Summer 2017

Coronary Artery Disease

Matthew Jarboe
matthew.jarboe@otterbein.edu

Coronary Artery Disease

Matthew Jarboe BSN

Otterbein University, Westerville, Ohio

Introduction

Coronary artery disease (CAD) is a highly prevalent disease in the United States populous. U.S. healthcare cost related to CAD was over $126,000,000,000 annually in 2010. The cost is expected to rise 41% by 2040 (Odden, et al., 2011, p 829). CAD is a major burden for the U.S. health care system projected to worsen with the aging of the population.

Coronary artery disease directly impacts my current practice in the Cardio-Vascular Intensive Care Unit (CVICU). Working in the CVICU, a large percentage of the patient population has undergone Coronary Artery Bypass Grafting (CABG). CABG surgery is performed on patients who have developed CAD. Furthering my pathological knowledge of this disease state will improve the quality of education I can deliver to my patients. This project should also improve the level of care I deliver by deepening my insight into this disease.

Additionally, I have a strong family history of CAD, so it is of personal interest.

Signs and Symptoms

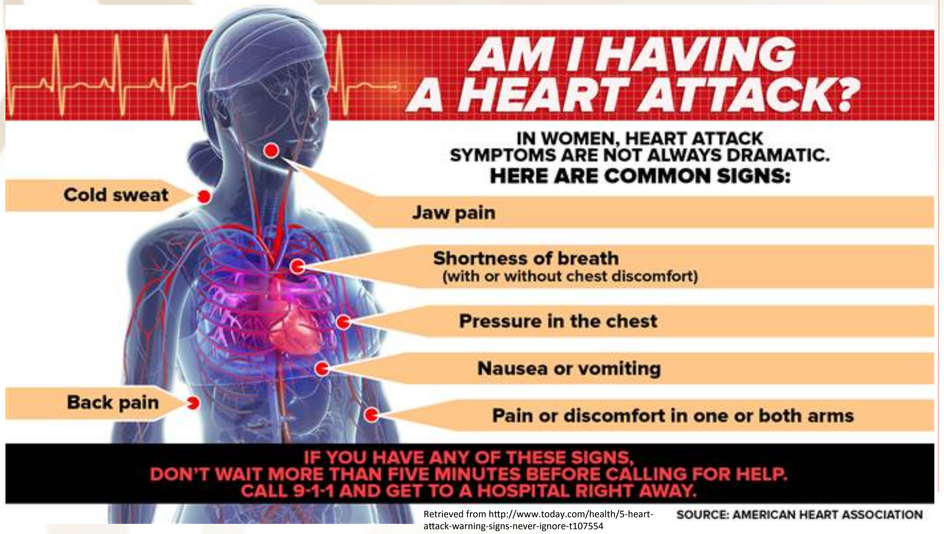
Coronary artery disease is a silent disease process until it progresses to a symptomatic stage. According to the National Heart, Lung and Blood Institute (2016), some people with CAD show no signs or symptoms until they are having a heart attack, arrhythmia, or heart failure. Other people develop signs and symptoms before they develop these related conditions. CAD causes myocardial ischemia, which can present as stable angina or prinzmetal angina.

Signs and symptoms of myocardial ischemia may include left sternal chest pain, chest tightness or pressure, shortness of breath, diaphoresis, and pallor (McCance & Huether, 2014, p1154). After coronary artery disease has progressed; heart failure, arrhythmia, and myocardial infarction (heart attack) can cause more severe signs and symptoms.

Myocardial infarction (MI) is a serious consequence of advanced CAD. The signs and symptoms of a heart attack include sudden crushing chest pain, described as “an elephant sitting on my chest” (McCance & Huether, 2014, p1160).

Nausea and vomiting can be caused from the pain nerve fibers stimulating the vomiting centers. Pain radiates to the back, jaw, neck, left arm, and shoulder. A feeling of relentless indigestion has also been described (McCance & Huether, 2014, p1160). The National Heart, Lung and Blood Institute (2016) list these signs and symptoms of a heart attack: nausea, vomiting, light-headedness, sleep problems, fainting, cold sweats, lack of energy, fatigue, and bilateral arm pain.

