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Risk-Adjusted Analysis of Outcomes Following Elective Open Abdominal Aortic Aneurysm Repair

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The purpose of this study was to describe a method to analyze outcomes following open abdominal aortic aneurysm (AAA) repair while considering the variability in patients' preoperative risk. Consecutive patients undergoing elective open infrarenal AAA repair during a 4-year period (2000-2003) were reviewed. Thirty-day or in-hospital mortality was the major outcome variable. Preoperative mortality risk was estimated for each patient using a validated scoring system that considers age, renal dysfunction, and coronary artery and cerebrovascular disease. A risk-adjusted cumulative sum method was used to compare observed versus predicted outcomes by assigning a risk-adjusted score, based on log-likelihood ratios, to each patient. These cumulative scores were sequentially plotted with preset control limits to allow for "signaling" when results were substantially different than expected (doubling or halving of odds ratios). Four hundred and sixty-three patients were studied with an overall early mortality rate of 4.5% (n = 21). Patients were allocated to three different preoperative risk groups (low, n = 89; medium, n = 160; high, n = 214) according to a medical comorbidity-based scoring system. Predicted (P) and observed (O) mortality rates for each group were as follows: low, 2.4% (P) and 2.2% (O); medium, 4.1% (P) and 4.4% (O); high, 9.3% (P) and 5.6% (O). The resulting risk-adjusted scores for each patient were plotted sequentially. This plot was flat for the first year and then adopted a negative slope crossing the lower control limit after 266 patients, indicating improved results compared to those expected. This coincided with the adoption of routine intraoperative cell saver use in our practice. This form of analysis allows for the prospective evaluation of results while considering patient-mix variabilities.

INTRODUCTION

Abdominal aortic aneurysm (AAA) surgery continues to be a prominent component of most vascular surgery practices and an extensively described area of research in the literature. With the introduction of endovascular technologies, several centers have reported studies comparing the various treatment modalities and their outcomes. Previously, population-based studies of nonemergent open AAA repair from Canada and the United States reported early mortality rates of 4.8%[1] and 8.4%,[2] respectively. Numerous single-center studies have reported lower rates and it is widely agreed that the endovascular approach can result in further reductions in early morbidity and mortality.[3]

A problem with many reports of surgical outcomes is the post hoc analysis of these results following their achievement. Although these studies have value with respect to their descriptive ability,

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their applicability to continuing quality assurance is limited. Of more value would be a prospective analytical tool allowing for early identification of results that differ from those that are acceptable, resulting in early intervention. One such instrument is the cumulative sum (CUSUM) failure method that our group has previously implemented in the examination of outcomes following emergent[4] and endovascular[5] AAA repair.

A significant shortcoming with the standard CUSUM method is its inability to consider differences in preoperative risk among patients. This results in the somewhat simplified and idealized situation in which an adverse outcome in a good-risk patient is considered in an identical fashion as one in a poor-risk patient. As a result, the risk-adjusted cumulative sum (RA-CUSUM) method was introduced with the ability to consider differences in patients' preoperative risk status.[6,7] The purpose of the present study was to describe a method allowing the analysis of a single center's contemporary experience with nonemergent open AAA repair while taking into account patient variability in medical comorbidities and preoperative risk.

METHODS

Our database, maintained by our research nurse, was reviewed to identify all patients who underwent AAA repair during a recent 4-year period (January 2000 to December 2003). The study cohort consisted of consecutive patients who underwent elective open repair of an asymptomatic AAA, excluding those with suprarenal extent or those who received emergent or endovascular repairs. Patient demographics were recorded along with medical comorbidities, length of stay, and intraoperative variables including transfusion requirements. The primary outcome variable was early mortality, defined as deaths occurring during hospital admission or within 30 days of surgery.

An estimated risk of mortality for each patient was formulated by noting the presence of several preoperative medical comorbidities and implementing the Glasgow Aneurysm Scoring (GAS) system. Briefly, a score was calculated for each patient according to the following formula: GAS = age in years + 7 points for myocardial disease + 10 points for cerebrovascular disease +14 points for renal dysfunction.[8] Myocardial disease refers to previous myocardial infarction or ongoing angina, while cerebrovascular disease refers to a history of neurological events including transient ischemic attacks, amaurosis fugax, or stroke. Renal dysfunction is defined as a history of acute or chronic renal failure and/or a preoperative serum urea level >20 mmol/L and/or serum creatinine level >150 μmol/L.

