Inflammatory abdominal aortic aneurysm: Predictors of long-term outcome in a case-control study

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Background. *Inflammatory abdominal aortic aneurysms (AAAs) are relatively rare clinical entities. The aim of this study is to ascertain the risk factors in patients with inflammatory AAAs and clarify which feature plays a role in determining the long-term outcome in these patients.*
Methods. *Between 1990 and 1997, 238 patients underwent surgery for an AAA at our institution, 17 (7.1%) of whom had the diagnosis of inflammatory type AAA. This group was matched in a case-control fashion to a group of 35 patients with similar characteristics of age, gender, and preoperative risk factors who were operated on for a noninflammatory AAA. All available clinical, pathologic, and postoperative variables were retrospectively reviewed, and the 2 groups were compared. In the inflammatory group, risk factor analysis was performed for poor outcome.*
Results. *All operations were performed through a standard transperitoneal median laparotomy. The 2 groups did not differ significantly in terms of clinical characteristics and preoperative risk factors Patients with inflammatory AAAs were significantly more symptomatic (100% vs 31%,* $P = .03$*) and had larger aneurysm size on admission (8.2 ± 1.2 cm vs 6.1 ± 0.4 cm,* $P = .04$*). In inflammatory AAAs, preoperative erythrocyte sedimentation rate was found to be significantly elevated (mean, 48 ± 14 mm/h vs 8 ± 3 mm/h,* $P = .01$*). Surgical morbidity and mortality rates did not differ between 2 groups. The intensive care unit and hospital stay periods were similar in both groups (2.2 days vs 1.8 days,* $P = .25$*, and 9.2 days vs 8.1 days,* $P = .35$*). Eight-year survival rates of inflammatory and noninflammatory groups were 60% and 74%, respectively (*$P = .01$*). Results from Cox proportional hazards model analysis showed that a high sedimentation rate after surgical intervention (*$P = .02$*), cardiovascular disease (*$P = .01$*), postoperative persisting fibrosis with ureteral entrapment (*$P = .01$*), and postoperative chronic renal failure (*$P = .02$*) were independent risk factors for death. Other surgical variables did not prove to be risk factors for long-term mortality.*
Conclusions. *Although the preoperative characteristics may differ in patients with inflammatory type AAAs, they can be treated today with low morbidity and mortality rates. However, inflammatory process may continue postoperatively because of unknown reasons, and the study documented that persisting, postoperative inflammatory process with or without retroperitoneal fibrosis may place a patient at high risk for poor outcome. This is important information for the long-term management of these patients, and we believe that their follow-up protocols should be more comprehensive to further improve their long-term survival rates. (Surgery 2007;141:83-9.)*

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Inflammatory abdominal aortic aneurysm (AAA) is considered a distinct variant of AAA and accounts for 2% to 14% of all cases.[1-5] Although this type of aneurysm is still accepted as a unique challenge because of its characteristic dense periaortic inflammation involving adjacent organs, very satisfactory results have been reported.[6-8] However,

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because of its rarity, even large cardiac centers will see only a handful of cases each year. Because there are limited number of studies evaluating the long-term outcome of these patients, we do not know clearly which factor play a role in determining long-term outcome of this subgroup of patients. Postoperative development of visceral organ complications in previously involved or uninvolved structures attributable to ongoing fibrosis after surgical intervention are well-known complications, but the impact of ongoing inflammation on long-term survival has not been largely described in the literature. The aim of this retrospective study is to ascertain the risk factors in patients with inflammatory AAAs and clarify which features play a role in determining the long-term outcome of these patients. We also hypothesized that follow-up protocols of this group of patients would differ from those of noninflammatory types and aimed to clarify which factors play a role in determining the long-term outcome in this group of patients.

METHODS

We reviewed hospital records to determine the patients who had operative repair of an AAA at Bezm-I Alem Vakif Gureba Hospital, Cardiovascular Surgery Service. The study was approved by the ethical committee of the institution. Between 1990 and 1997, 238 patients underwent an elective surgical intervention for a nonruptured AAA, 17 (7.1%) of whom had the diagnosis of inflammatory type AAA. Only symptomatic but nonruptured AAA were included in the study, and patients with a ruptured aneurysm were excluded from the study population. In all patients, clinical assessment, electrocardiogram, and biochemical analysis were performed as an initial diagnostic workup. The definitive diagnosis was established by using standard imaging modalities including ultrasonography (USG), contrasted or uncontrasted computed tomography (CT), and magnetic resonance imaging (MRI). The aneurysm was accepted as inflammatory if all of the following were present: (1) marked thickening of the aneurysm wall; (2) presence of periaortic fibrosis and inflammatory process involving adjacent organs; and (3) histopathologic evidence of inflammation at the biopsy material. Histologic confirmation was routinely obtained in all patients.

