

The profound impact of combined severe acidosis and malperfusion on operative mortality in the surgical treatment of type A aortic dissection

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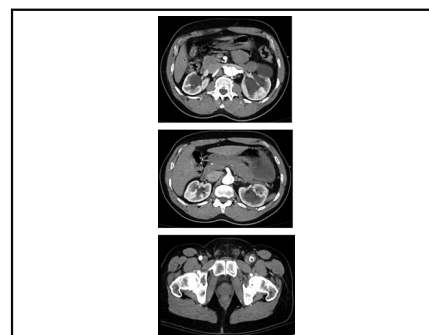
ABSTRACT

Objectives: Surgery for type A aortic dissection is associated with a high operative mortality, and a variety of predictive risk factors have been reported. We hypothesized that a combination of risk factors associated with organ malperfusion and severe acidosis that are not currently documented in databases would be associated with a level of extreme operative risk that would warrant the consideration of treatment paradigms other than immediate ascending aortic surgery.

Methods: Charts of patients undergoing repair of acute type A aortic dissection between January 1, 1996, and May 1, 2016, were queried for preoperative malperfusion, preoperative base deficit, pH, bicarbonate, cardiopulmonary resuscitation, severe aortic insufficiency, redo status, and preoperative intubation. Multivariable logistic analyses were considered to evaluate interested variables and operative mortality.

Results: Between January 1, 1996, and May 1, 2016, 282 patients underwent surgical repair of type A aortic dissection. A total of 66 patients had a calculated base deficit ≥ -5 or greater. Eleven of 12 patients (92%) with severe acidosis (base deficit ≥ -10) with malperfusion had operative mortality. No patient with severe acidosis with abdominal malperfusion survived. Multivariable analyses identified base deficit, intubation, congestive heart failure, dyslipidemia/statin use, and renal failure as predictors of operative death. The most significant predictor was base deficit ≥ -10 or greater (odds ratio, 9.602; 95% confidence interval, 2.649-34.799).

Conclusions: The combination of severe acidosis (base deficit ≥ -10) with abdominal malperfusion was uniformly fatal. Further research is needed to determine whether the identification of extreme risk warrants consideration of alternate treatment options to address the cause of severe acidosis before ascending aortic procedures. (J Thorac Cardiovasc Surg 2017; ■:1-8)



Malperfusion including renal artery, superior mesenteric artery, and left femoral artery.

Central Message

Significant operative mortality associated with severe acidosis combined with malperfusion warrants consideration of alternate treatment before ascending aortic replacement for type A dissection.

Perspective

Combined severe acidosis (base deficit ≥ -10) and malperfusion in the present study was associated with operative mortality in 92% of patients, and no patient with abdominal malperfusion and severe acidosis survived. The objective measurement of base deficit informs decisions regarding the treatment of patients with type A aortic dissection and should be collected in current databases.

Acute type A aortic dissection is associated with an in-hospital mortality rate of 12% to 35%.¹⁻⁷ Prompt surgical therapy is recommended to prevent the life-threatening

complications of rupture of the aorta into the pericardial or pleural space, rupture into a coronary ostium, acute aortic regurgitation, or neurologic compromise.^{8,9} The extent of the intimal tear is often the entire length of the aorta and jeopardizes blood flow to all organs downstream. The intimal flap may have multiple reentry points, and organs may become malperfused because of flap geometry that

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Abbreviations and Acronyms

CHF	= congestive heart failure
CPR	= cardiopulmonary resuscitation
HCO ₃	= bicarbonate
IRAD	= International Registry of Aortic Dissection
OR	= odds ratio
STS	= Society of Thoracic Surgeons

may be dynamic over time. The operative treatment and postoperative care consume multiple hospital resources, and the treatment is often referred to aortic centers or large, tertiary centers of care because of these concerns.¹⁰⁻¹²

We hypothesized that a number of preoperative variables not currently collected as part of the International Registry of Aortic Dissection (IRAD) or Society of Thoracic Surgeons (STS) databases, but related to organ malperfusion, would predict operative mortality. Identification of these factors would aid in the creation of algorithms for extremely high-risk patients.

MATERIALS AND METHODS

The records of all patients who underwent surgery for an acute Stanford type A aortic dissection between January 1, 1996, and May 1, 2016, at one institution with a designated Aortic Center and participation in IRAD were reviewed. Patients with the following characteristics were excluded: intraoperative dissection localized to the ascending aorta, intraoperative retrograde dissection caused by femoral cannulation, subacute or chronic (>2 weeks) type A dissection, and previous type B dissection or aneurysm of the descending aorta including previous repair or stent.

