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Abdominal Aortic Aneurysm: A Silent Killer

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Why AAA?

Rupture of an abdominal aortic aneurysm (AAA) is a significant cause of mortality in the United States. "Often asymptomatic, AAA is considered a silent killer because it frequently remains undiagnosed until the time of rupture or the patient's death" (Gordon & Toursarkinssian, p. 242). As healthcare professionals, being aware of the pathophysiology, risk factors and symptoms influencing this disease is important in aiding early diagnosis and treatment, helping to reduce complications and mortality rates of those affected.

Signs and Symptoms

Most patients with abdominal aortic aneurysms are completely asymptomatic and symptoms can go undetected for years. AAAs are often found incidentally, typically on a routine exam or a diagnostic test (Gordon & Toursarkissian, 2014). An aneurysm itself is not an emergency. However, as an aneurysm increases in size, there is an increase risk of rupture. Should a person experience symptoms, they may complain of chest, back or abdominal pain caused by the aneurysm putting pressure on surrounding organs. A pulsatile mass may also be identified in the periumbilical region and tenderness is present with deep palpation. Unfortunately, symptoms may not occur until rupture (Moennich & Mastracci, 2014).

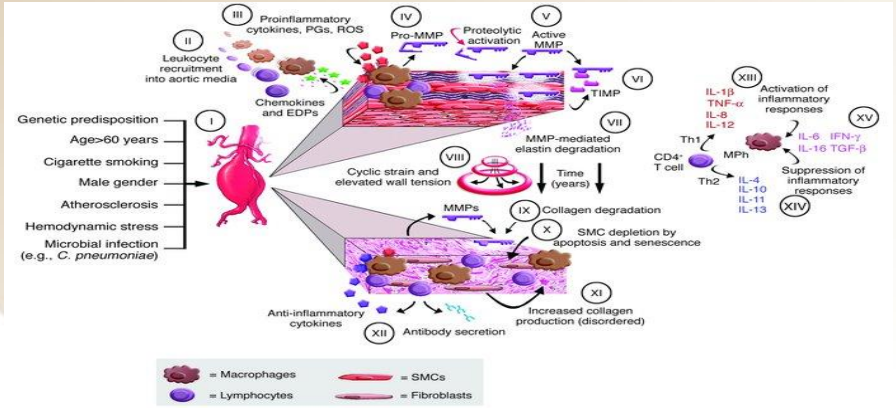
"Sudden, severe pain; symptoms of dizziness, nausea, or vomiting; cold, clammy skin; and a rapid heart rate when standing up should raise concerns about the possibility of a rapidly expanding aneurysm and impending rupture" (Gordon & Toursarkinssian, p.245). A ruptured abdominal aortic aneurysm presents with classic symptoms of sudden severe pain despite relief efforts and hypotension (Irwin, 2007). Pain is caused by expansion of the aortic wall and bleeding into the intravascular thrombotic edge (Gawenda & Brunkwall, 2012). Diminished or absent pulses may be noted along with blood pressure differences in extremities (Williams & Wilkins, 2013). An AAA rupture is life threatening and there is a high risk of severe complications and death (Moennich & Mastracci, 2014).

Significance and Underlying Pathophysiology

The aorta is the largest artery in the body and it originates from the left ventricle of the heart, supplying all of the body's arteries with oxygenated blood. Three layers comprise the wall of the aorta. The tunica adventitia, the outermost layer, is made up of connective and fibrous tissue that help to support the vessel. The middle layer known as the tunica media, is composed of smooth muscle and elastin capable of constricting related to the diameter of the vessel, while the tunica intima, the inner most layer, is elastic with a thin layer of endothelial cells bordering the blood (Irwin, 2007 & Woodrow, 2011). The hemodynamic properties of the cardiovascular system and its stressful forces can put the aorta at risk of a true aneurysm, affecting all three of these layers (Patel & Arora 2008).

An aneurysm is a condition that involves weakening in the walls of a blood vessel causing it to enlarge or dilate. Aneurysms can form in any blood vessel in the body, but are most commonly seen in the aorta and more specifically, the abdominal aorta (Moennich & Mastracci, 2014).

"An AAA is a permanent localized dilation of the abdominal aorta, beginning at the level of the diaphragm and extending to its bifurcation into the left and right common iliac arteries, that exceeds the normal diameter by 50% or is greater than three centimeters" (Li & Dai, p. 1). " The preferential site for AAA formation suggests potential differences in aortic structure, biology and stress along the length of the aorta. There is a natural reduction in the number of elastin layers in the aortic wall, with about half as many layers found in the infra-renal aorta compared to the proximal thoracic aorta. This is likely clinically relevant, since diminished elastin is associated with aortic dilation, while collagen degradation predisposes to aortic rupture" (DiMusto & Upchuch, p. 1).



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The formation of aneurysm involves a multifaceted process of destruction of the aortic media and supporting lamina through degradation of elastin and collagen (DiMusto & Upchurch, 2009). Elastin and collagen are important components of the aortic wall structure. "Elastin is easily stretched and provides the elastic recoil of large arteries, while aortic collagen is coiled such that the initial load in the aorta is borne by elastin'"(Crawford, Hurtgen-Grace, Talaric & Marley, p. 3). Collagen fibers eventually become load bearing at the vessel continues to stretch. Stiffness is created due to the loss of elastin. With years of pulsatile blood flow through degenerated vessel walls, this only exacerbates the process (Crawford et al, 2003). With disruptions in blood flow to vital organs such as the brain, heart, kidneys and GI tract, any complication related to the aorta can be life threatening (Baird, Keen, Swearingen, 2005).