Transperitoneal standard median laparotomy was the standard approach used in all cases. In the entire cohort, infrarenal abdominal aorta has been controlled safely with infrarenal clamping in 43 cases, but, for the remaining 9 patients, the suprarenal aorta was the site for clamping. We exposed the proximal neck of the aneurysmal sac by upward mobilization of the left renal vein. Following the control of distal aorta, the aorta was clamped and the sac was opened at the right anterolateral aspect. The aneurysmatic segment was replaced with an appropriately sized Dacron tube graft in 40 patients, whereas in the remaining 12 patients, an aortobifemoral bypass operation was performed by using a bifurcated graft.

Outcome variables and statistical analysis. The inflammatory group (n = 17) was matched in a case-control fashion to a group of 35 patients with similar characteristics of age, gender, and preoperative risk factors who were operated on for noninflammatory type AAAs. The operative reports were reviewed for the extension of the inflammatory process, organs involved, and suprarenal clamping. In-hospital morbidity and mortality, length of hospital stay, and postoperative survival rates were assessed. In-hospital mortality was defined as death occurring during hospitalization. Current smoking was tobacco use within 2 weeks of surgery. Patient follow-up was obtained by direct patient contact or telephone interview. Predictors of ongoing inflammation, especially family history of aneurysms, smoking history, inflammation markers, renal problems, and the typical signs of inflammation, were assessed. During the postoperative period, the erythrocyte sedimentation rate (ESR) was monitored serially at 3-month intervals. To determine the ongoing inflammation and fibrosis, we followed all patients regularly and evaluated with CT scan at 3 months, 6 months, and yearly thereafter.

Continuous data were expressed as means ± SEM, and categorical variables are reported as a percent. Continuous data were compared by paired samples *t* test and categorical data by the chi-square test. The primary outcome measure was death. The Cox proportional hazards model was used to investigate risk factors for death. A forward-step procedure was used to select prognostic factors. Risk factors with a *P* value $<.1$ in the univariate analyses were included in the multivariate models. In the multivariate models a *P* value $<.05$ was considered significant.

RESULTS

The demographic data of both groups is given in Table I. The mean age was 57.6 ± 5.2 years (range, 42-75 years) in the inflammatory group and 62.4 ± 6.8 years (range, 53-79 years) in the noninflammatory group. There was no significant gender difference between the 2 groups (82.3% men vs 80% men; $P = .45$). The prevalence of preoperative risk factors was similar, and no variable achieved statis-

Table I. Preoperative demographics of patients

	Inflammatory AAA (n = 17)	*Noninflammatory AAA (n = 35)*	P *value*
Coronary artery disease	8 (47%)	16 (45%)	.42
COPD 10	(59%)	19 (54%)	.34
PAOD	7 (41%)	15 (43%)	.32
Hyperlipidemia	16 (94%)	32 (91%)	.40
Diabetes mellitus	9 (53%)	14 (40%)	.25
Chronic renal failure	5 (29%)	7 (20%)	.15
Hypertension	13 (76%)	25 (71%)	.38
Current smoking	16 (94%)	32 (91%)	.40
Symptoms			
Abdominal pain	13 (76%)	11 (31%)	<.01
Lumbar pain	12 (70%)	7 (35%)	.01
Weight loss	14 (82%)	8 (23%)	<.01
Tenderness on palpation	14 (82%)	8 (23%)	<.001
Fever	9 (53%)	4 (11%)	.01
Aneurysm size (>7 cm)	17 (100%)	16 (45%)	.03
High ESR	16 (94%)	5 (14%)	<.001
Family history	6 (35%)	2 (6%)	.01

AAA, Abdominal aortic aneurysm; *COPD*, chronic obstructive pulmonary disease; *PAOD*, peripheral arterial occlusive disease; *ESR*, erythrocyte sedimentation rate.

tical significance except family history (35% vs 6%, $P = .01$).

