Probability of rupture of an abdominal aortic aneurysm after an unrelated operative procedure: A prospective study


It has been assumed by some authors that patients with abdominal aortic aneurysms may be at increased risk of rupture after unrelated operations. From July 1986 to December 1989, 33 patients (29 men, 4 women) with a known abdominal aortic aneurysm underwent 45 operations. Twenty-eight patients had an infrarenal abdominal aortic aneurysm, and five patients had a thoracoabdominal aneurysm. The abdominal aortic aneurysm ranged in transverse diameter from 3.0 to 8.5 cm (average 5.6 cm). Twenty-seven patients underwent a single operation, and six patients had two or more (range of 1 to 6). Operations performed were abdominal (13); cardiothoracic (9); head/neck (2); other vascular (11); urologic (7); amputation (2); breast (1). General anesthesia was used in 29 procedures, spinal/epidural in 6, and regional/local in 10. One postoperative death occurred from cardiopulmonary failure. One patient died of a ruptured abdominal aortic aneurysm at 20 days after coronary artery bypass (1/33 patients [3%]; 1/45 operations [2%]). Fourteen patients had repair of their abdominal aortic aneurysm at a later date, an average of 18 weeks after operation. Four patients had abdominal aortic aneurysm considered too small to warrant resection (average 5.6 cm). Four patients were considered at excessive risk for elective repair. The five thoracoabdominal aneurysm were not repaired. Four patients are awaiting repair. During this same 40-month period, two other patients, not known to have an abdominal aortic aneurysm, died of a ruptured abdominal aortic aneurysm after another operative procedure, at 21 days and 77 days. All three ruptured abdominal aortic aneurysms were 5.0 cm or greater in transverse diameter. We have found only speculative assumptions in the literature suggesting that an unrelated surgical intervention hastens the time of rupture of an abdominal aortic aneurysm. Our experience casts doubt that such a relationship exists, but it does not exclude its possibility. If there is any validity to the fear of postoperative aneurysm rupture, it will depend on the demonstration of biochemical changes in the aneurysm wall induced by surgical trauma.


Postoperative rupture of an abdominal aortic aneurysm (AAA) after operations other than AAA repair occurs occasionally. Swanson et al. reported 10 cases of large aneurysms (9.4 cm mean diameter) that ruptured within 5 weeks of exploratory laparotomy. They suggested that the exploratory laparotomy caused weakening of the aneurysmal wall by activation of collagenase and/or elastase. Others have noted sporadic cases of postoperative rupture of AAA. However, the incidence of postoperative rupture of AAs after unrelated surgical procedures remains unknown, and a causal relationship between surgery and increased risk of rupture has not been proved. In an attempt to document the incidence of postoperative aneurysmal rupture, we have followed all patients at the University of Pittsburgh who have had an AAA diagnosed before an unrelated, surgical procedure.

METHODS

From July 1986 to December 1989, 33 patients had an AAA identified before an unrelated procedure (30 patients) or during an exploratory laparotomy for an unrelated problem (three patients). The size of the AAs was measured by ultrasound, CT scanning, or direct measurement at exploratory laparot-
Table I. Range of size of aneurysm in 33 patients undergoing operation not related to their aneurysm

<table>
<thead>
<tr>
<th>Size</th>
<th>No. cases</th>
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<tbody>
<tr>
<td>3.0-4.4 cm</td>
<td>6</td>
</tr>
<tr>
<td>4.5-5.9 cm</td>
<td>11</td>
</tr>
<tr>
<td>≥6.0 cm</td>
<td>16</td>
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The patients were followed for 30 days, or until the patient's death, or until the AAA was resected.

