BLOOD PLATELET ACCUMULATION IN A DACRON GRAFT IMPLANTED IN A PATIENT WITH ABDOMINAL AORTA ANEURYSM: QUANTITATIVE SCINTIGRAPHIC ASSESSMENT USING INDIUM-111 LABELED PLATELETS – CASE REPORT*

MARIA M. JELEŃSKA¹, KRZYSZTOF BOJAKOWSKI¹, JOANNA MĄCZEWSKA², LESZEK KRÓLICKI², JACEK SZMIDT³

Department of General, Vascular and Transplant Surgery, Medical University of Warsaw¹
Kierownik: prof. dr hab. J. Szmidt
Department of Nuclear Medicine, Medical University of Warsaw²
Kierownik: prof. dr hab. L. Królicki

During routine screening after abdominal aorta aneurysm repair, thrombocytopenia accompanied by chronic activation of coagulation was detected in an 80-year old patient. Coagulation activity was assessed by the increased concentration of coagulation and fibrinolysis activation markers (prothrombin fragment F1+2 and D-dimer). After the most common causes of such activation were excluded using imaging and laboratory studies, Indium-111 labeled autologous blood platelet accumulation on a dacron graft was demonstrated using the scintigraphic technique. Thus, the implanted dacron graft was the cause of chronic thrombocytopenia and coagulation activation during the two years of follow up.

Key words: chronic thrombocytopenia, abdominal aorta aneurysm, vascular graft

An aortic aneurysm is one of the causes of local (1-5) and sometimes clinically overt, disseminated intravascular coagulation – DIC (6, 7, 8). In the majority of patients, surgical repair of an aneurysm is a definitive DIC treatment (6, 7, 8). However, in some patients, the increased coagulation activation persists after aneurysm repair surgery (10, 11). A patient, who developed thrombocytopenia with enhanced activation of coagulation and fibrinolysis for two years after abdominal aorta aneurysm (AAA) surgical treatment, is presented below.

CASE REPORT

An 80-year old man (K.N. medical history number 47512/01) with an AAA of 55 mm in diameter with arterial hypertension and ischemic heart disease in anamnesis underwent a classic surgical procedure using a knitted, gelatin-impregnated dacron graft (UniGraft, B. Braun Surgical GmbH). The bifurcated graft was anastomosed to the abdominal aorta below the renal arteries and with the common iliac arteries with an unabsorbable monofilament suture. After the procedure, therapy with acetylsalicylic acid (75 mg/day) was initiated.

Results of laboratory tests (4) performed 3, 9 and 24 months after surgical repair of the aneurysm are presented in tab. 1. Prothrombin time and APTT, fibrinogen and antithrombin concentrations were all within normal limits before and after surgery. Plasma concentrations of coagulation and fibrinolysis markers (prothrombin fragment F1+2 and D-dimer) were markedly increased after the procedure, as compared to preoperative values. The patient was not on drugs influencing platelet count; however 3 months after the procedure their number decreased by nearly 50% and did

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not improve in the subsequent two-year follow-up period (tab. 1).

Several tests were performed to detect the cause of coagulation activation. Results of chest computed tomography, abdominal ultrasound, echocardiography, chest X-ray, contrast barium enema and gastroscopy did not reveal any abnormalities. Red and white blood cell count, glucose, creatinine, urea, C-reactive protein and prostate specific antigen (PSA) were also within normal ranges. We suspected that thrombocytopenia and activation of coagulation were related to the implanted vascular graft since no other causes were identifiable.

Scintigraphic examination using autologous blood platelets labeled with Indium-111 provides direct evidence for blood platelet consumption in the vascular graft (12, 13, 14). After obtaining informed consent, this study was performed according to the described procedures (12). Indium-111 labeled autologous blood platelets (Mallinckrodt Medical B.V., Holland) with total radioactivity of approximately 800 MBq was administered intravenously. Chest and abdominal scintigrams were performed on a two-headed gamma camera (Varicam, Israel) equipped with a parallel, medium-energy collimator, using an energetic window of 20% width and energies 173- and 247-KeV at 2, 4, 24 and 48 hours after administration of labeled platelets.

