

PERIOPERATIVE PREVENTION AND MANAGEMENT OF MYOCARDIAL INFARCTION

Essay

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Anesthesia

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INTRODUCTION

Perioperative myocardial infarction (PMI) is one of the most important predictors of short- and long-term morbidity and mortality associated with non cardiac surgery. Prevention of a PMI is thus a prerequisite for an improvement in overall postoperative outcome. The design of effective preventive measures requires basic knowledge of the aetiology of PMI. Unfortunately, the exact nature of PMI remains an area of uncertainty and the subject of continued debate and controversy and the aetiology of PMI is multifactorial. The perioperative period induces large, unpredictable and unphysiological changes in sympathetic tone, cardiovascular performance, coagulation and inflammatory response (*Priebe, 2004*).

These changes induce, in turn, unpredictable alterations in plaque morphology, function and progression. Simultaneous perioperative alteration in homeostasis and coronary plaque characteristics may trigger a mismatch of myocardial oxygen supply and demand by numerous mechanisms. If not alleviated in time, it will ultimately result in MI, irrespective of its

aetiology (morphologically, haemodynamically, inflammatory, or coagulation induced). With these many and diverse factors involved, it is highly unlikely that one single intervention will successfully improve cardiac outcome following non-cardiac surgery (*Landesberg, 2003*).

Accordingly, the essay will address the aetiology of PMI, Pathophysiology of acute coronary syndromes, diagnosis, prevention either by coronary interventions or medical treatment especially β -blockers and anaesthetic management of at risk patients.

AIM OF THE WORK

The aim of the work is to show the aetiology of perioperative myocardial infarction (PMI), diagnosis, prevention either by coronary interventions or medical treatment especially β -blockers and anaesthetic management of at risk patients.

ANATOMY AND PHYSIOLOGY

Anatomical considerations:

The heart is the pump of the systemic and pulmonary circulations. Irregular in shape; it lies obliquely across the mediastinum behind the sternum, suspended by the great vessels (*Ellis and Feldman, 1996*).

Blood supply:

Arterial supply to the heart is via the left and right coronary arteries, so termed because they form a circle (corona) around the atrioventricular groove. While these arteries and their branches do anastomose with each other, the anastomoses are not sufficiently large to maintain a collateral circulation if a major branch is occluded. They are therefore functional, if not anatomical, end arteries. Thus the sudden occlusion of a major artery may result in sudden death, but more slowly developing occlusion of smaller branches may allow time for a collateral circulation to develop (*Ellis and Feldman, 1996*).

Arterial blood supply:

The right coronary artery arises from the anterior aortic sinus or right sinus of Valsalva, just above the anterior cusp of the aortic valve. It passes forward between the right atrium and pulmonary trunk and descends along the right atrioventricular groove to the inferior border of the heart where it turns round to the posterior surface and anastomoses with the left coronary artery at the posterior interventricular groove. It gives a marginal branch at the lower border of the heart, which runs to the left towards the apex, and in 80% of cases it terminates as the posterior interventricular branch, which supplies the interventricular septum. In view of the fact that the interventricular septum contains the bundle of His and its branches, the interventricular arteries are of particular importance (especially the inferior one). The right coronary artery supplies the sinoatrial node in 60% of individuals and, in 85%, the atrioventricular node and the posterior and inferior parts of the left ventricle. Conduction abnormalities are commonly associated with the occlusion of the right coronary artery (*Moffat, 1993*).

The left coronary artery arises from the posterior aortic sinus or left sinus of Valsalva. It passes forward

behind the pulmonary trunk and then divides in the space between the aorta and pulmonary artery into the left anterior interventricular artery (anterior descending artery) and the circumflex branch. The anterior interventricular artery passes along the anterior interventricular groove towards the apex, turns round the lower border and anastomoses with the posterior interventricular artery. The major branches of the left anterior interventricular artery are the diagonal branches, which supply the free wall of the left ventricle, and the septal branches, which supply the interventricular septum. The diagonal and septal branches are important landmarks in the description of lesions in the left anterior descending artery (*Lumely et al., 1995*).

