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A95

Title: CARDIAC SURGICAL PATIENTS PLASMA ENHANCES OXYGEN FREE RADICAL PRODUCTION IN NEUTROPHILS

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Phospholipase A_2 activity is enhanced during cardiac surgery by production from polymorphonuclear leukocytes (PMNs) with heparin administration [1]. Oxidant generation in circulating inflammatory cells and complement derived chemotactic factor might contribute to the increase in PLA_2 activity, which then liberate excessive quantities of oxygen free radicals[2]. In this study, we examined the effect of plasma from patients undergoing cardiac surgery on oxygen free radical production (OFR) in PMNs.

Eight adult patients scheduled for elective coronary artery bypass graft (CABG) surgery were studied with institutional approval and informed consent. Plasma samples were obtained before and after induction; prior to incision; before and after heparin; at 15, 30, 60 min on CPB; and 1 hour after CPB. Neutrophil superoxide production at 37°C was measured by the superoxide dismutase-inhibitable reduction of cytochrome c.Total amount of cytochrome c reduction after PMNs stimulation by A23187 or PMA were calculated from changes in absorbance at 550nm using the extinction coefficient $21x10^3/M/cm$.

Patient's plasma did not affect OFR in PMNs when cells were stimulated by A23187 (2.0±0.6, before anesthesia versus 1.9±0.9, 30 min on CPB). Significant changes in OFR occurred when PMA was used as agonist (Figure). OFR by PMA was 4.0±1.9 in plasma before anesthesia and 3.7±1.8 after induction. OFR increased significantly with plasma taken after incision, but not after heparin administration. CPB produced a further increase in OFR to 5.1±2.1 which decreased significantly to 3.6±1.8 one hour after CPB.

Plasma from cardiac surgical patients enhances PMA-induced OFR in neutrophils. Plasma endotoxin levels increase during cardiac surgery[3]. Together with activation of PLA_2 or complement, such factors in plasma might prime neutrophils for enhanced release of oxygen free radicals during cardiac surgery. Activation of neutrophils and enhanced release of oxygen free radicals by plasma from patients undergoing cardiac surgery under CPB may enhance tissue damage and result in some of the pathological phenomena observed in these patients after the operation.

References

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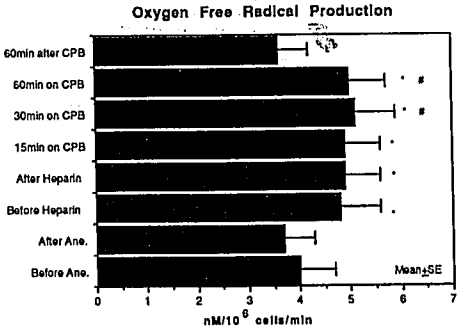

A96

Title: PERIOPERATIVE MYOCARDIAL ISCHEMIA IN RELATION TO ANATOMIC CHARACTERISTICS OF CORONARY ARTERIAL STENOSIS

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Several aspects of perioperative myocardial ischemia in patients (pts) undergoing coronary artery surgery (CABG), including an intraoperative recapitulation of a chronic preoperative ischemic pattern, suggests that ischemic episodes may be related to some characteristic of underlying coronary artery disease.[1,2] We, therefore, examined the relationship between angiographic location of coronary artery stenosis and perioperative myocardial ischemia in 44 patients undergoing CABG. After receiving IRB approval and informed consent 2 lead continuous ECG (Holter) monitoring was attached one day prior to surgery and continued intra- and postoperatively. Pts where categorized into 3 anatomic groups: Group I, 17 pts with steal-prone anatomy defined as occlusion of a major coronary artery and a ≥70% stenosis of vessel supplying collateral blood flow to myocardium distal to occlusion; Group II, 13 pts with stenosis ≥50% of the left main or ≥70% of proximal left anterior and circumflex coronary arteries (left main equivalent); Group III, 14 pts with ≥70% stenosis of major coronary arteries not fitting above groups. Myocardial ischemic events (MIE) were defined as ≥ 1mm shift in ST segment from baseline. All MIE were reviewed and required approval of two independent reviewers. Because the chances of detecting MIE increase with the duration of monitoring, MIE and duration of MIE were both normalized to hours of ECG monitoring for the pre-, intra- and postoperative monitoring periods. Chi-square and ANOVA were used to compare data between groups and values of $P<0.05$ considered significantly different.

There was no difference in the number of MIE between groups in the pre-, intra- or postoperative monitoring periods (table 1). Likewise, there was no difference in MIE per hr of monitoring or in the duration of MIE per hr of monitoring between groups for any of the three perioperative periods (table 1). That is, some pts displayed a pattern of preoperative ischemia that was no different intraoperatively. However, this chronic pattern of preoperative ischemia could not be detected in all pts with intraoperative MIE. Two patients from group II and two patients from groups III developed postoperative myocardial infarctions. Two of these patients had demonstrated MIE either intra- or post-operatively.

We conclude that while the trend for MIE appeared higher in pts with steal-prone anatomy this trend did not reach significance and, thus, perioperative MIE in patients undergoing CABG surgery do not seem to be related to pattern of coronary artery stenosis. In some but not all pts a pattern of chronic preoperative ischemia can be detected that is not changed perioperatively and is no different between anatomic groups tested.

Table 1. Characteristics of perioperative MIE (mean ± SD)

	Preop	Intraop	Postop
# of episodes			
Group I	1.23±1.75	0.35±1.06	1.11±1.53
Group II	0.75±1.48	0.25±0.62	0.92±1.73
Group III	0.80±1.60	0.13±0.35	0.73±1.28
MIE/hr of monitoring			
Group I	0.07±0.04	0.07±0.22	0.05±0.71
Group II	0.04±0.08	0.07±0.19	0.03±0.04
Group III	0.05±0.10	0.04±0.10	0.03±0.04
MIE (min)/hr of monitoring			
Group I	0.01±0.01	0.01±0.03	0.01±0.02
Group II	0.05±0.10	0.07±0.02	0.004±0.01
Group III	0.003±0.01	0.004±0.013	0.006±0.01

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