# Usefulness of the Hardman index in predicting outcome after endovascular repair of ruptured abdominal aortic aneurysms

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***Objectives:*** **The Hardman index, which has five variables, has been recommended as a predictor of outcome after open repair of ruptured abdominal aortic aneurysms (RAAAs). It has been reported that the presence of three or more variables is uniformly fatal. The aim of this study was to test the same model in an independent series of RAAA patients undergoing endovascular repair.**

***Methods:*** **A consecutive series of 41 patients undergoing endovascular repair for RAAA during an 8-year period was analyzed retrospectively. Thirty-day mortality and patient variables, including the five Hardman risk factors of age >76 years, serum creatinine >190 μmol/L, hemoglobin <9 g/dL, loss of consciousness, and electrocardiographic (ECG) evidence of ischemia, were recorded. The Hardman index and a revised version of the index with four variables (without ECG ischemia) were calculated and related to clinical outcome.**

***Results:*** **Operative mortality was 41% (17 of 41). On univariate analysis, only age >76 years (P = .01) and the use of local anesthesia (P < .0001) were statistically significant. Loss of consciousness (P = .05) showed a trend toward a higher mortality, albeit not statistically significant. On multivariate analysis, the use of local anesthesia was the only significant predictor of survival (odds ratio [OR], 0.03; 95% confidence interval [CI], 0.003-0.25, P = .001). Again, loss of consciousness showed an association with a higher chance of dying but did not achieve statistical significance (OR, 6.30; 95% CI, 0.93-42.51, P = .059). The original and revised versions of the Hardman index were both significantly associated with death (P = .02 and P = .001, $\chi^2$ test for trend). The cumulative effect of 0, 1, 2, and ≥3 risk factors on mortality was 0%, 27%, 36%, and 71% for the original index, and 12.5%, 21%, 60%, and 78% for the revised version, respectively. Four and two patients with a score of ≥3 in each version of the index survived endovascular repair.**

***Conclusions:*** **The Hardman index, with or without incorporating ECG ischemia, seems to be a simple and useful predictive tool in patients undergoing endovascular repair of RAAA, with the mortality rate increasing along with the Hardman score. However, the index cannot be used to accurately identify patients with no chance of survival after endovascular repair. (J Vasc Surg 2008;■■:■■■.)**

Despite advances in critical care medicine, the surgical treatment of ruptured abdominal aortic aneurysms (RAAAs) has remained largely unchanged during the last 50 years, with the mortality rate persisting in the 50% range. Although a recent meta-analysis of RAAA repair did show a gradual reduction in perioperative death, this gradual improvement in the literature may reflect reporting bias and patient selection.[1] Indeed, selecting patients who are likely to survive and abandoning treatment in those with no realistic chance of survival would improve the results of individual series. Furthermore, such a selective policy would spare unnecessary suffering and enhance use of scarce resources.

During the last decade, several studies had attempted to establish predictive tools for selecting patients for repair and rejecting those who are unlikely to survive. The Hardman index,[2] Physiological and Operative Severity Score for the enUmeration of Mortality and Morbidity (POSSUM),[3] and the Glasgow Aneurysm Score,[4] for example, have all been suggested to improve selection. The five-variable Hardman index is the simplest of these, and it has been proposed that treatment should be withheld in patients with three or more risk factors. However, more recent studies and a meta-analysis have questioned this because of problems with variability in scoring resulting from missing data, particularly in relation to electrocardiogram (ECG) results.[5,6] As a result, it has been suggested that the usefulness of the index as a predictive tool could be enhanced by incorporating only four variables—age, level of consciousness, hemoglobin, and renal function—and omitting ECG ischemia.[6]

Most of the more recent literature on RAAAs has also focused on endovascular aneurysm repair (EVAR). These techniques, which are increasingly used for elective AAA patients, could offer an attractive option in patients with RAAA because of the reduced physiologic stress. Whether

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the outcome of such patients could be improved by EVAR remains to be seen. To date, the application of the Hardman index has not been adequately studied in patients undergoing EVAR of RAAAs. The aim of this study was to test the usefulness of the Hardman index in an independent series of RAAA patients undergoing EVAR.

