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Case Report *Medical research*

A case report on management of coronary artery disease

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ABSTRACT

During the past decade, major improvements in the management of Acute Myocardial Infarction (AMI) patients have reduced AMI-related mortality by 50% in developed countries. Specifically, the introduction of intensive care units in the 1960s, improvement in pharmacologic reperfusion in the 1980s, and the widespread availability of percutaneous coronary intervention (PCI) in the 1990s have markedly reduced in-hospital mortality from AMI over the past 15 years.[5] Furthermore, the near-universal acceptance and prompt application of antiplatelet agents, β-adrenergic blockers, angiotensin-converting enzyme (ACE) inhibitors, and statins after an AMI have contributed to the improvement in long-term survival among AMI patients. Still, these great strides cannot diminish the impact of MI. First, the number of at-risk patients (e.g., genetic predisposition, smoking, poor dietary habits, or physical inactivity) is enormous and continues to increase, especially the population of diabetics, elderly patients, and other individuals with metabolic syndrome [5, 6].

One such case with high risk and in need of Coronary Bypass Grafting (CABG) has been treated by Dr. Appa Rao's Immunonutritive therapy and the result was beneficial to the patient. Patient was followed for a year and was found to be healthy.

Keywords: Coronary artery disease, Immunonutritive therapy.

INTRODUCTION

Myocardial infarction (MI) is defined as death of myocardial tissue. The extent and location of the infarction depend on the degree of ischemic burden, the availability of coronary collateral blood flow, the rapidity of reperfusion, and the location of the afflicted coronary artery [1].

During the past decade, major improvements in the management of AMI patients have reduced AMI-related mortality by 50% in developed countries. Specifically, the introduction of intensive care units in the 1960s, improvement in pharmacologic reperfusion in the 1980s, and the widespread availability of percutaneous coronary intervention (PCI) in the 1990s have markedly reduced in-hospital mortality from AMI over the past 15 years. [5] Furthermore, the near-universal acceptance and prompt application of antiplatelet agents, β-adrenergic blockers, angiotensin-converting enzyme (ACE) inhibitors, and statins after an AMI have contributed to the improvement in long-term survival among AMI patients. Still,

these great strides cannot diminish the impact of MI. First, the number of at-risk patients (e.g., genetic predisposition, smoking, poor dietary habits, or physical inactivity) is enormous and continues to increase, especially the population of diabetics, elderly patients, and other individuals with metabolic syndrome. [5, 6]

The underlying cause of coronary artery disease (CAD) is the formation of atherosclerotic plaques and ACS in most patients. The process of atherosclerosis starts early in life. Although atherosclerosis once was considered only a disease of cholesterol excess, it now is clear that inflammation also plays a central role in the genesis, progression, and complication of the disease. [8] In the earliest stage of atherosclerosis—endothelial dysfunction—induction and/or repression of several genes occurs in response to sheer stress of the blood flowing over the atherosclerotic plaque on the endothelial lining of the artery. In response to gene induction and repression, the endothelial cells decrease synthesis of nitric oxide, increase oxidation of lipoproteins and facilitate their entry into the arterial wall, promote the adherence of monocytes to the vessel wall and deposition of extracellular matrix, cause smooth muscle cell proliferation, and release local vasoconstrictor and prothrombotic substances into the blood; each action has a subsequent inflammatory response. [8] Taken together, all these factors contribute to the evolution of endothelial dysfunction to the formation of fatty streaks in the coronary arteries and eventually to atherosclerotic plaques. Therefore, the endothelium serves as an important autocrine and paracrine organ in the development of atherosclerosis.

A number of factors are directly responsible for the development and progression of endothelial dysfunction and atherosclerosis, including hypertension, age, male gender, tobacco use, diabetes mellitus, obesity, and dyslipidemias. [4]

The patient typically is in acute distress and may develop or present with cardiogenic shock. The classic symptom of ACS is midline anterior chest discomfort. Accompanying symptoms may include arm, back or jaw pain, nausea, vomiting, or shortness of breath. Patients less likely to present with classic symptoms include elderly patients, diabetic patients, and women. No signs are classic for ACS. However, patients with ACS may present with signs of acute heart failure, including jugular venous distension, rales, and S3 sound on auscultation. Patients may present with arrhythmias and therefore may have tachycardia, bradycardia, or heart block. Troponin I or T and creatine kinase MB are measured. Blood chemistry tests are performed with particular attention to potassium and magnesium, which may affect heart rhythm, and glucose, which when elevated places the patient at higher risk for morbidity and mortality. Serum creatinine level is measured to identify patients who may need dosing adjustments for some pharmacotherapy and patients who are at high risk for morbidity and mortality. Baseline complete blood count and coagulation tests (activated partial thromboplastin time and international normalized ratio) should be obtained because most patients will receive antithrombotic therapy, which increases the risk for bleeding.Fasting lipid panel is obtained. The 12-lead electrocardiogram is the first step in management. Patients are risk stratified into two groups: ST-segment elevation ACS and suspected non–ST segment elevation ACS.During hospitalization, measurement of left ventricular function, such as an echocardiogram, is performed to identify patients with low ejection fractions (<40%) who are at high risk for death following hospital discharge. Selected low-risk patients may undergo early stress testing.

