Fifteen-year Experience of Transperitoneal Management of Inflammatory Abdominal Aortic Aneurysms

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Objectives: to assess the long-term outcome of patients with inflammatory abdominal aortic aneurysms.

Materials and methods: over a fifteen-year period 598 cases of abdominal aortic aneurysm were treated and, of these, 32 cases (5.3%) were inflammatory in nature. The diagnosis was made on preoperative (CT) computed tomography in fifteen cases. Twenty-six patients were symptomatic on presentation and ten cases were repaired on an emergency basis. Only six were repaired electively. The transabdominal transaortic approach without dissection on the nearby adherent structures was used routinely.

Results: there was one postoperative death from a respiratory arrest leading to a thirty-day mortality of 3.1%. Early graft thrombosis occurred in three cases (9.3%) and all underwent successful thrombectomy. Colonic ischaemia was encountered in one patient who later developed an aortoenteric fistula. Two patients suffered a non-fatal myocardial infarction postoperatively leading to an overall morbidity of 18.7%.

Conclusions: patients with inflammatory aortic aneurysms fare worse than patients with aortic aneurysms in general. Preoperative suspicion assists in planning surgery. We believe that the transperitoneal approach with an anterolateral aortotomy and minimal dissection of adherent structures offers excellent results in dealing with this difficult group of patients.

Key Words: AAA; inflammatory; transperitoneal.

Introduction

Inflammatory abdominal aortic aneurysms are a distinct subgroup accounting for 3–10% of all aortic aneurysms. The triad of thickened aneurysm wall, extensive perianeurysmal and retroperitoneal fibrosis and dense adhesions of adjacent abdominal organs define the inflammatory abdominal aortic aneurysm (IAAA). These criteria are identical to the original descriptions by Walker who described a “thick, firm, smooth wall of the aneurysm, which is shiny white in appearance, with dense fibrosis, which extends to involve adjacent structures”. They present a unique challenge to the surgical team because of the difficulty in dissection and controlling the aorta and relevant neighbouring structures.

The aim of this paper is to review our management of this subgroup of aneurysms.

Materials and Methods

Over a fifteen-year period, 598 cases of abdominal aortic aneurysm were treated; of these, 32 cases (5.3%)

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<table>
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<th>Table 1. Patient risk factors.</th>
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<td>No.</td>
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<tr>
<td>Tobacco use</td>
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<tr>
<td>Hypertension</td>
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<tr>
<td>COPD</td>
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<tr>
<td>Ischaemic heart disease</td>
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<td>Positive family history</td>
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Twenty-six patients were symptomatic on presentation, with abdominal or back pain being the most frequent finding. Two presented with shock, one with fever and malaise, and one had lower limb symptoms (pain and swelling). Weight loss was an additional finding in three cases (9.1%). Eleven patients (34.3%) were tender on abdominal examination. Two patients, a 49- and 67-year-old, had an acute rupture of a 6.5 cm and 7.9 cm AAA, respectively. They presented with circulatory shock. A further patient in their sixties had contained rupture of a 10-cm AAA identified at laparotomy.

Compromised renal function was identified in five patients (15.4%), four of whom had hydronephrosis, suggestive of chronic ureteric obstruction. Three of these patients (9.1%) had ureteric stents placed preoperatively, with return of normal renal function postoperatively.

Ten cases were repaired on an emergency basis, 16 on an urgent basis, and 6 electively, based on clinical signs. Laparotomy and transperitoneal transaortic approach was used in all cases with 31 mid-line incisions and one transverse incision. As a routine, the transperitoneal transaortic approach (TTA) without dissection on the nearby adherent structures was used. We exposed the proximal neck of the aneurysm by ligating both the left gonadal vein and the descending lumbar tributary of the left renal vein, allowing upward mobilisation of the renal vein, thus facilitating exposure of the proximal neck. The distal end of the aneurysm is then exposed, the aorta clamped, and the sac opened at the left anterolateral aspect, the main advantage of this being that the aortotomy site is well-removed from the duodenum, and hence the difficult dissection of the duodenum from the aorta is avoided. Secondly, being anterolateral, the line of minimal dissection is away from the thickest anteriorly located zone of inflammation. However, in spite of this manoeuvre, we were still forced to clamp seven cases (22%) suprarenally to avoid the inflammatory reaction. This was because of involvement of the suprarenal aorta in the aneurysmal process in three cases, and due to dense adhesions around the aorta in four cases. In two cases it was necessary to divide, and then subsequently repair, the left renal vein to allow infrarenal clamping.

