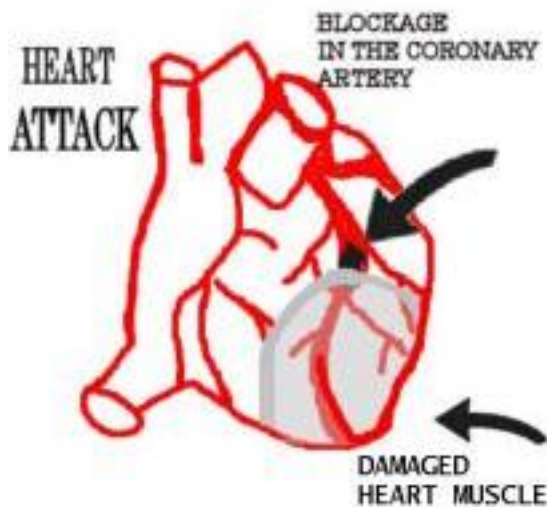




Detection of Myocardial Infarction

A.J. Michael

Fig. 1. Heart Attack



Ischemia is the leading cause of death worldwide and myocardial infarction (MI) causes well over 12 million deaths per year internationally. However, with better detections and treatments, morbidity and mortality of this disease has been lessened.

When experiencing a myocardial infarction, the most evident symptom is the pain or pressure in the chest. Other symptoms include sweating, clamminess, nausea, shortness of breath and vomiting. A study done on differences in symptoms of myocardial infarction between genders found that men reported more chest pain and diaphoresis while women had more atypical syndromes (Patel et al 2004). Atypical syndromes include increased nausea, vomiting, dyspnea, palpitations, indigestion, dizziness, fatigue, loss of appetite and syncope.

Therefore in confirming diagnosis, an electrocardiogram or measuring the serum cardiac markers is a better detection method than just depending on physical syndromes described by the patients. Common cardiac markers include C-reactive pro-

tein (CRP), cardiac troponin I and troponin T, fatty acid binding protein (FABP), myoglobin and B-type natriuretic peptide (BNP). CRP is used frequently to detect general inflammation whereas troponin is the best and most accurate for detecting myocardial damage. Cardiac markers are able to confirm diagnosis of an myocardial infarction within 3 hours by measuring leakages of myocardial enzymes. The healing process begins within 6 to 12 hours following an infarction. Coagulated necrosis occurs in which the muscle fibers become intensely eosinophilic. The infarct is then penetrated by neutrophils by the first day and then the process of replacing and repairing is done by macrophages and reparative cells within the next few days. The healing process is ultimately completed when the damaged heart muscle is replaced by a mature scar tissue.

REFERENCES

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