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Short Communications

Retroperitoneal Fibrosis and Infection of an Aortic Graft Prosthesis: Diagnosis and Therapeutic Problems

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Summary: The occurrence of a retroperitoneal fibrosis (RPF) after an aorto-bifemoral bypass is unusual. A case of RPF as a complication of an infection of the graft prosthesis is reported. Computed tomography and magnetic resonance imaging prove useful in diagnosing graft infection: they may reveal periprosthetic gas or perigraft fluid persisting for several months after surgery. However, they may fail when they are performed too early, and repeat performance is suggested.

Key words: retroperitoneal fibrosis, aortic graft prosthesis, computed tomography, magnetic resonance imaging

Introduction

Retroperitoneal fibrosis (RPF) is a rare, usually idiopathic disease in which an etiological factor can sometimes be found. This paper reports a case of RPF that occurred in the vicinity of an infected aortic graft prosthesis and raised difficult diagnostic and therapeutic problems.

Case Report

A 72-year-old man, suffering from hypertension and coronary heart disease, was referred for a fever of several weeks' duration, and for an inflammatory syndrome. Five months earlier, the patient had undergone an aorto-iliac bypass for an aneurysm of the subrenal abdominal aorta. There was no evidence of RPF at the time of surgery. At referral, abdominal ultrasonography showed a bilateral ureterohydronephrosis, and the intravenous pyelogram showed an antero-medium attraction of the ureters. Retroperitoneal fibrosis was suspected and then confirmed by computed tomography (CT) (Fig. 1). Endoscopic ureteral catheterization restored normal renal function and reduced the ureterohydronephrosis. However, the patient remained cachectic and with a slight fever. Biology showed a sedimentation rate (RCSR) of 100 after 1 h, C reactive protein of 100 mg/l (n<5 mg/l), white cell count of 9000 cells per mm^3 with 80% neutrophils, creatinine level of 13 g/l (n<12), and urea of 0.38 g/l (n<0.4 g/l). Blood cultures were sterile.

The poor general condition of the patient and the presence of RPF led us to start a regimen of steroids, using Prednisolon® at a dose of 1 mg/kg/day (60 mg/day). The patient's condition improved rapidly and the inflammatory syndrome partly receded. The RPF seemed to improve and, a few weeks later, the ureteral catheters were removed. However, a reduction by 20 mg in the dose of steroids was followed by a recurrence of the inflammatory syndrome. To reduce the daily intake of Prednisolon, a bolus treatment was started at a dose of 15 mg/kg Prednisolon on three consecutive days. Twenty-four h after the first bolus dose, the patient presented with acute pain in the right low abdominal quadrant. On CT, there were bilateral abscesses in the psoas muscles which were surgically drained. Samples taken during surgery proved positive for *Staphylococcus aureus*. The steroid administration was reduced to 20 mg/day and a double antibiotics course was started with Pristinamycin® 50 mg/kg/day and Rifampicin® 30 mg/kg/day. These abscesses led us to look again for a neighboring infection. On a new CT scan, gas could be seen between the graft and the remaining aneurysmal layer (Fig. 2). Magnetic resonance imaging (MRI) (Fig. 3) with gadolinium infusion showed a contrast intake in the remaining aneurysmal layer, which spread as a continuum to the left psoas muscles, where an abscess could be seen. These findings suggested a perigraft infectious phenomenon. Thus, the patient first underwent an axillo-bifemoral bypass, then removal of the graft prosthesis. Postoperative findings confirmed the diagnosis of a graft prosthesis infection. Subsequently, the patient remained apyretic and the inflammatory syndrome receded, with a Prednisolon dose of 15 mg/day.

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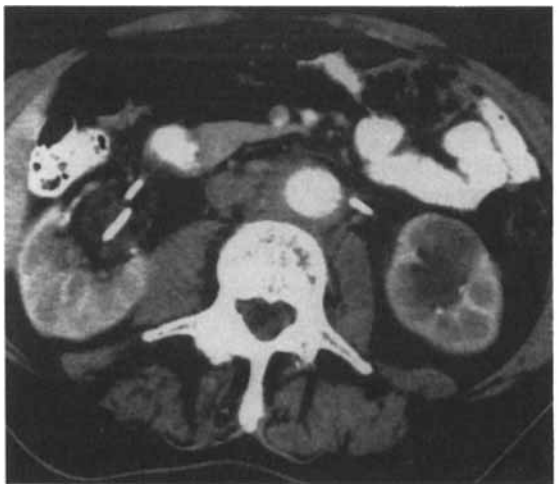

FIG. 1 Computed tomography scan. A soft tissue mass is present around aortic prosthesis.