## METHODS

Consecutive patients who underwent EVAR of a RAAA between March 1998 and August 2006 were enrolled. The study was held in a single vascular center that is based at a university hospital and provides on-call vascular services for northern Greece and certain parts of central Greece every fourth day. The elective EVAR program started back in 1995, and the vascular specialists of the unit have been involved in more than 1500 cases. The EVAR protocol for treating RAAAs was established in early 1998. There were no specific hemodynamic inclusion criteria for EVAR; however, the surgeon in charge had the option of sending the patient for immediate open repair in cases of severe hemodynamic instability. Nevertheless, unlike others, we did not consider hemodynamic instability as being a contraindication for EVAR.

The anatomic criteria for EVAR in RAAA patients were broadly similar to those used in the elective setting, the most important morphologic factor being the infrarenal neck. With growing experience, however, our selection criteria for both elective and RAAAs had expanded to the extent that more and more technically challenging cases had been treated by endovascular means. As a result, patients with difficult iliac anatomy, such as bilateral severe iliac occlusive disease, tortuosity, or aneurysmal involvement, had been considered as potential candidates. In addition, RAAAs with shorter (<15 mm), flared, or severely angulated proximal necks had also been accepted for EVAR, depending on the attending surgeon.

Once the diagnosis of a RAAA was suspected, access to computed tomography (CT) was quite quick, the whole process not lasting more than 15 to 20 minutes. As a result, the total time in such patients from admission to the scanning room and, subsequently, to the operating room was usually ≤30 minutes. In those being transferred from peripheral hospitals with the diagnosis already made on CT, the time from admission to the start of the endovascular procedure was minimal, because the patient was taken directly into the operating room, bypassing the emergency room and the radiology department. In the latter patients, the delay from the onset of symptoms to aneurysm exclusion depended on several factors beyond our control, such as how quickly the patient sought medical advice, how quickly the diagnosis of a RAAA was made by the referring physician, and the distance the patient had to travel by ambulance from the peripheral hospital to our center.

During the study period, 69 patients with the diagnosis of RAAA were admitted to our unit. Two died on arrival before any assessment and treatment was possible. CT scanning was used to assess 67 patients, and 42 (63%) were eligible for EVAR. The main reasons for exclusion were a problem proximal neck with either an inadequate length or a large diameter. Of the 25 patients who were unsuitable for stent grafting, 17 proceeded to open repair, six died before operation, and two were treated conservatively. Of the 42 patients who were anatomically suitable, all but one underwent EVAR, and no patient was denied EVAR for logistic or clinical reasons. In the latter patient, an attempt at EVAR was halted and converted to a standard open repair because of mechanical C-arm failure early during the operation. The remaining 41 patients were the focus of this study.

Patients who were thought to be suitable for EVAR were immediately transferred to a dedicated vascular operating room with endovascular facilities. The procedure was usually performed under local anesthesia. When needed, intravenous sedation was administered to minimize patient movement and discomfort. Intravenous fluid and blood resuscitation were kept to a minimum before aneurysm exclusion to allow hypotensive hemostasis. EVAR was then performed in standard fashion by cannulation of both femoral arteries with suitably sized sheaths.

Several types of stent grafts, including some homemade stent grafts, were used during the study period. In the early days, custom-made in-house devices were available in tube, aortouniiliac, and bifurcated design for use in elective AAAs. Our experience with these has been previously described in detail.[7] In brief, these consisted of a continuous self-expanding multiple Z-stent stainless steel structure covered with a pre-expanded polytetrafluoroethylene (PTFE) tube (commercial thin-wall 8 to 10 mm Impra [Impra Inc, Tempe, Ariz] and Gore [W. L. Gore and Assoc, Flagstaff, Ariz] pre-expanded grafts). The grafts were compressed into a 16F peel-away tube and were advanced into position with the help of a pusher through a long 16F sheath with distal radiopaque ring initially placed above the level of the renal arteries. Graft deployment was performed by withdrawal of the sheath. During that period, an inventory of such homemade endografts was initially kept in our hospital for use not only in elective cases but also in patients with symptomatic or RAAAs and other acute arterial lesions, such as ruptured aortas, pseudoaneurysms, or trauma cases. Two of the patients included in this series received an aortouniiliac custom-made endograft with a 32-mm proximal diameter.

