Pathophysiology

Unstable Angina Pectoris

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**Unstable Angina Pectoris**

**Background**

While walking up a flight of stairs, eating a heavy meal, sleeping or reading a book, a feeling of excruciating pain can be felt in the chest. Many people who have experienced it say it feels like an elephant is sitting on their chest, some say its as if a car was dropped on their chest, but the one question that is on everybodies mind is, am I having a heart attack? Anginal pain mimics a myocardial infarction because the fundamental etiology is the same, the oxygen demand by the myocardium is in an imbalance with the supply. Angina is described as a shortage of oxygen, further defined as ischemia and under the umbrella of acute coronary syndrome, due to decreased blood flow or perfusion to an artery providing blood flow to the heart. The major cause of angina is atherosclerosis. It is estimated that 6 million people have angina in the united states, with an addition of 350,000 newly diagnosed patients per year(1). Due to the similarities in the root of the symptoms in angina and myocardial infarction, a myocardial infarction is a common cause of death in patients suffering from progressive angina. The government and the Center for Disease Control(CDC) are working together to lower the risk of mortality related to these acute coronary syndromes(1).

**Pathophysiology**

In order to understand the pathophysiology, it is first necessary to understand the anatomy. The myocardium is located slightly left and behind of the sternum. On average it pumps 70 beats per minute with 3 ounces of blood being pushed and circulated through the entire body per beat. The myocardium’s blood supply is very oxygen rich due to the location its in and the artery it uses for the blood supply to meet its oxygen demand. The arteries that supply it are the coronary arteries that are the first to branch out of the aorta that is the main pipeline from the left ventricle to the body. Blood passes through the arteries when the myocardium is in relaxation phase, when the right and left ventricles are filling(2). The essential idea is to keep a balance between oxygen supply and demand.

There are several risk factors that lead to atherosclerosis including smoking, high cholesterol diet, alcohol consumption, sedentary life style and family history. An excess of these risk factors leads to the repetitious inflammatory response causing the arteries to narrow decreasing perfusion to the myocardium(2). For example, an excess of low density lipo-protiens can accumulate in the arterial wall and be modified by oxidation this would inflame the endothelial cells leading to the secretion of adhesion molecules so that leukocytes will adhere to the modified cell and penetrate into the intima of the arterial wall. Here, the leukocytes will turn the modified cells into foam cells and will take up the form of the fatty streak causing the wall to grow outwardly. At this point it is considered a lesion, the cells of the lining of the intima start to divide to compensate for the growing lesion and secrete extracellular matrix molecules including collogen, to make this compensatory process at a faster rate, the smooth muscle cells from the media of the artery pass the lesion and situate themselves in the intima forming a fiborous and stiff cap locking in the lipid rich lesion. In the event that there is no modification to the lifestyle and there is no decrease or removal of the source of the risk factor then the lipid lesion would grow further accumulating the foam cells leading to a cascade inflammatory response. T-lymphocytes send a chemical message to the macrophages to solicit activation. Upon macrophage activation it releases cell mediators that are enzymes, such as matrix metalloproteinase, which allow for the fibrous cap of collagen on the intima to weaken and eventually rupture(4). If there is a break or rupture it can cause a thrombus accumulation at which point there is an increase in fibrin causing more clots to develop in that localized area leading to complete blockage causing an ST segment elevation myocardial infarction(3). The majority of those who ask “am I having a heart attack?” actually have an unstable angina, this may occur suddenly during rest. It is an incomplete blockage (70% or less) which shows an ST segment depression. The problem with the incomplete blockage of the thrombus is that when it reabsorbs, it reabsorbs into the layer of the intima as a healing response, leading the intima to further thicken, but this time inwardly causing the lumen to narrow(3). If the risk factors are removed at this point it will allow time for the fatty steak to dissolve by apoptosis and other mechanisms, since the intima is already thickened it might cause stable angina which is during periods of increased oxygen demand, heavy meal, jogging, climbing stairs.

**Research**

Journal of Thrombosis and Haemostasis published a peer reviewed meta-analysis article that gathered results from 16 studies that indicated that the Mean Platelet volume (MPV) be the predictor of coronary artery disease(5). The article defined that based on the pathophysiology of atherosclerosis there is an increase in the activity of platelet aggregation and adhesion, thereby increasing the MPV in unstable angina patients. Due to its cascaded inflammatory response, the higher the level of the MPV marker the closer the individual is to sustaining a myocardial infarction. With an increase in the rate of acute coronary syndrome cases, there is a potential importance in utilizing MPV as a prognostic biomarker and as an indicator for lifestyle modification or further follow up with a cardiologist(5). This would be most beneficial if used in conjunction with the standard risk screening tools, such as, LDL levels. Further implication for this study is to research the effects of modification of the MPV marker would lead to improved outcomes in stable and unstable angina patients.

