Inflammatory Abdominal Aortic Aneurysm

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There is no disease more conducive to clinical humility than aneurysm of the aorta.
William Osler

Abdominal aortic aneurysm (AAA), the most common type of aneurysm, develops in 4% to 10% of people older than 60 years.1-12 Physicians are increasingly likely to encounter patients with AAA because the number of seniors is increasing rapidly and because recently published guidelines call for performing ultrasound examination to screen for AAA in men aged 65 to 75 years who have ever smoked.13

Abdominal aortic aneurysm can have grave consequences for patients: currently, the disorder accounts for 15,000 deaths per year.14 Although common forms of AAA are frequently discussed in the general medical literature, an unusual but important variant of AAA, inflammatory AAA, has received little attention. The symptoms and signs of inflammatory AAA are so protean that patients may present to a wide range of different physicians, including general internists, family physicians, nephrologists, urologists, radiologists, rheumatologists, emergency medicine physicians, gastroenterologists, and general and vascular surgeons.

See also Patient Page.
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Inflammatory abdominal aortic aneurysm (AAA) accounts for 5% to 10% of all cases of AAA and differs from typical atherosclerotic AAA in many important ways. Although both inflammatory and atherosclerotic AAA most commonly affect the infrarenal portion of the abdominal aorta, patients with the inflammatory variant are younger and usually symptomatic, chiefly from back or abdominal pain. Unlike patients with atherosclerotic AAA, most with the inflammatory variant have an elevated erythrocyte sedimentation rate or abnormalities of other serum inflammatory markers. Computed tomography and magnetic resonance imaging are both sensitive for demonstrating the cuff of soft tissue inflammation surrounding the aneurysm that is characteristic of inflammatory AAA. In contrast to atherosclerotic AAA, the inflammatory variant is characterized pathologically by marked thickening of the aneurysm wall, fibrosis of the adjacent retroperitoneum, and rigid adherence of the adjacent structures to the anterior aneurysm wall. An extraordinary expansion of the adventitia due to inflammation also distinguishes inflammatory from atherosclerotic AAA. Although the pathogenesis of inflammatory AAA appears to involve an immune response localized to the vessel wall, the etiology of the inflammatory reaction is unknown. Inflammatory AAA is almost never associated with inflammation of other arteries. Male sex and smoking, the main risk factors for atherosclerotic AAA, are even stronger risk factors for the inflammatory variant. Smoking cessation is the first step of medical therapy. Corticosteroids or immunosuppressive therapies may also have roles. Although inflammatory AAA appears less likely to rupture than atherosclerotic AAA, surgical intervention appears prudent once the diameter of the aneurysm exceeds 5.5 cm. Knowing the features of inflammatory AAA should allow physicians to distinguish it from atherosclerotic AAA or from systemic vasculitis and to treat it with the appropriate combination of medical and surgical therapies.

JAMA. 2007;297:395-400
www.jama.com

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(Reprinted) JAMA, January 24/31, 2007—Vol 297, No. 4 395

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PATIENT PRESENTATION

**Dr Hellmann:** Mr A is a 42-year-old man who has smoked 20 to 40 cigarettes daily since his teens and has been treated for familial hypercholesterolemia (for more than a decade) and for mild hypertension (for 1 year). He felt perfectly well until about 2 years ago. Mr A, please describe what you noticed first.

**Mr A:** Gradually I developed mild back pain located on both my sides. It was nothing severe. When I was growing up my mother didn’t take me to a doctor unless I had a bone or an organ showing. So I don’t go to doctors on a regular basis and didn’t see anyone about this pain; I just rode it out and after a few weeks it went away.

**Dr Hellmann:** But the pain in your flanks came back?

**Mr A:** That’s right. And this time I noticed not only the side pain but also difficulty and burning when I would begin to urinate. I saw a urologist who thought my prostate was enlarged. He gave me Flomax [tamsulosin], and my pain and difficulty urinating again went away.

**Dr Hellmann:** Please describe the third episode of back pain.

**Mr A:** That was a killer. It hit suddenly, hurt only my low back over my kidneys, and was so severe that I couldn’t go to work for a few days.