As a result of the rapid developments made in the field of stent graft technology that occurred later on in the study, we moved away from these homemade devices in favor of the new-generation, commercially available stent grafts, such as the Talent (Medtronic World Medical, Sunrise, Fla), the Excluder (W. L. Gore and Assoc, Flagstaff, Ariz), the AneuRx (Medtronic AVE, Santa Rosa, Calif), and the Endofit (Endomed Inc, Phoenix, Ariz) systems. The type of stent graft was chosen depending on the surgeon's preference and expertise, the anatomic characteristics of the aneurysm, and, most importantly, the device availability.

We defined "primary technical success" according to the reporting standards for endovascular aortic aneurysm repair of the Society for Vascular Surgery/American Association for Vascular Surgery (SVS/AAVS). This requires the successful introduction and deployment of the device in

the absence of surgical conversion or death, type I or III endoleaks, or graft limb obstruction, and extends through the first 24-hour period. When unplanned endovascular procedures were required, the term "assisted primary technical success" was used.

Demographic data were recorded together with the five preoperative variables included in the Hardman index: age of patient (>76 years), serum creatinine (>190 μmol/L), hemoglobin (<9 g/dL), loss of consciousness, and ischemia on preoperative ECG. Loss of consciousness was defined in a similar way to that used in the original Hardman report[2] as documented evidence of unresponsiveness or even cardiorespiratory arrest after presentation to our unit, or the referring hospital, if the patient had been transferred from an outlying unit. ECG ischemia was defined as presence of Q waves, ST segment elevation of depression or inverted T waves in more than one lead, or both. A patient may score between 0 (no Hardman variables present) and 5 (all five Hardman variables present). It has been reported that the presence of three or more variables is uniformly fatal. We also applied a revised version of the Hardman index which had incorporated only the first four variables of the original index and omitted ECG ischemia, as suggested by Acosta et al.[6] The revised index score could range between 0 and 4. Each of the five risk factors, preoperative variables, and the original and revised Hardman index scores were all related to mortality.

Statistical analysis was performed using StatsDirect 2.6.2 statistical software (StatsDirect Ltd, Altricham, UK). The univariate association of the five index factors and other preoperative variables was assessed by the Fischer exact or $\chi^2$ test, as appropriate. Multivariate analysis of risk factors was done by logistic regression. The $\chi^2$ test for trend was used to compare the trend in the actual mortality rate according to increasing Hardman score. The Fischer exact test was used to compare mortality in patients with low (<3) and high (≥3) Hardman scores.

## RESULTS

During the study period, 41 patients (39 men, 2 women), with a median age of 73 years (range, 59-90 years), underwent EVAR of a RAAA. Table I summarizes the patient characteristics and comorbidities. On arrival, 21 patients (51%) were hypotensive (<90 mm Hg), and 21 (51%) presented with hemodynamic instability, defined as a reduced level of consciousness or a systolic blood pressure <80 mm Hg, or both. The initial hemoglobin was a median 9.7 g/dL (range, 5.5-13.8 g/dL). Free rupture, defined as intraperitoneal blood or contrast on preoperative radiologic imaging, was present in nine (22%) patients. However, because none of these patients underwent laparotomy, free rupture could not be documented reliably and could have been underestimated.

The operations were with local anesthesia in 27 patients, general anesthesia in three patients, and the procedure started with local anesthesia in the remaining 11 patients but had to be converted to general anesthesia either because of loss of consciousness and severe hypovolemic shock, or severe discomfort.

