Inflammatory aortic aneurysm is associated with increased incidence of autoimmune disease

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Objective: **It has been suggested that certain genetic risk factors indicative of an autoimmune mechanism can be identified in patients with inflammatory aortic aneurysm (IAA). We therefore investigated whether there was a higher incidence of autoimmune diseases in patients with IAA. Further, we explored risk factors, need for in-hospital resources, and early results of treatment, in a case-control study in a university hospital setting.**

Material and methods: **From 1983 to 1994, 520 patients were operated because of abdominal aortic aneurysm (AAA). Thirty-one patients had IAA. Control subjects were matched for aneurysm rupture, emergency or elective hospital admission, and date of operation. Two noninflammatory AAA were included for every IAA.**

Results: **Of the 31 patients with IAA, 6 patients (19%) had autoimmune disease, compared with none of the control subjects ($P = .0017$). Two patients had rheumatoid arthritis, 2 patients had systemic lupus erythematosus, 1 had giant cell arteritis, and 1 patient had an undifferentiated seronegative polyarthritis diagnosed as rheumatoid arthritis. Nineteen patients (61%) with IAA had involvement of the duodenum, and 8 patients (26%) had hydronephrosis with ureteral involvement. Operating time was longer in the IAA group, which also had a higher need for blood transfusion. Hospital stay, intensive care unit stay, and 30-day mortality were similar in the two groups.**

Conclusion: **Except for longer operating time and more need for blood transfusions in the IAA group, use of hospital resources was similar after operations to treat IAA or noninflammatory AAA. The study findings indicate an association between IAA and autoimmune disease. This is in accordance with other reports that showed a genetic risk determinant mapped to the human leukocyte antigen (HLA) molecule in these patients. Further research is necessary to explore whether IAA might be a separate entity with a role of antigen binding in the origin of the disease. (J Vasc Surg 2003;38: 492-7.)**

Inflammatory aortic aneurysm (IAA) is a special variant of abdominal aortic aneurysm (AAA) first described by Walker et al.[1] Hydronephrosis in AAA, described by James[2] in 1935 and DeWeerd et al[3] in 1955, was probably caused by the same condition. Other investigators have stated that one can observe, to varying degree, the same type of inflammation in all atherosclerotic aneurysms.[4,5] IAA is characterized by a gray-white appearance observed during operation. Thickened aneurysm wall, perianeurysmal fibrosis, and dense adhesions to adjacent abdominal organs such as the duodenum and ureter are characteristic of IAA.[1,6] In particular, the anterior and lateral walls of the aneurysm are thickened, and the inflammatory tissue includes abundant infiltration of lymphocytes and plasma cells.[5,7,8] There is a similarity to idiopathic retroperitoneal fibrosis or periaortic fibrosis.[9] The histologic appearance of IAA is almost identical to giant cell arteritis.

The association between AAA, giant cell arteritis, and Takayasu disease has been described.[10] In contrast, Ehrenfeldt et al[11] did not find any association between aortic aneurysm or aortic dissection and occurrence of giant cell arteritis or polymyalgia rheumatica. In review of our cohort of AAA, it was striking that several patients with IAA had autoimmune disease, eg, rheumatoid arthritis, systemic lupus erythematosus (SLE), and giant cell arteritis. Further, it has been suggested that certain genetic risk factors can be identified in patients with IAA, indicating an autoimmune mechanism.[6,12]

The purpose of our study was to investigate whether there is a higher incidence of autoimmune disease in patients with IAA. Further we explored risk factors, need for in-hospital resources, and early results after treatment in patients with IAA compared with patients with noninflammatory AAA.

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Table I. Age, sex, and prevalence of comorbidity in 31 patients with IAA and 62 control patients with AAA

	IAA		*Controls*		
	N	*%*	*N*	*%*	*Significance*
Age (y, median)	69.9		70.6		NS
Gender					
Male	25	81	48	77	NS
Female	6	19	14	23	NS
Comorbidity					
Coronary heart disease	11	35	20	32	NS
Chronic obstructive pulmonary disease	2	6	9	15	NS
Diabetes	2	6	3	4.8	NS
Renal failure	7	23	8	13	NS
Hypertension	8	26	17	27	NS

IAA, Inflammatory aortic aneurysm; *AAA,* abdominal aortic aneurysm; *NS,* not statistically significant.