**Dr Hellmann:** Did you have fever, chills, or blood in the urine?

**Mr A:** No.

**Dr Hellmann:** Did position affect the pain, did you have pain shooting down your leg, or have any other symptoms?

**Mr A:** No.

**Dr Hellmann:** Please tell us what happened next.

**Mr A:** After a few weeks the pain resolved. When the side pain came back a fourth time several weeks later, I finally decided something must be wrong and went to see my regular doctor.

**Dr Hellmann:** Although you live out of state, I have the records from your doctor indicating that your physical examination was normal except for the old xanthomas over your elbows and achilles tendons. Your complete blood count, comprehensive metabolic panel, and urinalysis were normal. And your erythrocyte sedimentation rate [ESR] was 16. Because your doctor was concerned about the recurrent episodes of flank pain, he requested a CT [computed tomography] scan of your abdomen, which will be reviewed by Dr David Grand.

**Dr Grand:** These (FIGURE 1) are 2 reformatted coronal images from a contrast-enhanced CT scan. Both images demonstrate an infrarenal abdominal aortic aneurysm with extensive soft tissue surrounding the aorta and with inflammation extending into the peri-aortic fat. Accurate differentiation of inflammatory AAA from the more common atherosclerotic AAA depends on demonstration of perianeurysmal fat stranding indicative of inflammation rather than simply the enlarged aortic caliber seen in atherosclerotic AAA. This can be demonstrated by either CT or MRI [magnetic resonance imaging]. When these changes are present, it is critical to exclude the possibility of aortic leak or rupture. First, one must be certain that, as in this case, there is no evidence of blood within the retroperitoneum and pelvis. Second, enhancement of the perianeurysmal abnormal soft tissue, as seen in these images, indicates inflammation as opposed to a leak.

**Dr Hellmann:** Because of this intense inflammation around the aneurysm, you were initially diagnosed as having aortitis secondary to vasculitis, referred to a rheumatologist, and started on prednisone, approximately 40 mg daily. Did that medication help?

**Mr A:** Immediately I felt nearly complete relief. But as the prednisone dose was lowered over the next few weeks, the pain over my low back returned. When the prednisone was increased back above about 20 mg a day, my pain went away.

**Dr Hellmann:** A CT scan repeated about 2 months after the initiation of prednisone was reported to show a marked decrease in the retroperitoneal inflammation. Your rheumatologist continued the prednisone 20 mg per day and then started you on weekly oral doses of methotrexate (15 mg per week) and referred you to the Johns Hopkins Vasculitis Center. At presentation here, your vital signs were normal. Blood pressures in both arms were equal. Your carotid artery pulses and all of your extremity pulses were normal.
There were no bruits in the neck, chest, axilla, or abdomen. You had no flank tenderness or abdominal mass. Your ESR was 3. An MRI was done and will be reviewed by Dr Grand.

DR GRAND: The MRI in this case (FIGURE 2) again demonstrates fusiform dilatation of the infrarenal abdominal aorta that measures up to $3.1 \times 4.1$ cm. Additionally, the anterior wall of the infrarenal abdominal aorta was thickened up to 8 mm. The renal arteries, superior mesenteric artery, and celiac arteries are normal.

**DISCUSSION**

The case of Mr A is instructive because it illustrates the presenting features and management issues of inflammatory AAA, an important variant of AAA that is uniquely characterized by the presence of extensive periaortic inflammation.

**Terminology and History**

An aneurysm is a focal dilatation of a blood vessel. Normally, the aorta diminishes in size as it extends from the diaphragm to the bifurcation of the iliac vessels. Abdominal aortic aneurysm is usually defined as an aortic dimension greater than 1.5 times the normal diameter measured at the level of the renal arteries. While the normal size of the abdominal aorta varies with age, sex, and body size, the abdominal aortic diameter averages roughly 2 cm at the renal level. Therefore, an aortic diameter greater than 3 cm is usually considered an aneurysm. The abdominal aorta is the most common location of aneurysm. Abdominal aortic aneurysm occurs in 4% to 10% of people older than 60 years but is 30% to 400% more common in men than in women. Smoking is the single greatest risk factor for developing AAA, so among smokers the sex ratio may become more equal as the number of women approaches that of men. The risk of rupture is proportional to the diameter of the aneurysm, and the risk is especially high when the diameter exceeds 5.5 cm. Approximately 65% to 85% of patients whose aneurysm ruptures die suddenly. In the United States, AAA is 1 of the 15 leading causes of death among seniors.