**Table I.** Patients characteristics

| *Characteristics* | *No. (%) or median (range)* |
|---|---|
| Sex | |
| Male | 39 |
| Female | 2 |
| Age, median years | 73 (59-90) |
| Coronary artery disease | 16 (39) |
| Hypertension | 33 (80) |
| Diabetes | 7 (17) |
| Chronic renal impairment[a] | 4 (10) |
| Cerebrovascular disease | 3 (7) |
| Hostile abdomen | 5 (12) |
| Obesity | 14 (34) |

[a]Defined as serum creatinine level >190 μmol/L.

The median time from admission to starting the EVAR was 45 minutes (range, 30-150 minutes). Twenty-eight bifurcated (15 Talent [Medtronic, Minneapolis, Minn], 12 Excluder [W. L. Gore and Associates], and 1 AneuRx [Medtronic]) and 14 aortouniiliac (2 Talent, 10 Endofit [Endomed Inc, Phoenix, Ariz]), and 2 custom-made stent grafts were implanted. The discrepancy between the number of patients (n = 41) and endografts (n = 42) was because one patient received two endografts. In this patient, the main body of a bifurcated Talent (32 mm in proximal diameter) was placed in a lower position than initially planned due to technical error related to poor image (obesity) and neck angulation. Because of concerns about the possibility of distal migration, we revised our original plan and, instead of deploying the contralateral limb, advanced an Endofit aortouniiliac device (34-mm proximal diameter) inside the previous stent graft with a more proximal fixation.

The aortouniiliac stent grafts were supplemented by a femorofemoral crossover bypass in all but one patient with a pre-existing chronic occlusion of the contralateral iliac system. In this patient, chronic ischemia was well tolerated due to the several collaterals that had not been jeopardized by the stent graft, and the circulation remained similar to the preoperative status. An aortic occlusion balloon was used in two patients (5%). There were no conversions to open graft repair.

A total of 17 patients died during the first 30 postoperative days, 15 during the hospital stay and two after hospital discharge, giving a 30-day mortality rate of 41%. Immediate mortality (≤24 hours) was 11 patients, of whom two died during reconstruction, one from hemorrhagic shock and the other from a fatal arrhythmia and ongoing hemorrhage. In the latter patient, completion angiogram revealed a type Ia endoleak, and the patient sustained cardiac arrest before implantation of an aortic cuff and aneurysm sealing was possible. Another five patients died in the operating room at the completion of the repair and four within a few hours in the intensive care unit. All nine aneurysms had been successfully excluded, but by that

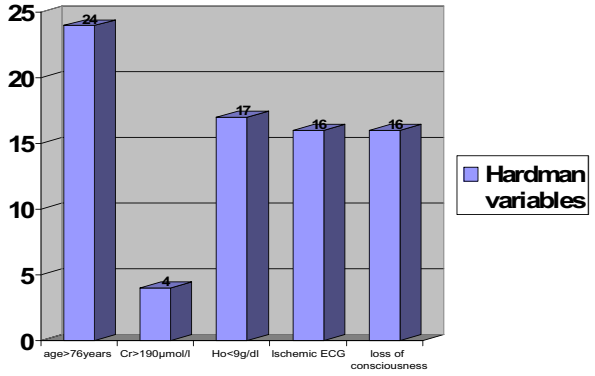


**Fig.** Number of patients with each one of the five Hardman risk factors. *Cr,* Creatinine; Hb, hemoglobin; *ECG,* Electrocardiogram.

time, it was presumably too late for these patients to survive. They were all moribund and died of intractable multiple organ failure despite aggressive resuscitation and after having received transfusion of a large amount of blood, blood products, and inotropes. Of the remaining six patients, two died on the second postoperative day, both from severe cardiopulmonary failure; one patient died on postoperative day 6 as a result of pulmonary edema and acute renal failure due to bilateral renal artery occlusion by the stent graft that necessitated dialysis; one died from respiratory infection on postoperative day 27 having sustained an intraoperative stroke; one died on postoperative day 28 of multiple organ failure and sepsis; and, finally, one patient died on postoperative day 29, 10 days after hospital discharge, of myocardial infarction. No patients presented with colonic ischemia, but one patient had abdominal compartment syndrome along with suspected bowel ischemia requiring decompression. This patient underwent a laparotomy on postoperative day 2, but the bowel was viable and resection was not performed. He eventually died on postoperative day 28 from multiple organ failure and sepsis.

