

Visualization of a Prosthetic Vascular Graft Due to Platelet Contamination During ¹¹¹In-Indium-labeled Leukocyte Scintigraphy

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A prosthetic axillo-femoral bypass graft was visualized during ¹¹¹In-labeled leukocyte scintigraphy in a patient referred for possible abdominal abscess. The presence of significant cardiac blood-pool activity raised the possibility that this uptake was due to deposition of contaminating labeled platelets rather than labeled leukocytes. An analysis of a small sample of the patient's blood confirmed that the circulating activity was due to labeled platelets. Increased activity along prosthetic vascular grafts in patients undergoing ¹¹¹In-labeled leukocyte scintigraphy may be due to adherent platelets and not indicative of infection.

Although its incidence is low (2%–6%), graft infection is associated with a high mortality approaching 75%, especially in aorto-femoral grafts. Graft infection is often difficult to diagnose since it can occur years after surgery, and localizing signs and symptoms can be absent (1,2). Several studies support the usefulness of indium-111 (¹¹¹In)-labeled leukocyte scintigraphy in detecting prosthetic vascular graft infections (2–5). The reported sensitivity in these clinical studies ranges from 85%–100% with a specificity range of 76%–93%.

We present a case in which a prosthetic axillo-femoral vascular graft was visualized during ¹¹¹In-labeled leukocyte scintigraphy in a patient referred for possible abdominal abscess. This finding was subsequently considered to be most likely due to adherence of contaminating platelets and not related to infection.

Case Report

A 63-yr-old female, 15 yr status post pelvic exenteration for ovarian carcinoma, presented with abdominal pain, right lower quadrant tenderness, and decreased colostomy output for one week. She had been operated on three times in the past for adhesions secondary to radiation enteritis. Unrelated but of note was the fact that she had an 8-mm Gortex right axillo-femoral bypass graft 10 yr previously.

The white blood cell count was normal at 9.7 (k/ul) (normal, 4.5–10.0) with a slight neutrophilic predominance of 72% (normal, 33–71%).

The diagnostic evaluation began with an abdominal CT scan with intravenous contrast that showed a normal, patent bypass graft and no intra-abdominal abscess. An upper gastrointestinal x-ray series showed narrowed loops of distal ileum with

proximal dilatation consistent with a partial small bowel obstruction.

In order to further evaluate the possibility of intra-abdominal abscess, an ¹¹¹In-labeled leukocyte scan (Fig. 1) was performed. Although no intra-abdominal focus was identified, modest (greater than blood pool), nonuniform, linear uptake of activity along a long segment of the vascular graft suggested graft infection. On further evaluation, as discussed below, the conclusion was reached that the graft uptake in the case presented was most likely related to deposition of contaminating labeled platelets, not leukocytes.

The patient remained afebrile with negative blood cultures. She was discharged following conservative therapy for partial small bowel obstruction.

DISCUSSION

Imaging with ¹¹¹In-labeled leukocytes is generally considered a reliable, noninvasive technique for localizing a variety of infectious processes (1,2). Meticulous technique is crucial not only to isolate and radiolabel the desired cell population, but also to maintain sterility and cell viability. In our laboratory, the patient's blood is collected in ACD (1:6) to a total volume of 40 ml. Leukocyte-rich plasma is created by gravity sedimentation, and the leukocyte pellet is formed by centrifuging the plasma at 250 g for 5 min. The pellet is resuspended in saline and incubated with ¹¹¹In-oxyquinoline* for 30 min. At the end of incubation, plasma is added to the suspension prior to recentrifugation. The labeled cells are resuspended in autologous plasma for injection. Imaging is routinely performed at 24 hr with little or no blood-pool activity.

Further review of the images in this case revealed significant cardiac blood-pool activity, which implied an excess of circulating labeled erythrocytes or platelets. Platelet uptake has been found on synthetic grafts in patients up to 10 yr post-implantation (6). To determine if labeled platelets were the source of the activity, the labeling procedure on this patient was reviewed. It was discovered that the rheostat on the centrifuge had malfunctioned and that spins of less than 250 g had not been possible. It was postulated that in forming the leukocyte pellet the cells were subjected to higher-than-usual gravitational forces resulting in contamination of the leukocytes with an excess number of platelets.