RESULTS

Thirty-three patients with documented AAAs underwent 45 operations. Twenty-eight patients had an infrarenal AAA, and five patients had a thoracoabdominal aneurysm. There were 29 men and 4 women, and their average age was 74.3 years. The AAAs ranged in transverse diameter from 3.0 to 8.5 cm (average 5.6 cm). Six measured between 3 to 4.4 cm, 11 between 4.5 to 5.9 cm, and 16 greater than or equal to 6 cm (Table I). Twenty-seven patients had a single procedure, and six had two or more procedures. The operations performed were abdominal (13), cardiothoracic (9), other vascular (11), urologic (7), amputation (2), and breast (1) (Table II). Thirty major operations and 15 minor were performed. General anesthesia was used in 29 procedures, spinal/epidural in 6 procedures, and regional/local in 10.

One patient died after surgery of a ruptured AAA at 20 days after coronary artery bypass grafting (1/33 patients, 3%; 1/45 procedures, 2%; 1/30 major procedures, 3%). Fourteen patients had their AAAs repaired at a later date, an average of 18 weeks after the previous procedure. Four patients are awaiting repair of their AAA. Four patients had AAAs that were considered too small to warrant resection (average 3.6 cm). Four patients were considered to have too great a perioperative risk for elective aneurysm repair. The five thoracoabdominal aneurysms (average 5.5 cm) were not resected. One patient died after surgery of multiorgan systems failure after coronary artery surgery.

During this same 40-month period, two additional patients, not known to have AAAs, and thus not followed in our prospective study, died of a ruptured AAA after an unrelated procedure at 21 days and 77 days. The AAAs of the three patients who died of postoperative aneurysm rupture measured greater than 5 cm and ruptured at an average of 40 days after the unrelated surgical procedure. The procedures were coronary artery bypass grafting (20 days), nasal reconstruction (27 days), and forearm arteriovenous fistula (77 days).

DISCUSSION

The risk of aneurysm rupture after surgery for an unrelated problem is not known and difficult to quantify. Indeed an aneurysm may rupture at any time and at any size; there are no data that accurately predicts when this might occur.

Size appears to be the best predictor of rupture. In two studies Szilagyi et al.7,8 have shown that AAAs greater than 6 cm have a greater chance of rupture: 31% for small AAAs and 43% for large AAAs. Nevitt et al.9 reviewed 176 patients with AAAs and found 16 to have had ruptured AAAs, all with aneurysms larger than 5 cm. Although the larger aneurysms (>6 cm) have a greater chance of rupture, small aneurysms (<5 cm) have ruptured without warning.

Clinical and laboratory evidence exists to support and contradict the hypothesis that surgical procedures precipitate aneurysm rupture. The review by Swanson et al.1 of 10 patients with ruptured AAAs after exploratory laparotomy was the first to propose this relationship. In their paper they describe a mechanism of enzyme activation in the aortic wall by the...
laparotomy. These enzymes act to weaken the already dilated aorta and thus precipitate rupture. This clinical study did not include supportive laboratory data. Others have noted anecdotal and sporadic rupture of AAA after unrelated procedures.

Clinical studies of AAAs and concomitant diseases such as coronary artery disease, cholelithiasis, and colon carcinoma corroborate the low incidence of postoperative AAA rupture in patients who undergo an unrelated operation. Many patients with AAAs have severe coronary artery disease and require coronary artery bypass grafting before aneurysm repair. In the review of Hertz to the knowledge of the patient, only two (3%) had a postoperative rupture of their AAA. Acinapura et al. confirmed this result with 20 patients with AAAs who had coronary artery revascularization without a single postoperative rupture. Seven of our patients had coronary artery surgery. One died of a ruptured AAA. One died of multisystem organ failure. Three have not had the AAAs repaired. Of the remaining two, one has had successful repair, and the other is awaiting repair.

Five percent of patients with AAAs also have gallstones. Several investigators state that concomitant cholecystectomy and AAA repair can be safely done and that if cholecystectomy is not performed, there will be a high incidence of postresection cholecystitis. Fry and Fry presented 13 patients with AAA and who had a biliary procedure before the AAA repair. One patient had a postoperative rupture of a thoracoabdominal aneurysm 8 days after a cholecystectomy. Moir and Litherland also documented the safety of cholecystectomy in patients with AAAs. Thirty-four patients with symptomatic cholecystitis and AAA (average 6.3 cm) underwent cholecystectomy without a postoperative rupture.

