Aortic Dissection (Dissecting Hematoma) or dissecting aneurysm

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Dissection of blood between & along the laminar planes within the media, with the formation of blood filled channel within the wall of aorta.

Age distribution

Two groups of patients:

I. Men 40 to 60 years of age, in hypertensive patient, form 90% of cases.

II. Younger patient, associated with connective tissue abnormality like Mar fan syndrome.

Etiology of aortic dissection:

1. Mostly unknown (pregnancy).
2. Hypertension.
3. Mar fan syndrome.
4. Complication of arterial cannulation or catheterization, (Iatrogenic aortic dissection).

Important notes:

- ✓ Aortic dissection is unlike the ATH & SYPHILITIC aneurysm, not cause dilatation of aortic wall.
- ✓ Dissection is unusual in presence of ATH & syphilis in wall of aorta, (because scarring of these lesions are protector against the formation of dissecting hematoma).

Types of aortic dissection: according to the site of dissection:

1. Type A. proximal lesions involving either ascending aorta only or both ascending & descending aorta.
2. Type B. distal lesions involving the aorta distal to subclavian artery.

Complications of aortic dissection:

1. Rupture, this is the most common cause of death in patient with aortic dissection, usually rupture into one of body cavities.
2. Retrograde dissection into the aortic root cause disruption of aortic valve, lead to cardiac failure & cardiac temponade.
3. Extension of dissection into great arteries of neck, coronary, renal, mesenteric & iliac arteries causing vascular obstruction & ischemia of these organs.
4. Sometimes, the blood ruptures into the lumen of the aorta, producing a second or distal intimal tear and a new vascular

channel within the media of the aortic wall (to produce a "double-barreled aorta" with "false channel").

5. Compression of spinal arteries causing transverse myelitis.

Clinical features of aortic dissection:
Sudden onset of severe pain, usually at the anterior chest, then radiate to back & moving downward with direction of dissection, (differential diagnosis is myocardial infarction).

Mechanisms of aortic dissection:
- ✓ Aortic dissection is usually due to an intimal tear (original lesion), which extends into but not through the media of the ascending aorta, usually within 10 cm of the aortic valve.
- ✓ Such tears are usually transverse or oblique and 1 to 5 cm in length, with sharp but jagged edges.

Inflammatory diseases (Vasculitidis):
- ✓ Can affect any vessels of any type in any organ.
- ✓ Mechanisms of vasculitis:
  1. Immune-mediated vasculitis (noninfectious vasculitis).
  2. Infectious vasculitis.

Immune mediated (Noninfectious) vasculitis: either
1. Systemic necrotizing vasculitides, affect the aorta and medium-sized vessels.
2. Small vessel vasculitis, affect small vessels, such as arterioles, venules, and capillaries.

Classsification of vasculitidis depend on pathogenesis:
1. Infectious:
   - ✓ Bacterial (e.g., Neisseria), Spirochetal (syphilis), Fungal (aspergillosis) & viral.

2. Immunologic:
   - ✓ Systemic lupus erythematosus and rheumatoid arthritis.
   - ✓ Drug-induced.
   - ✓ Wegener granulomatosis.
   - ✓ Microscopic polyangiitis (microscopic polyarteritis).
   - ✓ Goodpasture syndrome (anti-glomerular basement membrane antibodies).
   - ✓ Kawasaki disease (antiendothelial antibodies).
   - ✓ Inflammatory bowel disease.
   - ✓ Paraneoplastic vasculitis.

3. Unknown:
Giant cell (temporal) arteritis, Takayasu arteritis,
Polyarteritis nodosa (classic polyarteritis nodosa)

Giant Cell (Temporal) Arteritis

- The most common form of the vasculitides.
- Age: older individuals, rare before the age of 50.
- Sites: temporal arteries but also the vertebral and ophthalmic arteries (blindness), can involve any arteries throughout the body (aorta).

