aortic aneurysms

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**Aortic Aneurysm**
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**What is an aneurysm?**
- Aortic aneurysms are characterized by local inflammation with degeneration around the aorta, which leads to weakening and widening of the vessel.
- Can be congenital or acquired and occur at different locations of the thoracic or abdominal wall.
- Wall rupture is ultimately a mechanical failure that occurs when intramural stresses exceed wall strength.

**Why Aneurysm**
- Working in a cardiac surgery intensive care has allowed me to take care of patients with aneurysm repairs and it generated an interest to take care of patients with aneurysm.
- Thoracic or abdominal wall aneurysm can occur at different locations of the vessel.

**Pathophysiological process**
- IL-6 has already been suggested as a prognostic biomarker for AAA.
- Elastic strength and displacement under stress depend on the smooth muscle cells in the aortic wall.
- In the early stage of aneurysm, endothelial cells, under hemodynamic shear stress, secrete major attractant protein, MCP-1, and IL-6.
- This triggers recruitment of monocytes from the blood into the media layer of the arterial wall.
- The monocytes mature into macrophages.
- Macrophages from the adventitia are also chemoattracted by MCP-1, IL-6, and IL-8.
- T cells are activated by contact with macrophages in the presence of IL-12, and macrophages are activated by IFN-γ produced by the T cells.
- Fibro- blasts produce collagen, and the collection of ECM, TGF, and Elastase weakens the ability of the adventitia layer to withstand stress.
- Macrophages are known to cause apoptosis in SMCs, and this leads to reduction in elastin, thus weakening the elastic strength of the media.

**Significance of pathophysiology**
- Fatigued hemorrhage, paraplegia caused by interruption of anterior spinal artery, abdominal ischemia stroke, myocardial ischemia, lower extremity ischemia, renal failure, impotence, and cardiac tamponade.
- Sudden, intense and persistent abdominal or back pain, which can be described as a tearing sensation.
- Pain that radiates to your back or legs.
- Sweating.
- Clumminess.
- Dizziness.
- Nausea.
- Vomiting.
- Low blood pressure.
- Fast pulse.

**Risk Factors/ findings**
- The central histological findings in non-syndromic Abdominal thoracic aortic aneurysm (ATAA) analyses are the loss of smooth muscle cells (media degeneration) and the alteration of elastic fiber structures.
- Major factors are: the MMP2/9-TIMP system, smooth muscle stress and cell death, aging processes (telomere length), alterations in genes and protein expression and function.
- The major risk factor for non-syndromic ATAA formation is hypertension, and anti-hyper-tensive therapy is a gold standard.

**Nursing Interventions**
- Monitor for signs and symptoms of spinal cord ischemia such as pain, numbness, paresis, and weakness caused by dissection.
- Monitor for signs of stroke or cardiac tamponade caused by dissection.
- Check extremities for sensation, temperature, pulses, color, capillary refill, and petechiae.
- Teach patient about blood pressure medications and the importance of taking them as prescribed.
- Teach the patient to recognize and report signs and symptoms of an expanding aneurysm or rupture.

**Conclusion**
- Aortic aneurysms result from degeneration of the medial wall, which occurs as a normal part of the aging process as well as with hypertension, atherosclerosis, trauma or infection, immunologic conditions, and as a complication of Marfan Syndrome.
- Thoracoabdominal aortic aneurysm may originate in the ascending aorta and aortic arch (frequent site of dissection) or in the lower descending thoracic aorta and upper abdominal aorta.
- Aortic aneurysms develop by various pathophysiological processes and is clinically significant for life.
- Surgical Intervention is the only way to fix these processes once a certain circumference develops.

**References**
- References