Finally, AAA and colon carcinoma occur simultaneously in 2% of the patients with AAA. Trueblood et al. reviewed 27 patients with an intraabdominal malignancy and AAA. Three of 27 patients (11%) died of ruptured aneurysms within 3 days of the operation. Nora et al. had 2 of 14 patients with colon carcinoma die of a ruptured AAA (6 and 9 cm, respectively) (14%). In this study four of the patients had exploratory laparotomy for colon carcinoma with no postoperative rupture of an AAA.

The role of collagen, elastin, and their proteolytic enzymes, collagenase and elastase, in the pathophysiology of AAAs remains uncertain. Collagen content in AAAs as compared with aortas with occlusive disease decreases. Collagenase activity was found in AAAs but not in aortas with occlusive disease. A second proteolytic enzyme, elastase, has also been found to be elevated in AAAs. This evidence was confirmed by Cohen et al. who also showed that alpha-1-antitrypsin was decreased in AAAs, and that the ratio of elastase to alpha-1-antitrypsin was higher in patients with ruptured AAAs. However, the significance of elastin and elastase has been questioned by Dobrin et al. He treated human arterial vessels with elastase and collagenase and found that only the collagenase treated vessels ruptured. Menashi et al. could not document either decreased collagen content or increased collagenase activity in AAAs.

Finally the practical application of these enzyme studies is unclear. Cohen et al. studied the effect of surgery on arterial wall enzyme activity in a rabbit model. He measured collagenase activity in the aortas of rats that had cecal resection, or laparotomy, of aortic mobilization or aortotomy. Only aortotomy caused an increase in the activity of collagenase within the aortic wall. We have found a low risk of postoperative rupture of an AAA in patients who undergo a procedure other than aneurysm repair. We included all surgical procedures both large and small (Table I). Our only postoperative rupture occurred in a patient with an aneurysm of 5.0 cm who remained dependent on a ventilator in the intensive care unit 3 weeks after his coronary artery surgery. Two other patients whose AAA had nor been documented before creation of an arteriovenous fistula and nasal reconstruction died of ruptured AAA. By ultrasound one of the AAAs measured 6.7 cm and the others measured 5 cm at laparotomy.

REFERENCES

7. Sziglavy DE, Elliot JP, Smith RF. Clinical fate of the patient

DISCUSSION

Dr. Ronald Stoney (San Francisco, Calif.). This is an interesting report on a topic that has been brought to this Society before, and it raises our interest on the probability of rupture of an aneurysm after an unrelated operation. However, neither this paper today nor ours preceding this, or the literature that was cited can really predict the actual rupture risk. There are probably many factors at work. For example, in the present study, the risk is extremely low if you consider there was one rupture among 45 procedures. On the other hand, it may be high if you consider one rupture among seven coronary artery bypass graft patients, and it may be intermediate if you consider there was one rupture among 23 patients who had aneurysms greater than 4 cm in diameter.

Regarding the follow-up of patients who are known to have an aneurysm and who undergo an unrelated operation, I think we can make some observations and perhaps ask the authors a question. They selectively operated on their patients with infrarenal AAAs during the follow-up, as you saw. I am not sure what their exact indications were for which patient should undergo aneurysm repair after an unrelated operation. They might be able to tell us that. Second, what are their reasons not to operate on thoracoabdominal aneurysms that were in the size range for possible risk of rupture after the unrelated operation.

The authors provide no real data on the biochemical behavior of the aortic wall, but they do discuss previous literature much of which centers around proteases and protease inhibitors in the aortic wall. Probably this is of less value today, because colleagues in our wound healing lab point out that most of these older studies were based on assays that are probably inaccurate by today's standards. So we may not have the proper information from some of the older literature to make some judgments about the behavior and the metabolism and the degradation of the aortic wall.