Patients in the inflammatory group were significantly more symptomatic; all patients in this group had at least 1 symptom at the time of diagnosis. In contrast, there were only 11 (31%) symptomatic patients in the noninflammatory group ($P = .03$). Weight loss (82% vs 23%, $P < .01$) and abdominal pain (76% vs 31%, $P < .01$) were the most common findings in the study group. Fever was reported more frequently in patients with inflammatory aneurysm (53% vs 11%, $P = .01$). An elevated ESR was observed in 94% of patients with an inflammatory AAA and in only 14% of patients with a noninflammatory AAA ($P < .001$). Mean ESR levels of inflammatory and noninflammatory AAAs were 48 ± 14 mm/h and 8 ± 3 mm/h, respectively ($P = .01$). Similarly, white blood count was also elevated in 82% of patients with an inflammatory AAA, compared with 11% of patients with a noninflammatory AAA ($P < .001$). The patients had larger aneurysm size on admission (8.2 ± 1 cm vs 6.1 ± 0.4 cm). All patients from the inflammatory group had an aneurysm size >7 cm, whereas in the other group only 16 patients had an aneurysm size >7 cm ($P = .03$).

All inflammatory AAA patients had the presence of periaortic fibrosis and an inflammatory process involving adjacent organs. The aneurysm was infrarenal in all cases. Although the duration of operation was longer in the inflammatory group because of dense adhesions (234 ± 15 min vs 189 ± 17 min), this difference did not reach statistical significance ($P = .12$). Average aortic cross-clamp time was similar in both groups (57 ± 12 min vs 52 ± 9 min, $P = .51$). In the biopsy material obtained perioperatively, the aneurysm wall was found to be thicker in inflammatory aneurysms (9.8 ± 1.7 mm vs 3.7 ± 1.2 mm, $P = .02$). Inflammatory AAAs showed typical dense perianeurysmal fibrosis involving duodenum in all cases, whereas this percentage was 14% in the other group ($P < .001$). The left renal vein was also involved by the inflammatory process in 7 cases in the entire cohort including 6 (35%) patients in the inflammatory group and 1 (3%) patient in the noninflammatory group ($P < .01$). In addition, in the inflammatory group, there were varying degrees of inferior vena cava and left ureteral involvement in 5 (29%) and 8 (47%) patients, respectively.

Early postoperative outcome. In-hospital mortality in the inflammatory AAA series was 2 of 17 (11%) patients. Acute renal failure was the cause of early mortality in these 2 patients.

Preoperative ureteral entrapment was the possible cause of postoperative acute renal failure in these patients. These 2 patients died at the fifth and ninth days, respectively. There was 1 early death in the other group because of myocardial infarction in a patient with a previous history of a coronary artery bypass grafting operation.

In the entire cohort, 5 patients required early reoperation because of postoperative bleeding (2 patients in the inflammatory group and 3 in the noninflammatory group, $P = .42$). All patients were

Table II. Peri- and postoperative data of both groups

	Inflammatory AAA (n = 17)	*Noninflammatory AAA (n = 35)*	P *value*
Operation time	234 ± 15 min	189 ± 17 min	.12
Cross-clamp time	57 ± 12 min	52 ± 9 min	.51
Aneurysm wall thickness	9.8 ± 1.7 mm	3.7 ± 1.2 mm	.02
Early mortality	2 (11%)	1 (3%)	.20
Extubation time	6.3 ± 1.2 h	5.9 ± 1.4 h	.30
Intensive care unit stay	2.2 d	1.8 d	.25
Intermittent renal failure	5 (29%)	3 (8%)	.04

AAA, Abdominal aortic aneurysm.

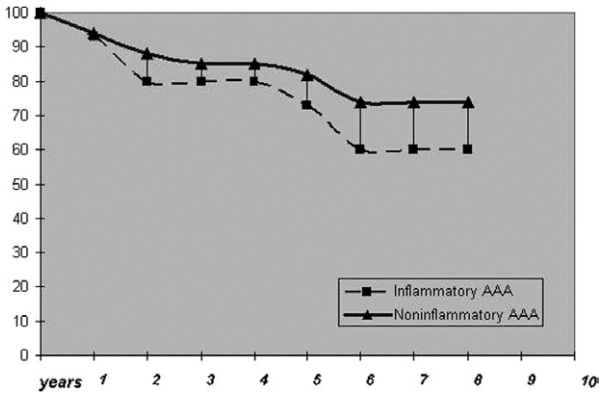

Fig 1. Eight-year survival rates in all patients. *AAA*, Abdominal aortic aneurysm.

extubated within the operation day. Eight patients (5 in the inflammatory group and 3 in the noninflammatory group) had intermittent renal failure, but dialysis was required in 3 patients only for 2 weeks. Patients with inflammatory AAAs tended to have higher rates of renal failure ($P = .04$) than did patients with noninflammatory AAAs. All patients requiring postoperative dialysis were in the inflammatory group. The intensive care unit and hospital stay periods were similar in both groups (2.2 days vs 1.8 days, $P = .25$, and 9.2 days vs 8.1 days, $P = .35$). The peri- and postoperative data are listed in Table II.

