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Serum Lactate and Base Deficit as Predictors of Mortality After Ruptured Abdominal Aortic Aneurysm Repair

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Objective. *Whole body hypoperfusion and lower torso ischaemia-reperfusion contribute to post-operative organ dysfunction in patients undergoing repair of ruptured abdominal aortic aneurysm (AAA). Serum lactate and base deficit are markers of tissue ischaemia and are used to assess the adequacy of resuscitation. This study examines the prognostic value of immediate post-operative levels of serum lactate and base deficit in ruptured AAA.*

Methods. *Thirty patients (24 men and 6 women of median age 74, range 51–85, years) who survived to at least 12 h after ruptured AAA repair were studied retrospectively. The relationship between immediate post-operative lactate, base deficit and mortality was determined.*

Results. *Fifteen patients (50%) died, all from organ failure. An elevated lactate (>2.1 mmol/l) and base deficit (<-2 mmol/l) were present in 20 (67%) and 27 (90%) patients, respectively. Lactate ($p<0.001$) and base deficit ($p=0.003$) were significantly higher in non-survivors compared with survivors. Lactate ($p=0.021$) and base deficit levels ($p=0.028$) were independently significant for predicting mortality and a significant interaction existed between lactate and base deficit levels for predicting mortality ($p=0.027$). The sensitivity and specificity of lactate ≥ 4.0 mmol/l was 13 of 15 (87%) and 12 of 15 (80%), respectively, and base deficit ≤ -7 mmol/l was 12 of 15 (80%) and 12 of 15 (80%), respectively. The likelihood ratios for a positive result with the defined cut-off values for lactate and base deficit were 4.3 and 4.0, respectively. Lactate ≥ 4.0 mmol/l and base deficit ≤ -7 mmol/l were associated with a 94.5% probability of death while lactate ≤ 4.0 mmol/l and base deficit ≥ -7 mmol/l were associated with a 4% probability of death.*

Conclusion. *These data demonstrate that an immediate post-operative serum lactate ≥ 4.0 mmol/l and base deficit ≤ -7 mmol/l are good predictors of outcome after ruptured AAA repair. The prognostic value of these simple and inexpensive tests require corroboration in a larger prospective study.*

Keywords: Lactate; Base deficit; Ruptured aortic aneurysm.

Introduction

Approximately 40% of patients who fail to survive repair of ruptured abdominal aortic aneurysm (AAA) die intra-operatively from cardiac arrest or uncontrollable haemorrhage while the remainder of deaths occur in the late post-operative period from multiple organ dysfunction.[1] Many scoring systems have been proposed to assist in identifying those patients with ruptured AAA who are at increased risk of peri-operative mortality, but these scoring systems may be cumbersome and the data required are frequently unavailable.[2–4] Serum lactate and base deficit are the most commonly used markers for assessing the adequacy of resuscitation in resolving tissue ischaemia in critically ill patients. Lactate is a by-product of anaerobic metabolism and is most commonly elevated secondary to tissue hypoxia. Base deficit is calculated by an arterial blood gas analyser using pCO_2, pH and HCO_3^- levels and represents the quantity of base required to normalize the pH of 1 l of blood. Many studies have demonstrated a correlation between elevated lactate, base deficit and mortality in critically ill patients[5–7] but there are no studies examining the prognostic value of these markers in patients with ruptured AAA. The aim of the present study was to examine the prognostic value of immediate post-operative serum lactate and base deficit on arrival in the intensive therapy unit (ITU) in patients undergoing emergency repair of ruptured AAA.