MATERIAL AND METHODS

From 1983 to 1994, 520 patients were operated on because of AAA. All patient records were reviewed, and the cohort was reexamined in the outpatient clinic or by questionnaire to surviving subjects 5 to 16 years after the operation. Thus 100% follow-up was obtained. In 31 patients (6%) the operating surgeon diagnosed IAA. The macroscopic criteria for IAA used in our department include white glistening perianeurysmal fibrosis, thickened aneurysm wall, and dense adhesions of adjacent abdominal organs.[13] In 21 cases the diagnosis was confirmed at histologic examination with hematoxylin-eosin-saffran staining. Characteristic findings at microscopy were loss of smooth muscle cells and elastic tissue in the media, which in most cases was extensively replaced with fibrous tissue. The periaortic tissue exhibited granulation tissue with fibrosis and a lymphoplasmocytic inflammatory infiltrate, often with follicular aggregations of mature lymphoid cells. Granulomas were not observed.[7] Aneurysm wall thickening was recorded in all patients either intraoperatively, on the histologic specimen obtained during surgery, or on preoperative computed tomography scans.

Six patients (19.4%) with IAA were women; median patient age was 70 years (Table I). Twelve IAA (39%) were asymptomatic, 15 IAA (49%) were symptomatic without rupture, and 4 IAA (13%) were ruptured.

The study design was case-control within a cohort. The control group was matched for rupture, emergency or elective hospital admission, and date of operation. Control subjects were selected from a group of patients with noninflammatory AAA, and 2 control subjects were included for every patient with IAA, for a control group with 62 patients. Control subjects included the last patient operated on before and the first patient operated on after each patient with IAA. For patients with ruptured IAA, the last patient with ruptured non-IAA before and the first patient after served as controls. Review of medical records and follow-up were performed similarly in both groups. The diagnosis of autoimmune disease was made before surgery in all cases, and was verified by a rheumatologist from clinical investigation, serologic tests, and biopsy findings, when needed. We also compared the patients with IAA with the total group of 489 patients operated on to treat non-IAA during the study period. Because the control group was matched for rupture, and type of admission was related to occurrence of symptoms, we compared the incidence of symptoms with the total group of 341 patients with nonruptured AAA.

Table II. Prevalence of autoimmune disease in 31 patients with IAA and 62 control patients with noninflammatory AAA

	IAA	*Controls*	P
Autoimmune diseases	6 (19.3%)	0	.0017
Rheumatoid arthritis	3	—	
Systemic lupus erythematosus	2	—	
Giant cell arteritis	1	—	

IAA, Inflammatory aortic aneurysm; *AAA,* abdominal aortic aneurysm.

In-hospital resources were estimated from patient records. Length of stay, stay in the intensive care unit or intermediate care unit, operating time, anesthesia time (total time in the operating room), and need for assisted ventilation and blood transfusions were recorded. From preoperative investigations, concomitant diseases such as coronary heart disease (angina, previous myocardial infarction, percutaneous coronary intervention, or aortocoronary bypass grafting), hypertension treated medically, renal failure (creatinine concentration >140 μmol/L), chronic obstructive pulmonary disease, and diabetes were also noted.

The investigation was approved by the local ethics committee. Data were entered into the statistical database Medlog, with which analysis also was performed. For statistical analysis the nonparametric Wilcoxon rank sum test and χ^2 test, with appropriate corrections, were used. Observed differences with $P < .05$ were considered statistically significant.

