Intra-Mucosal Acidosis as a Predictor of Cardiac Outcome Following Abdominal Aortic Aneurysm Surgery

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Objective. To assess if sigmoid ischaemia is a prognostic indicator of early and late post-operative cardiac morbidity and mortality.

Materials and methods. Patients undergoing elective abdominal aortic aneurysm repair (AAAR) were included in the study. Demographic details and risk factors for heart disease were recorded. Sigmoid pH$_I$ was measured at the time of surgery using a silicone tonometer and perioperative morbidity and mortality were recorded in all patients. Seven years following surgery the patients and their general practitioners were contacted to determine the patient’s health.

Results. Thirty-eight patients were included in the study. Within the follow-up period, 22 (58%) had died. Eight patients died of cardiac failure or myocardial infarction. The pH$_I$ in patients with cardiac related deaths [6.99 (6.84–7.10)] was significantly lower than those with non-cardiac related deaths [7.11 (7.04–7.21), p < 0.05]. Similarly, patients who suffered acute cardiac events (within 30 days following AAA repair) had lower pH$_I$ [7.01 (6.88–7.12)] compared to those who did not [7.09 (6.90–7.19), p < 0.05].

Conclusion. The results show that sigmoid ischaemia is more frequent amongst patients that develop cardiac events after AAAR and is associated with a worse long term outcome. This suggests that global hypoperfusion as a result of an under performing heart may be partly responsible for the sigmoid ischaemia in patients following AAAR. Therefore, low sigmoid pH$_I$ may predict an increased risk of cardiac complications in these patients.

Keywords: Aortic aneurysm; Sigmoid ischaemia; Intra mucosal acidosis.

Introduction

During abdominal aortic aneurysm repair (AAAR) colonic ischaemia may occur which can be detected by measuring the intramucosal pH (pHi) of the sigmoid colon by tonometry, a technique that has been well validated and is widely used.¹,² As a result of this ischaemic insult it has been suggested that the gut produces mediators such as myocardial depressant factor (MDF)³ and tumour necrosis factor (TNF),⁴ which have been shown to cause myocardial dysfunction. Although this is the widely accepted normal sequence of events, it is possible, however, that primary myocardial dysfunction may initiate global hypoperfusion and cause intestinal ischaemia.

The aim of this study is to assess if sigmoid intramucosal acidosis during aortic surgery predicts myocardial dysfunction, therefore suggesting that sigmoid ischaemia may be a manifestation of an under performing heart rather than the cause.

Materials and Methods

This study was approved by the Research Ethics Committee of the Queens University of Belfast. Thirty-eight non-consecutive patients undergoing elective abdominal aortic aneurysm surgery in accumulated over a period of a few years were evaluated. Demographic details, risk factors for cardiovascular disease, past history of cardiac events and presence of co-existing morbid states were recorded.

All patients were pre-medicated 2 h prior to undergoing routine thiopentane/morphine induction and muscle relaxation with pancuronium. A straight or bifurcated sealed Dacron graft was used to replace the aneurysmal aorta depending upon the findings at operation. All patients received 5000 IU of intravenous heparin 5 min prior to the application of the aortic cross clamp.
Sigmoid intramucosal pH measurement was performed using the silicone tonometer (Actamed Ltd, Wakefield, UK). The balloon of the tonometer was inserted into the rectum immediately after anaesthesia and then advanced into its sigmoid position at laparotomy. The position of the tonometer balloon was confirmed again just before closure of the abdomen. Once in place, 2.5 ml of normal saline was instilled into the semi permeable balloon via one of the injecting ports. After allowing a suitable equilibration time of no less than 15 min, the saline was analysed for the partial pressure of CO2. Together with an arterial time of no less than 15 min, the saline was analysed for injecting ports. After allowing a suitable equilibration instilled into the semi permeable balloon via one of the abdomen. Once in place, 2.5 ml of normal saline was confirmed again just before closure of the sigmoid colon was calculated using the Henderson Hasselbach equation. This calculation was simplified by the use of the slide rule provided by Actamed Ltd, Wakefield, UK. Both partial pressure of CO2 of the saline and standard bicarbonate concentration in blood were measured using the blood gas analyser. Instrumentation Laboratory, BGE set at 37 °C. The pHi was calculated at regular intervals and the minimum value used for subsequent analysis.

The tonometer was kept in situ over a period of 8–24 h depending on the rate of spontaneous expulsion by the patients. If not expelled by the patient within 24 h, the balloon was manually removed.

Post-operatively, the patients were managed in high dependency beds in the Vascular Surgery Unit. These patients had their cardiac rhythm, rate, central venous pressure, and arterial blood pressure monitored (Hewlett-Packard HP78566A, Andover USA) routinely for at least three days after surgery. Occurrence of angina, myocardial infarction (MI), congestive cardiac failure (CCF) and arrhythmias requiring medical attention were recorded during the post-operative hospital stay. Angina was diagnosed from ECG changes in the presence of chest pain, MI from ECG changes or enzyme rise, and CCF from shortness of breath and chest X-ray findings of pulmonary congestion.