Late postoperative outcome. After a follow-up of up to 8 years, 15 (30%) of 49 patients have died. Among these, 6 patients were in the inflammatory group (6/15) and 9 were in the noninflammatory group (9/34). Survival from the first procedure to 8 years for all 49 patients undergoing an operative procedure was 60% in the inflammatory group and 74% in the noninflammatory group ($P = .01$) (Fig 1).

Table III. Risk factors analyzed by means of univariate analysis in patients with inflammatory AAA

Variable	*Hazard ratio*	*95% CI*	P *value*
Cardiovascular disease	2.43	0.97-3.12	.03
Preop renal insufficiency	0.72	0.38-1.63	.06
Size on admission	0.57	0.26-1.02	.08
Aneurysm wall thickness	0.81	0.34-1.53	.10
Preop ESR level	1.12	0.47-2.12	.07
Postop ESR level	2.18	1.12-3.76	.018
Preop ureteral entrapment	1.43	0.89-2.97	.06
Early intermittent renal failure	0.92	0.73-1.44	.07
Postop chronic renal failure	2.24	1.14-3.36	.02
Postop persisting fibrosis with ureteral entrapment	3.87	2.16-11.81	.017

Variables are shown only if *P* value is .10 or less.
AAA, Abdominal aortic aneurysm; *CI*, confidence interval; *ESR*, erythrocyte sedimentation rate; *postop*, postoperative; *preop*, preoperative.

After surgical repair, the inflammatory process had completely resolved at late follow-up in only 33% (5/15) of patients. The remaining 10 patients showed a persistent inflammatory process on scans obtained at the late postoperative follow-up period. In these patients, computed tomography scans obtained at late follow-up revealed varying degrees of ureteral entrapment, renal atrophy, and hydronephrosis. In addition, the continuation of an elevated ESR level was also noticed when compared with those of patients in whom the inflammatory process had resolved (45 ± 10 mm/h vs 9 ± 5 mm/h, $P < .01$). In the inflammatory group, among 6 late deaths, there was only one cardiac-related mortality. Interestingly, all remaining late deaths in this group were observed in patients with persistent inflammatory process and ongoing fibrosis. One patient died at the end of the first year because of urosepsis. The cause of mortality in 2 patients was renal failure, and we unexpectedly noted development of ureteral stricture late after surgery in previously asymptomatic patients, all of whom had a significant rise in ESR postoperatively. The exact cause of mortality in the remaining 2 patients cannot be clearly determined. In the noninflammatory group, 2 patients had cardiac infarction (2 and 5 years later, respectively), 2 patients had cancer of the lung, and 5 patients died of unknown reasons that were not related to surgical intervention.

Univariate analysis of risk factors for subsequent death in the inflammatory group is shown in Table III. To understand further whether the

Table IV. Risk factors analyzed by means of multivariate analysis in patients with inflammatory AAA

Variable	*Hazard ratio*	*95% CI*	P *value*
Cardiovascular disease	2.25	0.86-2.72	.02
Postop ESR level	2.07	1.23-3.19	.01
Postop chronic renal failure	2.04	1.10-3.04	.02
Postop persisting fibrosis with ureteral entrapment	6.57	2.27-8.31	.01

AAA, Abdominal aortic aneurysm; *CI*, confidence interval; *ESR*, erythrocyte sedimentation rate; *postop*, postoperative.

risk factors for late death differ within the group, we did a subgroup analysis. We appreciated that the ongoing inflammatory process is an important factor and that it had clinical relevance. The results from the multivariate Cox proportional hazard model analysis are displayed in Table IV. Of all risk factors included in this study, 4 variables are identified as independent predictors of late death: elevated ESR after surgery, cardiovascular disease, postoperative chronic renal failure, and postoperative persistent fibrosis with ureteral entrapment. Persistent inflammatory process and fibrosis are strongly associated with the risk of death in patients undergoing surgery for an inflammatory AAA.

DISCUSSION

With the recent refinements in vascular surgery, cardiac anesthesia, and postoperative care, inflammatory AAAs can be surgically treated with very low morbidity and mortality rates.[1,4] On the other hand, our study documented that the late survival rate of this subgroup is lower than expected, especially in patients with an ongoing inflammatory process.

In our study, the incidence of inflammatory type AAAs was 7.1%, which is quite comparable with the literature. It is not certain whether they have an independent pathogenesis or simply represent the extreme end in the spectrum of inflammatory changes present in all aortic aneurysms. Although various factors including pathogenic B-cell response, cytomegalovirus infection, extracellular matrix modification, cytokines and HLA-DR1B gene association all have been proposed in the etiology, the exact cause of the inflammation is still a subject considerable debate.[9-13] Familial predilection has also been described in atherosclerotic AAAs,[14,15] but few studies address specifically the inflammatory type aneurysms. In parallel with other series,[1] we also observed a familial tendency in our patient group.