RESULTS

Of the 31 patients with IAA, autoimmune disease was diagnosed in 6 before surgery. In contrast, no autoimmune

Table III. Symptoms and signs in patients with IAA compared with control subjects with noninflammatory AAA

	IAA (N = 30)		*Controls (N = 62)*		
	n	*%*	*n*	*%*	P
General symptoms	7	23	3	4.8	.02
Joint phenomena	2	6.7	0	0	NS
ESR (median) (mm/L)*	28.0		16.5		<.001
Hematocrit (median)*	0.38		0.43		.011
Aneurysm size (median) (mm)	65		60		NS
Duodenal involvement	19	61	—		—
Aortoduodenal fistula	1	3.0	—		—
Distal involvement†	9	27	—		—
Hydronephrosis	8	26	1	1.6	.0008
Right side	2		1		
Left side	3		0		
Bilateral	3		0		

IAA, Inflammatory aortic aneurysm; *AAA,* abdominal aortic aneurysm; *ESR,* erythrocyte sedimentation rate; *NS,* not significant.
*Patients and control subjects with ruptured aneurysms excluded.
†Inflammation extending onto common iliac arteries.

disease was found in the control subjects ($P = .0017$; Table II). Two patients (1 seropositive, 1 seronegative) had rheumatoid arthritis,[14] and 2 patients had SLE.[15] These diagnoses were in accordance with the criteria set forth by the American College of Rheumatology. Both patients with SLE also had antiphospholipid syndrome. One of these patients had low titer of antinuclear antibody, low titer of antideoxyribonucleoprotein, anti–Sjögren's syndrome A antibody, high titer of antiribonucleoprotein, and high titer of anti-Smith antibody, and medium titers of anti-ribonucleoprotein and anticardiolipin antibodies. This 39-year-old woman had an aneurysm with diameter of 65 mm. The other patient with SLE and antiphospholipid syndrome had an autoantibody profile consisting of positive antinuclear antibodies, medium titer of positive anticardiolipin antibodies, low titer of anti–Sjögren's syndrome A antibody, high titer of anti–Sjögren's syndrome B antibody, low titer of anti-Smith antibody. One patient had undifferentiated seronegative polyarthritis, which was diagnosed as rheumatoid arthritis. One patient had giant cell arteritis, documented by characteristic inflammatory lesions in the temporal artery. Microscopy of the biopsy specimen showed pronounced expansion of the internal lamina elastica, necrotic and hyalinized lamina media, infiltration of mononuclear leukocytes in all layers, and giant cells in the internal lamina elastica. American College of Rheumatology criteria for classification of giant cell arteritis were fulfilled.[16] In the IAA group, 1 patient with rheumatoid arthritis, 1 patient with SLE, and 1 patient with giant cell arteritis used oral corticosteroid therapy; no patients in the control group used such treatment.

In 3 of 489 patients (0.6%) with noninflammatory AAA autoimmune disease was diagnosed at operation. This incidence is significantly lower than in the patients with IAA ($P < .001$). One patient had sarcoidosis, 1 patient had SLE, and 1 patient had giant cell arteritis.

Patients with nonruptured IAA (n = 27) more often had abdominal pain, compared with patients with nonruptured AAA (n = 341)(51.9% vs 30.2%; $P = .04$). In turn, this seemed to cause a slight tendency toward urgent admission to hospital in the IAA group (48% vs 34%), but this difference did not reach statistical significance ($P = .20$). There was no significant difference in reports of back pain or tenderness at clinical examination in the two groups.

In the IAA group, median aortic wall thickness was 11.2 mm (range, 4.2-25 mm). There were no significant difference in maximal aneurysm diameter in the two groups, with median diameter of 60 and 65 mm, respectively. In patients with IAA, the duodenum was involved in 19 patients (61%) and the left renal vein in 5 patients (16%), and in 9 patients (29%) the inflammatory reaction extended beyond the aortic bifurcation. Eight patients (26%) in the IAA group had hydronephrosis from involvement of the urether, which was bilateral in 2 patients. Erythrocyte sedimentation rate was significantly higher in patients with IAA compared with control subjects (28 vs 16 mm; $P < .01$). General symptoms such as malaise and weight loss were more often observed in patients with IAA than in the control group (Table III).