Although AAA has been recognized for centuries, the unique variant Mr A had—inflammatory AAA—was not described until 1972, when Walker et al reported that 10% of 187 patients undergoing resection of AAs at the University of Manchester demonstrated the 3 key distinguishing features of the inflammatory variant: (1) marked thickening of the aneurysm wall, (2) fibrosis of the adjacent retroperitoneum, and (3) rigid adherence of the adjacent structures to the anterior aneurysm wall.

Since this initial description 35 years ago, it has become clear that inflammatory AAA is one member of a family of disorders often referred to as “chronic periaortitis.” This family includes 3 members: (1) inflammatory AAA, (2) idiopathic retroperitoneal fibrosis, and (3) a combination of the 2 disorders. In inflammatory AAA, the inflammation and fibrosis surround the aorta, usually without obstructing the bowel or ureters. In idiopathic retroperitoneal fibrosis, inflammatory fibrosis of the retroperitoneum occurs in the absence of aortic aneurysm. Since nature does not always observe these artificial boundaries, patients may present with variations within this spectrum of inflammation and fibrosis. Indeed, while Mr A most strikingly had inflammatory AAA, he did, at onset, have CT evidence of retroperitoneal inflammation. Our discussion will focus on inflammatory AAA and not on the other manifestations of chronic periaortitis.

**Epidemiology**

Although few physicians are familiar with inflammatory AAA, it is not uncommon. Most large series indicate that the inflammatory variant is seen in 5% to 10% of all cases of AAA. The vast majority of patients with inflammatory AAA are men (TABLE). Smoking, present in at least two thirds of those with atherosclerotic AAA, is virtually universal in those with the inflammatory variant. Most series on inflammatory AAA have not examined medical comorbid conditions in detail, but the variant appears to be associated with other risk factors for atherosclerosis. Indeed, Mr A had had hypertension and hypercholesterolemia.

![Image](3 cm)

*Image obtained after anti-inflammatory treatment demonstrates a fusiform aortic aneurysm without evidence of significant wall thickening or periaortic inflammation.*

<table>
<thead>
<tr>
<th>Feature</th>
<th>Inflammatory Variant</th>
<th>Atherosclerotic Variant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>~5</td>
<td>~95</td>
</tr>
<tr>
<td>Sex ratio, men:women</td>
<td>6:1 to 30:1</td>
<td>5:1</td>
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<tr>
<td>Age at diagnosis, y</td>
<td>66</td>
<td>71</td>
</tr>
<tr>
<td>Smoking</td>
<td>80-100</td>
<td>75</td>
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<tr>
<td>Family history of aortic aneurysm</td>
<td>15</td>
<td>1.5</td>
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<td>Symptoms from aneurysm</td>
<td>84</td>
<td>9</td>
</tr>
<tr>
<td>Risk of aneurysm rupture</td>
<td>4</td>
<td>20</td>
</tr>
</tbody>
</table>

*Data from Pennell et al, Linblad et al, and Nitecki et al.*
The original observation by Walker et al that patients with inflammatory AAA were 5 to 10 years younger than those with atherosclerotic AAA has been verified by subsequent series.3,7 Family history also appears to be a more powerful factor in inflammatory than in atherosclerotic AAA.7,11 It is, therefore, relevant that Mr A recalled that his grandfather had experienced some form of AAA.

**Surgical Findings and Pathology**

Walker et al described the operative findings of inflammatory AAA this way:

The inflammatory aneurysm has a thick, firm, smooth wall which is shiny white in appearance although there is an obvious increase in the vascularity of the wall with multiple small vessels traversing it. The adjacent dense fibrosis is marked and may involve adjoining tissues and structures.2

Subsequent studies have confirmed that the triad of gross pathologic findings in inflammatory AAA is (1) a thickened aneurysm wall, (2) intense perianeurysmal and retroperitoneal fibrosis, and (3) extensive adhesions involving surrounding organs.9,15 The thickening of the aortic wall ranges from 0.5 to 3.0 cm.6 In most cases, the perianeurysmal fibrosis occurs most prominently around the ventral and lateral walls of the aneurysm; for reasons that are not known, the posterior wall of the aorta is usually spared in inflammatory AAA.8

