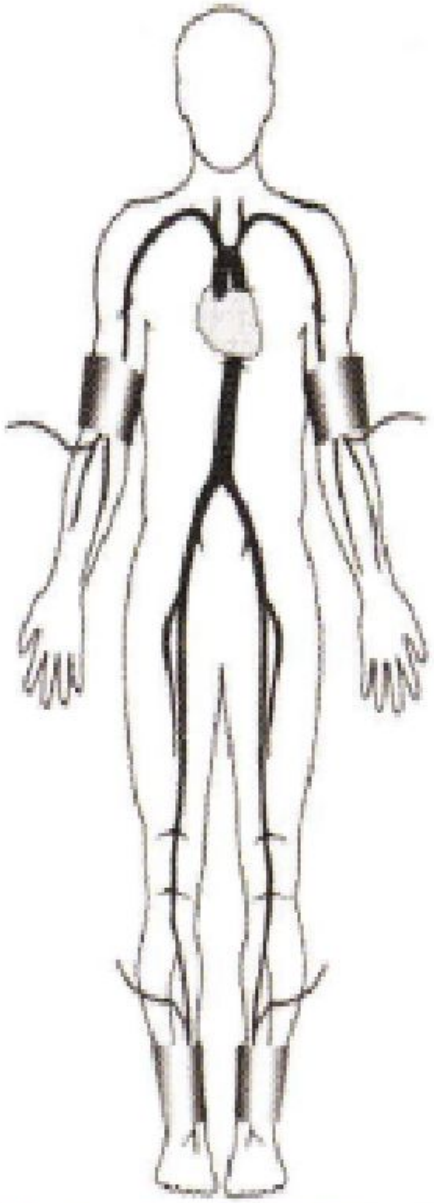
4. Mechanism of injury:

a) Hemodynamic (rupture of plaque and reduce the lumen to more than 70%, causing symptom)
b) Thrombotic (occlusion of long standing may be asymptomatic due to presence of collaterals but acute occlusion may cause severe consequences)
c) Atheroembolic (carotid artery and TIA, or stroke and peripheral arterial plaque causing blue toe or finger syndrome)
d) Thromboembolic (usually secondary to AF)

Recommendation for ankle brachial index,ABPI, screening to detect PAD (In health, the ABPI is 0.9-1.3).

1. Patient with exertional leg symptom
2. Non healing wound
3. Age 65 y or older
4. Age 50 or older with history of smoking or DM
5. TBI should be used to establish PDA in non-compressible vessels

Measurement of the Ankle Brachial Index



Right ABI = ratio of

Higher of the right ankle systolic pressures (posterior tibial or dorsalis pedis)

Higher arm systolic pressure (left or right arm)

Left ABI = ratio of

Higher of the left ankle systolic pressures (posterior tibial or dorsalis pedis)

Higher arm systolic pressure (left or right arm)

Chronic lower limb arterial disease:

- PAD affects the leg eight times more than the arm.
- Lower limb ischaemia presents as two distinct clinical entities: intermittent claudication (IC) and critical limb ischaemia (CLI).
- The presence and severity of ischaemia can be determined by clinical examination and measurement of ABPI.
- In health, the ABI is 0.9-1.3,
- in IC typically 0.5–0.9
- in CLI and severe PAD usually below 0.5.

Intermittent claudication (IC)

- IC describes ischaemic pain affecting the muscles of the leg upon walking.
- The pain is usually felt in the calf because the disease most commonly affects the superficial femoral artery.
- The pain may be felt in the thigh or buttock if the iliac arteries are involved.
- Typically, the pain comes on after a reasonably constant 'claudication distance' and rapidly subsides on stopping walking. Resumption of walking leads to a return of the pain.
- Most patients describe a cyclical pattern of exacerbation and resolution due to the progression of disease and subsequent development of collaterals.

Critical limb ischaemia CLI:

* Defined as rest (night) pain, (requiring opiate analgesia), and/or tissue loss, present for more than 2 weeks, & ankle BP of less than 50 mmHg.

* Rest pain only, with ankle pressures above 50 mmHg, is known as subcritical limb ischaemia (SCLI).

* The term severe limb ischaemia (SLI) is used to describe both CLI and SCLI.

* IC is usually due to single-segment plaque, SLI is always due to multilevel disease. In contrast to patients with IC, those with SLI are at high risk of losing their limb without surgical bypass or endovascular revascularisation (EVR by angioplasty or stenting.

* Imaging is performed using Doppler, MRI or CT. Intra-arterial digital subtraction angiography (IA-DSA) is usually reserved for those undergoing EVR.

Clinical features of CLI:

1. Pulse: diminished or absence
2. Bruits: denote turbulent blood flow
3. Reduce skin T.
4. Pallor on elevation and rubber on dependency (Buerger`s sign)
5. Sluggish filling of superficial veins
6. Muscle wasting
7. Skin and nails: dry, thin , and brittle
8. Loss of hair

:Best medical therapy (BMT) for PAD

Secession of smoking .1
2. Regular exercise (30 m of walking, 3 times / wk)
3. Antiplatelete agents (aspirin 75 mg, clopidogrel 75 mg daily)
4. Peripheral vasodilator, cilostazol, to improve walking distance
Reduce of cholesterol (by diet control and statine therapy) .5
Dx and treatment of DM .6
7. Dx and treatment of frequently associated condition (HT, anemia, HF

Surgical intervention with angioplasty, stenting, ** endarterectomy or bypass is usually only considered after BMT has been given at least 6 months with no symptomatic .improvement, and patients who are severely disabled

Diabetic vascular disease

- Approximately 5–10% of patients with PAD have diabetes and 30–40% in patients with SLI.
- Diabetes does not cause obstructive microangiopathy at the capillary level
- Many diabetic patients present late with extensive tissue loss, which accounts for the high amputation rate.
- Diabetic foot characterized by: heavy arterial calcification, presence of immune compromization, multi-system arterial diseases, distal involvement, sensory neuropathy, motor neuropathy and autonomic neuropathy

Buerger's disease (thromboangiitis obliterans)

- This is an inflammatory obliterative arterial disease that is distinct from atherosclerosis and usually presents in young (20–30 years) male smokers.
- It affects distal arteries, giving rise to claudication in the feet or rest pain in the fingers or toes.
- Wrist and ankle pulses are absent but brachial and popliteal pulses are present.
- Disease also affects the veins, giving rise to superficial thrombophlebitis.
- It often remits if the patient stops smoking; sympathectomy and prostaglandin infusions may be helpful.
- Amputation is frequent outcome if patients continue to smoke.

Chronic upper limb arterial disease

The subclavian artery is the most common site of disease & may manifest as:

- *Arm claudication* (rare)
- *Atheroembolism* (blue finger syndrome). Small emboli lodge in digital arteries and may be confused with Raynaud's phenomenon but, in this case, the symptoms are unilateral.
- *Subclavian steal syndrome*. When the arm is used in the presence of subclavain artery lesion, blood is 'stolen' from the brain via the vertebral artery. This leads to vertebro-basilar ischaemia, which is characterized by dizziness, cortical blindness and/ or collapse. Where possible, subclavian artery disease is treated by means of angioplasty and stenting.


THANK YOU