The circumflex artery passes laterally around the left border of the heart to reach the posterior interventricular groove. It supplies the sinoatrial node in 40% of individuals and the lateral wall of the ventricle via the marginal arteries (*Lumely et al., 1995*).

Generally, therefore, the left coronary artery supplies the left ventricle and the right coronary artery supplies the right ventricle: both supply the

interventricular septum and the atria are supplied in a variable manner. Variations in this anatomical pattern do occur. In less than 20% of individuals, the circumflex artery give rise to the posterior descending artery and the left coronary artery supplies the whole of the interventricular septum and atrioventricular node. Thus the left coronary artery is the most important supply for the left ventricle unless the posterior descending artery arises from the right coronary artery (*Lumely et al., 1995*).

Venous drainage:

The veins of the heart follow the pattern of the arteries although they have different names. Approximately two-thirds of the myocardial venous drainage is via the coronary sinus and anterior cardiac veins into the right atrium. The remaining blood drains by means of small veins (venae corde minimae) that open directly into the cavities of the heart. The coronary sinus, a continuation of the great cardiac vein, is formed at the left border of the heart and passes to the right in the posterior interventricular groove. It enters the right atrium near the orifice of the inferior vena cava (*Lumely et al., 1995*).

The coronary sinus receives the following:

- Great cardiac vein drains both ventricles; it ascends in the anterior interventricular groove, turns around the left border of the heart and becomes the coronary sinus.
- Middle cardiac vein lies in the posterior interventricular groove.
- Small cardiac vein, this a continuation of the right marginal vein, which runs along the lower border of the heart.
- Oblique vein, this descends obliquely on the back of the left atrium and opens near the left extremity of the coronary sinus, it is a remnant of the left superior vena cava.

The anterior cardiac vein crosses in the anterior cardiac groove. It drains the anterior surface of the heart and opens directly in the right atrium (*Hawng and Sinclair, 1997*).

The coronary venous blood draining (via thebesian. anterior sinusoidal and the anterior luminal veins) directly into the left ventricle constitutes a fixed shunt and contributes to the dilution of oxygenated blood (*Hawng and Sinclair, 1997*).

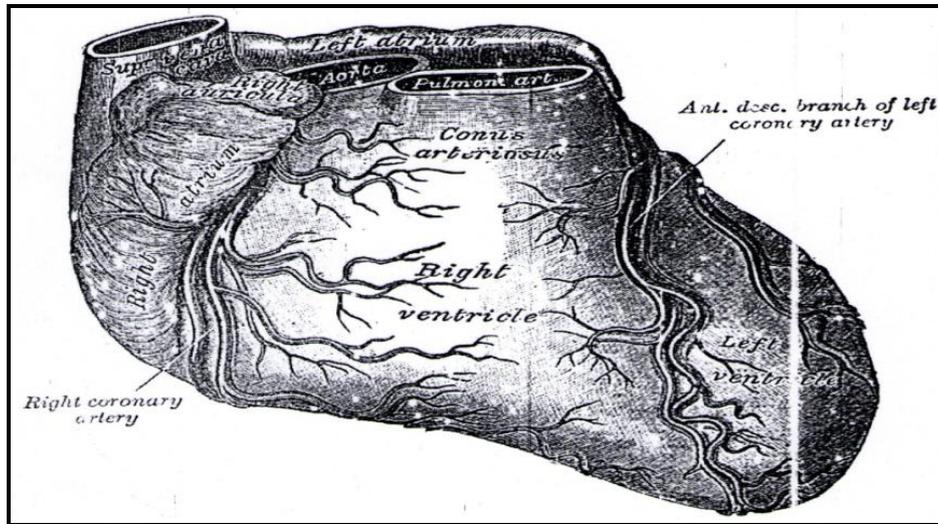


Fig. (1): Anatomy of coronary circulation.

Physiological considerations:

Coronary blood supply:

Myocardial cells receive an abundant blood supply, contain numerous mitochondria in which energy is produced, and have an increased myoglobin content which act as oxygen store. The great majority of the energy used by cardiac cells is provided by aerobic metabolism in the form of ATP. At rest, less than 1% of energy is produced anaerobically although this proportion can increase to 10% under hypoxic conditions. The main metabolic substrates are free fatty acids, glucose and ketones (*Katz, 1975*).

