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Ultrasonographic evaluation of abdominal organs after cardiac surgery



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ABSTRACT

Background: Disturbances of the hepatosplanchnic region may occur after cardiac operations. Experimental studies have implicated impairment of splanchnic blood supply in major abdominal organ dysfunction after cardiopulmonary bypass (CPB). We investigated the impact of the cardiac operation and CPB on liver, kidney, and renal perfusion and function by means of ultrasonography and biochemical indices in a selected group of cardiac surgery patients.

Materials and methods: Seventy five patients scheduled for a major cardiac operation were prospectively included in the study. Criteria for selection were moderate or good left ventricular ejection fraction and absence of previous hepatic or renal impairment. Ultrasonography examination of the hepatic and renal vasculature and examination of biochemical parameters were performed on the day preceding the operation (T_0), on the first postoperative day (T_1), and on the seventh postoperative day (T_2).

Results: Portal vein velocity and flow volume increased significantly, whereas hepatic artery velocity and flow volume decreased at T_1 in comparison with T_0 . Hepatic vein indices remained unaffected throughout the observation period. Renal artery velocity and flow decreased, whereas renal pulsatility index and renal resistive index increased at T_1 as compared with T_0 . Aspartate aminotransferase and alanine aminotransferase values were increased as compared with baseline values 24 h postoperatively. All parameters displayed a trend to approach preoperative levels at T_2 . Strong negative correlations between alanine aminotransferase values at T_1 and hepatic artery velocity and flow volume at the same time point were also demonstrated ($R = 0.638$, $P < 0.001$ and $r = 0.662$, $P < 0.001$, respectively).

Conclusions: The increase in portal vein flow and velocity and the decrease in hepatic artery flow and velocity in the period after CPB might be attributed to the hypothermic bypass technique and the hepatic arterial buffer response, respectively. The decrease in renal blood flow and velocity and the parallel increase in Doppler renal pulsatility index and

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renal resistive index could be considered as markers of kidney hypoperfusion and intra-renal vasoconstriction. Maintaining a high index of suspicion for the early diagnosis of noncardiac complications in the period after CPB and institution of supportive care in case of compromised splanchnic perfusion are warranted.

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1. Introduction

Cardiopulmonary bypass (CPB) is a fundamental component of most heart surgery procedures. With notable advances in equipment and the integration of sophisticated technology, CPB allows the safe performance of a variety of open cardiac operations. However, there is evidence from experimental data and clinical series that CPB can have a profound impact on the hepatosplanchnic region [1]. Specifically, there have been numerous reports of liver function impairment after open-heart surgery, which often occurs irrespective of preoperative hepatic reserve [2]. Hyperbilirubinemia and transient elevation of liver enzymes are a common occurrence after CPB procedures [3,4]. Similarly, the spectrum of renal injury after CPB can vary from subclinical alterations of renal function indices to acute renal failure necessitating dialysis [5,6]. Establishment of renal failure after CPB can unduly prolong hospital stay, while at the same time it is considered an independent risk factor of death [7,8].

The mechanism of postbypass hepatic and renal injury can be multifactorial in origin; and although it is possible that microembolism and free-radical generation mediated by extracorporeal circulation are a contributing factor, it is also quite possible that derangements in hepatic and renal blood flow play a pivotal role in the pathophysiology of specific splanchnic injury [9–13]. Reductions of renal and hepatic blood flow during CPB have been documented with the aid of invasive techniques mostly in reports from experimental studies [10,14,15]. This reduction can be present in spite of the use of inotropic support after CPB and can be evident even several hours postoperatively. Pancreatic injury can also occur after CPB and is usually attributed to splanchnic hypoperfusion or extracorporeal circulation-mediated inflammation [10,16,17].

In case of postbypass liver or renal dysfunction, trans-abdominal ultrasound can provide useful information, as it allows both morphologic and functional evaluation of intra-abdominal organs. Ultrasound, used as a valuable bedside tool for the assessment of blood flow in many vascular territories, provides noninvasive and accurate assessment of the splanchnic circulation and can be used for measurements in the postoperative setting, with minimal nuisance for cardiac surgery patients.

The aim of the present prospective observational study was the evaluation of intra-abdominal organ function post-bypass, based on commonly used biochemical parameters and ultrasound findings in a typically mixed cardiac surgery adult population. A correlation between biochemical indices indicative of organ dysfunction and ultrasound-derived measurements was also attempted to establish a possible association between biochemical disturbances and intra-abdominal organ blood flow alterations.

2. Materials and methods

The protocol of this prospective observational study was approved by the Institutional Review Board of the Attikon University Hospital and took place according to the Helsinki Declaration. After evaluation of eligible patients during the preoperative visit and after obtaining written informed consent from each participant, 75 patients scheduled for a major cardiac operation were enrolled in the study. Exclusion criteria were known previous hepatic or renal impairment, as assessed by the patient's history and routine biochemical markers, previous major gastrointestinal surgery, poor left ventricular function preoperatively (ejection fraction less than 45%), unstable angina requiring nitrates, emergency procedures, overt hemodynamic instability postoperatively with a requirement for inotropic support and/or vasoconstrictive agents, or a requirement for an intra-aortic balloon pump either preoperatively or postoperatively. Patients were additionally excluded if, during the ultrasound examination, there was poor echo transmission, like in case of meteorism, or poor cooperation in maintaining apnea.

The patients' usual medications, including β -blockers and calcium channel blockers, were continued preoperatively until the morning of surgery. Premedication consisting of oral lorazepam and intramuscular morphine was administered on the night before surgery and 1 h before surgery, respectively.

Techniques of anesthesia and CPB were standardized. Specifically, anesthesia was induced with midazolam 0.3 mg kg^{-1} , etomidate $0.15\text{--}0.2 \text{ mg kg}^{-1}$, and fentanyl $4\text{--}7 \text{ }\mu\text{g kg}^{-1}$, and tracheal intubation was facilitated with rocuronium 0.8 mg kg^{-1} . Ventilation was performed with an oxygen in air mixture, with partial pressure of carbon dioxide (PCO_2) maintained at 35–45 mm Hg during mechanical ventilatory support and CPB. Anesthesia maintenance was ensured with sevoflurane at an end-tidal concentration of 1.5%–2%. Once the patient was anesthetized, invasive hemodynamic monitoring was established with an arterial catheter and a pulmonary artery catheter inserted through the right internal jugular vein for measurement of cardiac output and oximetry. After systemic heparinization (300 UI kg^{-1}), extracorporeal circulation was initiated when an activated clotting time of longer than 480 s was achieved. During extracorporeal circulation, cardiac arrest was induced and maintained by intermittent anterograde administration of cardioplegia solution. Nonpulsatile hypothermic CPB was used, consisting of a membrane oxygenator and a roller pump, after priming with a crystalloid-colloid solution, with a pump flow of $2.3 \text{ L min}^{-1} \text{ m}^{-2}$, a mean arterial pressure of 50–80 mm Hg by phenylephrine infusion, and moderate hemodilution with a hematocrit of over 20% by transfusion of packed red blood cells when required. Nasopharyngeal temperature of approximately 32°C was maintained throughout.

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