Atrial Fibrillation
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Introduction
Atrial fibrillation is one of the most common heart arrhythmias (Schumacher et al., 2018). From diagnosis to treatment, some risk factors that are common causes of heart arrhythmia is atrial fibrillation. So many patients are not knowledgeable about the risk factors and can misdiagnosis as other diseases. The practitioner can further assist patients by educating patient’s on actions of fatty acids, calcium homeostasis, PR interval, atrioventricular conduction, and how sinus node dysfunction can contribute to atrial fibrillation (Schumacher et al., 2018).

Presentation of Case
A 45-year-old male with no prior history of cardiovascular disease comes to the emergency department with complaints of shortness of breath and bilateral lower extremity edema for one month. He stated that brought him to the ED was his chest pain that did not go away after he rested today. He is at home with a heart rate of 160, blood pressure 120/80, and respiratory rate of 28. His lungs have some rales/fibrosis at the bases. There is noted pitting edema in the ankles and on the legs. ECG results read atrial fibrillation. The physician orders an IV line and to start a normal saline drip titrating his heart rate to under 120. Also, a bias of lifting of Cardiac Sinus is administered. Cardiurus is an antithrombotic drug to help control the heart rate and keep the heart rhythm under control by keeping the heart rate under 120 beats per minute (BPM) (2017). The patient is admitted to the hospital. She had a cath lab test, and the catheter will evaluate him. The echocardiogram and EKG of trigeminal nerve pain. The patient is at the risk of blood clots the physicians order “Paracetamol based per patient’s weight” will be administered. The cardiologist considers stopping calcium loading, normalization the rhythm back into sinus rhythm. Cardiologist decides to wait one month on blood thinners to do a cardiac test to see if the patient is in atrial fibrillation and obtain new cardiac rhythm. Echocardiogram is ordered to see if a conduction of the rhythm system (Zaman, 2017) (Zaman, 2022). This surgery works by牢牢t (614x136 to 808x269) with the tissue to disrupt the Purkinje-electrical signal causing the atrial fibrillation (Jackson, 2017).

Pathophysiological Process

Pathophysiology
Atrial Fibrillation occurs when the atria become so irritable that it no longer is beating, but mostly quivering ineffectively. Electrical impulses rapidly start in the sinoatrial node, and a cardiac action potential is traveling throughout the atrial muscle and down to a ventricular node. Action potential is then redirected to the atrophic ventricular node because of the fibrillar tissue. This fibrillar tissue has a more rapid and chaotic, this increases the ventricular volume and creates a cardiac conversion. Calcium enters atrial cells with each action potential. When atrial potentials are rapid and chaotic, this increases calcium entry and causes calcium production and calcium loading. Homeostasis is initiated by protective mechanisms that reduce calcium entry and promote conversion of calcium-potassium current (Denham et al., 2018). Calcium remodeling at the atrial fibrillation by releasing calcium from the sarcomere in the channels. Calcium remodeling is a protective process that can eventually develop atrial fibrillation. This action shows the strong influence of calcium remodeling abilities to initiate atrial fibrillation (Denham et al., 2018). Calcium and other electrolytes can affect the conduction by prolonging the PR interval. Prolonging of the PR interval happens with the result of the fibrillar tissue. When PR interval prolongation happens, it can lead to a mis firing of the systemic can cause atrial fibrillation or other heart conditions (Schumacher et al., 2018).

Significance of Underlying Pathophysiology
The exact mechanisms by which cardiovascular risks predispose patients to atrial fibrillation are unknown. Some common risks factor associate with atrial fibrillation is neurotransmitter excess, homocysteine stress, atrial electrical, atrial inflammation, metabolic stress, and neurohormonal cascade activation is thought to cause this arrhythmia. The risks are concluded that the grouping of fatty acids plays a part in risk factors of atrial fibrillation. Fatty acids patterns in adipose tissue have diverse sources and metabolic pathways. These increase the chances of atrial fibrillation to develop (Dinesen et al., 2018). Generally, with atrial fibrillation will develop congestive heart failure (Carlisle, 2019).

Underlying Pathophysiology
Depolarization facilitates a small amount of calcium in each cell from the cardiac calcium channel at the beginning of each contraction. Calcium enters atrial cells with each action potential. When atrial potentials are rapid and chaotic, this increases calcium entry and causes calcium production and calcium loading. Homeostasis is initiated by protective mechanisms that reduce calcium entry and promote conversion of calcium-potassium current (Denham et al., 2018). Calcium remodeling at the atrial fibrillation by releasing calcium from the sarcomere in the channels. Calcium remodeling is a protective process that can eventually develop atrial fibrillation. This action shows the strong influence of calcium remodeling abilities to initiate atrial fibrillation (Denham et al., 2018). Calcium and other electrolytes can affect the conduction by prolonging the PR interval. Prolonging of the PR interval happens with the result of the fibrillar tissue. When PR interval prolongation happens, it can lead to a mis firing of the systemic can cause atrial fibrillation or other heart conditions (Schumacher et al., 2018).

Signs and Symptoms
Most common symptom is quivering or fluttering heartbeat

• Rapid or irregular heartbeat (rapid, flapping, or pounding)
• General fatigue
• Dizziness
• Shortness of breath
• Weakness
• Tachypnea or confusion
• Fatigue when exercising
• Sweating
• Chest pain or pressure (CDC, 2017)

Different types of atrial fibrillation Paroxysmal: less than seven days (time over may become more often and last longer leading to chronic Persistent: lasts longer than seven days. Persistent: does not go away no matter the treatment Long-standing: heart rate is irregular and lasts longer than twelve months Nonvalvular: not caused by the heart valve issue All types can increase risk for stroke. (Association, 2020)

AFib: Wait-and-See or Early Cardioversion to Obtain Normal Sinus Rhythm?

Conclusions
Atrial fibrillation can have a range of symptoms and treatment. Best results of atrial fibrillation are knowledge of what to look for to minimize symptoms. Atrial fibrillation is not fatal but needs treatment. Early control of the treatment is to get patient back into a sinus rhythm by medications, cardioversion or surgical intervention if chronic atrial fibrillation.