labeled leukocytes in a patient subsequently shown to have purulent pericarditis. Experience with ^{111}In leukocyte imaging in cardiac disorders is limited. Martin et al[6] reported focal accumulation of isotope around the left border of the heart in a patient after left ventricular aneurysmectomy who developed an abscess of the left ventricular wall and pericardium with a sinus draining to the chest wall. McDougall et al[7] have noted diffuse myocardial uptake in one patient with clinical myocarditis, but they have generally found no uptake in a small number of patients who had endocarditis or myocardial infarction. In contrast, other investigators have demonstrated myocardial uptake of ^{111}In leukocytes in experimental and clinical myocardial infarction.[8] Gallium-67 scanning has also detected a case of unsuspected purulent pericarditis;[9] however, ^{67}Ga uptake by the heart is nonspecific for purulent pericarditis and occurs in noninfective pericarditis,[10] metastatic pericardial disease, endocarditis,[10] and other cardiac disorders.[10,11] Further experience is needed to determine whether indium-111 leukocyte imaging is more specific than gallium-67 for purulent pericarditis.

In conclusion, we have demonstrated that striking pericardial uptake on a whole-body indium-111 leukocyte scan can occur with purulent pericarditis and relates to the presence of leukocytes in the pericardial fluid. Since indium-111 leukocyte imaging may become the procedure of choice for occult infection,[8] others may be confronted with this finding on scan. Since purulent pericarditis may present insidiously in debilitated or immunosuppressed patients,[3] indium-111 leukocyte imaging may be useful in making this diagnosis.

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Airway Compression by a Right Aortic Arch in the Absence of a Vascular Ring*

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This case describes the diagnosis and successful management of an unusual form of respiratory distress in an infant with tetralogy of Fallot. Severe compression of the airway resulted from a large right aortic arch in the absence of a vascular ring.

A right aortic arch can cause obstruction of the airway when it forms a vascular ring around the trachea and esophagus with either a patent or atretic left arch or a left ductus arteriosus or ligament. In this report, we describe an infant with tetralogy of Fallot whose associated large right aortic arch in the absence of a vascular ring compressed the right main-stem bronchus, causing hyperinflation of the right lung and respiratory failure. To our knowledge, this is the first report of such a case and its successful surgical repair.

Case Report

An infant was seen at one day of age with a cardiac murmur and cyanosis. A chest x-ray film disclosed normal cardiac size, a right aortic arch, and no deviation of the mediastinum. Cardiac catheterization confirmed the presence of tetralogy of Fallot and demonstrated a right aortic arch with mirror-image branching of the brachiocephalic arteries. During the infant's initial hospitalization, hyperinflation of the right lung developed. A barium esophagogram excluded the diagnosis of a vascular ring. The arterial pH was 7.39, the arterial carbon dioxide tension ($PaCO_2$) was 39 mm Hg, and the arterial oxygen pressure (PaO_2) was 65 mm Hg. There was no clinical evidence of respiratory compromise, and no further work-up was performed.

Between one and two months of age, the infant developed progressive dyspnea, increasing cyanosis, and difficulty in feeding. Physical examination revealed a cyanotic and tachypneic infant with grunting respirations but without stridor. The respiratory rate was 60/min, and breath sounds over the right side of the chest were diminished. A grade 2/6 systolic ejection murmur was heard at the left upper sternal border. The edge of the liver was palpable 4 cm below the right costal margin. Arterial blood on admission showed a pH of 7.18, a $PaCO_2$ of 68 mm Hg, and a PaO_2 of 21 mm Hg. A chest x-ray film revealed further hyperinflation of the right lung, with a shift of the mediastinum to the left and atelectasis of the left lung (Fig 1).

Intubation to the level of the carina and administration of oxygen failed to relieve the respiratory failure. Subsequent low-pressure mechanical ventilation and, eventually, muscular paralysis with

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Occult Purulent Pericarditis Detected by Indium-111 Leukocyte Imaging*

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Leukocyte imaging with indium-111 is a relatively new technique which, to this point in time, has been discussed almost exclusively in the radiologic literature. Although this procedure has been used mainly to detect intra-abdominal infection, the thorax is routinely imaged along with the abdomen, and therefore detection of cardiac disease may be feasible. This case report is of a young woman after liver transplantation who developed occult purulent pericarditis initially detected by a leukocyte scan with indium-111. This case demonstrates that striking pericardial uptake on a whole-body indium-111 leukocyte scan can occur with purulent pericarditis, and it reemphasizes how insidiously purulent pericarditis may present in an immunosuppressed patient.