Many of the signs and symptoms of heart failure are associated with the hearts inability to pump adequately and the associated fluid build up. The National Heart, Lung and Blood Institute (2016) states that heart failure leads to tiredness, shortness of breath, fatigue, trouble breathing, swelling in the feet, legs, and stomach, and neck vein distention.



Retrieved from http://www.today.com/health/5-heart-attack-warning-signs-never-ignore-t107554

Pathophysiology

Coronary artery disease is the result of atherosclerosis in the arterial vessels of the heart. Atherosclerosis is also responsible for renal artery stenosis, carotid artery stenosis, and lower extremity peripheral artery disease (Imori et al., 2014). Atherosclerosis narrows the lumen of arterial vessels, leading to a decreased supply of oxygen and nutrition to the myocardium. This deprivation in blood flow and the resulting lack of oxygen and nutrients, leads to myocardial ischemia. If ischemia is prolonged or the blood vessel becomes totally occluded it can lead to acute coronary syndrome (McCance & Huether, 2014, p. 1148).

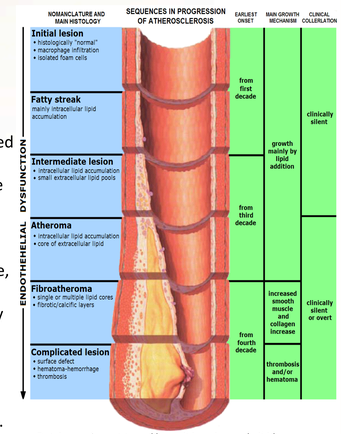
The pathophysiological process of atherosclerosis is important in the development CAD. Listed are the pathophysiological steps leading to atherosclerosis as discussed by McCance and Huether (2014) on pages 1145-1147:

1. Low-density lipoprotein (LDL) accumulates within arterial vessel walls.
2. Injury occurs to the endothelium of the arterial vessel wall. Hypertension, diabetes mellitus, smoking, autoimmune response, and dyslipidemia contribute to this injury.
3. Once damaged, the endothelial cells stop producing adequate amounts of vasodilator and antithrombotic cytokines.
4. Pro-inflammatory cytokine are released by damaged endothelial cell allowing macrophage and leukocyte adherence to the endothelium.
5. Macrophage release toxic oxygen radicals and enzymes that result in oxidative stress, causing LDL to oxidize, further injuring the vessel wall.
6. Oxidized LDL undergo phagocytosis by macrophages creating foam cells.
7. Foam cells accumulate in the endothelium creating fatty streaks. A collagen layer forms over top of the fatty streaks creating a fibrous plaque.
8. Fibrous plaques can decrease blood flow causing signs and symptoms ischemia.

The development of fibrous plaque is the foundation of atherosclerosis. The majority of fibrous plaques have the potential to rupture. Protienases causes apoptosis within the fibrous plague, which leads to bleeding from the plaque. The clotting cascade is activated and a rapid thrombus forms occluding the vessel. Plaque rupture is known as a complicated plaque. “Other causes of thrombosis include plaque erosion which is less frequent than plaque rupture but is a common cause of thrombosis in young individuals especially women <50 years of age” (Sakakura et al., 2013).

Occlusion of the lumen is responsible for many of the signs and symptoms caused by CAD. Calcified plaques are the least likely to rupture and occlude the lumen.

Autoimmune ideologies for decreased lumen size cannot be ruled out. A study by Patel, Anzalone, Buja, and Elghetany (2014) found that Immunoglobulin G4–related disease can result in fibrotic overgrowth in the endothelium causing severe lumen obstruction.



Retrieved from https://en.wikipedia.org/wiki/Atherosclerosis#/media/File:Endo_dysfunction_Athero.PNG

Significance of Pathophysiology

Prevention of atherosclerosis should be the primary goal in treatment of CAD. One of the most widely used medications in the prevention and treatment of CAD are HMG-CoA reductase inhibitors.