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Methods

Between March 2002 and December 2003, a total of 45

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patients presented to this institution with a diagnosis of suspected ruptured AAA. Ten patients did not undergo operation: 3 patients declined surgery and 7 patients were in such a poor clinical condition that they were considered unlikely to survive attempted repair. Thirty-five patients underwent attempted repair of ruptured AAA. Five patients died during attempted repair or within 12 h of surgery from cardiac events or uncontrollable haemorrhage and were excluded from analysis. Thirty patients (24 men and 6 women of median age 74, range 51–85, years) who survived to at least 12 h after ruptured AAA repair were studied retrospectively. The cut-off of 12 h was used as it was felt that patients who died subsequent to this time point were more likely to succumb from the effects of hypoperfusion and organ dysfunction (which may be preceded by changes in lactate and base deficit) rather than sudden events such as cardiac arrest and haemorrhage. The following clinico-pathological data were retrieved from the case notes: co-morbidity; clinical condition on presentation to the Emergency Department including lowest mean arterial pressure (MAP) and pre-operative haemoglobin (Hb) and serum creatinine; operative details including lowest intra-operative MAP, total operation time, measured blood loss and blood transfusion requirement; immediate post-operative Hb, serum creatinine, serum lactate and base deficit on arrival in the ITU; and post-operative complications and outcome.

Ruptured AAA was defined as the presence of retroperitoneal and/or intra-peritoneal blood in the absence of any other identifiable cause other than an aortic aneurysm. Operative mortality was defined as death within 30 days of surgery or during the same hospital admission. The normal laboratory range for serum lactate was >2.1 mmol/l and for base deficit was >-2 mmol/l.

The Mann–Whitney *U*-test, Spearman rank correlation and binary logistic regression were used where appropriate. A probability value of less than 0.05 was considered statistically significant.

Results

Nine of 30 (30%) patients were hypotensive (systolic blood pressure less than 100 mmHg) in the Emergency Department and one patient sustained a cardiac arrest prior to transfer to the operating room. Co-morbidity data are shown in Table 1. All patients underwent transperitoneal open aneurysm repair under general anaesthesia. Infra-renal aortic cross-clamping was required in 20 patients and two patients with suprarenal AAA required suprarenal aortic cross-clamping. One patient underwent repair of a ruptured proximal para-anastomotic aortic aneurysm with supracoeliac aortic clamping. In six patients, a supracoeliac aortic clamp was initially applied for 5–20 min before obtaining control by infrarenal aortic clamping. A dacron aorto-aortic graft was inserted in 20 patients and a bifurcated graft in nine patients. One patient with a ruptured mycotic infrarenal AAA underwent axillo-bifemoral bypass and aneurysm resection.

Table 1. Co-morbidity in 30 patients operated for ruptured AAA

Co-morbidity	Number of patients
Hypertension	15
Ischaemic heart disease	6
Previous myocardial infarction	6
Previous stroke	1
Peripheral arterial occlusive disease	4
Chronic renal dysfunction	4
Chronic obstructive pulmonary disease	11
Cigarette smoking	
Current	14
Reformed	6
Prescribed medication	
Anti-platelet therapy	6
β-receptor antagonist	5
Statin	4
Calcium-channel antagonist	9
Diuretic	10
Nitrate	5
Angiotensin-converting enzyme inhibitor	7
Warfarin	2
Bronchodilator	8

Fifteen (50%) patients died in the post-operative period. Sixteen patients developed organ failure [cardiac failure ($n=2$), respiratory failure ($n=7$), renal failure ($n=5$), coagulopathy ($n=1$) and multi-organ failure ($n=5$)] and 15 of these patients died. Five patients underwent re-operation: 2 patients had colectomy for ischaemic bowel, 1 required laparostomy for abdominal compartment syndrome and two patients had a negative laparotomy. The median (range) post-operative stay was 16 (1–55) days.

Lowest pre- ($p=0.002$) and intra-operative MAP ($p=0.026$) and immediate post-operative Hb ($p=0.029$) were significantly lower, and total operation time ($p=0.035$), measured operative blood loss ($p=0.05$), intra-operative blood transfusion requirement ($p<0.001$) and immediate post-operative creatinine ($p=0.006$) were significantly higher in non-survivors compared with survivors (Table 2).

In the immediate post-operative period, an elevated lactate (>2.1 mmol/l) and base deficit (<-2 mmol/l) were present in 20 (67%) and 27 (90%) patients, respectively. A significant correlation existed between lactate and base deficit ($r=-0.53$, $p=0.003$). Lactate