GAS values were subsequently converted to estimates of individual patient mortality risk by using a previously validated population-based analysis of this risk scoring system.[9,10] Prior to implementation these estimated risks were compared with our 1999 aneurysm outcome data as a form of internal validation. Proportions were compared using the chi-squared test with differences considered significant at the $p < 0.05$ level.

Subsequently tertiles of the study cohort's GAS values were converted to estimates of early mortality, as per the following[9]: GAS <69 – mortality risk = 0.024; 69 < GAS <77 – mortality risk = 0.041; GAS >77 – mortality risk = 0.093.

RA-CUSUM Method[6,7,11]

Mathematically a RA-CUSUM chart is created by plotting X_t versus patient number t where $X_t = \max(0, X_{t-1} + w_t)$. The patient's score ($w_t$) depends on estimated risk of early mortality (p_t), the patient's outcome (y_t), where $y_t = 0$ for survival and $y_t = 1$ for mortality, and OR_A, or an alternative level of performance to be detected. In the present study each patient's risk, p_t, was derived after formulation of a GAS risk score and allocation of an individual mortality risk, as described previously. Because each patient's mortality risk varies, OR_A is set as an odds ratio to detect a doubling or halving in the observed mortality rate compared to those expected.

Each patient's score (w_t) is derived using a log-likelihood ratio. This is defined by the logarithm of the ratio of the probability of the observed outcome to that expected by the estimated risk defined as p_t if the outcome is mortality and $(1 - p_t)$ if the outcome is survival.

$$w_t = log(OR_A / 1 - p_t + OR_A p_t), \text{ if } y_t = 1 (\text{mortality})$$

$$w_t = log(1 / 1 - p_t + OR_A p_t), \text{ if } y_t = 0 (\text{survival})$$

When plotting the RA-CUSUM chart to detect changes in the early mortality rate the w_t scores associated with mortality are positive and those associated with survival are negative. With this risk-adjusted analysis the "penalty" for the death of a low-risk patient is larger than that for the death of a high-risk patient. To complete the design of the chart a control limit (h) is set. For the present study designed to detect increases or decreases in early

mortality rate, setting the control limit (h) at ±3 would result in a "signal" when the odds ratio has not changed (false alarm) once every 1919 patients on average.

A plot of this patient cohort's outcomes, with respect to early mortality, is displayed in Figure 1. Negatively valued scores, designed to identify decreases in mortality, are plotted below positively valued scores designed to identify increases in mortality. On the RA-CUSUM chart a negative slope represents improved results compared to those predicted while deterioration in performance results in a positive slope. The procedure is designed to "signal" when X_t falls below the lower control limit ($h = -3$) or above the upper control limit ($h = 3$), indicating a halving or doubling of the odds ratio, respectively. The plot is reset to zero following each signal. As a form of continuing quality assurance, each "signal" in this process will prompt an investigation and evaluation of factors that may be contributing to this deviation of observed outcomes from those predicted.

RESULTS

Between the beginning of 2000 and the end of 2003, 463 patients underwent nonemergent open AAA repair by one of four vascular surgeons at our university-affiliated medical center, with an overall early mortality rate of 4.5% ($n = 21$). This study cohort consisted of 390 males (84%) with a mean age of 72.1 years (Table I). The mean aneurysm diameter was 62 mm. Proximal clamp position was immediately infrarenal in all patients, with the vast majority treated via the transperitoneal route. Tube or bifurcated grafts were used as indicated by the patient's anatomy.

The mortality rates used to predict risk in this study were derived from and validated in European population-based studies.[9,10] These rates were compared with those our 1999 nonemergent open AAA repair group (116 patients). In this group GAS scores were determined and each patient was allocated to one of three risk groups based on the GAS tertiles previously mentioned. The overall mortality rate in 1999 was 4.3% ($n = 5$), with each risk group's mortality rate being as follows: low, 3.7% ($n = 1$); medium, 2.9% ($n = 1$); and high, 7.6% ($n = 3$). These did not differ significantly when compared to the rates (2.4%, 4.1%, 9.3%) previously derived from the literature and subsequently used in this analysis ($p = 0.075$).