> “HMG-CoA reductase inhibitors, also known as statins, selectively inhibit an enzyme called HMG-CoA reductase (3-hydroxy-3-methylglutaryl coenzyme A reductase) that is involved in the synthesis of mevalonate, a precursor of sterols including cholesterol. By inhibiting this enzyme, cholesterol and LDL-cholesterol production is decreased. Statins also increase the number of LDL receptors on liver cells, which enhances the uptake and breakdown of LDL-cholesterol. Most of the effects of statins occur in the liver. Research has shown that elevated levels of total cholesterol, LDL-cholesterol, and apolipoprotein B are risk factors for developing cardiovascular disease.” (Anderson, Stewart, Thornton, Wilson, Fookes, & Puckey, (Eds.). 2017).

Apolipoprotein B (apoB) levels are the best predictor of fatal myocardial infarction. ApoB is a better predictor than LDL at screening for future ischemic cardiovascular events, but LDL is the current standard in the U.S. (Benn, Nordestgaard, Jensen, & Tybjaerg-Hansen, 2007, p. 661). Decreasing the circulating LDL level forces the body to pull the LDL out of fibrous plaques in the arterial walls. Plaque passivation is the process by which statins stabilize active plaques at risk for rupture (Pradka, 2000, p. 43).

According to Bibbins-Domingo (2016) “USPSTF recommends initiating use of low- to moderate-dose statins in adults aged 40 to 75 years without a history of CVD who have 1 or more CVD risk factors (dyslipidemia, diabetes, hypertension, or smoking) and a calculated 10-year CVD event risk of 10% or greater (B recommendation).” While the benefits of statins in the prevention of CAD has been well documented; research conducted by Rothschild, Novak, and Rich (2016) found that statin therapy did not significantly improve mortality in individuals over the age of 80 who were hospitalized with myocardial infarction.

Treatment of the other underlying causes of CAD is important as well. Controlling hypertension and diabetes mellitus are crucial components in preventing CAD. Tobacco cessation should also be recommended. Surprisingly, a study by Reis et al. (2017) found that Marijuana use is not associated with an increase risk of CAD. Understanding the root causes of CAD will lead to better treatment recommendations from providers. Unfortunately, genetic testing has not been found to be economically viable in detecting CAD (Hochheiser, Juusola, Monane, & Ladapo, 2014). Developing better testing and treatment options could mitigate the future cost of this highly prevalent disease.

Implications for Nursing Care

Listed are the steps needed to reduce the risk of cardiac events (Pradka, 2000, p. 43):

1. smoking cessation
2. hypertension control
3. regular physical activity
4. aggressive lowering of elevated low-density lipoprotein (LDL) values
5. losing weight
6. making healthier dietary choices

A study by Dmitrieva, and Burg (2015), found that elevated serum sodium levels, even within the normal range, is associated with vascular changes that facilitate CAD. The U.S. Food and Drug Administration (2016) recommends 2300mg of sodium per day for U.S. adults and 1500mg per day for hypertensive and pre-hypertensive individuals.

Medication education is another important area of nursing care. Patients need to understand why they are taking their medications, and the importance these medications have in treating their condition. When patients do not understand why they are taking medications, they are more likely to stop taking them without consulting their provider.

Conclusion

Coronary artery disease affects millions of Americans. The consequences of untreated CAD can be detrimental to ones quality of life. There are modifiable risk factors such as dyslipidemia, hypertension, and diabetes mellitus that should be controlled when possible. Lifestyle changes can also have a significant impact on the course of CAD. As healthcare providers, we should do our best to minimize the impact that coronary artery disease has on our patient population.

References

Benn, M., Nordestgaard, B. G., Jensen, G. B., & Tybjaerg-Hansen, A. (2007). Improving prediction of ischemic cardiovascular disease in the general population using apolipoprotein B: the Copenhagen City Heart Study. *Arteriosclerosis, Thrombosis, And Vascular Biology, 27*(3), 661-670.