Various studies revealed that a significantly higher proportion of patients with inflammatory AAAs are symptomatic on admission. Von Fritschen and colleagues[3] reported that 74% of patients with inflammatory AAA were symptomatic, whereas this percentage was 100% in our group. In our inflammatory series, all patients were symptomatic at the time of diagnosis. The presence of signs and symptoms in the group may be related to higher aneurysm size in our patients. Preoperative diagnosis of an inflammatory aneurysm requires a high index of suspicion and comprehensive evaluation of individual patient. Nowadays, current imaging modalities may possess 85% sensitivity and 100% specificity in detecting an inflammatory process.[16] Especially in cases with a marked thickening of aneurysmal wall, the presence of fibrotic changes in retroperitoneal area together with clinical and laboratory data should raise the suspicion for an inflammatory process.

The incidence of obstructive uropathy in inflammatory AAA was approximately 20%.[4,17] In our series, the incidence of preoperative chronic renal failure was 29%, but preoperatively, there were only 3 patients with obstructive uropathy. However, our study showed that the patients with inflammatory aneurysms were at an increased risk for postoperative renal failure. Postoperative computed tomographic evaluation revealed that, despite surgical intervention, the fibrotic process did not resolve or even progress in some patients. Therefore, we believe that postoperative persistence or progression of ureteral entrapment was the underlying cause of renal dysfunction. The postoperative renal failure in this patient group was possibly due to the ureteral involvement. Five of 17 patients had postoperative renal failure, and 3 required renal replacement therapy for 2 weeks. More importantly, at the late follow-up, various degrees of renal atrophy and renal stricture were observed in our patients.

Another important finding is the evidence of high ESR level in these patients. We believe that the presence of an elevated ESR preoperatively should alert the surgical team to prevent the iatrogenic events that may occur during the surgical procedure. Previous studies have generally focused on the preoperative level of ESR; however, the significant observation of this study concerns postoperative elevation of ESR. Although the number of patients used for statistical analysis was small, the presence of a relationship between high postoperative ESR and ongoing inflammation should be considered an important preliminary finding, and ESR may be used as an important parameter in the follow-up of these patients.

This study showed that the surgical repair of an inflammatory AAA might not result in complete

resolution of inflammatory process. The inflammatory process had completely resolved at late follow-up in only 33% of patients. The study reported by Nitecki[1] agrees with our observations. In their series, Nitecki stated that complete resolution was observed in slightly more than half of the patients. More importantly, we demostrated a significant association between the ongoing inflammatory process and subsequent death. Although inflammatory AAAs have similar perioperative mortality rate in our series, the late outcome is impaired in comparison with the noninflammatory counterpart. In the light of this finding, we can conclude that the follow-up protocols of these patients should differ from patients with an aneurysm of arteriosclerotic origin.

Endovascular repair of inflammatory AAAs has been proposed by some authors,[18,19] but the effect of endograft placement on perianeurysmal inflammation is not clearly defined. Endograft placement may induce a systemic inflammatory response in these patients, but the cause of this local response remains unknown. Vallabhaneni[20] reported that an endograft did not reverse the fibrotic process and even induced this condition in approximately one sixth of their patients without evidence of preoperative fibrosis. Although it is proposed that endovascular techniques may be an alternative, indications possess some controversy, especially in inflammatory lesions.

There are some limitations of the study. First, the study population is small because this is a single-center experience and even large cardiac centers will see only a handful of cases each year. However, in the statistical analysis, the levels of significance were generally strong. In addition, because of its retrospective design, the findings were made on the basis of data available in the medical records. Advances in patient management during the study period may also influence the postoperative results.

In conclusion, patients with an inflammatory AAA are usually symptomatic on admission and have an elevated ESR. Although the wall of the aneurysm sac is thicker, the huge size of the aneurysm is associated with an absolute risk of rupture and death. Early mortality and morbidity rates are identical to those encountered in the arteriosclerotic aneurysms. However, the long-term outcome of these patients is impaired because of adjacent organ involvement. This population-based study has shown that the presence of elevated postoperative ESR level, cardiovascular disease, postoperative chronic renal failure, and an ongoing inflammatory process are all independent risk factors that place a patient at high risk for poor outcome. This information is important for the long-term management of these patients, and we believe that their follow-up protocols should be more comprehensive to further improve their long-term survival rates.

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