As described previously, age and the presence of renal dysfunction and coronary and cerebrovascular disease were taken into account to calculate a GAS for each patient. Patients were then allocated to one of three preoperative risk groups (low, $n = 89$, medium, $n = 160$, and high, $n = 214$) as described in Table II. Predicted (P) and observed (O) mortality rates for each group were as follows: low, 2.4% (P) and 2.2% (O); medium, 4.1% (P) and 4.4% (O); high, 9.3% (P) and 5.6% (O), for an overall predicted and observed mortality rate of 6.2% and 4.5%, respectively. Differences between observed and predicted rates proved insignificant except for those of the high-risk group ($p = 0.025$).

Each patient was then plotted sequentially using a two-sided risk-adjusted CUSUM graph, represented in Figure 1. The upper graph was designed to detect a doubling of the odds of mortality while the lower graph was set to detect a halving of this odds ratio. When the RA-CUSUM graph crosses the

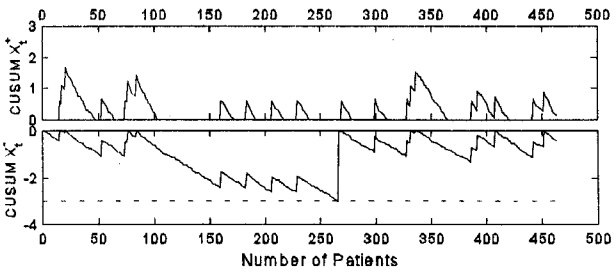

Fig. 1. RA-CUSUM of early mortality following elective open AAA repair, 2000-2003. The control limit h was set at ±3. The upper curve is set to "signal" when there is a doubling of the odds ratio of early mortality, whereas the lower curve is set to "signal" with a halving of the odds ratio. This plot "signals" at patient 266 by crossing the lower control limit. The graph is reset at zero and monitoring then continues.

Table I. Patient demographics

Factor	No. or mean	Range
Patients (n)	463	
Male gender [n (%)]	390 (84)	
Age (years)	72.1	47–88
Aneurysm diameter (mm)	62	50–128
Length of stay (days)	8.9	2–13

Table II. Predicted and observed mortality rates

Risk category	No.	GAS	Predicted mortality (%)	Observed mortality (%)
Low	89	<69	2.4	2.2
Medium	160	69–77	4.1	4.4
High	214	>77	9.3	5.6
Total	463	All	6.2	4.5

control limit ($h = \pm 3$) the observed data differ sufficiently from those predicted to prompt a "signal" or "alarm." Subsequently, the RA-CUSUM plot is reset to zero and monitoring is continued.

For the first 83 patients in Figure 1, both curves remain relatively flat about the baseline with no evidence of change in the odds of early mortality from that which is predicted by the GAS-based model. Following patient 83 the lower curve adopts a negative slope such that by patient number 266 the graph decreases below the lower control limit ($h = -3$), indicating a halving of the odds of mortality. Following this "signal" the RA-CUSUM graph was reset to zero and there were no further signals for the duration of this patient cohort.

Although any such analysis is only suggestive of a causal relationship, a deviation in this odds ratio should prompt an investigation into possible contributing factors irrespective of patient-related factors or comorbidities adjusted for during the analysis itself. Several procedure-related factors were reviewed, including time of year, operating surgeon, length of operation, graft configuration, and intraoperative cell saver use, which eventually proved to be the sole factor to change during this time period. Otherwise, patients were treated in an identical fashion throughout the study period. During the treatment of the first 83 patients the cell saver was used intraoperatively 38% ($n = 32$) of the time. Coinciding with the eventual halving of the mortality odds over the next 184 patients was a statistically significant increase in cell saver use ($n = 96$ or 52%; $p = 0.025$).

DISCUSSION

The overall early mortality rate of 4.5% experienced in this study compares favorably to those reported in the literature. Although these rates may differ according to whether they were derived from a population-based study or a single institution, a recent review of the literature found an average mortality rate of 5.5%.[12] This is very similar to the 4.8% and 5.6% mortality rates observed by participants in the Canadian Society for Vascular Surgery Aneurysm Study Group[1] and the United Kingdom Small Aneurysm Trial,[13] respectively.