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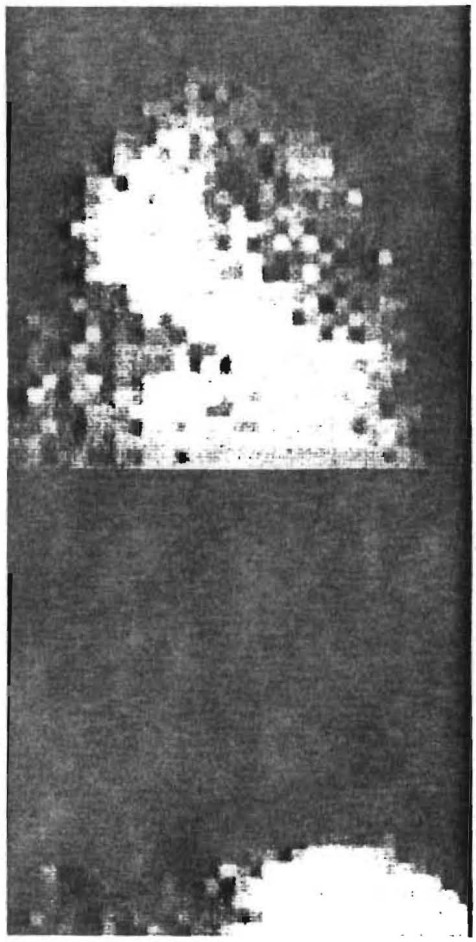

FIGURE 1. A *(upper)*. Computer display of anterior indium-111 leukocyte image showing abnormal uptake around heart. Below this is seen normal uptake by liver and spleen. 1B *(lower)*. Repeat indium-111 leukocyte image three weeks later. Abnormal pericardial activity has disappeared.

Purulent pericarditis is an uncommon[1,2] but life-threatening illness. It is essentially 100 percent fatal in untreated patients, and even appropriately treated patients have approximately a 40 percent mortality.[1,2] The early recognition of purulent pericarditis may be thwarted by the absence of typical symptoms and signs of pericarditis,[3] especially in debilitated patients. Antemortem diagnosis of this entity was made in only 60 percent (28) of a combined total of 46 patients in two recent clinical series[1,4] and in 18 percent of 55 autopsies.[5]

We present the findings in a patient who developed a spiking fever following liver transplantation. An indium-111 (^{111}In) leukocyte scan was obtained because of a suspected intra-abdominal abscess. Striking uptake around the heart was seen instead and was the first clue that the patient had purulent pericarditis.

Case Report

A 26-year-old woman was admitted for liver transplantation for cryptogenic micronodular cirrhosis and end-stage hepatic disease. There was no prior history of cardiovascular disease. Physical examination on admission revealed an afebrile patient with respirations of 18/min, blood pressure of 130/45 mm Hg, and pulse rate of 112 beats per minute without orthostatic change. She was deeply jaundiced with multiple spider angiomas. Cardiopulmonary ex-

amination revealed clear lungs, a hyperdynamic precordium, a grade 3/6 midsystolic murmur, and bounding pulses. The liver was palpable two fingerbreadths below the right costal margin, and a small amount of ascites was present. Laboratory data on admission included a hematocrit reading of 34 percent, a white blood cell (WBC) count of 11,800/cu mm (65 percent polymorphonuclear leukocytes, 5 percent band forms, 14 percent lymphocytes, and 16 percent monocytes), and a prothrombin time of 20 seconds (control, 12 seconds). The chest x-ray film revealed clear pulmonary fields and a normal cardiac silhouette. An electrocardiogram was unremarkable except for sinus tachycardia.

Eight days after admission, the patient underwent liver transplantation. The immediate postoperative period was complicated by adult respiratory distress syndrome, disseminated intravascular coagulation, and acute allograft rejection. The patient's pulmonary function improved, and she received a second liver transplant one week later. Immunosuppressive therapy included prednisone and cyclosporine.

Six days after the second transplant, the patient developed daily temperature spikes as high as 39.4°C. She initially had no complaints of chest discomfort. The findings from physical examination were notable only for a sinus tachycardia (130 beats per minute) and a blood pressure of 150/100 mm Hg. The WBC count rose to 22,700/cu mm (63 percent polymorphonuclear leukocytes, 27 percent band forms, 2 percent lymphocytes, 4 percent monocytes, and 4 percent eosinophils). Several chest x-ray films were obtained, but no definite infiltrate was demonstrated. The patient's cardiac silhouette appeared grossly unchanged as compared to prior films. Multiple cultures of blood, sputum, and urine did not reveal the source of the fever. Computerized tomography of the abdomen and pelvis did not demonstrate a definite abscess.

Approximately 12 days after the onset of fever, the patient developed pleuritic, right-sided chest pain near the costal margin. A ventilation-perfusion lung scan showed no indication of a pulmonary embolus. Autologous leukocytes were labeled with 0.5 mCi of indium-111 and were reinjected in an attempt to identify a suspected intra-abdominal abscess. Scintillation camera imaging performed at 24 hours demonstrated a normal abdomen but strikingly abnormal uptake around the heart (Fig 1A). This finding suggested pericardial or diffuse myocardial blood pool uptake. At this time the patient demonstrated a normal venous pulse and a weak apical impulse without any murmur, gallop, or pericardial rub. The ECG showed diffuse ST-segment elevation compatible with pericarditis.