Bibbins-Domingo, K., Grossman, D. C., Curry, S. J., Davidson, K. W., Epling, J. J., García F. R., & ... Pignone, M. P. (2016). Statin Use for the Primary Prevention of Cardiovascular Disease in Adults: US Preventive Services Task Force Recommendation Statement. JAMA: Journal Of The American Medical Association, 316(19), 1997-2007. doi:10.1001/jama.2016.15450

Dmitrieva, N. I., & Burg, M. B. (2015). Elevated Sodium and Dehydration Stimulate Inflammatory Signaling in Endothelial Cells and Promote Atherosclerosis. *Plos ONE, 10*(6), 1-22. doi:10.1371/journal.pone.0128870

Anderson, L. A., Stewart, J., Thornton, P., Wilson, K., Fookes, C., & Puckey, M. (Eds.). (2017). *Statins: What are statins.* Retrieved from Drugs.com website: https://www.drugs.com/drug-class/hmg-coa-reductase-inhibitors.html

Hochheiser, L. I., Juusola, J. L., Monane, M., & Ladapo, J. A. (2014). Economic Utility of a Blood-Based Genomic Test for the Assessment of Patients with Symptoms Suggestive of Obstructive Coronary Artery Disease. *Population Health Management, 17*(5), 287-296. doi:10.1089/pop.2013.0096

Imori, Y., Akasaka, T., Ochiai, T., Oyama, K., Tobita, K., Shishido, K., & ... Saito, S. (2014). Co-existence of carotid artery disease, renal artery stenosis, and lower extremity peripheral arterial disease in patients with coronary artery disease. *American Journal Of Cardiology, 113*(1), 30-35. doi:10.1016/j.amjcard.2013.09.015

McCance, K., & Huether, S. (2014). The cardiovascular and lymphatic systems. In V. Brasher, & R. Neals (Eds.), *Pathophysiology: The biologic basis for disease in adult and children.* (pp. 1145-1163, 7th ed.). St. Louis, MO: Elsevier.

Odden, M. C., Coxson, P. G., Moran, A., Lightwood, J. M., Goldman, L., & Bibbins-Domingo, K. (2011). The Impact of the Aging Population on Coronary Heart Disease in the U.S. *The American Journal of Medicine, 124*(9), 827-833. Retrieved from http://doi.org/10.1016/j.amjmed.2011.04.010

Patel, N. R., Anzalone, M. L., Buja, L. M., & Elghetany, M. T. (2014). Sudden Cardiac Death Due to Coronary Artery Involvement by IgG4-Related Disease. *Archives Of Pathology & Laboratory Medicine, 138*(6), 833-836. doi:10.5858/ arpa.2012-0614-CR

Pradka, L. (2000). Lipids -- how low do you go: plaque regression and passivation. *Journal Of Cardiovascular Nursing, 15*(1), 43-53.

Reis, J. P., Auer, R., Bancks, M. P., Goff, D. C., Lewis, C. E., Pletcher, M. J., Rana, J. S., Shikany, J. M., & Sidney, S. (2017). Cumulative Lifetime Marijuana Use and Incident Cardiovascular Disease in Middle Age: The Coronary Artery Risk Development in Young Adults (CARDIA) Study. *American Journal Of Public Health, 107*(4), 601-606. doi:10.2105/AJPH.2017.303654

Rothschild, D. P., Novak, E., & Rich, M. W. (2016). Effect of Statin Therapy on Mortality in Older Adults Hospitalized with Coronary Artery Disease: A Propensity-Adjusted Analysis. *Journal Of The American Geriatrics Society, 64*(7), 1475-1479. doi:10.1111/jgs.14207

Sakakura, K., Nakano, M., Otsuka, F., Ladich, E., Kolodgie, F. D., & Virmani, R. (2013). Pathophysiology of Atherosclerosis Plaque Progression. *Heart, Lung & Circulation, 22*(6), 399-411. doi:10.1016/j.hlc.2013.03.001

U.S. Department of Health and Human Services, National Institutes of Health, National Heart, Lung, and Blood Institute. (2016) *What are the signs and symptoms of coronary heart disease?* Retrieved from https://www.nhlbi.nih.gov/health/health-topics/topics/cad/signs

U.S. Department of Health and Human Services, U.S. Food and Drug Administration. (2016). *Sodium in your diet: Use the nutrition facts label and reduce your intake.* Retrieved from https://www.fda.gov/food/resourcesforyou/consumers/ucm315393.htm

OTTERBEIN UNIVERSITY