Aortic Aneurysm

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Aortic Aneurysms
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Aortic Aneurysm Overview
- Aorta is the major blood vessel that supplies blood to the body.
- An aneurysm is a localized dilation or outpouching of a vessel wall.
- Aneurysms can be located in ascending aorta, aortic arch or descending aorta.
- Aneurysms can lead to dissections in the artery wall.
- Three layers of aorta wall (external to internal): tunica adventitia, tunica media, and tunica intima.
- True aneurysm: involves all three layers.
- False aneurysm: does not involve all three layers, extravascular hematoma.
- Stanford Classification of Dissection: Type A: dissection involves ascending aorta.
- Type B: dissection involves descending aorta.
- Aneurysms can cause life-threatening bleeding.

Signs and Symptoms
Non-ruptured Aneurysm
- Can be asymptomatic until pressure on other organs, or ruptures.
- Tenderness/pain in the chest or abdomen.
- Vague persistent back pain.
- Shortness of breath.
- Ruptured Aneurysm
- Borin pain in chest, back or abdomen.
- Tachycardia.
- Loss of consciousness.
- Internal bleeding from rupture.
- Hypovolemic shock.
- Hypotension.
- Cyanosis.
- Skin mottling.
- Altered mental status.

Elastin and collagen type I, III are key structural components of the aortic wall.
Elastin and collagen degradation by proteases leads to vessel dilation and increased strain on medial collagen fibers.
Proteases cause an induction and overexpression of inflammatory mediators.
Early stage: endothelial cells secrete MCP-1 and IL-6.
MCP-1 and IL-6 cause recruitment of monocytes that mature into macrophages.
T-cells are activated by contact with macrophages and IL-12.
Accumulation of MMP, TIMP and collagen weaken adventitia.
Macrophages cause apoptosis of smooth muscle cells leading to reduction in elastin and weakening media.

Pathophysiology
- Elastic degradation and increased strain on medial collagen.
- Vague peritoneal pain.
- Radiating pain to abdomen.
- Tenderness/pain in the chest or abdomen.
- Adenomia soft without pulsatile mass, n tenderness present.
- Aortic CTA showed classic aortic dissection type B from thoracic aorta to distal iliac artery with an intramural hematoma type B.
- Medical management included: HR goal 60 bpm, systolic BP 100-120 mmHg.
- BP treated with IV labetalol and nitroglycerin (nitropusside was not available).
- OR scheduled after control of V/S and aneurysm.
- Repair of aorta was completed with tubular graft.
- Patient was discharged on day 18 and had no complications at follow up appointments.

Aortic mural inflammatory cell infiltration is a central pathologic feature.
- Activated macrophages are strongly related to increased rupture of vessel wall.
- Large inflammatory process promotes the further degradation of vessel wall.
- As elastin and collagen breakdown the aneurysm diameter increases.
- Larger diameter aneurysms increase the risk of dissection and rupture, and places more strain on aortic vessel wall.

Case Study
- 51 year old male with complaints of bilateral flank pain radiating to abdomen.
- History of HTN and 35 pack-year smoking history.
- Vitals: HR 95 BPM, RR 20, BP 230/120, SpO2 94% on room air.
- Peripheral pulses equal, bilateral and full.
- Abdomen soft without pulsatile mass, no tenderness present.
- Aortic CTA showed classic aortic dissection type B from thoracic aorta to distal iliac artery with an intramural hematoma type B.
- Medical management included: HR goal 60 bpm, systolic BP 100-120 mmHg.
- BP treated with IV labetalol and nitroglycerin (nitropusside was not available).
- OR scheduled after control of V/S and aneurysm.
- Repair of aorta was completed with tubular graft.
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Why Aortic Aneurysms
I have taken care of many patients preoperative and postoperatively with aortic aneurysms while working in the cardiovascular intensive care unit. Caring for these patients provided an interest in learning the pathophysiology behind the disease process.

Significance of Pathophysiology
- Aortic mural inflammatory cell infiltration is a central pathologic feature.
- Activated macrophages are strongly related to increased rupture of vessel wall.
- Large inflammatory process promotes the further degradation of vessel wall.
- As elastin and collagen breakdown the aneurysm diameter increases.
- Larger diameter aneurysms increase the risk of dissection and rupture, and places more strain on aortic vessel wall.

Figure 1. Anurysms

Figure 2. CEC.gov

Risk Factors
- Smoking.
- High blood pressure.
- Male gender.
- Age > 60 years old.
- Hereditary link.
- Hypercholesterolemia.
- Connective tissue disorders: Marfan’s syndrome and Ehlers-Danlos syndrome.
- Trauma.
- Inflammatory vasculitis.

Figure 3. Vascular.org

References

Conclusion
- The major risk factors for aneurysm development are high blood pressure, smoking, and male gender.
- Aneurysms are weakened areas in the aorta caused by degradation of elastin and collagen.
- Inflammatory mediators are the major cause of decreased elastin and collagen.
- Type A dissections - ascending aorta, type B dissections - descending aorta.
- Aneurysms progress in size leading to rupture which is associated with a high mortality rate.
- A ruptured aneurysm displays signs of hypovolemic shock with a rapidly falling blood pressure.