Selecting evidence-based therapies described in the ACC/AHA guidelines for patients without contraindications results in lower mortality. [10, 11] General treatment measures for all STE ACSs and high- and intermediate-risk NSTE ACS patients include admission to hospital, oxygen administration (if oxygen saturation is low, i.e., <90%), continuous multilead ST-segment monitoring for arrhythmias and ischemia, glycemic control, frequent measurement of vital signs, bedrest for 12 hours in hemodynamically stable patients, avoidance of Valsalva maneuver (prescribe stool softeners routinely), and pain relief. [2, 3, 9] Patients with STE ACS are at high risk of death, and efforts to reestablish coronary perfusion should be initiated immediately. Reperfusion therapy should be considered immediately and adjunctive pharmacotherapy initiated.[3] Patients at low risk for death, MI, or the need for urgent coronary artery revascularization typically are evaluated in the emergency department, where serial biochemical marker tests are obtained. If these test results are

negative, the patient may be admitted to a general medical floor with ECG telemetry monitoring for ischemic changes and arrhythmias, undergo a noninvasive stress test, or be discharged from the emergency department. Moderate- and high-risk patients are admitted to a coronary intensive care unit, an intensive care step-down unit, or a general medical floor in the hospital depending on the patient's symptoms and perceived level of risk. High-risk patients should undergo early coronary angiography (within 24–48 hours) and revascularization (with PCI or CABG) if a significant coronary artery stenosis is found[2]. Moderate-risk patients with positive biochemical markers for infarction typically also undergo angiography and revascularization during hospital admission. Moderate-risk patients with negative biochemical markers for infarction also may undergo angiography and revascularization or first undergo a noninvasive stress test; only selected patients with a positive stress test proceed to angiography. Following risk stratification, pharmacotherapy for NSTE ACS is initiated.

CASE PRESENTATION

A 68year old female patient is a known case of Coronary artery disease. She has a history of Anterior Myocardial infarction in 2007, she underwent Coronary angiogram in March'2007 which showed single vessel disease and PTCA/Stent was placed at the right circumflex artery (RCA) and left PTRA. She had severe left ventricular dysfunction and recurrent ventricular failure. She was found to be DSE positive in the month Feb'2008. She again underwent Coronary angiogram in the month of March'2008 which showed triple vessel disease. She suffered with cavernous sinus thrombosis in 2009. Again for the third time she underwent coronary angiogram in the month of May'2014 which revealed severe triple vessel disease. She was admitted to Krishna Institute of Medical sciences Hyderabad with complains of shortness of breath FC II-III with uneasiness since 1 month, history of Paroxysomal Nocturnal dyspnea and no complaints of chest pain/ palpitation/ syncope. She is a known case of Hypertension, Diabetes mellitus and hypothyroidism.

Latest coronary angiogram report and 2D Echo report suggested LVEF to be 35%, with diffuse calcific atherosclerosis, and triple vessel disease. She was advised to undergo bypass surgery i.e., CABG and was declared as a high risk case.

At this juncture the patient visited Dr. Appa Rao and she decided not to go for CABG as her two sons who had already experienced CABG had no benefit out of it. Dr. Appa Rao started his immunonutritive therapy in the months of May'2014.

Patient was followed up until March'2015 and was found to be comfortable within two weeks of starting immunonutritive therapy with no complains and was also found to be healthy

DISCUSSION

The underlying cause of coronary artery disease (CAD) is the formation of atherosclerotic plaques and ACS in most patients. The process of atherosclerosis starts early in life. Although atherosclerosis once was considered only a disease of cholesterol excess, it now is clear that inflammation also plays a central role in the genesis, progression, and complication of the disease. [8]

Dr. Appa Rao's Immunonutritive therapy focuses mainly on Inflammation. In this patient Immunonutritive therapy was initiated, when the patient's Angiogram and 2D Echo report suggested LVEF to be 35%, with diffuse calcific atherosclerosis, and triple vessel disease and she was advised to undergo bypass surgery i.e., CABG and was declared as a high risk case. Immunonutritive therapy has shown beneficial results in this patient.

CONCLUSION

A 68 year old female patient who is a known case of coronary artery disease and had undergone coronary angiogram and PTCA/ Stenting was performed. Later coronary angiogram performed for the second time in 2009 revealed triple vessel disease. Again in 2014 when coronary angiogram was performed it suggested severe triple vessel disease and she complained of shortness of breath FC II-III with uneasiness since 1 month, history of Paroxysomal Nocturnal dyspnea and no complaints of chest pain/ palpitation/ syncope. She is a known case of Hypertension, Diabetes mellitus and hypothyroidism.

The angiogram and 2D Echo reports suggested LVEF to be 35%, with diffuse calcific atherosclerosis, and triple vessel disease. She was

advised to undergo bypass surgery i.e., CABG and was declared as a high risk case.

Patient was not willing to undergo CABG as she could not find any benefit with it when her two sons have undergone the same. She decided to visit Dr. Appa Rao and take his immunonutritive therapy.

She was started on immunonutritive therapy in the month of May'2014 and was followed until March'2015 and was found to be comfortable and health.

Treatment schedule and follow up

Injection Human normal immunoglobulin (12 mg) and histamine dihydrochloride (0.15 mcg), (Belonging to any manufacturer). Two vials once in three days (3 doses) followed by two vials once in a week until 8 weeks. Aceclofenac 100mg twice a day for one month. Prednisolone tapered and maintained 5 mg per day. Ranitidine 150 mg once a day in the morning.

Tomato, Banana fruit, Prawns and milk were restricted in nutrition.

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