There were no differences between groups with regard to incidence of preoperative coronary heart disease, hypertension, diabetes, or chronic obstructive pulmonary disease. Thus, with the exception of local fibrotic changes, there were no differences in preoperative risk factors in the two groups. Median operating time was significantly longer in the IAA group compared with the control group (203 vs 160 minutes; $P = .003$; Table IV). There was also a higher need for blood transfusion in the IAA group (median, 6 vs 3 units of saline adenine glucose mannitol blood; $P = .009$). However, there was no statistically significant difference in median postoperative hospital stay or time in the intensive care unit (108 vs 92 hours; $P = .209$). Neither was the need for assisted ventilation different (median, 0 hours). Thirty-day mortality among patients with IAA was 25% in those with ruptured aneurysm and 7.4% in patients

Table IV. Hospital resources used after operation in 31 patients with IAA compared with 62 control subjects with noninflammatory AAA

	IAA		*Controls*		
	Median	*Range*	*Median*	*Range*	P
Operating time (min)	205	110-320	160	65-381	.003
Anesthesia time (min)	300	185-455	274	125-535	.04
Postoperative hospital stay (h)	216	48-720	216	0-4416	NS
Intensive care/intermediate care unit stay (h)	108	36-542	92	5-4416	NS
Postoperative artificial ventilation (h)	0	0-144	0	0-3648	NS
Blood transfusion (median units SAG)	6		3		.009

IAA, Inflammatory aortic aneurysm; *AAA,* abdominal aortic aneurysm; *SAG,* saline adenine glucose mannitol blood; *NS,* not significant.

with nonruptured aneurysm, and there was no difference compared with the control subjects.

DISCUSSION

Walker et al[1] found no evidence of a systemic collagen disorder in their patients with IAA. However, other authors have proposed an autoimmune predisposition in such patients.[6] Prevalence of aortitis has been decribed in 10% of patients with ankylosing spondylitis,[17] and aortitis has also been found in a proportion of patients with rheumatoid arthritis.[9] Further, accelerated atherosclerosis, including plaque formation in the carotid arteries, and higher incidence of death due to cardiac disease have also been observed in this group.[18-20] Although AAA has been described in patients with various autoimmune diseases,[21] to our knowledge this is the first report to show a higher incidence of autoimmune disease in patients with IAA compared with matched control subjects with noninflammatory AAA. Evidence of a genetic predisposition for development of IAA exists. Thus a genetic risk determinant mapped to the HLA-DR molecule, and in particular to the HLA-DRB1 locus and the alleles B1*15 and B1*0404, suggests a role for genetic risk factors in IAA.[6,11] Of interest, substitution of a glutamine for a negatively charged aspartic acid at the entrance to pocket 4 significantly changes the binding of the pocket and therefore changes antigen selection. This suggests a critical role for antigen binding in the origin of the disease, and its distinct location on the HLA-DR molecule suggests disease specificity, compared with giant cell arteritis. HLA typing was not possible in our patients, but is done in prospective studies. Various environmental factors, eg, viruses, may have a role in the triggering mechanisms for development of inflammatory rheumatic disease, connective tissue diseases, and primary vasculitis. Tests for microbial antigens such as herpes simplex virus and cytomegalovirus[22-24] have not been performed. However, detection of these viruses in serum and in biopsy specimens of arterial wall can be done in forthcoming studies. Microscopy of biopsy specimens may be important to reveal cellular composition and deposits that reflect both extent and different types of immunologic activity in the vascular wall.[24]

Inasmuch as the question of increased incidence of autoimmune disease in IAA first was discussed after the study had been completed, there was no chance that the surgeons were biased and looked for autoimmune disease more diligently in patients with IAA. Furthermore, our criteria for IAA were the same during the entire study. In a case-control study, matching of control subjects is important. We decided to match for rupture and urgency of admission when we identified the control group. Selecting control subjects from the group of patients operated on directly before or after the index patients was done to provide comparable data, which compensated for any possible temporal differences in treatment policy. Primarily, we approached the data via a nested case-control design. A limitation of that design is not that we selected two control subjects per index case, but that the number of patients with the index condition, IAA, was restricted. It is recommended that the number of control subjects be increased,[25] as in our study, but not beyond 4:1. We choose 2:1 as a compromise, ie, the last eligible patient before and the first patient after each index case. However, to test our hypothesis further, we compared the occurrence of autoimmune diseases in the IAA group with that in the entire noninflammatory AAA group. A strength of the study was complete follow-up of patients throughout the entire period before introduction of endovascular procedures to treat AAA at our hospital.

