Postoperative Renal Function After Elective Abdominal Aortic Aneurysm Repair Requiring Suprarenal Aortic Cross-Clamping

Fabio A. Kudo¹, Toshiya Nishibe¹,², Keiko Miyazaki¹, Toshifumi Murashita¹, Keishu Yasuda¹, Motomi Ando², and Masayasu Nishibe³

¹Department of Cardiovascular Surgery, Hokkaido University School of Medicine, Sapporo, Japan
²Department of Surgery, Division of Cardiovascular and Thoracic Surgery, Fujita Health University, Toyoake, Japan
³Department of Surgery, Eniwa Midorino Clinic, Eniwa, Japan

Abstract

Purpose. To examine postoperative renal function after suprarenal aortic cross-clamping performed without renal hypothermia in patients undergoing elective abdominal aortic aneurysm (AAA) surgery.

Methods. Between 1991 and 2000, 18 patients underwent surgery for a juxtarenal AAA, which required a suprarenal aortic cross-clamp. All AAAs were repaired with a proximal anastomosis just below the renal arteries. We divided the patients into two groups according to the duration of the renal ischemia: <45 min (n = 12) and ≥45 min (n = 6). The postoperative changes in renal function were analyzed.

Results. There were no hospital deaths and none of the patients needed permanent hemodialysis. The postoperative peak in the serum creatinine level after suprarenal cross-clamping for ≥45 min was significantly higher than that after cross-clamping for <45 min. The percentage changes in serum creatinine and blood urea nitrogen were correlated positively with the duration of renal ischemia, and were significantly greater in the group with renal ischemia of <45 min than in the group with prolonged renal ischemia (≥45 min).

Conclusions. Suprarenal aortic cross-clamp without performing renal hypothermia is safe and able to be tolerated well by the patient during elective AAA surgery, although careful attention must be paid to limiting the period of renal ischemia.

Key words Abdominal aortic aneurysm · Suprarenal cross-clamp · Renal function

Introduction

More than 90% of abdominal aortic aneurysms (AAAs) develop below the level of the renal artery, leaving the interposed healthy part of the aorta to allow for infrarenal placement of the aortic clamp. Juxtarenal AAAs account for 2%–20% of all infrarenal AAAs.¹⁻³ The higher risk associated with juxtarenal AAA repair is related to the necessity for suprarenal aortic cross-clamping, temporarily excluding blood circulation from the kidneys,²⁻⁴⁻⁶ which may cause acute ischemic injury of the kidney. The aim of this study was to examine the early postoperative renal function of patients in whom a suprarenal aortic cross-clamp was applied during elective abdominal aortic surgery, without renal hypothermia.

Patients and Methods

Between January 1991 and March 2000, 18 patients underwent elective repair of an abdominal juxta- or infrarenal aortic aneurysm requiring suprarenal aortic cross-clamping, at Hokkaido University Hospital. We divided the patients into two groups according to the duration of the renal ischemia: <45 min (n = 12) and ≥45 min (n = 6). These times were based on the findings of previous investigations, which established that permanent renal damage begins after about 20 min of warm ischemia (37°C) and is invariably present after more than 45 min of warm ischemia.⁷⁻⁸ Any patients who suffered rupture or required renal artery reconstruction were excluded from this study. The group of patients subjected to a renal ischemia for <45 min consisted of ten men and two women aged 66.2 ± 9.0 (mean ± SD) years. The other group, subjected to prolonged renal ischemia, consisted of six men aged 70.3 ± 4.3 years.

Preoperative evaluation routinely included aortography or computed tomography, or both, but confirma-
tion of the proximal extension of the lesion and, more importantly, of its relation to the renal arteries was made intraoperatively. None of the patients had a serum creatinine concentration of >2.0 mg/dl, indicative of renal dysfunction.