Histopathological examination shows that most of the thickening of the aortic wall results from expansion of the adventitia by a marked inflammatory reaction.2 The intima in inflammatory AAA reveals atherosclerosis, much as is seen in atherosclerotic AAA. The media shows atrophy and loss of elastic tissue.18 The striking inflammatory reaction in the adventitia consists of plasma cells, lymphocytes (mostly B cells and smaller numbers of CD4+ T cells), and macrophages.7,18,19 Occasional giant cells and small granulomas are sometimes seen.18 Infrequently, arteritis of the vasa vasorum is evident. Many of the lymphocytes are activated, and germinal centers are common.20 Eosinophils are present infrequently and neutrophils rarely.5 Immunohistochemical studies of the adventitia also demonstrate up-regulation of a wide range of inflammatory cytokines, including interleukin 2, interleukin 4, interleukin 1α, and adhesion molecules.7,19 Thus, the extraordinary expansion of the adventitia due to inflammation is the major feature that distinguishes inflammatory from atherosclerotic AAA.

**Etiology and Pathogenesis**

Although the etiology of inflammatory AAA is unknown, the immunohistopathological findings suggest that this variant results from an immune response to an antigen localized to the adventitia.7,9 The precise antigen target has not been defined. One hypothesis is that the inciting antigen is a lipid or a product of lipid oxidation found in an atherosclerotic aorta.7,17,21 The finding that the distribution of class II human leukocyte antigens is the same in patients with atherosclerotic and inflammatory AAA but different from that of controls suggests that genes that regulate antigen presentation influence aneurysm formation.22

In addition, inflammatory cells, which are absent in the normal aorta, are present to varying degrees in all patients with AAA, reinforcing the notion that deposition of lipid in blood vessels frequently provokes some level of inflammatory response. Indeed, the current concept of the pathogenesis of coronary artery disease emphasizes the central role of vascular inflammation triggered by lipids. Since an intact media constitutes an immunoprivileged site, the ability of lipids deposited in the media to elicit an inflammatory response may depend on thinning or disruption of the media caused by proteases, which in turn may be uniquely dysregulated or dysfunctional in patients who develop inflammatory AAA.7,14 Reports of retroperitoneal fibrosis resolving in some cases after surgery for inflammatory AAA have underscored the possibility that the inflammatory variant may result from an unusually severe local inflammatory reaction to atherosclerotic lipids deposited in the aorta.6 Although inconclusive, these pieces of evidence indicate the possibility that an inflammatory response to vascular lipids plays a role in the pathogenesis of inflammatory AAA.

There is some, but less compelling, evidence that inflammatory AAA is a systemic autoimmune disease.7,9,19,23 Unlike Mr A, many patients with the inflammatory variant have systemic symptoms. In addition, many patients with inflammatory AAA have elevated levels of systemic inflammatory markers such as the ESR or C-reactive protein. Some studies have reported a high frequency of certain autoantibodies in patients with inflammatory AAA, including antinuclear antibodies and antineutrophil cytoplasmic antibodies.7,23 There have been several reports of other autoimmune diseases accompanying the inflammatory variant, including Wegener granulomatosis, Henoch-Schönlein purpura, polyarteritis nodosa, and autoimmune thyroid disease.19 One study of 31 patients with inflammatory AAA found that 19% had an associated autoimmune disease, compared with none of the control patients.23

Some investigators have theorized that inflammatory AAA is caused by an infectious disease. Most of the speculation has centered on cytomegalovirus and Chlamydia pneumoniae.24,25

**Presenting Symptoms, Signs, and Laboratory Abnormalities**

Many studies have confirmed the impression of Walker et al that, in contrast to patients with atherosclerotic AAA, the majority of patients with inflammatory AAA are asymptomatic at presentation. Pain—usually either back pain, as in Mr A, or abdominal pain—develops in 80% of patients.7 In contrast, only 8% to 18% of patients with atherosclerotic AAA have symptoms.9 As was the case with Mr A, the back pain begins insidiously and chiefly affects the lower lumbar area but may extend to or primarily involve the abdomen. Constitutional symptoms, such as fever, malaise, and weight loss, although absent in Mr A, are
reported in approximately 20% to 50% of patients.\textsuperscript{2,7,9} When inflammatory AAA is accompanied by extensive retroperitoneal fibrosis, then symptoms and signs of duodenal obstruction, ureteral colic, or inferior vena caval obstruction may develop. Fortunately, the lifetime risk of rupture posed by the inflammatory variant is less than 5%,\textsuperscript{8} so very few patients present with acute abdominal pain and circulatory collapse.