The pathogenesis of AAAs are still unclear, however inflammation is most likely the leading process responsible for the development of changes in the extracellular matrix of the aortic wall, which leads to obliteration of elastin and alteration of collagen fibers. According to Dawson et al. 2006, "studies suggest that aneurysm-derived cytokines perpetuate the cycle of inflammation and proteolysis that is the pathological hallmark of abdominal aortic aneurysms" (p. 324).

The inflammatory cells produce matrix metalloproteinase (MMP) with the ability to degrade the extracellular matrix proteins in the aortic wall, thus the wall gradually loses its elasticity and strength. The process of matrix degradation is regulated by cytokines, which are produced by various cells and are mainly inflammatory. (Vladislav et al, 2002). Additionally there is a depletion of smooth muscle cells due to apoptosis, which assists in worsening of the aortic walls and leads to AAA degeneration (Malekzadeh, Fraga-Silva, Montecucco, Mach, & Stergiopulos, 2013). This deterioration of the aortic wall also leads to inflammatory release from the wall itself, including transforming growth factor-beta (TGF-beta) and vascular endothelia growth factor (VEGF) that lead to further inflammation (DiMusto & Upchurch, 2009).

The initial assault of the aortic wall by inflammatory cells is endothelia injury by various causes including smoking, hypertension, hyperlipidemia, viruses and oxidized lipids (Vladislav et al, 2002).

With advancing age, the elastic in the aorta is decreased and further weakens the wall of the vessel. Acute hypertension may decrease flow into the media, leading to ischemia, which further weakens this layer (Baird, Keen, Swearingen, 2005).

Implications for Nursing Care

The goal from a nursing prospective is to recognize, manage and prevent aortic aneurysm rupture. A considerable amount of interest has been on improving interventions such as surgery, however this cannot benefit those with an undetected aneurysm and certainly those who's aneurysm ruptures and die before reaching the hospital (Scott, R., 2007).

Major risk factors include history of smoking, age, race, male sex, atherosclerosis, hypertension, family history and genetic conditions such as Marfan syndrome (Gordon & Toursarkinssian, 2014). Because AAAs are difficult to diagnose, it is important as healthcare professionals to be aware of these risk factors.

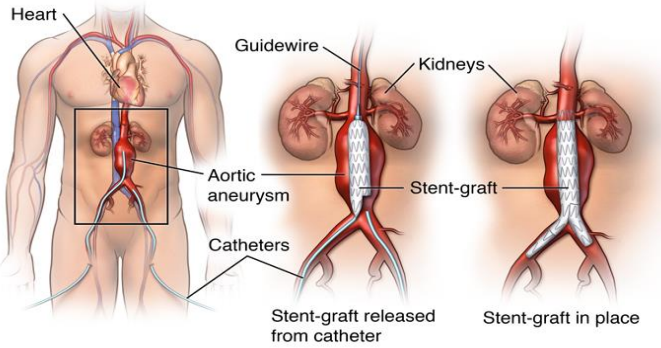
Aneurysm diameter remains the most important clinical determinant for risk of rupture. Due to high mortality rates with surgical intervention, people with elevated BMI over the age of 55 with either hypertension, coronary artery disease, peripheral vascular disease or family history should be routinely screened by ultrasound (Crawford et al. 2003). If an aneurysm is less than 4 cm in diameter, it can be monitored every one to two years and if it's greater than 4 cm, monitoring should occur every six months to a year for safety (Gordon & Toursarkinssian, p. 245).

"The annual risk of rupture is low in patients who have an AAA that is less than 5.5 cm in diameter and size and has been stable, thus intervention is not recommended. However patients who have an AAA that is greater than 5.5 cm or an annual expansion of 1 cm or greater in a year are at high risk of rupture and are candidates for aneurysm repair" (Gordon & Toursarkinssian, p. 245).

Antihypertensive such as beta-blockers or ACE-inhibitors should be started as soon as the diagnosis is suspected. The goal is to reduce the systemic arterial pressure to an appropriate level without compromising vital organ perfusion (Olusen, Jacobs & Amanullah, 2008). Statins, macrolides, diet, and smoking cessation all play a role in slowing the progression of an AAA (Baxter, Terrin & Dalman, 2008).

Surgical intervention for an AAA is required when the aneurysm is 5 cm or more in diameter. Surgery is definitive but risky. The aneurysm is resected and the damaged portion of the aorta is replaced with a graft. A less invasive option is an endovascular repair in which the walls of the aorta are reinforced to prevent enlargement and rupture (Williams & Wilkins, 2013).

Endovascular aneurysm repair (EVAR), abdominal aortic aneurysm (AAA)



Conclusion

Abdominal aortic aneurysms carry a mortality rate of nearly eighty percent (Gordon & Toursarkinssian, 2014). The diagnosis of an aneurysm itself is not an emergency, but when the vessel ruptures it becomes a life-threatening situation with a high probability of death. It is crucial to the survival of these patients that recognition, prevention, diagnosis, safe and effective medical management and surgical techniques assist in reducing morbidity and mortality rates of those affected by this silent killer disease.

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