Patients were followed in out-patients for the first two years after surgery, unless complications arose. Subsequent follow-up was obtained through their general practitioner. Mean follow-up was 21.1 months (range 3–49 months).

Results

CT scans showed thickening of both adventitia and media in all patients and posterior sparing of the aortic wall was present in all, while calcification of the media central to the adventitia fibrosis was present in five cases. The aneurysm was infrarenal in 29 cases (90.6%) and in three cases (9.4%) involved the renal arteries. Average aortic clamp time was 50.5 minutes (range 23–150 mins). Operative findings were characterised by a thickened aortic wall from 0.5 to 3.0 cm and a shiny white periaortic and retroperitoneal inflammatory reaction. The inflammatory adhesions involved the duodenum in 17 cases (53.1%), the inferior vena cava in nine cases (28.1%) and the left renal vein in five instances (16%). Other structures involved in the inflammatory process were the ureters 33%, small bowel 20% and sigmoid colon 5%. In one of these cases an anomalous inferior vena cava (IVC) crossed the aneurysm anteriorly, and had to be divided and repaired. A thrombosed 4-cm aneurysm was found in one case. The iliacs were involved in eighteen cases and the distal anastomosis was to the common iliac artery in thirteen cases and to the common femoral artery in the remaining five instances.

The mean diameter of the aneurysms was 7.15 cm (range 4–10 cm). Aortic grafts were placed in 30 cases (11 tube grafts and 19 bifurcated grafts). One patient had a primary axillary-bifemoral graft after ligation of an infected contained rupture. An 80-year-old patient with a 10-cm and bilateral 8-cm iliac aneurysms had deteriorating cardiac function on the table, and the procedure was abandoned in favour of treating the patient with oral steroids.

There was one postoperative death from a respiratory arrest on day 26 leading to a 30 days’ mortality of 3.1%. Eight further deaths occurred during follow-up (Table 2).

Early graft thrombosis occurred in three cases (9.3%); two bifurcated and one axillary-bifemoral graft. Successful thrombectomies were performed in all three cases. One patient had a second laparotomy one week postoperatively, at which a gangrenous portion of the sigmoid colon was resected. Fifteen months later he developed a late graft-enteric reaction with an aorto-enteric fistula and a graft infection, necessitating an axillo-bifemoral bypass which remained patent for 4 years until death from renal failure secondary to multiple myeloma. Two patients suffered postoperative

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<th>Table 2. Five-year mortality.</th>
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<td>No.</td>
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<tr>
<td>Respiratory</td>
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<tr>
<td>Malignancy</td>
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<tr>
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<td>Total</td>
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Table 3. Operative complications.

<table>
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<th>No.</th>
<th>%</th>
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<tbody>
<tr>
<td>Graft thrombosis</td>
<td>3</td>
<td>9.3</td>
</tr>
<tr>
<td>Colonic ischaemia</td>
<td>1</td>
<td>3.1</td>
</tr>
<tr>
<td>Cardiac and respiratory</td>
<td>2</td>
<td>6.2</td>
</tr>
<tr>
<td>Death (30 days)</td>
<td>1</td>
<td>3.1</td>
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myocardial infarctions leading to an overall morbidity of 18.7% (Table 3).