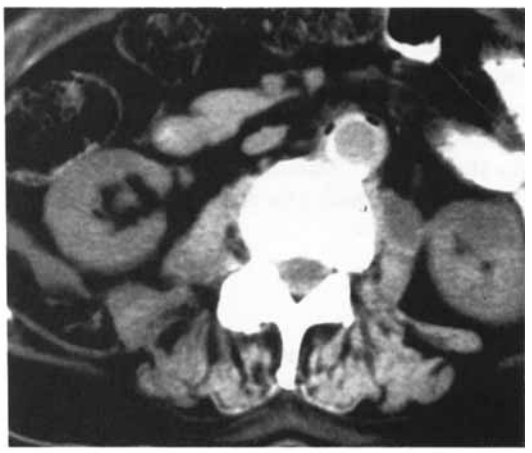

FIG. 2 Computed tomography scan. Gas is seen between the graft and the remaining aneurysmal layer.

Discussion

Macroscopic RPF is a possible complication in 5 to 23% of abdominal aortic aneurysms.[1] A review of postoperative pathologic examinations showed that all aneurysms can be responsible for some level of fibrous reaction. In abdominal aneurysms, some periadventitial inflammation could be present; it was more severe when the atheromatous plaques had broken and dilacerated the media layer. Retroperitoneal fibrosis would be the most severe form of this perivascular inflammation.[2] The hypothesized pathogenic mechanism would be an immunoallergic response to certain components of the atheromatous plaque, which would constitute a true vasculitis. Insoluble lipids (ceroids) would penetrate the periaortic tissues in the vicinity of the media layer which had been damaged by atheromatous plaque. Presence in the serum of anticeroid antibodies would constitute a good argument for an immunoallergic origin of RPF.[2] Some authors would suggest that there should be systematic investigations of aortic extasia in case of RPF.[2]

Fibrous reactions can also be observed after any type of aortofemoral graft surgery. It is difficult to estimate their frequency as they are often clinically silent. In a prospective study of 20 patients, Heard described four cases with partial ureterohydronephrosis in the early postoperative phase, which receded in most cases without any treatment within 1 year.[3] There are nevertheless true cases of RPF as a complication of vascular surgery. These cases are rare, as only 20 have been reported so far.[1,4,5] They should be distinguished from ureterohydronephrosis, which occurs because of a mechanical compression of the ureter that lies behind the graft prosthesis.[4] Postsurgical cases of RPF can benefit from a general course of steroid administration or from surgery.[2] Among these published cases, only two well-documented case reports[1,5] mention an inflammatory syndrome. These patients suffered from periarteritis nodosa, which, in one case, was known before surgery; in the other, it was diagnosed 9 months after surgery through the occurrence of a neurological condition, Raynaud disease, and an inflammatory syndrome.[1,5] Our observation showed that the inflammatory syndrome was related to an infection of the graft prosthesis. This is the first report of RPF related to an infection of a graft prosthesis. In such a context, diagnosis of an infection is difficult, and CT and especially MRI prove beneficial in the investigation of a delayed graft infection. One of the most reliable signs on CT scanning to indicate infection is the demonstration of perigraft gas, which in normal circumstances has been shown to persist for only a few weeks after surgery. The diagnosis of graft infection by MRI in the postoperative period is complicated by the possibility of operative alterations in the retroperitoneum, such as hema-

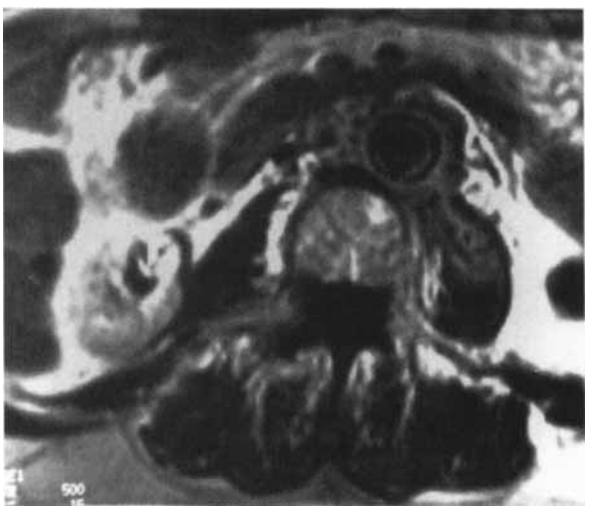

FIG. 3 Gadolinium enhanced T_1 weighted image (SE . TR500/15) shows a communicating perigraft fluid accumulation with a psoas abscess.

toma or seruma, but in patients with inflammatory syndrome the presence of fluid in MRI in the last postoperative phase is associated with graft infection. Furthermore, MRI can also evaluate the inflammatory involvement of surrounding structures. However, both examinations can fail to diagnose an infection in the first week or even sometimes in the first 3 months after surgery.[6] Thus they should be performed again before wrongly eliminating the diagnosis of infection.

Conclusion

After the insertion of an aortic graft prosthesis, fibrosis reaction can be observed in the early postoperative phase. It is usually moderate, with no compression or inflammatory syndrome, and it needs no treatment. The occurrence of a true postoperative RPF is far less frequent. When it is associated with an inflammatory syndrome, one should systemically look for an underlying systemic disease or, as in our case, a graft infection. Performing MRI is thus a necessity before starting a course of steroids.

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