Although these descriptive data are informative, additional analytical tools are often necessary to assess changes in outcomes over time and for the analysis of data in a prospective fashion. The CUSUM failure method was developed with this in mind as a method of recognizing change in a series of clinical outcomes prior to the point when such change would be recognizable by standard retrospective analysis.[14] Briefly, each patient's outcome is translated into a required success or failure format and a target or acceptable level of performance is chosen. Patients are plotted sequentially with the resulting graph being observed until it reaches a threshold. A generally positive slope indicates poorer results compared to the target rate, whereas a negatively sloped graph indicates improved results. Because this method can consider physician experience as a variable and can assess results over time, it has a distinct applicability for and has been applied to several surgical disciplines predominantly in the cardiac surgery field.[15] More recently, our vascular surgery group has applied this method to the analyses of learning curves associated with ruptured[4] and endovascular[5] AAA repair.

Although the use of the standard CUSUM technique involves simple mathematical calculations that result in a readily interpretable graph, it does have several significant shortcomings. These include its inability to determine the specific time period between cases that would result in optimal results. Other statistical tools, such as first-order differential equations, are necessary for this.[16] Additionally, because this traditional CUSUM method derives its origins from the extensive experience with process monitoring in industry, it makes no adjustment for differences in risk, as industrial inputs are usually consistent and adjustments are not required.[7] When applied to the surgical field this results in the somewhat unrealistic and idealized situation in which an adverse outcome in a good-risk patient is treated identically to one in a poor-risk patient. This method fails to take into account the intuitively obvious surgical principle that the probability of a successful outcome may vary across different patient risk profiles, irrespective of a surgeon's clinical acumen or skill.

In an initial effort to incorporate a case-by-case risk adjustment, variable life-adjusted displays (VLAD) were developed and used in assessing outcomes following cardiac surgery in the United Kingdom.[17] With VLAD plots, risk adjustment results in a surgeon's score equaling the difference between the predicted risk and the observed outcome (net lives saved). This would result in a surgeon who is performing as expected having a score of zero. This technique allows for easily interpretable plots for trends and patterns but, despite attempts to improve statistical interpretation,[18] does not provide a means for significance testing.[19] The RA-CUSUM method, as developed by Steiner's group,[6,7] offers risk adjustment and allows for signaling when there is sufficient statistical evidence that the observed results are significantly worse or

better than expected. Prior to our use of this technique with aneurysm surgery it had been previously described in the analysis of an Australian intensive care unit's outcomes.[11]

Each individual patient must be allocated a specific preoperative risk of mortality in order to proceed with such a risk-adjusted analysis. Although early mortality may not be the optimal determining factor of success following aneurysm repair, it is a widely used surrogate and is easily measurable. It has been well documented that early mortality rates following nonemergent open AAA repair are closely correlated with the number and complexities of coexisting medical comorbidities. In the Canadian Aneurysm Study an overall 4.8% early mortality rate was observed, but this increased to 6% in those patients with coronary artery disease and 12% in those with preexisting renal dysfunction.[1] Other studies have documented significantly increased odds of mortality associated with increased age, pulmonary dysfunction, coronary artery disease, congestive heart failure, and renal dysfunction.[20,21] The challenge for the present study is to translate these readily recognized medical risk factors into a valid preoperative risk scoring system. Prior attempts at such scoring systems include the Physiology and Operative Severity Score for the enUmeration of Mortality and morbidity (POSSUM),[22] the Glasgow Aneurysm Score,[8] and a prediction rule formulated at a Dutch hospital.[21]

We reviewed these scoring systems to identify one most applicable to our own patient population. The GAS, first described in 1994, was initially formulated after a review of 500 randomly selected patients.[8] Univariate and subsequent multivariate analysis identified age, myocardial disease, cerebrovascular disease, and renal disease as independent risk factors for early mortality. The regression coefficients were then used to create a simple risk score (as described in the Methods section) incorporating these risk factors. The ability of the GAS to accurately predict mortality was further confirmed in studies from the United Kingdom[23] and Finland.[10] Receiver operating characteristic curve and logistic regression analyses were used to allocate specific risk levels to various score intervals and validated with data from the Finnvasc registry of over 1900 patients.[9] These score intervals and their associated mortality rates were compared to those from our 1999 aneurysm cohort and found to be statistically similar. With this measure of external and internal validity, the GAS system and its respective mortality rates were used to develop a preoperative mortality risk for each individual patient in our study cohort. Study data displayed a positive correlation between the observed mortality rate and GAS, as would be expected (Table II).