In order to verify that the greater-than-normal circulating

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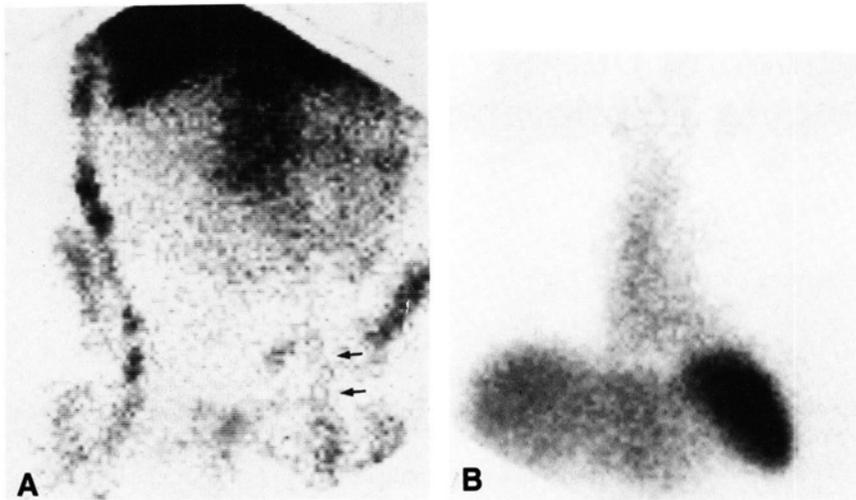


FIG. 1. (A) Anterior view of the abdomen and pelvis from the ^{111}In -labeled leukocyte scan showing modest, nonuniform linear uptake conforming to the patient's 10-yr-old right axillo-femoral prosthetic bypass graft. This activity was felt to represent adhered labeled platelets, not leukocytes. Blood pool is seen in the left iliac vessels (small arrows). Activity in the left abdomen corresponds to the colostomy site. (B) Anterior chest image from same study showing significant cardiac activity due to circulating platelets.

TABLE 1. Method for Characterizing Circulating Activity

1. Collect 5 cc blood in ACD.
2. Spin whole blood (250 g \times 5 min).
3. Separate platelet-rich plasma from packed cells.
4. Count packed cells in well counter.
5. Spin platelet-rich plasma (150 g \times 10 min).
6. Count platelet pellet in well counter.
7. Count platelet-poor plasma in well counter.

activity visualized as blood pool was due to excess labeled platelets and not red blood cells, a 5-cc blood sample was obtained from the patient. The platelets, plasma, and packed red and white cells were isolated from the sample by differential centrifugation and assayed for radioactivity (Table 1). In the patient presented, over 78% of the circulating activity was found to be platelet-associated; 21% was associated with packed cells, and 1% with platelet-poor plasma. In another patient referred for a possible infected prosthetic vascular graft (whose scan did not demonstrate blood-pool activity), the same analysis yielded the following distribution: 24% in the platelet fraction, 58% in packed cells, and 18% in the plasma.

Labeled leukocytes accumulate along an infected vascular bypass graft in a readily recognizable pattern of linear uptake that follows its configuration and course (3). No leukocyte accumulation has been reported on noninfected grafts (7). Deposition of ^{111}In -labeled platelets on grafts occurs and can produce a similar pattern (6).

A recent report (8) described two cases of false-positive uptake in noninfected pseudoaneurysms during ^{111}In -labeled leukocyte scintigraphy. The authors postulated that the uptake seen on those scans represented platelet or erythrocyte accumulation on pathologically proven thrombi. In the case presented here, uptake along the patent axillo-femoral bypass graft was similarly postulated to represent adherence of contaminating platelets since platelet (and not erythrocyte) reactivity of vascular prostheses has been well documented (6,9,10).

Indium-111-labeled leukocyte scintigraphy depends on careful isolation and labeling techniques. Although the potential for labeling platelets increases with a decreasing white cell count, the major factor in reducing platelet contamination is the separation of the two cell types. A small increase in the centrifugal force will greatly increase the number of platelets in the leukocyte pellet. Contaminating platelets will be visualized as circulating activity on the resulting scan and may accumulate at sites of vascular surgery. Therefore, in the presence of blood-pool activity the possibility of contaminating platelets adhering to prosthetic vascular grafts should be considered before a definitive diagnosis of graft infection is made.

NOTE

*Amersham International, Inc., Arlington Heights, IL

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