Five to seven years following surgery, the patient’s general practitioners were contacted and the medical notes reviewed to obtain follow-up information. If the patients were still alive, development of any cardiac events such as angina, MI, CCF (defined as above), and arrhythmias were recorded. If the patient had died, cause of death as recorded on the death certificate and the others were due to carcinoma, renal failure or stroke and are classified as non-cardiac. The demographic details and risk factors of the cardiac and non-cardiac deaths are presented in Table 1. There were significantly more patients with pre-operative angina in the cardiac death group. There also were a larger number of pre-operative MIs in this group but it did not quite reach statistical significance.

There was a significantly lower minimum pH in those who subsequently died from cardiac causes [6.99 (6.84–7.10)] in comparison to the non-cardiac group [7.11 (7.04–7.21), p<0.05]. As expected there was a strong positive correlation observed between those who died of cardiac related causes and those with pre-operative angina (r = 0.76). There was no correlation between pre-operative angina and pH.

When all 38 patients were considered together, 17 developed cardiac complications such as arrhythmias, ischaemic heart disease or congestive cardiac failure within the follow-up period. The demographic details and risk factors are in Table 2.

Again there was a significantly lower minimum pH in those with cardiac complications [7.01 (6.88–7.12)] vs. 7.09 (6.90–7.19), p<0.05]. There was also a significant negative correlation between preoperative angina and pH (r = −0.4). There was no correlation between pH and aortic crossclamp time.

**Discussion**

The detection of intramucosal acidosis by tonometry has been used to demonstrate intestinal ischaemia in patients following major cardiac and non-cardiac surgery, including abdominal aortic aneurysm repair.6,7 Although intestinal infarction following aortic surgery is rare, sub-clinical splanchnic ischaemia, detectable only as intramucosal acidosis, may occur in a greater proportion of patients.5,7–10 The mechanism for the decrease in intramucosal pH (pHi) following aortic surgery is complex and in most cases, is probably due to a combination of local and systemic factors causing hypotension. The normal compensatory response to the hypotensive insult is an increase in contractility and rate of the heart with redistribution of the circulating volume away from less dependent organs such as the gut and skeletal muscles. Usually homeostasis is rapidly restored and splanchnic perfusion re-established, but when cardiac function is
A consequence of intestinal ischaemia is the loss of mucosal barrier function with an increase in intestinal permeability.\textsuperscript{11,12} This will in turn lead to permeation of luminal contents across a disrupted intestinal wall and subsequently endotoxaemia and generation of proinflammatory cytokines. This putative cascade of events is supported by the significant correlation found between endotoxaemia, the production of interleukin-6 and tumour necrosis factor and intramucosal acidosis in patients following AAA repair.\textsuperscript{13} The production of tumour necrosis factor and interleukin-6 has been associated with the development of intramucosal acidosis during abdomino-peritoneal mortality following AAA repair.\textsuperscript{17} This is assumed to be due to impaired reversal of the hypotensive episode may be delayed and splanchic ischaemia prolonged.

Although the sample size in this study is small the development of intramucosal acidosis was found to be associated with a greater incidence of cardiac morbidity and mortality. These results suggest that the development of intramucosal acidosis during abdominal aortic aneurysm repair could have longer-term implications as it may infer an underperforming or unstable myocardium. It has been shown that although surgery to repair an abdominal aortic aneurysm may reduce the risk of sudden death from rupture of the aneurysm, the life expectancy of these patients is still significantly less than those of an age-matched control.\textsuperscript{17} This is assumed to be due to cardiac deaths as the identification of those with significant cardiac disease and correction of their cardiac instability can lead to an improvement in longevity. Hertzler et al.\textsuperscript{18} demonstrated that nearly two thirds of patients with AAA had angiographically significant coronary artery disease. A third of these patients will have significant reversible myocardial ischaemia, which may be detectable by dobutamine stress echo or exercise stress test. It may be that the insult of repairing the abdominal aortic aneurysm can induce sufficient myocardial strain and identify those with significant cardiac compromise, similar to a stress echocardiogram. In this case an underperforming heart is demonstrated as intramucosal acidosis due to failure of the normal cardiac regulatory mechanism. The bowel ischaemia may therefore represent a global ischaemic event manifested as tissue acidosis.

Therefore, in conclusion, atherosclerosis is a progressive, systemic disease and although aortic abdominal aortic aneurysm repair can be accomplished safely and prevent death from a ruptured aneurysm, the operation will not prevent late death from progressive disease in other organs, especially that of the heart.\textsuperscript{19} Tonometric measurement of intramucosal pH is an accepted means of monitoring systemic oxygenation and outcome in critically ill patients and in this study it was found to be associated with future cardiac complications. As we have shown these cardiac events to occur up to 7 years following surgery, it is more likely that sigmoid ischaemia is an indicator of an underperforming heart than the acidosis causing myocardial dysfunction as has been previously suggested.

These results therefore demonstrate that the development of intramucosal acidosis may allow the identification of vulnerable patients and provide a golden opportunity to further investigate and treat those with cardiac instability so premature demise may be avoided.
References


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