Successful endograft deployment was achieved in 38 patients, whereas primary technical success, as defined by SVS/AAVS, occurred in 23 (56%) of 41. Nevertheless, this increased to a primary-assisted technical success rate of 71%, because six patients required unplanned additional endovascular maneuvers intraoperatively to secure successful completion of the repair. These included 2 proximal and 2 distal stents, 2 proximal and 2 distal graft extensions, and conversion of a bifurcated to an aortouniiliac stent graft.

The numbers of patients with each index variable are graphically presented in the Fig. A full data set of the five scoring variables was obtained in all 41 patients. Five patients had no risk factors, 11 patients had one, 11 patients had two, and 14 patients had three or more risk factors. In the latter group, two patients had four risk factors, but no patient had all five Hardman risk factors. After omitting ECG ischemia as a variable, the distribution of the other four factors was 8 patients with 0 risk factors, 14 patients with 1, 10 patients with 2, and 9 patients with 3. No patient presented with all 4 risk factors.

**Table II.** Univariate associations between 30-day mortality and patient variables, including the five Hardman risk factors

| *Variable* | *Patients, No.* | *Deaths* | P |
|---|---|---|---|
| History of cardiac disease | | | |
| Yes | 16 | 7 | |
| No | 25 | 10 | >.99 |
| Hypertension | | | |
| Yes | 33 | 14 | |
| No | 8 | 3 | >.99 |
| Current or ex-smokers | | | |
| Yes | 32 | 15 | |
| No | 9 | 2 | .3 |
| Chronic obstructive pulmonary disease | | | |
| Yes | 11 | 6 | |
| No | 30 | 11 | .5 |
| Cerebrovascular disease | | | |
| Yes | 3 | 1 | |
| No | 38 | 16 | >.99 |
| Hostile abdomen | | | |
| Yes | 5 | 2 | |
| No | 36 | 15 | >.99 |
| Diabetes | | | |
| Yes | 7 | 3 | |
| No | 34 | 14 | >.99 |
| Hypotension on admission (<90 mm Hg) | | | |
| Yes | 21 | 10 | |
| No | 20 | 7 | .5 |
| Hemodynamic instability | | | |
| Yes | 21 | 11 | |
| No | 20 | 6 | .2 |
| Bifurcated graft | | | |
| Yes | 27 | 12 | |
| No (aortouniiliac) | 14 | 5 | .7 |
| Type of anesthesia | | | |
| Local | 27 | 5 | |
| General, or local to general | 14 | 12 | <.0001[a] |
| Age >76 years | | | |
| Yes | 24 | 14 | |
| No | 17 | 3 | .01[a] |
| Loss of consciousness | | | |
| Yes | 16 | 10 | |
| No | 25 | 7 | .05 |
| Hemoglobin <9 g/dL | | | |
| Yes | 17 | 10 | |
| No | 24 | 7 | .1 |
| Serum creatinine >190 µmol/L | | | |
| Yes | 4 | 3 | |
| No | 37 | 14 | .2 |
| Ischemic electrocardiogram | | | |
| Yes | 16 | 7 | |
| No | 25 | 10 | .9 |

[a] $P < .05$, Fisher exact or $\chi^2$ test as appropriate.

Univariate associations between risk factors and mortality are summarized in Table II. Of the five Hardman variables, only age >76 years ($P = .01$) proved to be a statistically significant predictor. Loss of consciousness showed a trend towards a higher mortality, albeit not a

**Table III.** Multivariate analysis of risk factors using logistic regression

| *Variable* | *OR (95% CI)* | P |
|---|---|---|
| Age >76 years | 4.45 (0.64-30.86) | .13 |
| Loss of consciousness | 6.30 (0.93-42.51) | .059 |
| Local anesthesia | 0.03 (0.003-0.25) | .001 |

*CI,* Confidence interval; *OR,* odds ratio.

statistically significant one ($P = .05$). Of the remaining Table II variables, the use of local anesthesia ($P < .0001$) was also statistically significant. These three potentially significant factors were entered into a multivariate model using logistic regression (Table III). The use of local anesthesia remained the only significant predictor of survival (odds ratio [OR], 0.03; 95% confidence interval [CI], 0.003-0.25, $P = .001$). Again, loss of consciousness seemed to be associated with a higher chance of dying but did not achieve statistical significance (OR, 6.30; 95% CI, 0.93-42.51; $P = .059$).

