



PRE-HOSPITAL MANAGEMENT OF MYOCARDIAL INFARCTION IN DENTAL SETUP-A BRIEF OVERVIEW

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Article Info	ABSTRACT
<p>Received 15/01/2015 Revised 27/02/2015 Accepted 25/03/2015</p> <p>Key words: GTN, dental management, hypertension, oxygen.</p>	<p>Cardiovascular diseases are one of the main causes of death in the developing world. In addition to their associated morbidity, such disorders are important due to the number of affected individuals and the many patients subjected to treatment because of them. The drug treatments used by these patients can give rise to oral manifestations in the form of xerostomia, lichenoid reactions, burning mouth sensation, loss of taste sensation, gingival hyperplasia and bleeding, as well as extraoral manifestations such as sialadenosis. An inadequately controlled cardiological patient constitutes a risk case in dental practice; dental professionals therefore, must take a series of aspects into account before treating such patients, in order to avoid complications.</p>

INTRODUCTION

In the dental clinics during dental procedures, whenever patient complains of chest pain, any kind of dental procedures should be stopped immediately. Patient should be made to relax and if the pain persists for more than 10 minutes then anginal pain should be accounted for. Myocardial infarction occurs when myocardial oxygen demand exceeds available oxygen supplied by the blood for an extended period of time [1]. Management of a patient with acute myocardial infarction (AMI) is a medical emergency. Local guidelines for the management of myocardial infarction should be followed where they exist.

Pathology

Myocardial infarction is defined by pathology as myocardial cell death due to prolonged ischaemia. It takes several hours before myocardial necrosis can be identified by macroscopic or microscopic post-mortem examination.

Myocardial infarctions are usually *classified by size: microscopic* (focal necrosis), *small* [$<10\%$ of the left ventricular (LV) myocardium], *moderate* (10–30% of the LV myocardium), and *large* ($>30\%$ of the LV myocardium) [2].

Myocardial infarction can be defined pathologically as acute, healing, or healed. Acute myocardial infarction is characterized by the presence of polymorphonuclear leukocytes. The presence of mononuclear cells and fibroblasts, and the absence of polymorphonuclear leukocytes characterize healing infarction. Healed infarction is manifested as scar tissue without cellular infiltration [3].

Patients who suffer sudden cardiac death with or without ECG changes suggestive of ischaemia represent a challenging diagnostic group as it is difficult to say with certainty whether these patients succumbed to a myocardial infarction or to an ischaemic event that led to a fatal arrhythmia. The mode of death in these cases is



sudden. These individuals may die before blood samples for biomarkers can be obtained, or these individuals may be in the lag phase before cardiac biomarkers can be identified in the blood [4].

Clinical relevance

Therefore, the management of patients with suspected MI should include control of cardiac discomfort, rapid identification of patients who are candidates for urgent reperfusion therapy, triage of lower-risk patients to the appropriate location in the hospital, and avoidance of inappropriate discharge of patients with MI. Complications may occur immediately following the heart attack (in the acute phase), or may need time to develop (a chronic problem). Acute complications may include heart failure; aneurysm or rupture of the myocardium; mitral regurgitation; arrhythmias, such as ventricular fibrillation, ventricular tachycardia, atrial fibrillation, and heart block. Longer term complications include heart failure, atrial fibrillation, and the increased risk of a second MI [2].

Aims of management

While the primary concern of physicians is to prevent death, those caring for victims of myocardial infarction aim to minimize the patient's discomfort and distress and to limit the extent of myocardial damage. The care can be divided conveniently into four phases [5]:

1. *Emergency care* when the main considerations are to make a rapid diagnosis and early risk stratification, to relieve pain and to prevent or treat cardiac arrest.
2. *Early care* in which the chief considerations are to initiate reperfusion therapy, to limit infarct size and to prevent infarct extension and expansion and to treat immediate complications such as pump failure, shock and life-threatening arrhythmias.
3. *Subsequent care* in which the complications that usually ensue later are addressed.
4. *Risk assessment* and measures to prevent progression of coronary artery disease, new infarction, heart failure and death.

PRE-HOSPITAL MANAGEMENT

Arrange an emergency ambulance if an AMI is suspected. Take an electrocardiogram (ECG) as soon as possible, but do not delay transfer to hospital as an ECG is only of value in pre-hospital management if pre-hospital thrombolysis is being considered. Advised to call for an emergency ambulance if the chest pain is unresponsive to glyceryl trinitrate (GTN) and has been present for longer than 15 minutes or on the basis of general clinical state - eg, severe dyspnoea or pain [4].

Oxygen Administration

Do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry, as soon as possible, ideally before hospital admission [3,5].

Pharmacotherapy

Pain relief with GTN sublingual/ spray and/ or an intravenous opioid 2.5-5 mg diamorphine or 5-10 mg morphine IV with an antiemetic should be achieved. Avoid intramuscular injections, as absorption is unreliable. Aspirin 300 mg should be given orally (dispersible or chewed) [6].

The National Institute for Health and Clinical Excellence (NICE) recommends using intravenous bolus (reteplase or tenecteplase) rather than an infusion for pre-hospital thrombolysis [7].

