Postimplantation Syndrome After Endovascular Aortic Aneurysm Repair in an Elderly Patient

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Endovascular procedures have been proposed as minimally invasive alternative treatments, allowing safe and effective aortic aneurysm repair. Despite the potential benefits, endovascular stent grafting may elicit an unexpected systemic inflammatory response, called postimplantation syndrome (PIS). The main features of PIS include fever, elevated C-reactive protein levels, leukocytosis and/or coagulation disturbances, perigraft air on abdominal computed tomography, and no evidence of infection. The main management of PIS is supportive care. Antibiotics have no clinical benefit. We report a case of PIS after endovascular aortic aneurysm repair in an elderly patient.

Key Words: Endovascular aortic aneurysm repair, Postimplantation syndrome

INTRODUCTION

Endovascular aortic aneurysm repair (EVAR) has been widely used because of its advantages compared with conventional laparotomy. When abdominal aortic aneurysm (AAA) patients were suitable for EVAR, in a previous report1, the success rate approached 98%. However, EVAR also has complications2. Among them, postimplantation syndrome (PIS) is characterized by systemic inflammation after abdominal aortic stenting3. The syndrome is frequently observed following endovascular treatment of aortic disease, PIS manifests clinically as a systemic inflammatory response syndrome (SIRS), meeting two of the four criteria for SIRS: fever and leukocytosis4. However, negative blood culture results are characteristic of PIS5. We report a case of PIS after EVAR in an elderly patient.

CASE REPORT

A 75-year-old man with a history of hypertension was admitted to the hospital for asymptomatic AAA, He had an infrarenal abdominal aneurysm (maximal diameter, 5.5 cm) (Fig. 1) and was treated with an Endurant stent (Medtronic Minimed Inc., Dublin, Ireland). On postoperative day 1, his body temperature was 38.2℃. Laboratory find-
Table 1. Comparison of body temperature and laboratory findings on different postoperative days

<table>
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<tr>
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<th>Postoperative day (day)</th>
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<tbody>
<tr>
<td></td>
<td>0</td>
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<tr>
<td>Body temperature (°C)</td>
<td>36.5</td>
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<tr>
<td>WBC (mm$^3$, 4.0–10.0×10$^3$/mm$^3$)</td>
<td>5,100</td>
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<tr>
<td>CRP (mg/L, 0–5 mg/L)</td>
<td>2</td>
</tr>
<tr>
<td>Platelet (mm$^3$, 140–400×10$^3$/mm$^3$)</td>
<td>157,000</td>
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WBC, white blood cell; CRP, C-reactive protein.

Findings included elevated C-reactive protein (CRP) levels and low platelet (PLT) counts (Table 1). Abdominal computed tomography (CT) on postoperative day 5 showed focal air densities in the abdominal aorta (outside of the stent). Initially, we suspected abscess formation (Fig. 2). We considered a systemic infection and treated him with intravenous empirical antibiotics (third-generation cephalosporin+ciprofloxacin). Finally, we diagnosed him with PIS because of the presenting continuous pyrexia, abnormal lab values (including elevated CRP and relatively low PLT counts) despite antibiotic therapy, and negative blood culture results. Additionally, abdominal CT showed peri-graft air in the abdominal aorta. Thus, we quickly stopped the intravenous antibiotics and began treatment with oral nonsteroidal anti-inflammatory drugs (NSAIDs) for symptom improvement. The fever resolved and laboratory findings soon normalized. He was discharged with improved symptoms on postoperative day 8. Follow-up abdominal CT on postoperative day 30 showed no visualization of focal air density (Fig. 3).

**DISCUSSION**

AAA is a frequently encountered disease; EVAR has gained acceptance for selected patients as an alternative to traditional open surgery and has become a standard procedure for treating AAA. Previous standard AAA management consisted of prosthetic graft interposition requiring open laparotomy, bowel manipulation, and aortic cross-clamping. This approach had severe comorbidities, particularly among older patients, and increased the surgical risk. Conversely, endovascular procedures have recently been proposed as minimally invasive alternative treatments and allow safe and effective AAA repair. Since EVAR was introduced in 1991, several authors have reported their experiences. The advantages relate to the minimally invasive nature of the new modality, faster recuperation, and decreased procedure-related morbidity and mortality. PIS is frequently observed following endovascular treatment of aortic disease. A syndrome of fever and leukocytosis after stentgraft implantation was incidentally noted in prior clinical trials performed in Germany, and it was defined in
accordance with the definition of SIRS by the presence of fever with a temperature >38°C and leukocytosis despite antibiotic therapy and negative blood culture results. The etiology of PIS is unknown, but it does not appear to be called by infection. It is believed to result from complex interactions between the vascular endothelium and the endoprosthesis fabric. Potential theoretical mechanisms of PIS are extensive local endothelial activation following stent application, contact activation of blood components by the graft fabric, and thrombus adhesion. Consecutive release of cytokines and arachidonic acid metabolites, together with leukocyte activation, may explain the systemic symptoms. The main features of PIS include fever, elevated CRP, leukocytosis and/or coagulation disturbances, perigraft air on abdominal CT, and no evidence of infection. The main management of PIS is supportive care. Antibiotics have no clinical benefit. Early management with oral NSAIDs proved to be an adequate treatment modality in many patients. In our case, PIS occurred after EVAR. The patient suffered from persistent fever and simultaneous changes in various laboratory parameters. His blood culture results were negative, and follow-up abdominal CT scan on postoperative day 5 showed several perigraft air densities suggesting PIS. After 1 month, an abdominal CT scan showed nearly complete absorption of the previous lesions. Our case raises the question as to whether there is a place for antibiotic use with PIS. Antibiotic use is controversial. According to the original article, prolonging antibiotic treatment beyond the day of endovascular intervention did not provide any short- or long-term clinical benefit in patients with PIS. In some patients, the initial inflammatory response after EVAR is not always spontaneously attenuated and could lead to the development of SIRS even several days after the operation. It seems reasonable to keep patients who develop PIS after EVAR under surveillance for a while. In one study, some patients showed features of mild SIRS with recurrent fever, tissue edema, anorexia, fatigue and weakness, and it was symptomatically helpful to treat with oral NSAIDs. Other patients needed intravenous corticosteroids. Researchers have reported an increased risk of AAA in elderly patients, and the AAA prevalence was 4–9% in a population aged >65 years. PIS can occur in elderly patients similar to our patient. The treatment for those patients does not require antibiotics.

In conclusion, PIS is a relatively common complication of EVAR used to treat AAA, manifesting features of a systemic inflammatory response even in elderly patients similar to our patient. Although the pathophysiology of PIS is unknown, antibiotic treatment is controversial. PIS mostly improves over time without antibiotic use. Thus, ongoing injudicious antibiotic use is thought to be unnecessary. Further studies are needed to understanding the underlying pathophysiology and evaluate effective preventive strategies.

REFERENCES

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