Anterior scintigrams were used for further analysis (fig. 1a). Quantitative analysis in the regions of interest (denoted by squares; fig. 1b) was conducted as previously described (15) and the number of counts was corrected for the half-life of Indium-111.

Results of scintigraphic examinations revealed accumulation of Indium-111 labeled pla-

<table>
<thead>
<tr>
<th>Test</th>
<th>Before surgery</th>
<th>After surgery</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prothrombin time (s)</td>
<td>14,2</td>
<td>14,9</td>
<td>15,1</td>
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<tr>
<td>APTT (s)</td>
<td>36,5</td>
<td>35</td>
<td>35,5</td>
</tr>
<tr>
<td>Fibrinogen (mg/dl)</td>
<td>347</td>
<td>415</td>
<td>357</td>
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<tr>
<td>Antithrombin (%)</td>
<td>85</td>
<td>105</td>
<td>103</td>
</tr>
<tr>
<td>D-dimer (ng/ml)</td>
<td>2,931</td>
<td>5,426</td>
<td>6,351</td>
</tr>
<tr>
<td>Prothrombin fragment F1+2 (nM)</td>
<td>2,24</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Blood platelets (x10^9/μl)</td>
<td>211</td>
<td>130</td>
<td>114</td>
</tr>
</tbody>
</table>

Table 1. Hemostatic parameters in AAA patients before and after classic open repair

Fig. 1. Chest and abdominal scintigram (AP projection) performed 48 hours after administration of autologous Indium-111 labeled blood platelets (a). The same scintigram with regions of interest denoted by squares (b)

1 – heart, 2 – liver, 3 – spleen, 4 – aorta, 5 – proximal anastomosis, 6 – vascular graft, 7 – distal right anastomosis, 8 – distal left anastomosis
telets in the vascular graft that increased over time (fig. 2). The accumulation of Indium-111 labeled platelets in the dacron graft was also confirmed by the ratios of accumulation of Indium-111 labeled platelets in the graft relative to accumulation in the heart, aorta, liver and spleen (fig. 3).

DISCUSSION

Parameters of haemostasis presented in this paper performed 3, 9 and 24 months after AAA surgical repair indicate higher coagulation activation accompanied by thrombocytopenia, as compared to pre-surgery (tab. 1). Scintigrams revealed that blood platelet consumption in the studied patient results from their accumulation in the knitted dacron graft (fig. 1). It means, that chronic thrombocytopenia and activation of coagulation over two years of follow-up resulted from the implanted vascular graft.

![Fig. 2. Accumulation over time of Indium-111 labeled blood platelets in the vascular graft. Calculations are based on the number of counts obtained in regions of interest (see fig. 1b). Proximal anastomosis; vascular graft; distal right anastomosis; distal left anastomosis.](image)

![Fig. 3. Ratio of accumulation of Indium-111 labeled blood platelets in the graft versus accumulation in the aorta, heart, liver and spleen. Calculations are based on the number of counts obtained in regions of interest (see fig. 1b). Proximal anastomosis; vascular graft; distal right anastomosis; distal left anastomosis.](image)
Our results demonstrate the most significant accumulation of blood platelets in the proximal anastomosis. This effect may be a result of local blood flow disturbances.

The activation of coagulation after a year post-AAA surgical repair and graft implantation (10, 11) has been reported, but the clinical scenario was different from what has been presented in this paper. In the groups of patients analyzed by the authors, the medians levels of coagulation and fibrinolysis activation markers were increased, but to values lower than pre-surgery. However, in the presented case, the concentration of coagulation and fibrinolysis markers after surgery was higher than prior to surgery. Also, the median blood platelet count was slightly increased after the surgical procedure in the previous reports, but it was decreased in our experience.

Scintigrams obtained using Indium-111 labeled autologous blood platelets performed more than a year after AAA surgical repair, which are described in (13, 14), demonstrated blood platelet accumulation in the vascular graft, but determination of platelet number in the peripheral blood was not performed and coagulation and fibrinolysis activation markers were not measured.

Local coagulation activation in K.N. after AAA surgical repair was not accompanied by any clinical symptoms. However, the local activation of coagulation can always be transformed into life-threatening DIC, which has been reported in several AAA patients following vascular graft implantation (16, 17).

REFERENCES


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Adress correspondence: 02-097 Warszawa, ul. Banacha 1a