Aspirin is essential in the management of patients with suspected MI and is effective across the entire spectrum of acute coronary syndromes. Rapid action is achieved by buccal absorption of a chewed 160–325-mg tablet in the dental clinical set up. This measure should be followed by daily oral administration of aspirin in a dose of 75–162 mg [8,9].

For control of discomfort: Sublingual nitroglycerin can be given safely to most patients with MI. Up to three doses of 0.4 mg should be administered at about 5-min intervals [10].

Therapy with nitrates should be avoided in patients who present with low systolic arterial pressure (<90 mmHg) [11].

Morphine is a very effective analgesic for the pain associated with MI. Morphine is routinely administered by repetitive (every 5 min) intravenous injection of small doses (2–4 mg), rather than by the subcutaneous administration of a larger quantity, because absorption may be unpredictable by the latter route [9,12].

Intravenous beta blockers are also useful in the control of the pain of MI. There is evidence that intravenous beta blockers reduce the risks of reinfarction and ventricular fibrillation [13].

Prevention

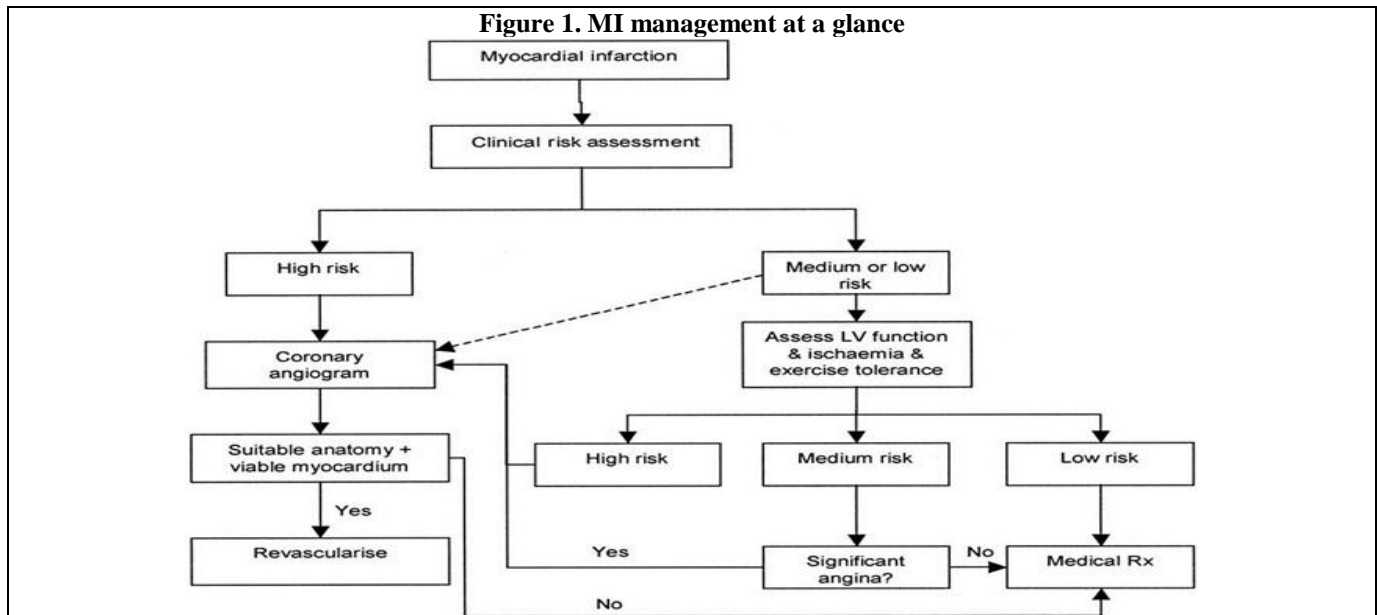
Dental professionals should routinely take complete medical history of the patient. In the cases where the patient gives a history of previous angina pain or MI, stress management protocol should be followed first. The risk of a recurrent MI decreases with strict blood pressure management and lifestyle changes, chiefly smoking cessation, regular exercise, a sensible diet for those with heart disease, and limitation of alcohol intake [13,14].

Aspirin is first-line, owing to its low cost and comparable efficacy, with clopidogrel reserved for patients intolerant of aspirin. Beta blocker therapy such as metoprolol or carvedilol should be started. These have been particularly beneficial in those who are high-risk such as those with left ventricular dysfunction and/or continuing cardiac ischaemia. β -blockers decrease mortality and morbidity. They also improve symptoms of cardiac ischemia in MI [15,16]. Statin therapy has been shown to reduce mortality and morbidity. The general consensus is that statins have plaque stabilization effects that may



prevent myocardial infarction [17]. Evidence supports the consumption of polyunsaturated fats instead of saturated

fats as a measure of decreasing coronary heart disease [18,19].



CONCLUSION

The development of coronary heart disease is attributed to several risk factors such as high serum cholesterol concentration, low serum high density lipoprotein cholesterol concentration, smoking, hypertension, and diabetes. Some dental professionals are not aware of this emergency vital clinical condition.

Therefore, dentists should develop their knowledge on the myocardial management, in order to prevent this life threatening event in their clinical setups. Many aspects of the treatment of MI should be initiated in the dental setup itself and then continued during the in-hospital phase of management.

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