Our hospital serves a well-defined geographic area. Autoimmune disease would have been recorded if the patient had received any treatment at a hospital in the region, because all patient records were thoroughly reviewed and 100% follow-up was obtained. We therefore find it unlikely that any autoimmune disease in the control group was missed.

The technical challenges represented by the inflammatory changes are reflected in the longer operating time and greater need for blood transfusion. This may be an argument in favor of endovascular therapy for IAA.[26-28] Endovascular repair is feasible in these cases,[29] but both regression and proliferation of the inflammatory changes[30] have been reported. Therefore this treatment method remains controversial. Thirty-day mortality of 7.4% in the patients

with nonruptured IAA seems high, but includes two patients who underwent emergency surgery. One patient had acute ischemia of the left lower limb after arteriography. Closed compartment syndrome developed, and fasciotomy was required. Severe multiorgan failure developed, including renal failure, and the patient died on the tenth postoperative day. The other patient underwent emergency surgery because of pain, but there was no rupture. The immediate postoperative course was uneventful, but on the fifth postoperative day a large myocardial infarction occurred, which was verified at autopsy.

We investigated data for patients operated on from 1983 to 1994 because at that time all AAAs were treated with open surgery with a standardized technique that was the same for all operating surgeons. In February 1995 endovascular treatment was introduced in our hospital, and this technique has since been used in about 40% of cases. Thus patients who underwent open surgery since 1995 were selected.

Mitchinson[31] claimed that IAA, perianeurysmal fibrosis, and idiopathic retroperitoneal fibrosis are all a manifestation of the same process, and therefore proposed the common name periaortitis. This was later supported by Martina et al[32] in a series in which idiopathic retroperitoneal fibrosis seemed to be related to a high incidence of aortic atherosclerosis. Stella et al[33] also showed that degree of postoperative regression not unexpectedly is related to cell-fibrosis ratio, in which fibrosis preponderance is related to poor response with regard to regression of periaortic thickening after graft implantation. The results are not uniform, however,[34-38] and progression after open repair as well as endovascular repair have been reported.[31,35] Several reports of IAA and perianeurysmal fibrosis include a combination of both conditions,[1,37] and IAA, idiopathic retroperitoneal fibrosis, and perianeurysmal fibrosis are often clinically indistinguishable when complicated with uretheral involvement.

In conclusion, our study has demonstrated an association between IAA and autoimmune disease. This is in accordance with other reports that showed a genetic risk determinant mapped to the HLA molecule in these patients. The findings seem to contradict the theory that IAA is only an end stage of an inflammatory process present in all aortic aneurysms, and supports the theory that IAA is a separate entity. In this series there was longer operating time and higher need for blood transfusion after surgery to treat IAA, compared with operations to treat noninflammatory AAA. However, use of other hospital resources and early mortality were similar in both groups.

