Abdominal Aortic Aneurysm: A Silent Killer

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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 undetected until the time of rupture or the patient’s death (Gordon & Toursarkissian, 2014). A ruptured aortic aneurysm increases the patient’s chance of death dramatically. Early diagnosis and treatment are critical to reduce mortality and morbidity associated with AAA.

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 intima, the endoluminal 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 elastic tissue forming the wall of the aorta, with a layer of elastic tissue. The intima is a lumen with the diameter of the outermost layer of endothelial cells bordering the lumen (Irwin, 2007; Woodcock, 2011).

The hemodynamics 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 et al. 2008). AAA begins as a localized dilation of the abdominal aorta, beginning at the level of the diaphragm and extending to its common iliac arteries (Crawford et al. 2012). The most likely cause of AAA is atherosclerosis. In the aorta, this is borne by smooth muscle and only a small amount of collagen. As the aorta is stretched beyond its normal limits, it leaks blood into the surrounding tissue, creating a pulsatile mass. The symptoms may be noted along with blood pressure changes or palpitations. The true aneurysm is a condition that involves weakening in the walls of a blood vessel causing it to enlarge or dilate. An aneurysm itself is not a medical emergency, however, if it is not treated, a rupture can cause severe complications and death (Irwin, 2007).

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 common iliac arteries. At the level of the lumbosacral spine, the AAA is approximately 6-10 cm in diameter. There is a plethora of research investigating the diagnosis and treatment of AAA (Moennich & Gawenda, 2014). The wall of the aorta is made up of three layers: the inner layer known as the tunica intima, the middle layer comprised of the tunica media, and the outer layer of the tunica adventitia (Patel et al. 2008). The tunica adventitia is made up of collagen and elastin, which are responsible for the elasticity and strength of the vessel wall. The tunica media is the middle layer and is made up of smooth muscle cells with elastic fibers. The tunica intima is composed of the inner lining of the vessel. The elastic fibers of the tunica media are responsible for the elasticity of the vessel wall (Crawford et al. 2012). Type I and Type II aneurysms are most commonly found in the infrarenal aorta (Crawford et al. 2012). A type I aneurysm is found in the infrarenal aorta, whereas a Type II aneurysm is found in the suprarenal aorta (Crawford et al. 2012).

The aorta is composed of three layers: the inner lining of the vessel, which is the tunica intima; the middle layer, comprised of the tunica media; and the outer layer, the tunica adventitia (Moennich & Gawenda, 2014). The tunica intima is composed of the inner lining of the vessel. The tunica media is the middle layer and is made up of smooth muscle cells with elastic fibers. The tunica intima is the innermost layer of the aorta and is composed of endothelial cells and smooth muscle cells (Gawenda et al. 2014). The tunica media is the middle layer of the aorta and is composed of smooth muscle cells and elastic fibers (Baird, Keen, Swearingen, 2005). The tunica adventitia is the outermost layer of the aorta and is made up of connective tissue (Baird, Keen, Swearingen, 2005).

Stiffness is created due to the loss of elastin. With years of pulsatile blood flow through degenerated vessel walls, this elastin may be replaced with a graft. A less invasive option is an endovascular repair in which the walls of the aorta are replaced with a graft. An aneurysm can be routinely screened by ultrasound (Crawford et al. 2003). If an aneurysm is less than 4 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 for rupture. 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)

Heal or kill?

EVAR is performed to prevent the rupture of an aneurysm and to reduce the chance of death from rupture of the aneurysm. EVAR involves the insertion of a graft into the abdominal aorta through a small incision in the groin to repair an abdominal aortic aneurysm (AAA). EVAR is a minimally invasive procedure that replaces the aneurysm sac with a synthetic graft. The graft is attached to the wall of the aorta, and the aneurysm is clamped off, allowing the blood flow to continue through the native aorta (Crawford et al. 2012). EVAR is performed on an outpatient basis and is less invasive than open surgical repair.

References