Eur J Vasc Endovasc Surg **30**, 263–266 (2005)
doi:10.1016/j.ejvs.2005.04.003, available online at http://www.sciencedirect.com on SCIENCE DIRECT®

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.

Methods

Between March 2002 and December 2003, a total of 45

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1078–5884/000263 + 04 $35.00/0

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

Table 2. Clinico-pathological variables in survivors and non-survivors of ruptured AAA repair

	Median (range)		*P* *
	Survivors	Non-survivors	
Lowest pre-operative MAP (mmHg)	104 (65–132)	63 (30–101)	0.002
Pre-operative Hb (g/dl)	10.9 (3.7–15.5)	11.4 (4.2–14.6)	NS
Pre-operative creatinine (μmol/l)	119 (81–263)	134 (101–476)	NS
Lowest intra-operative MAP (mmHg)	63 (47–87)	52 (33–63)	0.026
Intra-operative blood loss (l)	2.6 (0.4–8.0)	6 (2.5–25.0)	0.05
Intra-operative blood transfusion (units)	7 (0–12)	12 (6–38)	<0.001
Total operation time (mins)	180 (105–360)	230 (135–390)	0.035
Post-operative Hb (g/dl)	10.9 (8.0–13.0)	9.0 (6.8–13.9)	0.029
Post-operative creatinine (μmol/l)	126 (79–235)	161 (118–385)	0.006
Post-operative base deficit (mmol/l)	−4.1 (1.2 to −15.5)	−10.3 (1.3 to −16)	0.003
Post-operative lactate (mmol/l)	1.9 (0.5–10.0)	7 (2.5–11.7)	<0.001

MAP, mean arterial pressure; Hb, haemoglobin.
* Mann–Whitney test.

($p<0.001$) and base deficit ($p=0.003$) were significantly higher in non-survivors compared with survivors (Table 2). There was no significant difference in lactate and base deficit in patients who had a supracoeliac aortic clamp compared with patients who did not.

The sensitivity and specificity of lactate ≥ 4.0 mmol/l was 13 of 15 (87%) and 12 of 15 (80%), respectively. The sensitivity and specificity of base deficit ≤ -7 mmol/l was 12 of 15 (80%) and 12 of 15 (80%), respectively, (Fig. 1). Lactate ≥ 4.0 mmol/l and base deficit ≤ -7 mmol/l were the cut-off values with the best sensitivity and specificity. 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 ($p=0.021$) and base deficit levels ($p=0.028$) were independently significant for predicting mortality and there was also a highly significant interaction between lactate and base deficit levels for predicting mortality ($p=0.027$). Using binary logistic regression analysis, lactate $\geqslant 4.0$ 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 mortality.

Significant correlations existed between lactate and lowest pre-operative MAP ($r=-0.55$, $p=0.002$), lowest intra-operative MAP ($r=-0.60$, $p=0.001$), intra-operative blood transfusion requirement ($r=0.5$, $p=0.006$) and immediate post-operative Hb ($r=-0.36$, $p=0.05$). Significant correlations also existed between base deficit and lowest pre-operative MAP ($r=0.62$, $p<0.001$) lowest intra-operative MAP ($r=0.44$, $p=0.025$), intra-operative blood transfusion requirement ($r=-0.39$, $p=0.037$), immediate post-operative Hb ($r=0.40$, $p=0.027$) and post-operative creatinine ($r=-0.46$, $p=0.014$).

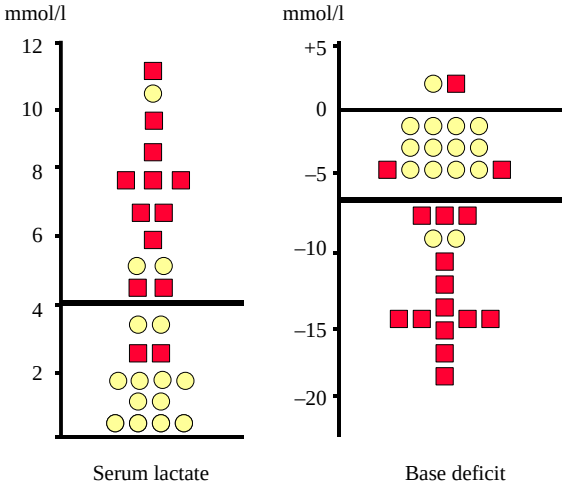

Fig. 1. Serum lactate and base deficit in survivors and non-survivors of ruptured AAA repair. The arbitrary cut-off levels for predicting outcome are shown by the double horizontal lines. Survivors are represented by the light circles and non-survivors by the dark squares.

Discussion

The principal findings of the present study are (a) immediate post-operative serum lactate and base deficit levels on admission to the ITU are significantly higher in non-survivors compared with survivors of ruptured AAA repair, and (b) an elevated lactate and base deficit are good predictors of subsequent mortality from organ failure.

In the present study, both lactate and base deficit were elevated in non-survivors and a significant correlation existed between the two markers. By contrast, a recent study in critically ill patients with shock and multiple trauma demonstrated significantly higher lactate levels in non-survivors compared with survivors on admission to the ITU with no significant difference in base deficit and no correlation between the two markers.[7]

Elevated serum lactate is a manifestation of severe and irreversible whole body hypoperfusion. This is confirmed in the present study by the strong correlations between lactate and peri-operative blood pressure, blood transfusion requirements and post-operative Hb, all of which were also significantly different in survivors and non-survivors.

The fact that elevated lactate and base deficit immediately post-operatively can predict outcome suggests that the resuscitative measures taken in the Emergency Department and the operating theatre may be sub-optimal. For example, hypotensive resuscitation before definitive control of haemorrhage,[8–10] the use of warm and lactate-containing crystalloid for resuscitation (in the absence of blood),[11,12] and intra-operative red blood cell salvage techniques (which were not used in the present study) may all contribute to improving the patient's clinical condition before and during surgery. Elevated lactate and base deficit levels at such an early stage after surgery may identify the onset of other complicating factors associated with poor outcome in ruptured AAA repair, namely cardiovascular instability, renal impairment, mesenteric ischaemia and ultimately multiple organ dysfunction.

The present study was retrospective and has obvious limitations. For example, previous studies have demonstrated that clearance of serum lactate to normal levels within 12–24 h is associated with improved prognosis, but so few patients in the present study had lactate levels recorded at 24 h that this relationship could not be determined. The small number of patients precluded multivariate statistical analysis and so it was not possible to prove that early post-operative lactate and base deficit are independent predictors of mortality. Nevertheless, using binary logistic regression we have demonstrated that, in our institution, a combination of pre-determined cut-off values for lactate and base deficit detected immediately post-operatively can be used to accurately predict subsequent mortality after ruptured AAA repair. A prospective audit of resuscitative measures and lactate and base deficit in a larger series of patients undergoing ruptured AAA repair is currently underway in our institution. If the findings of the present study are confirmed, then this would provide objective evidence to assist clinical decision-making regarding early withdrawal of life support in selected patients after ruptured AAA repair.

In conclusion, the present study has demonstrated that immediate post-operative serum lactate and base deficit are good predictors of outcome after ruptured AAA repair. Further research is required to determine whether these simple, reproducible and inexpensive tests have a role in the risk stratification of patients with ruptured AAA.

Acknowledgements

We thank Dr Tim Marshall, Senior Lecturer in Public Health and Epidemiology, University of Birmingham, for performing the statistical analyses, and the Consultant Vascular Surgeons of Birmingham Heartlands and Solihull NHS Trust (Teaching) for allowing us to study patients treated under their care.

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Accepted 4 April 2005
Available online 2 June 2005