Regulation of coronary blood flow:

At rest, approximately 250 ml min⁻¹ of blood perfuse the coronary arteries, 5% of cardiac output. Compared with other tissues, the extraction of oxygen from blood passing through the coronary circulation is high and can only be increased by about a further 20%. Because of the limited capacity of the heart to provide energy anaerobically, any increased myocardial oxygen demand must be met by improving the oxygen supply by altering coronary blood flow (*Seely et al., 1999*).

At rest, the coronary vasculature is relatively constricted. Increased metabolic demands are therefore met by appropriate coronary vasodilatation, which increases coronary flow up to five folds. There are two mechanisms involved in the regulation of coronary blood flow, localized metabolic control and neurohumoral control (*Seely et al., 1999*).

Metabolic control of coronary blood flow:

Inadequate blood supply to areas of the heart results in hypoxia and accumulation of myocardial metabolites (such as carbon dioxide, phosphate, adenosine, prostaglandins, hydrogen ions and potassium ions). Some of these metabolites have a

major effect on the coronary vasculature, dilating small arterioles and precapillary sphincters to increase local coronary blood flow. Local metabolism thus has a major role in regulating coronary blood flow (*Seely et al., 1999*).

Neurohumoral control of coronary blood flow:

The coronary vessels are innervated by both sympathetic and parasympathetic fibres, but the role of the autonomic nervous system in controlling coronary blood flow is probably minor compared with local effects (*Seely et al., 1999*).

Distribution of coronary blood flow:

Differences in intraventricular pressures created during cardiac systole result in significant differences in the pattern of perfusion between right and left ventricles. During left ventricular systole, myocardial contraction results in the production of a large intraventricular pressure. This pressure is transmitted across the ventricular wall, progressively increasing from the epicardium to the endocardium to the subendocardium. The pressure in the subendocardial layer of the myocardium exceeds systolic arterial blood pressure. Consequently, there is no subendocardial blood flow during systole, and flow

occurs in the arteries supplying the subendocardium of the left ventricle only during diastole. Low pressures in the outer muscle layers of the left ventricle result in some systolic blood flow. In the right ventricle, the lower intraventricular pressures result in flow to all areas of the heart throughout the cardiac cycle (*Seely et al., 1999*).

PATHOPHYSIOLOGY OF MI

Incidence of perioperative myocardial ischaemia and infarction:

Incidence of perioperative myocardial ischaemia:

In patients with or at risk of coronary artery disease, the reported incidence of perioperative myocardial ischaemia varies considerably, i.e. between 20 and 63% (*Landesberg et al., 2002*).

In studies that examined the incidence of myocardial ischaemia throughout the entire perioperative period, postoperative myocardial ischaemia was consistently found to occur considerably more often than preoperative (ratio approximately 3:1) and intraoperative ischaemia (ratio approximately 5:1) (*Mangano et al., 1991*).

Postoperative myocardial ischaemia seems to be the best predictor of in-hospital and long-term cardiac morbidity and mortality. It increased the relative risk of experiencing an early postoperative cardiac event (e.g. myocardial infarction, unstable angina, congestive heart failure) (*Mangano et al., 1991*). Postoperative myocardial ischaemia also increased the

odds for long-term (30 days to 2 years after surgery) cardiac events (unstable angina, non-fatal myocardial infarction, cardiac death, surgical coronary revascularization) 2.2-fold. In-hospital postoperative myocardial ischaemia preceded long-term adverse cardiac outcome in up to 70% (*Mangano et al., 1991*).

Incidence of perioperative myocardial infarction:

The reported incidence of postoperative cardiac events (the combined incidence of non-fatal myocardial infarction, unstable angina, congestive heart failure, cardiac death) varies between 5.5 and 53% and that of postoperative myocardial infarction between 1.4 and 38% (*Krumholz et al., 1994*).

Postoperative myocardial infarction increased the odds for long-term cardiac events 20-fold. The various findings would suggest that postoperative cardiac outcome is more affected by postoperative factors that cause myocardial ischaemia than by the patient's preoperative cardiac status (*Krumholz et al., 1994*).