The original and revised versions of the Hardman index were both significantly associated with death ($\chi^2$ test for trend, $P = .02$ and $P = .001$, respectively; Tables IV and V). In addition, a significant association was found between the presence of three or more risk factors and death (Fisher exact test, $P < .01$ and $P < .02$, respectively). The cumulative effect of 0, 1, 2, and ≥3 risk factors on mortality was, respectively, 0%, 27%, 36%, and 71% for the original index and 12.5%, 21%, 60%, and 78% for the revised version. Four and two patients with a score ≥3 in each version of the index survived EVAR.

**Table IV.** Thirty-day mortality in relation to the original five-variable Hardman index

| *Risk factors, No.* | *Patients, No.* | *Mortality, No. (%)* | P |
|---|---|---|---|
| 0 | 5 | 0 (0) | |
| 1 | 11 | 3 (27) | |
| 2 | 11 | 4 (36) | |
| ≥3 | 14 | 10 (71) | .02[a] |
| 0-2 | 27 | 7 (26) | |
| 3-5 | 14 | 10 (71) | <.01[b] |

[a]$\chi^2$ test for trend.
[b]Fisher exact test.

**Table V.** Thirty-day mortality in relation to the revised four-variable Hardman index after omitting ischemic electrocardiogram as a factor

| *Risk factors, No.* | *Patients, No.* | *Mortality, No. (%)* | P |
|---|---|---|---|
| 0 | 8 | 1 (12.5) | |
| 1 | 14 | 3 (21) | |
| 2 | 10 | 6 (60) | |
| ≥3 | 9 | 7 (78) | .001[a] |
| 0-2 | 32 | 10 (31) | |
| 3-4 | 9 | 7 (78) | .02[b] |

[a]$\chi^2$ test for trend.
[b]Fisher exact test.

## DISCUSSION

In the current era of clinical governance, risk stratification has become an important aspect of daily vascular practice. This is particularly true for patients presenting with a RAAA. In some countries, such as the United Kingdom, vascular surgeons advocate a selective approach in the management of patients with RAAA, whereas in other countries, such as Greece, an "all-comers" policy is more commonly adopted. Selecting patients for emergency surgery and rejecting those with little or no chance of survival is controversial and a matter for ethical debate. If this policy were to be implemented, the use of reliable risk scoring systems to facilitate such decisions would be extremely important. The Hardman index is the simplest of the predictive tools that have been used to select RAAA patients and is the only system that was originally developed from a cohort of patients undergoing surgery specifically for RAAA and not vascular or abdominal surgery in general. All five variables are readily available preoperatively, and the simplicity of the index makes its use in the preoperative setting very appealing. Furthermore, it has been proposed that open surgical treatment should be withheld in patients with a Hardman score ≥3.

Until recently, the index has been reported to be accurate and has been supported by four independent series.[2,8-10] Subsequent studies, however, yielded inconsistent results.[6,11-17] Some found the index to be a poor predictor and failed to show a statistical association with death,[12-14] whereas others found a strong correlation.[6,12,15] Nevertheless, even in the latter case, the presence of ≥3 risk factors was not found to be associated consistently with a 100% mortality, and, therefore, a score ≥3 could not be used as an absolute limit for denial of surgery. A recent meta-analysis of nine series with 970 patients did confirm the usefulness of the Hardman index in the prediction of death after surgery for RAAA.[6] The presence of 0, 1, 2, and ≥3 Hardman variables was associated with a mortality rate of 25%, 42%, 64% and 77%, respectively. In the pooled analysis, however, the authors included 71 EVARs that might have influenced the overall results of open surgery.