All surgery was performed via the transabdominal or retroperitoneal approaches with the patient under general anesthesia. Arterial blood gases, electrolytes, blood counts, and coagulation profiles were monitored serially and corrected as needed. Intravenous heparin (70 U/kg) was given to all patients 3 min before aortic cross-clamping. Renal function was protected by limiting the period of warm ischemia, ensuring adequate circulating blood volume before surgery with preoperative intravenous fluid hydration and adequate blood volume replacement during and immediately after surgery, avoiding repetitive or prolonged renal ischemia, and maintaining maximal cardiac performance. Additional measures included the use of diuretics (mannitol and furosemide) and dopamine in vasodilatory dosages (1.5–3.0 µg/kg per minute). We did not induce renal hypothermia with cold renal perfusion or topical cooling during suprarenal clamping. All aneurysms were repaired with proximal anastomosis just below the renal arteries, minimizing the renal exclusion time as much as possible. Once proximal anastomosis was completed, the aortic clamp was moved distal to the anastomosis to restore flow in the renal arteries, while the distal reconstruction of the aorta or the iliac arteries was being performed.

**Statistics and Definitions**

Postoperative renal damage was evaluated by the percentage change in serum creatinine or blood urea nitrogen (BUN), calculated as the peak postoperative creatinine value/preoperative creatinine value × 100 or the peak postoperative BUN/preoperative BUN × 100, respectively. Linear regression was used to determine the relationship of variables, and Student’s t-test and the chi-squared test were used where appropriate (StatView 5.0, SAS Institute, Cary, NC, USA). All data are expressed as mean ± SD, and differences were considered significant at a 5% probability level.

**Results**

There were no hospital deaths, and none of the patients required permanent hemodialysis or suffered paraplegia. The operative data are shown in Table 1. In the group subjected to renal ischemia of <45 min, surgery was performed via a transabdominal incision in eight patients and via a retroperitoneal incision in four patients. In the group subjected to prolonged renal ischemia, surgery was performed via a transabdominal incision in three patients and via a retroperitoneal incision in three patients. Extension to the iliac arteries with a bifurcated graft was performed in all patients. The operative time, estimated blood loss, autologous blood replacement, homologous blood transfusion, and cell-saver blood replacement did not differ between the two groups. The average suprarenal aortic cross-clamp time was significantly shorter in the group subjected to renal ischemia of <45 min than in the group subjected to prolonged renal ischemia.

The preoperative and postoperative renal function values are summarized in Table 2. There were no significant differences in the preoperative creatinine clearance rate (CCR), serum creatinine, and BUN between the two groups. The postoperative peak serum creatinine level after suprarenal cross-clamping for ≥45 min was significantly higher than that after cross-clamping for <45 min. The percentage change in the serum creatinine and BUN was correlated positively with the duration of renal ischemia (Fig. 1a,b), and was significantly greater in the group subjected to renal ischemia of ≥45 min than in the group subjected to renal ischemia of <45 min. Only one patient required temporary dialysis postoperatively after 45 min of suprarenal aortic cross-clamping.

**Discussion**

Juxtarenal AAAs are defined as aneurysms that involve the infrarenal abdominal aortic segment extending up to, and sometimes including, the lower margin of the origins of the renal arteries. According to previous reports, juxtarenal aneurysms account for between 2% and 20% of all infrarenal AAA. The higher risk associated with juxtarenal AAA repair is related to the necessity for suprarenal aortic cross-clamping, leading to the temporary exclusion of blood circulation through the kidneys, with greater hemodynamic consequences.

Insufficient postoperative renal function is a common complication after surgery of the upper abdominal aorta, which usually occurs secondary to ischemia-induced acute tubular necrosis. There are two types of acute ischemic injury caused by either temporary periods of interrupted renal perfusion or periods of systemic hypoperfusion associated with AAA surgery and its pathophysiologic mechanisms. First, swelling of the tubular cells occurs after reperfusion, depending on the magnitude and duration of ischemia, which causes tubular obstruction, and further reduction or cessation of glomerular filtration in the nephron. Second, tubular cells can either lose their basement membrane attachment secondary to the interstitial ischemia that