A minority of patients with inflammatory AAA have related physical findings. Fever has been reported exceptionally.\textsuperscript{9} Abdominal tenderness with or without a pulsatile abdominal mass is the most common finding but is present in only approximately one third of patients.\textsuperscript{2,5,10} Thus, it is not surprising that findings from Mr A’s abdominal examination were normal.

Laboratory abnormalities reflecting systemic inflammation are common. All of the original patients in the study by Walker et al, for example, had elevated ESRs, vs only 29% of those with atherosclerotic AAA.\textsuperscript{9} In subsequent studies, the percentages of patients with an elevated ESR have ranged from 40% to 90%.\textsuperscript{5} The elevation is often modest, with most values in the 20- to 50-mm/h range.\textsuperscript{2} Levels of C-reactive protein are also frequently elevated. Anemia is less common, and leukocytosis is rare.\textsuperscript{9}

**Imaging**

Routine abdominal radiographs are usually unremarkable except for showing extensive aortic calcification seen in 75% of patients,\textsuperscript{3} which does not distinguish inflammatory from atherosclerotic AAA. Computed tomography or MRI are the best imaging modalities for diagnosis and follow-up. As with Mr A (Figure 1), CT scanning displays well the aneurysm and the thickened aortic wall, as well as the surrounding peri-aortic inflammation and fibrosis. The peri-aortic inflammation of inflammatory AAA is isodense to muscle but enhances with administration of intravenous contrast material.\textsuperscript{3} As was seen with Mr A, corticosteroid therapy can reduce that degree of peri-aortic enhancement. The cuff of soft tissue surrounding the aorta in inflammatory AAA can usually be distinguished from tumor or lymph nodes. Bladder cancer and liposarcoma are 2 cancers that are capable of provoking an intense periaortic reaction. A useful finding in this regard is that the periaortic inflammation of inflammatory AAA, in contrast to tumor, rarely displaces the aorta from the vertebrae.\textsuperscript{3} In the inflammatory variant, the aortic wall thickening is limited to the anterior and lateral aspect; the posterior aortic wall is spared.\textsuperscript{6} Computed tomography may also show extensive calcification of the media. One study of 355 patients with AAA estimated that CT scanning was 83.3% sensitive and 99.7% specific in diagnosing the inflammatory variant.\textsuperscript{26}

Magnetic resonance imaging is also very sensitive in demonstrating the aneurysm, the thickening of the wall (even better than CT), and the surrounding periaortic inflammation. Magnetic resonance imaging also avoids the radiation exposure and administration of nephrotoxic intravenous contrast material required with CT.\textsuperscript{19} The periaortic mass in inflammatory AAA is hypointense on T1-weighted images, but hyperintense on T2-weighted images.\textsuperscript{19} The periaortic cuff of inflammation enhances strikingly with administration of intravenous gadolinium.\textsuperscript{7} Some physicians, noting that MRI provides less information about the ureters, prefer CT imaging and reserve MRI for patients who have renal insufficiency.\textsuperscript{7}

Ultrasoundography has the well-known advantages of being relatively inexpensive and of not exposing the patient to ionizing radiation. However, it is inferior to CT and MRI in demonstrating the aortic wall thickening or the peri-aortic changes of inflammatory AAA.\textsuperscript{7}

Recent studies show that positron emission tomography may turn out to be the most sensitive means of assessing the metabolic activity of periaortic inflammation.\textsuperscript{27,28}

**Differential Diagnosis**

There is no other condition apart from inflammatory AAA that produces an abdominal aneurysm with intense periaortic and retroperitoneal fibrosis and inflammation. The initial question in this case was whether Mr A had systemic vasculitis. Vasculitis of the aorta occurs in Takayasu aortitis, but that disease chiefly strikes young women and most commonly affects the thoracic aorta and its branches. Temporal arteritis can affect large arteries including the aorta, but patients with that disease are almost always older than 50 years and have headache, polymyalgia, jaw claudication, and visual symptoms.\textsuperscript{20}