REFERENCES

1. Walker DI, Bloor K, Williams G, Gillie I. Inflammatory aneurysms of the abdominal aorta. Br J Surg 1972;59:609-14.
2. James TGI. Uremia due to aneurysm of the abdominal aorta. Br J Urol 1935;7:157-61.
3. DeWeerd JH, Ringer MG, Pool TL, Gambill EE. Aortic aneurysm causing bilateral ureteral obstrution: report of a case. J Urol 1955;74: 74-81.
4. Rijbroek FA, Moll FLV, Dijk HA, Meijer R, Janssen JW. Inflammation of the abdominal aortic aneurysm wall. Eur J Vasc Surg 1994;8:41-6.
5. Rose AG, Dent DM. Inflammatory variant of abdominal aortic aneurysm. Arch Pathol Lab Med 1981;105:409-13.
6. Rasmussen TE, Hallett JW, Metzger RLM, Richardson DM, Harmsen WS, Goronzy JJ, et al. Genetic risk factors in inflammatory abdominal aortic aneurysms: polymorphic residue 70 in the HLA-DR B1 gene as a key genetic element. J Vasc Surg 1997;25:356-64.
7. McMahon JN, Davies JD, Scott DJA, Tennant WG, Powell JE, Hughes AO, et al. The microscopic features of inflammatory abdominal aortic aneurysms: discriminant analysis. Histopathology 1990;16:557-64.
8. Parums DV, Dunn DC, Dixon AK, Mitchinson MJ. Characterization of inflammatory cells in a patient with chronic periaortitis. Am J Cardiovasc Pathol 1990;3:121-9.
9. Hackett E. Idiopathic retroperitoneal fibrosis: a condition involving the ureters, the aorta and the inferior vena cava. Br J Surg 1958;46:3.
10. Gravallese EM, Corson JM, Coblyn JS, Pinkus GS, Weinblatt ME. Rheumatoid aortitis: a rarely recognized but clinical significant entity. Medicine (Baltimore) 1989;68:95-106.
11. Ehrenfeld M, Bitzur R, Schneiderman J, Smolinsky A, Sidi Y, Gur H. Aortic aneurysm and dissection are not associated with an increased risk for giant cell arteritis/polymyalgia rheumatica. Postgrad Med J 2000; 76:409-11.
12. Rasmussen TE, Hallett JW, Schulte S, Harmsen WS, O'Fallon WM, Weyland CM. Genetic similarity in inflammatory and degenerative abdominal aortic aneurysms: a study of human lekocyte antigen class II disease risk genes. J Vasc Surg 2001;34:84-9.
13. Rasmussen TE, Hallett JW. Inflammatory aneurysms of the abdominal aorta: new perspectives in pathogenesis and management. Perspect Vasc Surg 1999;10:1-21.
14. Arnett FC, Edsworthy SM, Bloch DA, Mcshane DJ, Fries JF, Cooper NS. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum 1988;31: 315-24.
15. Tan EM, Cohen AS, Fries JF. The 1982 revised criteria for the classification of systemic lupus erythematosus. Arthritis Rheum 1982;25: 1271.
16. Hunder GG, Bloch DA, Michel BA, Stevens MB, Arend WP, Calabrese LH, et al. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. Arthritis Rheum 1990;33:1122-8.
17. Ball J. Symposium on the spondylarthritides: aspects of pathology. Rheumatol Rehabil 1979;18:210-3.
18. Van Boornum S, McColl G, Wicks IP. Accelerated atherosclerosis: an extraarticular feature of rheumatoid arthritis? Arthritis Rheum 2002;46: 862-73.
19. Kumeda Y, Inaba M, Goto H, Nagata M, Henmi Y, Furumitsu Y, et al. Increased thickness of the arterial intima-media detected by ultrasonography in patients with rheumatoid arthritis. Arthritis Rheum 2002;46: 1489-97.
20. Goodson N. Coronary artery disease and rheumatoid arthritis. Curr Opin Rheumatol 2002;14:115-20.
21. Ohara N, Miyata T, Sato O, Oshiro H, Shigematsu H. Aortic aneurysm in patients with autoimmune diseases treated with corticosteroids. Int Angiol 2000;19:270-5.
22. Tanaka S, Toh Y, Mori R, Komori K, Okadome K, Sugimachi K. Possible role of cytomegalovirus in the pathogenesis of inflammatory aortic diseases: a preliminary report. J Vasc Surg 1992;16:274-9.
23. Tanaka S, Komori K, Okadome K, Sugimachi K, Mori R. Detection of active cytomegalovirus infection in inflammatory aortic aneurysms with RNA polymerase chain reaction. J Vasc Surg 1994;20:235-43.
24. Jennette JC, Falk RJ. Small-vessel vaculitis. N Engl J Med 1997;337: 1512-23.
25. Hennekens CH, Buring JE. Epidemiology in medicine. Boston, Mass: Little Brown & Co, 1987.
26. Lacquet JP, Lacroix H, Nevelsteen A, Suy R. Inflammatory abdominal aortic aneurysms: a retrospective study of 110 cases. Acta Chir Belg 1997;97:286-92.
27. Deleersnijder R, Daenens K, Foruneau I, Maleux G, Nevelsteen A. Endovascular repair of inflammatory abdominal aortic aneurysms with