The association between death and the Hardman index in published series of open RAAA repair is summarized in Table VI.[2,6,8-13,16,17] These figures confirm the significant predictive effect of the index but also its failure to consistently identify a subgroup of patients with 100% mortality in which surgery should be withheld. The authors of the meta-analysis speculated that the utility of the index seemed to be impeded by variability in scoring resulting from missing or nondiagnostic data, particularly with regards to ECG.[6] As a result, they suggested that ECG ischemia should be omitted from the index.

In the present study, we specifically tested the same model in a consecutive series of patients with a RAAA undergoing EVAR using both a four- and five-variable

**Table VI.** Mortality (%) in relation to the number of the Hardman variables in 11 series of patients undergoing open repair of ruptured abdominal aortic aneurysm[a]

| | | *Number of risk factors, No. (%)* | | | |
|---|---|---|---|---|---|
| *First author* | *No.* | *0* | *1* | *2* | *≥3* |
| Hardman[2] | 154 | 10/62 (16) | 19/52 (36) | 23/32 (72) | 8/8 (100) |
| Prance[8] | 69 | 3/16 (19) | 5/18 (28) | 13/27 (48) | 8/8 (100) |
| Neary[9] | 188 | 23/66 (35) | 40/73 (55) | 29/39 (74) | 9/10 (90) |
| Boyle[10] | 79 | 2/24 (8) | 7/29 (24) | 11/20 (55) | 6/6 (100) |
| Calderwood[11] | 136 | 21/52 (40) | 19/41 (46) | 23/30 (77) | 12/13 (92) |
| Gatt[12] | 59 | 4/9 (44) | 10/18 (56) | 15/22 (68) | 7/10 (70) |
| Tambyraja[13] | 82 | 4/26 (15) | 17/31 (55) | 6/16 (38) | 3/9 (33) |
| Leo[15] | 114 | 4/43 (9) | 17/34 (50) | 23/29 (79) | 7/8 (87.5) |
| Acosta[6] | 106 | 5/27 (19) | 13/34 (38) | 13/22 (59) | 17/23 (74) |
| Sharif[16] | 74 | 9/22 (41) | 11/24 (46) | 11/18 (61) | 7/10 (70) |
| Sharif[17] | 178 | 12/27 (44) | 18/39 (46) | 21/31 (68) | 15/18 (83) |
| Total | 1239 | 95/374 (25) | 176/393 (45) | 188/286 (66) | 99/124 (80) |

[a]One published study[14] that reported both open and endovascular repair was excluded because no separate data on open repair could be extracted.

**Table VII.** Mortality (%) in relation to the number of the Hardman variables in the four series reporting patients undergoing endovascular repair of ruptured abdominal aortic aneurysm

| | | *Number of risk factors, No. (%)* | | | |
|---|---|---|---|---|---|
| *First author* | *No.* | *0* | *1* | *2* | *≥3* |
| Larzon[14] | 15 | 0/5 (0) | 0/5 (0) | 2/3 (67) | 0/2 (0) |
| Acosta[6] | 56 | 1/13 (8) | 6/23 (26) | 7/12 (58) | 5/8 (62.5) |
| Sharif[16] | 52 | 2/13 (15) | 6/22 (27) | 7/14 (50) | 2/3 (66) |
| Present study | 41 | 0/5 (0) | 3/11 (27) | 4/11 (36) | 10/14 (71) |
| Total | 164 | 3/36 (8) | 15/61 (25) | 20/40 (50) | 17/27 (63) |

version of the index. We excluded patients undergoing open repair or those who were treated nonoperatively. Offering EVAR depended only on anatomic criteria, the proximal neck being the most decisive factor, and unlike other centers, we applied no specific hemodynamic criteria. We hypothesized that because of its minimally invasive nature, EVAR may be better tolerated by fragile patients who are unlikely to survive open surgical repair. The results of our study suggest that similar to open repair, either version of the Hardman index is a strong predictor of outcome after EVAR. Patients with a high index score (≥3) had a significantly higher operative risk than those with lower scores (<3). Furthermore, the mortality rate increased with the number of positive Hardman criteria. This is similar to the results of nine of the 12 previously published series of open repair.[2,6,8-17]