Cogan syndrome is a rare cause of vasculitis affecting the aorta that causes visual and vestibulolauditory symptoms. Inflammation of the aorta, almost exclusively the thoracic aorta, occurs rarely with other diseases, such as ankylosing spondylitis and rheumatoid arthritis. Other nonrheumatic diseases such as Marfan syndrome, Ehlers-Danlos syndrome, cystic medial necrosis, and infections (salmonella, syphilis) can cause aortic aneurysms but without periaortitis.

**Management**

Medical and surgical approaches both have a role in managing patients with inflammatory AAA.\textsuperscript{5,7,30,31}

**Medical Treatment.** The possibility that inflammatory AAA results primarily from inflammation has prompted efforts to use corticosteroids or other anti-inflammatory or immunosuppressive therapies. Improvement of symptoms, signs, and CT or MRI evidence of inflammation has been described. Indeed, with prednisone treatment, Mr A’s back pain promptly resolved and CT evidence of the retroperitoneal inflammation decreased. Corticosteroid-sparing agents, such as methotrexate, cyclophosphamide, and azathioprine, have also been reported effective.\textsuperscript{3} However, the ultimate efficacy of the anti-inflammatory approach has not been proven.

Given that the preponderance of evidence suggests that inflammatory AAA results from a local inflammatory reaction to atherosclerotic lipids, great attention should be placed on minimizing atherosclerotic risk factors. In
Mr A—and, as is illustrated by the review of epidemiology, for most patients—smoking cessation is critically important. For Mr A, control of his diabetes (a late complication of his prednisone treatment), hypertension, and hyperlipidemia is also important.

**Surgical Management.** The aim of surgical treatment is to prevent rupture. Although data suggest that an inflammatory AAA is less liable to rupture than an atherosclerotic AAA,8 surgical intervention appears prudent once the diameter exceeds 5.5 cm.14 The surgical management of inflammatory AAA presents unique challenges that have been overcome by modifying the approach that was first tried by Walker et al 34 years ago.2 A hallmark of inflammatory AAA is the dense adhesions that surround the aneurysm and frequently involve the duodenum (97%-70%), the ureters (20%-44%), and left renal vein (48%-51%).9 Early surgical experience showed that adhesiolysis and mobilization of periaortic structures frequently caused complications, including enterotomies, especially of the duodenum; injuries to ureters and vena cava; and increased mortality.14 Minimizing dissection of these tissues has greatly reduced the likelihood of complications. Most investigators now favor a transperitoneal approach to inflammatory AAA.32 Infra renal aortic clamping is usually performed and every effort is made to avoid supraceliac clamping.32 Bifurcated or straight grafts are used equally frequently.32 Aortic fistula is more commonly found in rupture of inflammatory AAA than in rupture of atherosclerotic AAA.32 Despite the many technical difficulties, surgical results have greatly improved over the last 35 years. Postoperative mortality has decreased from 12.5% in the 1950s to approximately 5% at the turn of this century.7 Still, the postoperative mortality for inflammatory AAA probably remains slightly higher than that for atherosclerotic AAA.2

The degree to which inflammatory AAA affects retroperitoneal fibrosis is controversial. Despite early reports that repair of inflammatory aneurysms resulted in dramatic reduction in retroperitoneal fibrosis, more recent reports indicate that repair causes complete regression in only 23% to 53% of patients.7 Resolution of the periaortic fibrosis may occur less often after repair of endovascular aneurysm.7 Still, when the anatomical features are appropriate, endovascular repair appears to work as well for inflammatory as for atherosclerotic AAA.7

**REFERENCES**


FINANCIAL DISCLOSURES: None reported.

**FOUR-MONTH FOLLOW-UP**

Mr A happily reports that he has celebrated his 4-month anniversary of not smoking and that being in a room with 200 physicians and watching a surgeon point to images of his aneurysm at medical grand rounds provided a fresh and powerful incentive to stop smoking. His back pain has resolved. He has tapered off the prednisone and still takes weekly oral methotrexate.