Preliminary information from work in our own wound healing laboratory shows this group has continued to analyze human aortic tissue for both proteases and their inhibitors. In one study currently to be published, they found normal aortas, and in aortic specimens from unruptured aneurysm no collagenases could be identified. However, specific proteolytic activity, which can degrade, denature, or uncross link collagen, elastin, or other glycoproteins in the aortic wall were measured and accurately identified by use of their specific assay techniques.

The major protein synthesized in their laboratory by living human aortic tissue is a tissue inhibitor, metalloproteinase, that they call TIMP. Both TIMP and its enzymes were located in the vasovasorum of the human aneurysm wall. I believe this is the first time this has been observed.

We suspect that the major source of proteolytic activity of the human aorta may be the endothelium of the vasovasorum. These cells may respond to factors, as yet unknown, in the serum of patients with aneurysms who perhaps undergo an unrelated surgical operation or other stress. The reaction may cause an imbalance of the activities of the proteases and their inhibitors, which favor a net degradation of aortic connective tissue and leads to aneurysm rupture.

Finally, as I think about the problem now, there is not anything really new in the report you heard today from...
Dr. Durham, but I think the Society should be indebted to him for reminding us of the potentially lethal behavior of one of the vascular surgeon's most commonly encountered surgical diseases. We hope we will not be distracted from the aortic aneurysm in a patient when he or she undergoes another unrelated, though indicated, operation. Careful postoperative surveillance and timely reoperation for the aneurysm discovered should minimize the lethal sequences of aneurysm rupture in this setting.

Dr. Jon Cohen (New Hyde Park, N.Y.). The postoperative rupture of an AAA after an operative procedure continues to haunt the vascular surgeon. Although not common, its occurrence is frequent enough and the outcome disastrous enough to be a continuous dilemma. In a recent local New York meeting, an informal poll of 60 practicing vascular surgeons indicated that fully 80% of those surgeons had at least one or two patients with this very problem. Many others in this audience have had similar experiences, and this paper further indicates that this phenomenon may not just be merely a random spontaneous event.

In an animal study to specifically address this problem, we found that laparotomy with or without bowel resection causes a marked increase in aortic elastase activity, which fails to return to normal within 1 week of surgery. Furthermore, in a group of patients undergoing coronary artery bypass grafting and in other patients undergoing general surgery procedures, we found a marked increase in circulating proteolytic activity in the postoperative period that does not return to normal by 1 week after surgery.

The data presented by Dr. Durham are very interesting. If one excludes patients with aneurysms of less than 5 cm, then three of 22 patients had rupture for an overall rupture rate of 14%. My question for the authors is that given a rupture rate of 14%, do they treat their patients with large aneurysms any differently? Do they keep them in the hospital and operate on them earlier, or do they feel comfortable discharging them and readmitting them at a future date?

We now keep these patients in the hospital after their coronary or general surgery procedures and repair the aortic aneurysms before discharge, which is usually about 2 weeks after the other procedure.

Dr. Durham. To answer the first question from Dr. Cohen, it would be one of 24 patients (4%) that would give us an incidence of rupture similar to the 3% reported. The other two patients who had ruptured AAAs did not have these identified before operation or follow-up.

We attempt to repair the AAA in these patients within 2 to 3 weeks; however, the patient makes the final decision as to when and if it will be during the same hospitalization.

In answering Dr. Stoney, I found it difficult to identify the incidence of rupture from my review of pertinent articles. The AAAs at greatest risk for preoperative rupture have not been identified in the past because the articles do not identify the AAAs according to size. Indeed, it appears that the articles that do categorize the AAAs according to size show that the large AAAs have the greatest chance of rupture in the postoperative review.

We repair symptomatic AAAs, which range in size of greater than 4.5 cm. The thoracoabdominal aneurysms were not repaired either because of size (< 4 cm) or because the patient became ill.

During this study we did not gather biochemical data. The purpose was to establish or disprove clinical significance of postoperative rupture of AAAs.