**Discussion**

In 1972 Walker and colleagues were the first to use the term “inflammatory abdominal aneurysm” and their clinicopathologic findings were found in 10% of the aneurysms that they treated. Since then, a separate clinical entity, the inflammatory aneurysm has been well recognised.1-6

The prevalence of abdominal aortic aneurysms has been estimated at 4.3–8.8% in the male population over 65 years of age, and ruptured abdominal aortic aneurysms account for 1–2% of all deaths in this same population. Rijbroek et al.7 reported that patients with inflammatory aneurysms were significantly younger than patients with other aneurysms (p=0.013). At 66.1 years the average age of patients in this series is consistent with means of 64 to 66 years in three previous series reporting on inflammatory aortic aneurysms. The male predominance of aortic aneurysm is slightly higher in this category, with Linblad et al.5 reporting the proportion of females at 14% for inflammatory aneurysms and 20% for aortic aneurysms generally. The published epidemiological data puts the proportion of males with inflammatory aortic aneurysms generally at 80%, and this is somewhat lower than the 93% reported in this series. A familial tendency to the development of the aneurysms exists in 6.1 to 15.1% of non-inflammatory AAAs. Few series of inflammatory aneurysms mention family history. Pennell et al.8 found a family history of aneurysms in 7.6% of patients with inflammatory aneurysms. Four patients (12%) in this series had a positive family history, with a first-degree relative having been affected by AAA in the past, and this is in keeping with the finding that 15–20% of patients with abdominal aortic aneurysms report an affected first-degree relative.9

Rasmussen and Hallett in 19979 supported the theory that inflammatory AAAs arise from the same or similar antigenic stimulus, which is responsible for the non-inflammatory aneurysms. Genetic and chemical factors such as tobacco use predispose certain people to the development of non-inflammatory AAAs and others to develop the extreme end of an inflammatory spectrum. The percentage of patients with inflammatory aortic aneurysms who smoke is high, ranging from 77 to 100% in reported series.5,8,9

Sterpetti et al.9 have postulated that inflammatory aortic aneurysms may be caused by compression of the lymphatic vessels by the enlarging aneurysm, with resulting lymphatic stasis, oedema, and fibrosis. The lymphatic network is poorly developed in the posterior wall of the aneurysm, and one indeed would expect a reduced amount of fibrosis in the posterior and lateral region in this setting. Our CT-scan findings are in keeping with this pathologic explanation, and the average wall thickness in the anterior region of the aneurysm was almost twice that of the posterolateral wall. There were no cases of clinically significant oedema.

A recent Mayo clinic study found good sensitivity for detecting an inflammatory AAA by CT scan or ultrasound, 90% versus 60% respectively.10 Pennell et al.8 demonstrated the prospective ability of ultrasound to diagnose an inflammatory aneurysm in only 13.5% of patients. Retrospectively, these ultrasound studies were reinterpreted as having findings suggestive of an inflammatory AAA in 60% of patients.

Several reports found that a significantly greater proportion of inflammatory aneurysms are symptomatic (p<0.0001).5,7,9 Weight loss has also been reported as a common associated symptom and the rate of 9.1% here compares to rates of 20% of others. We also noted a high rate (22%) of chronic obstructive pulmonary disease which is considerably higher than the ten per cent reported by Crawford et al.11

Six patients (22.2%) were tender on clinical examination, a rate almost identical to rates of 22% and 23% in two previous series,1,6 which illustrates that tenderness on palpation is not a universal feature of inflammatory aneurysms and its absence is not significant in ruling out these lesions. As has been previously discussed in the literature, tenderness to palpation is a subjective finding and depends on the enthusiasm with which it is sought. It is also important to remember that not all tender aneurysms are either inflammatory or ruptured; however, when elicited, tenderness is an indication for urgent repair.

The extension of fibrosis to involve the ureters was responsible for compromised renal function in 18.5% of the patients, which is similar to other reported series. In 1994 Rijbroek et al.7 reported the absence of associated iliac or femoral aneurysms, which contrasts with the findings of Pennell where half their patients...
had additional aneurysms.\(^6\) In this series additional aneurysms were found in 9 patients (28%).

Two patients (6.3%) in this series had ruptured aneurysms at the time of operation: this is similar to the 6–12% rates of rupture reported by others\(^4,5,8\) who found a significantly lower rate of rupture with circulatory shock in inflammatory aneurysms than in aneurysms generally \((p<0.01)\). The average clamp time reported by Leseche \textit{et al.}\(^4\) was 48 min, which is almost identical to the present series. Linblad \textit{et al.}\(^5\) reported that the duration of aortic occlusion did not differ between inflammatory and non-inflammatory aneurysms.