Two-dimensional echocardiography demonstrated moderate pericardial effusion (Fig 2). Right cardiac catheterization showed normal pressures and a cardiac index of 6.4 L/min; there was no evidence of tamponade physiology. A pericardiocentesis was performed, with removal of 120 ml of turbid serosanguineous fluid. Gram stain revealed many polymorphonuclear leukocytes and many small intracellular Gram-negative rods. Data on the pericardial fluid included a glucose level of 146 mg/100 ml (serum glucose level was 214 mg/100 ml), lactic dehydrogenase level of 1,770 IU, and a total protein level of 3.5 g/100 ml. On the next day, 800 ml of purulent pericardial fluid was drained surgically. Culture of the pericardial fluid grew *Legionella pneumophila* (serogroup 6). Additional microbiologic and infectious aspects of this case will be reported elsewhere (Pasculle et al, unpublished observations). Following appropriate therapy a repeat leukocyte scan three weeks later showed complete resolution of the abnormal activity (Fig 1B).

Discussion

Considerable experience has accumulated in the past few years using indium-111 labeled leukocytes to detect abdominal inflammatory disease. A recent clinical study of 542 patients showed that the indium-111 leukocyte scan had a sensitivity, specificity, positive predictive value, and negative predictive value all over 90 percent.[5] Although gallium-67 citrate is still the most commonly used radionuclide for detection of abscesses,[5] it labels tumors as well as abscesses and therefore lacks the specificity of ^{111}In leukocyte imaging.[5] In addition, ^{111}In labeled WBCs do not accumulate in the normal gut, as does ^{67}Ga[5] and scanning can usually be completed within 24 hours (compared to 48 to 72 hours with gallium).

This case demonstrated marked pericardial uptake of ^{111}In

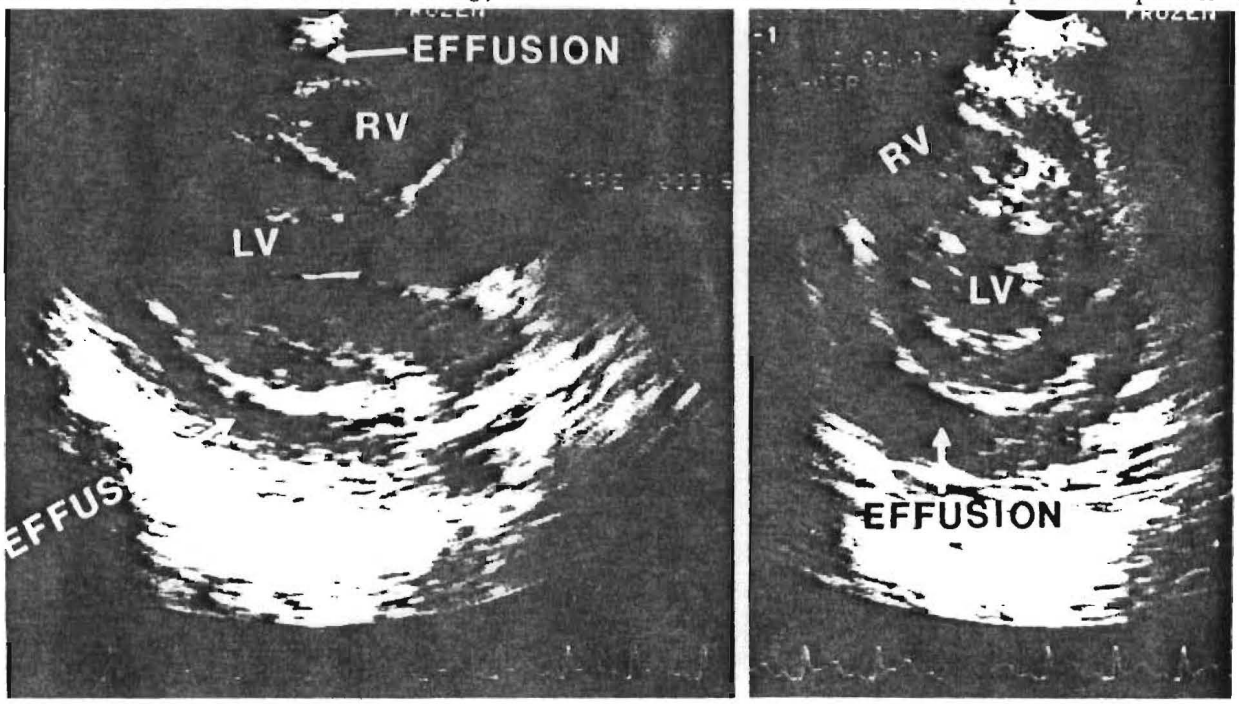

FIGURE 2. Long *(left)* and short *(right)* axis two-dimensional echocardiographic views. Echo-free space consistent with pericardial effusion surrounds heart. ST-segment elevation is seen on ECG. LV, Left ventricle; and RV, right ventricle.