

Perioperative strategies to shorten hospital stay : post operative strategies in vascular surgery

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The large amount of literature concerning postoperative outcome in vascular patients is a warrant to the importance of their perioperative morbidity which is the main factor which increases the length of hospital stay in these individuals, who have a high prevalence of concomitant disease confined to the heart, the vascular system, the lung and the metabolic system. Whether or not the humoral, mechanical, inflammatory and hypercoagulability phenomena which add up their deleterious effects following surgery lead to post operative coronary complication is by far the main determinant of the length hospital stay of these patients. With the introduction of biomarkers like troponin I (cTnI), our ability to identify and quantify myocardial damage in the postoperative period has been greatly enhanced, with small increases in cTnI being associated with both longer hospital stay and worse short and long-term outcomes.

This means that it is of the utmost importance to have an well-adapted postoperative management in order to shorten hospital stay by preventing cardiac complications. Consequently, the role of the anesthetist in charge of postoperative management for vascular patients goes beyond that of most other patient populations since there is a unique opportunity to limit long duration hospital stay associated with the occurrence of cardiac complication by applying refined postoperative management.

Taking into account the nature of postoperative cardiac complications and the moment when they occur during the operating period, it is possible to design perioperative management, which may improve the cardiac prognosis in vascular patients suffering undergoing non cardiac surgery and reduce the duration of their hospital stay.

Studies undertaken 15 years back when postoperative myocardial infarction (MI) was detected by means of electrocardiogram recordings every 12 hours following an operation suggested that this complication occurred often on the second or third postoperative day (1). More recent studies where postoperative myocardial necrosis has been detect-

ed by repeated troponin determinations have revealed that, in fact, postoperative myocardial infarction appears much earlier between 12 and 32 hours after the end of surgery (2-4). Therefore, acute postoperative myocardial necrosis appears not as a fatality on the third day, but rather as the consequence of episodes of myocardial ischemia occurring during surgery or in the first postoperative day.

Further understanding of the mechanisms underlying postoperative myocardial infarction, as well as their early identification, may contribute to the reduction of the incidence of postoperative myocardial infarction and its associated prolonged hospital stay and morbidity in the future. However, in spite of our improved ability to detect postoperative myocardial infarction and to assess its impact on outcome, the exact pathophysiology of postoperative myocardial infarction has yet to be elucidated. We still do not know whether postoperative myocardial infarction is due mostly to a sudden development of a thrombotic process associated with vulnerable plaque rupture, as is the case in most non-surgical myocardial infarction's, or is it due to increased patient vulnerability that is further complicated by the challenging postoperative conditions. Does postoperative myocardial infarction occur more often as a result of sudden coronary artery occlusion followed by massive necrosis, or is it mostly due to the cumulative effects of repeated and/or prolonged ischemic episodes, which by themselves may not cause extensive myocardial damage ? The answers to these questions may have both pathophysiological and therapeutic implications, which may potentially lead to the reduction of the still considerable cardiac morbidity following vascular surgery.

A recent study from our group (4) revealed 2 types of postoperative myocardial infarction that are different in their timing and biomarker pattern using intensive surveillance of troponine following surgery. The sudden myocardial infarction occurs in the early postoperative period and is not

preceded by sub-infarction myocardial damage, while the delayed myocardial infarction occurs later and is preceded by a long period (> 24 hours) of myocardial damage in which cTnI values are elevated.

About 60% of the patients that developed postoperative myocardial infarction had their infarction preceded by a prolonged period of myocardial damage infarction. The causative role of prolonged ischemia in the development of myocardial infarction has been previously postulated. Our findings do however suggest that, in addition to the well-recognized entity of delayed myocardial infarction there is another postoperative myocardial infarction entity, namely, the sudden myocardial infarction, which may have been hitherto unrecognized in the postoperative setting. The sudden myocardial infarction develops quite early in the postoperative period, is not preceded by myocardial damage, and is associated with a sudden rise in cTnI. Thus the cTnI profile of sudden myocardial infarction resembles that of acute non-surgical myocardial infarction, and is most probably due to acute coronary occlusion resulting from plaque hemorrhage, rupture and thrombus formation, as indeed has been observed in autopsies of fatal postoperative myocardial infarction's.

The identification of these 2 different types of postoperative myocardial infarction has some therapeutic implications to prevent postoperative coronary events.

In patients that are prone to develop postoperative myocardial infarction, and especially in those who are prone to develop sudden myocardial infarction, prevention can be achieved by better preoperative identification of the vulnerable plaque, and by a better plaque stabilization, either metabolically (e.g., statins) or by actual coronary stenting.

The patients who develop delayed myocardial infarction do present an additional opportunity for the prevention of postoperative myocardial infarction during the period of elevated biomarkers of myocardial injury. This period of sub-infarction myocardial damage lasts about 60 hours and should be regarded as a "Golden period" in which various interventions (e.g., intensive beta blockers therapy, adequate analgesia, correction of anemia) may potentially prevent postoperative myocardial infarction.

Consequently, monitoring troponin levels in the postoperative period following vascular surgery

enables the identification of patients with myocardial damage and the institution of early aggressive intervention in order to prevent the evolution of postoperative myocardial infarction during this 'golden period' that lasts about two days.

Postoperative surveillance of troponin following surgery is the cornerstone of postoperative management in these patients. Values between 0,2 and 1 ng/mL mean an evolution in the coronary disease, whether it is an unstable ischemia with myocardial damage or a limited sub-endocardial necrosis. Such values classify the patients as at high risk of developing significant postoperative myocardial infarction. Above 1 ng/mL, there is a myocardial infarction with cell loss implying an actual a decrease in medium term life span. In clinical practice even the slightest rise in troponin I reveals a perioperative myocardial damage caused by ischemia and warrants therapeutic interventions aiming at improving myocardial oxygen balance. It is thus necessary to admit the patient in an intensive care unit and control all factors influencing the myocardial energetic balance in order to limit damage to the heart. Administration of Beta-adrenergic antagonists will help decrease the heart rate and the administration of aspirin and continuous intravenous heparin will be useful if compatible with the type of surgery.

If acute myocardial infarction seems extensive (ST depression in several territories and high levels of Troponin I) or if signs of serious damage appear (circulatory shock, pulmonary oedema, polymorphic extrasystoles, prolonged hypotension or ST elevation), one must not hesitate to contemplate coronary angioplasty or bypass grafting.

References

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