special reference to concomitant ureteric obstruction. Eur J Vasc Endovasc Surg 2002;24:146-9.
28. Lindblad B, Almgren B, Bergqvist D, Eriksson I, Forsberg O, Glimåker H, et al. Abdominal aortic aneurysm with perianeurysmal fibrosis: experience from 11 Swedish vascular centers. J Vasc Surg 1991;13: 231-7.
29. Hinchliffe RJ, Macierewicz JA, Hopkinson BR. Endovascular repair of inflammatory abdominal aortic aneurysms. J Endovasc Ther 2002;9: 277-81.
30. Vallabhaneni SR, McWilliams RG, Anbarasu A, Rowlands PC, Brennan JA, Gould DA, et al. Perianeurysmal fibrosis: a relative contra-indication to endovascular repair. Eur J Vasc Endovasc Surg 2001;22:535-41.
31. Mitchinson MJ. Chronic periaortitis and periarteritis. Histopathology 1984;8:589-600.
32. Martina FB, Nuech R, Gasser TC. Retroperitoneal fibrosis and chronic periaortitis: a new hypothesis. Eur Urol 1993;23:371-4.
33. Stella A, Gargiulo M, Faggioli GL, Bertoni F, Cappello I, Brusori S, et al. Postoperative course of inflammatory abdominal aortic aneurysms. Ann Vasc Surg 1993;7:229-38.
34. Baskerville PA, Blakeney CG, Young AE, Browse NL. The diagnosis and treatment of peri-aortic fibrosis (inflammatory aneurysms). Br J Surg 1983;70:381-5.
35. Bitisch M, Nørgaard HH, Røder O, Schroeder TV, Lorentzen JE. Inflammatory aortic aneurysms: regression of fibrosis after aneurysm surgery. Eur J Vasc Endovasc Surg 1997;13:371-4.
36. Di Marzo L, Sapienza P, Bernucci P, Gallo P, Tedesco M, Cavarallo A. Inflammatory aneurysm of the abdominal aorta: a prospective clinical study. J Cardiovasc Surg (Torino) 1999;40:407-12.
37. Crawford JL, Stowe CL, Safi HJ, Hallman CH, Crawford ES. Inflammatory aneurysms of the aorta. J Vasc Surg 1985;2: 113-24.
38. Nitecki SS, Hallett JW, Stanson AW, Ilstrup DM, Bower TC, Cherry KJ Jr, et al. Inflammatory abdominal aortic aneurysms: a case-control study. J Vasc Surg 1996;23:860-9.

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CORRECTIONS

In: "Pixel distribution analysis of B-mode ultrasound scan images predicts histologic features of atherosclerotic carotid plaques" (Lal BK, Hobson RW II, Pappas PJ, Kubicka R, Hameed M, Chakhtura EY, et al. J Vasc Surg 2002; 35:1210-7).

The name *Ellie Y. Chakhtura* is spelled incorrectly. The correct spelling is *Elie Y. Chakhtoura*.

In: "Carotid artery stenting: analysis of data for 105 patients at high risk" (Hobson RW II, Lal BK, Chaktoura E, Goldstein J, Haser PB, Kubicka R, et al. J Vasc Surg 2003;37:1234-9).

The name *Ellie Y. Chaktoura* is spelled incorrectly. The correct spelling is *Elie Y. Chakhtoura*.

In: "Endothelial cell seeding fails to attenuate intimal thickening in balloon-injured rabbit arteries" (Conte MS, Choudry RP, Shirakowa M, Fallon JT, Birinyi LK. J Vasc Surg 1995;21:413-21).

The name *Choudry* is spelled incorrectly. The correct spelling is *Choudury*.