Thirty-five per cent of the grafts performed were tube grafts, while distal vessel disease necessitated bifurcated aortic grafts in 65%. None of the reviewed series reported the use of extra anatomical grafts, while in our experience these were used twice: in one instance as a primary procedure and the other as a secondary procedure after the primary graft had to be removed. Figures for operative complications have not been widely reported in previous series of inflammatory aneurysms, but the rate of operative complications in this series (18.7%) parallels that of two other review articles.\(^8,11,12\)

In early experience with inflammatory AAAs surgeons attempted extensive adhesiolysis of peri-aneurysmal structures. These attempts were complicated by enterotomies, especially of the duodenum, and injuries to the ureters and vena cava substantially affecting operative mortality. In 1978 Goldstone \textit{et al.}\(^13\) described the importance of a modified approach to the inflammatory AAA. They emphasised the hazards of attempted mobilisation of the duodenum from the anterior wall of the aneurysm and advised obtaining proximal and distal control of the aneurysm with "as little dissection as possible". Crawford\(^11\) advised against dissection of the surrounding structure and advocated aortic cross-clamping at the diaphragm until the proximal anastomosis is completed. With this technique of limited dissection he described operative mortality of 3–4%, matching those of non-inflammatory AAAs.

There was no operative mortality in the urgent or elective cases in this series and this compares favourably with the 9% mortality for elective repair of inflammatory aneurysms reported in a recent Swedish multicentre study\(^2\) and the 7.9% reported by Pennell \textit{et al.},\(^3\) which was found to be significantly higher than the 2.4% in the patients with non-inflammatory aneurysms. This difference was attributed to technical problems encountered as the result of the inflammatory process.

In a similar series collected over 17 years Moosa \textit{et al.}\(^6\) reported an operative mortality of 5%, which is almost identical to that of 3.1% in this series. These rates compare favourably with rates of 11–12% in other series of inflammatory aneurysms and more widely reported mortalities for all abdominal aneurysm repairs of between four and eight per cent. Rijbroek \textit{et al.}\(^7\) had zero per cent in-hospital mortality but did not report a 30-day figure.

The superiority of either a transabdominal or retroperitoneal approach for aortic surgery remains a source of controversy.\(^14\) Although it is generally agreed that the retroperitoneal incision offers advantages in selected patients with juxtarenal and suprarenal aortic aneurysms, it has failed to gain widespread acceptance for routine infrarenal aortic reconstruction. Since the resurrection of the retroperitoneal approach, numerous reports have claimed a clear superiority of this incision approach in terms of intraoperative fluid replacement, postoperative pulmonary complications, length of ileus, and length of stay in the intensive care unit. But significant right iliac aneurysms, diffuse retroperitoneal fibrosis, previous retroperitoneal dissection (e.g. left colectomy) all are contraindications to this approach. In a prospective trial Cambria \textit{et al.}\(^14\) found no significant difference between the transperitoneal approach and retroperitoneal approach.

Remarkable progress in the diagnosis and management of abdominal aortic aneurysms has been made since the first repair by Dubost in 1951. Recent clinical findings reveal strong links to current smoking and familial tendencies. Smoking and viruses may represent environmental-risk factors that accentuate an inflammatory response, while the familial tendency may represent a genetic effect in immune-responsiveness, tissue repair or structure proteins.

Preoperative suspicion of an inflammatory aneurysm assists in planning surgery that is made more hazardous by the presence of fibrosis and adhesions. A diagnosis of inflammatory aneurysm should be considered in any patient with an abdominal aortic aneurysm who is symptomatic in the absence of evidence of rupture. Computed tomography is the imaging tool of choice in assessment of such patients.

As all vascular surgeons are familiar with the transperitoneal approach, the anterolateral aortotomy with minimal dissection around adherent structures is a feasible option in dealing with technically difficult inflammatory aortic aneurysms.

\section*{References}

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