Individual patient scores reflect these mortality risks assessed preoperatively, resulting in the penalty for the death of a low-risk patient being more severe than that of a higher-risk patient. Although it can't be assumed that all possible risk factors are considered, this method is ideally suited for situations in which there is a variable mix of patients over time. To simplify presentation, scores designed to detect decreases in mortality are given a negative value and plotted underneath the positively valued scores designed to detect increases in mortality as illustrated in Figure 1.

In this study the plot was set to identify a doubling or halving of the odds of mortality. A measure of significance was used with the introduction of control limits ($\pm h$) which are formulated by considering the frequency and time period of cases, estimated mortality rates or performance levels, and patient mix variability. A Markov chain procedure was used to estimate the average run length of a chart for any given control limit.[6] The sensitivity of the chart can be altered, resulting in more frequent false alarms or quicker detection of changes in the mortality rate. With increased procedure frequency longer average run lengths are desirable. There is a tradeoff, however, with control limits further from zero giving longer average run lengths with respect to false alarms, and also to true alarms when the surgical performance has actually changed.[6,7] In the present example, setting the control limit at ±3 gives an average run length of around 1919 patients between false signals when the surgical performance is actually acceptable. Given the frequency of AAA surgery at our institution, this would result in a false alarm approximately once every 8 years.

In Figure 1 the plot adopts a negative downward trend following the first 83 patients, the last point at which the test statistic measured zero before the signal, cumulating in the crossing of the lower control limit at patient 266. This indicates a halving of the odds of early mortality. This apparent decrease in mortality may occur if either the actual mortality rate has decreased or if the initial estimates of mortality risk were too high. Although the predicted and observed mortality rates associated with the high-risk group were significantly different, overall rates and those of the other risk groups did not differ. Also, case mix was consistent, with higher-risk patients equally distributed during the 4-year study period. A likely explanation for this

reduction in mortality rate in higher-risk patients is the widespread increase in utilization of endovascular repair techniques in these higher risk individuals. Following signaling the plot in Figure 1 was reset and monitoring continued. If the mortality estimates were too high we would have expected another signal, given the consistency in case mix. This did not occur, indicating that the observed decrease in mortality was most likely unique to this patient interval (patients 83-266) as opposed to a function of the preoperatively predicted mortality rates. Another possibility is that this represents a false alarm. Although the control limit was deliberately set to minimize the chances of this possibility, it is difficult to assign a likelihood to such a false alarm.

Because this form of analysis considers case mix and patient variability, a signal prompts an investigation and identification of other factors that may have contributed to this change in results. In the present study, the only possible contributing factor to the observed reduction in mortality identified was the increased intraoperative use of the cell saver. This was somewhat unexpected as data (not reported) collected by our institution's Blood Conservation Committee have revealed a relatively minor reduction in transfusion requirements (approximately one unit per patient) with our routine cell saver use in elective aortic surgery. To our knowledge, no one has linked reductions in transfusion requirements resulting from autotransfusion device use with reductions in mortality following elective aortic surgery.

Given our relatively conservative reduction in autologous blood use, it is more plausible that the observed relationship between a reduction in mortality and increase in cell saver use is coincidental rather than causative. With this risk-adjusted analysis correcting for variability in patients' comorbidities and preoperative risks, additional factors, as yet to be determined, must be responsible for the reduction in early mortality observed during a segment of this study cohort. Because this risk-adjusted cumulative plot failed to signal subsequently, these causative factors must be unique to the patient interval during which this mortality reduction was identified.

CONCLUSIONS

Nonemergent open AAA repair can be performed with acceptable mortality rates that are directly related to patient comorbidities. With standard analytical tools it is difficult to consider case mix variability while monitoring results over time. The RA-CUSUM procedure has the ability to adjust for variability in patient characteristics. This is especially important at tertiary centers where referral patterns may change over time.

With its risk adjustment capabilities, this method is a valuable tool in the monitoring of surgical outcomes, allowing for the early detection of results that deviate from those expected. Such "signals" or "alarms" would lead to a review of these cases and appropriate intervention.

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