Morphology of Giant cell Arteritis: (Two patterns)
1. The commonest pattern characterized by:
   - Nodular thickenings of affected segment of arteries with reduction of the lumen and may become thrombosed.
   - Granulomatous inflammation of the inner half of the media.
   - Fragmentation of the internal elastic lamina.
   - Macrophages & both CD4 & CD8 lymphocytes are frequently seen in close proximity to the damaged elastic lamina.

2. Less common pattern,
   - Granulomas and giant cells are rare or absent.
   - Nonspecific panarteritis with a mixed inflammatory infiltrate (lymphocytes, macrophages, neutrophils and eosinophils).

In the later healed stage of both of these patterns reveals collagenous thickening of the vessel wall; organization of the luminal thrombus sometimes transforms the artery into a fibrous cord.

Pathogenesis: (2 hypotheses)

- ✓ T-cell-mediated immune response to an unknown vascular wall antigen.

(This hypothesis is supported by formation of a granuloma & presence of CD4+ T cells in the lesions).

- ✓ Some mode of inheritance.

Symptoms: Is either facial pain or headache, more intense along the course of the superficial temporal artery, or only vague, fever, fatigue, weight loss-without localizing signs.

Polyarteritis Nodosa

- Polyarteritis nodosa (PAN) is a systemic vasculitis of small or medium-sized muscular arteries (but not arterioles, capillaries, or venules), typically involving renal and visceral vessels but sparing the pulmonary circulation.

Morphology of PAN:

Classic PAN is characterized by

Gross

- Segmental trans mural necrotizing inflammation of arteries of medium to small size, in any organ (with the exception of the lung).
- Segmental erosion with weakening of the arterial wall may cause aneurysmal dilation or localized rupture.
- Impairment of perfusion, causing ulcerations, infarcts, ischemic atrophy, or hemorrhages in the area supplied by these vessels.

Mic:

1. Trans mural inflammation of the arterial wall (neutrophils, eosinophil's, and mononuclear cells and is accompanied by fibrinoid necrosis).
2. The lumen may become thrombosed.
3. Later, fibrous thickening of the vessel wall.
4. Particularly characteristic of PAN is that all stages of activity may coexist in different vessels or even within the same vessel.

Age: young adults, (can in children and elderly).
Course: acute, subacute, or chronic and is frequently remittent and episodic.

Symptoms:

a. Fever of unknown cause, and weight loss; hypertension, usually developing rapidly; abdominal pain and melena (bloody stool) caused by vascular lesions in the gastrointestinal tract; diffuse muscular aches and pains; and peripheral neuritis, which is predominantly motor.
b. Renal involvement is often prominent and a major cause of death.
c. About 30% of patients with PAN have hepatitis B antigen in their serum.
d. There is no association with ANCA.

Takayasu Arteritis:

- ✓ Granulomatous vasculitis of medium and larger arteries.
- ✓ Characterized principally by ocular disturbances and marked weakening of the pulses in the upper extremities (pulseless disease), related to fibrous thickening of the

aorta. (50% of cases have involvement of pulmonary arteries) .

- ✓ Predominantly in females younger than 40 years old.
- ✓ The cause and pathogenesis are unknown, although immune mechanisms are suspected.
- ✓ Mic: this disease is indistinguishable from giant cell (temporal) arteritis when involved aorta. (Distinctions based largely on the age of the patient).

Microscopic polyangiitis (Microscopic polyarteritis, Hypersensitivity, or Leukocytoclastic angiitis);

Differ from polyarteritis nodosa by:

1. Affects arterioles, capillaries, and venules (vessels smaller than those involved in PAN).
2. It typically involves lungs (can any organ).
3. All lesions of microscopic polyangitis tend to be of the same age in a single patient.

Possible etiology:

a. Immunologic reaction to an antigen such as drugs (penicillin).
b. Microorganisms (streptococci).
c. Tumor antigens.
d. Infiltration with neutrophils, (leukocytoclastic angiitis).

THANKS