Available patient records were carefully scrutinized for a wide spectrum of preoperative and intraoperative variables. In addition to the usual variables collected for the STS and IRAD databases, records were specifically scrutinized for documented evidence for clinical suspicion of brain, coronary, extremity, or abdominal malperfusion. Additional clinical variables collected included preoperative lowest pH, preoperative lowest bicarbonate (HCO₃), preoperative *nadir* base deficit, preoperative cardiopulmonary resuscitation (CPR), redo status, preoperative intubation, and aortic insufficiency. Malperfusion was diagnosed using imaging studies (computed tomography scan with intravenous contrast) and review of records. Findings included the following: neurologic deficit or coma (brain malperfusion), lower-extremity lack of pulse or discoloration and pain (extremity malperfusion), severe abdominal pain (abdominal organ malperfusion), or electrocardiogram evidence of coronary ischemia (coronary malperfusion). Records were surveyed with Washington University Institutional Review Board approval.

Operative technique was variable and largely surgeon dependent and has been described by Lawton and colleagues.⁵ The study end point was operative mortality rate. Operative mortality (30 days) was defined as death in the initial hospitalization or within 30 days of surgery.

Statistics

Statistical analysis. All statistical tests were 2-sided using an $\alpha = 0.05$ level of significance. SAS Version 9.3 (SAS Institute Inc, Cary, NC) was used to perform all statistical analyses.

Descriptive statistics (eg, frequencies, %, mean, and standard deviation) were completed on preoperative and intraoperative variables. To predict operative mortality, we fitted a multivariable logistic regression model

using the patient's preoperative and intraoperative characteristics. Univariate logistic regression model was used to model the binary outcome. Multivariable analysis through stepwise selection was presented to examine the relationship between binary outcome and independent predictors, a significance level of .3 is required to allow a predictor into the model, and a significance level of .15 is required for a predictor to stay in the model. For those selected predictors, additionally, the integrated discrimination improvement was used to assess a model. The macro %idmacro, which computes integrated discrimination improvement, was used to examine whether a new predictor should be added to a model. The significance of the predictors in the final model was examined by the likelihood ratio test, and the performance of the model was tested by C-statistics. The Hosmer-Lemeshow test was used to test goodness of fit.

Base deficit was included as a single variable and separately as a level (≥ 0 , -5 to 0 , -10 to -5 , or ≥ -10). Severe acidosis was defined as base deficit of -10 or greater.

RESULTS

Between January 1, 1996, and May 1, 2016, 282 patients underwent surgical repair of type A aortic dissection. A total of 51 patients (18%) died in the operating room or within the initial hospitalization (operative mortality). A total of 83 patients had an aortic valve procedure (16 valve replacements including 8 root replacement with valve conduits, 8 valve-sparing root replacements, and 59 aortic valve repairs with resuspension of the valve). Table 1 summarizes the clinical characteristics of the patients.

A total of 80 patients (28%; 80/282) had malperfusion of any type or a combination of types. Of those 80 patients with malperfusion of any type, 29 (36%, 29/80) died (operative mortality) and 51 (64%, 51/80) did not. In contrast, only 11% (22/202) of patients who did not have malperfusion died (operative mortality). Operative mortality was significantly higher in the group of patients with malperfusion than in patients with no malperfusion (29/80, 36% vs 22/202, 11%, respectively, $P < .001$).

A total of 66 patients had a calculated base deficit -5 or greater (Table 2). Of these patients, 42 had a base deficit between -5 and -10 , and 24 had a base deficit -10 or greater. Eleven patients (26%, 11/42) with a base deficit -5 to -10 had operative mortality, and 11 patients (46%, 11/24) with severe acidosis (base deficit ≥ -10) had operative mortality.

Twenty-one patients had a combination of base deficit -5 to -10 and malperfusion. Ten of 21 patients (48%) with base deficit -5 to -10 combined with malperfusion of any type died (operative mortality). Among these 21 patients, malperfusion was associated with 29%, 33%, 50%, 60%, or 75% operative mortality depending on the type: extremity only, brain only, abdominal only, abdominal + extremity, or coronary only, respectively.

Twelve patients had a combination of severe acidosis (base deficit ≥ -10) and malperfusion. Eleven of 12 patients (92%) with severe acidosis combined with malperfusion of any type died (operative mortality). Operative mortality in this group (base deficit ≥ -10 with malperfusion) was statistically greater than operative mortality in the group of

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