Table VII summarizes the mortality figures in relation to the Hardman index in four series of patients who underwent EVAR, including our own study. The overall mortality rate of EVAR in the group with the higher index score (≥3) was 63% (17 of 27), that is, 10 patients with ≥3 risk factors survived. Naturally, this represents an improvement from the pooled mortality of 80% encountered in the respective open repair group. This observation may imply two things: either that the index is not suitable for use in patients undergoing EVAR because it has been developed from studies of patients undergoing open repair; or that patients who have little or no chance of survival after open repair according to the index may survive EVAR. Of course, one should take into account the small number of patients, the retrospective nature of the studies, and the potential differences between the open and endovascular groups before interpreting the results, particularly with regards to hemodynamic instability.

The mortality of 41% seen in our series may seem rather high to represent progress compared with the best series of open RAAA repair. The proponents of open repair would argue that with such results, there is no strong reason to start an endovascular program for RAAAs at all. Nevertheless, it is worth remembering that only the "best" results of open repair are usually published. Reviewing the English language literature, one can find a huge variation in mortality rates after EVAR of RAAAs, with figures ranging from 0% to 54%.[18] There are several reasons for what appears to be a high mortality rate in our study: First, this represents the early phase of our learning curve, and despite being one of the largest published single-center experiences worldwide, it is still reporting on average five patients treated annually during an 8-year period.

Second, we only used anatomic and no hemodynamic criteria in patient selection. This would certainly affect our results. If patients were stratified according to their hemodynamic instability, the results of open repair in unstable patients would be equally dismal, or even worse.

Third, as experience with both elective and emergency endovascular cases increased, there was a tendency to take on cases with more challenging anatomy, increasing the operative difficulty. Similarly, it is also possible that we intuitively moved towards offering EVAR to some of the patients who before the introduction of endovascular techniques would have been normally turned down for open surgery. Naturally, the latter is indeed difficult to quantify.

Given the results of the present study, it becomes apparent that a selective approach in offering EVAR to patients with a RAAA, based on either a four- or a five-variable version of the Hardman index, is not appropriate, because no group with a mortality rate of 100% could be identified; therefore, rejecting a patient for EVAR based simply on the Hardman index would be incorrect. Other factors not included in the index should also be taken into account, such as the cardiorespiratory reserve, the presence of malignancy, mental status, and hemodynamic instability. The latter may be the single most important determinant of survival in RAAA patients; however, this failed to reach statistical significance in our series.[19]

The only significant prognostic factor was the use of local anesthesia. Patients who needed general anesthesia, either from the start or during the procedure, had a higher chance of dying. Strictly speaking, this is not a preoperative variable. However, because our intention was to treat all patients under local anesthesia, being able to perform the endovascular procedure under local anesthesia without conversion to general anesthesia implies a more stable hemodynamic condition. Finally, further factors, such as the technical difficulty in excluding the aneurysm, may also influence survival and should be included in the equation for when possible survival has been passed. However, these are intraoperative factors that cannot be incorporated in a preoperative scoring system to identify those with no chance of survival.

## CONCLUSION

The Hardman index seems to be a simple and useful predictive tool, not only as previously shown in patients undergoing open repair of RAAA but also in patients having emergency EVAR. In addition, the index appears to have a cumulative predictive significance, with the mortality rate increasing along with the Hardman score. The index cannot specify accurately a group of patients with no chance of survival after EVAR; therefore, clinical decision making solely on the basis of the index does not seem appropriate. Finally, omitting ECG ischemia from the index does not appear to improve the index performance. Further studies on risk scoring with prospective validation may help identify patients with RAAA for whom surgery, open or endovascular, is futile.

## AUTHOR CONTRIBUTIONS

Conception and design: CK, ASK, TG
Analysis and interpretation: CK, KP, ASK, ET, AGK, TG
Data collection: CK, DK, KP, ASK, ET, AGK
Writing the article: CK, DK, KP, ASK, ET, TG
Critical revision of the article: CK, DK, KP, ASK, ET, AGK, TG
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