Another recent research study enrolled 5656 subjects that helped to evaluate whether cardiac events in patients with anginal pain with or with out preexisting coronary disease presenting normal ECG readings would increase risk for further delaying treatment or lifestyle modifications(7). The reason why this study is important is due to its timeliness with the healthypeople2020 expectations. Since the health care industry took a massive shift towards preventative care and primary prevention, this involves education, disease awareness and screening(6). This very recent research study noted that one-third of the individuals who have had anginal pain with negative ECG reading or biomarkers were subsequently found to have acute coronary syndrome(7). Hospitals do not admit individuals that show no valid indication of an acute coronary syndrome. These individuals are treated for anginal pain and discharged and are relieved its not they are not having a heart attack, yet they are at even greater risk for one following a minor ischemic episode. This article can be generalized internationally and amongst all healthcare professionals, the focus is to encourage patients to modify their lifestyle and diet, and seek follow up care with a cardiologist or primary physician.

**Treatment**

Management would include immediate reversal of ischemia by means of nitrates and oxygen. Oxygen limits the injury by increasing the supply to the artery, the goal is to keep the saturation at least 90%. Oxygen alone would not be effective, it needs to be utilized in conjunction with vasodialating agents(8). Vasodialating agents sucha s nitrates, relaxes and dialates the smooth muscle of the arteries and other veins. Dialating the viens would decrease the preload, thereby decreasing the stress on the ventricals. They additionally reduce the afterload which all leads to a decreasing the demand of oxygen by the myocardium(8). Nitrates also have a direct dialating effect on the epicardial coronary arteries and arteriols, resulting in an improved blood flow to the heart supplying the ischemic area with oxygen makes the injury reversible if given without delay. Nitrates are paramount in the management of unstable angina (not in stable angina) and are to be given via sublingually dissolving, IV push or patch in a severe emergency case. Morphine sulfate may be given due to its effects on venodilation and analgesia, this would decrease pain and in-turn decrease the workload of the myocardium and decrease the heart rate due to lowering the demand for oxygen(3). Following nitrate therapy, additional drugs may be used after a work up of the labs to see if cardiac markers indicate an MI.

**Future Research Implications**

Further research needs to address the importance of stress reduction during an acute coronary syndrome. There is an extensive amount of research on pre-angina, management and post-angina, but there is a lack of research on the benefits of reduction in stress during the actual ischemic episode. Various qualitative studies noted that the majority of patients say that feel “impending doom” (4). Stress is a very good source of angina, but in an unstable angina the stress is increased due to a lack of knowledge by the patient. So the already increased demand for oxygen by the myocardium with a lack of supply, is actually increasing the demand for more oxygen because of the added stress. The direction research should take is to develop an understanding based on evidence on how to calm the patient and reduce the risk of progression to an MI. the added stress and feeling of impending doom also increases the heart rate lessening the amount of blood that is being pushed through the coronary arteries because coronary arteries fill during relaxation of the ventricles, but if relaxation is insufficient then the flow to the coronary arteries is, as well, compromised. This research would raise awareness in individuals who already seek treatment for atherosclerosis and encourage healthcare professionals to lower the anxiety of the patient by showing a positive and assertive manner and attitude. Subsequently, this attitude would project a sign of security in the patient regarding treatment and lower the level of stress thereby improving the outcomes and the prognosis.

**Personal Views from the Author**

Just staring my nursing profession, I was offered positions at geriatric facilities and home care visiting nurse agencies, I did not see this in my future. I have been interning at a hospital in the downtown Brooklyn area within the emergency room. I continuously ask to be placed in the cardiac unit of the ER. It is where the action is as well as where my heart lies. My father had chest pain a few times in his life and luckily the cardiac compromise was negative. I always wasnted to learn more about chest pain and the different possibilities it entails. In the cardiac ER, I tend to people with chest pain and I noticed that 1 out of 2 patients is crying and shaking and nervous about whats to come, the patients who have the same symptoms but are more relaxed have a decreased heart rate, get stable quicker and show almost no lingering effects as opposed to the patients who are forgetting to breath, and are scared. I stand by their side telling them exactly what is going to happen in the immediate future, we will draw labs, get an ECG reading and we will immediately get results and as soon as that happens, the doctor will be able to define what is causing this, in the mean time, you need to breath deeply and calmly. I noticed that those who I approached felt relaxed and decreased heart rate by 15 bpm and or systolic blood pressure by 5 to 8 points. What I concluded is that individuals fear what is unknown to them and need to be made aware of what is happening around them and to them in basic sentences so they can breath and decrease the demand of oxygen by the heart thereby decreasing the progression to an MI.

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