

and diagnostic biopsy [2, 3] are extremely important. Clinical improvement is uncommon but possible with early corticosteroid treatment [2, 5]. In this case, PTCA was tried but failed. Coronary artery bypass grafting was even considered but was not feasible because of underlying vasculitis. Eventually, we implanted the HeartMate LVAD because of the progressive deterioration of congestive heart failure.

The wound care of the LVAD patient is critically important, because it might progressively induce pump pocket infection, graft infection, or even tissue valve endocarditis and sepsis. In our patient, the exit site wound did not heal well in the early phase possibly due to poor nutrition, poor general condition, bad drive-line fixation, and relatively short subcutaneous tunnel (the patient's height is 165 cm). If there had been no infection of the exit site and pump pocket, mediastinitis may not have occurred. The importance of wound care of LVAD patients cannot be emphasized more.

Heart transplantation could be a rational and effective treatment of severe eosinophilic heart disease before its fatal event [6]. Yet, there was no literature found regarding the treatment of such disease with LVAD. Based on our experience in this extremely rare case, we found that the eosinophilic heart disease did relapse during the LVAD support and also impact the cardiac function during relapse when it resulted in the significant decrement of LVAD pump flow, though the condition could be controlled with steroid administration. While characteristics of the disease should be kept in mind, long-term low-dose steroid treatment should be considered in such patients. After heart transplantation, serial investigations including echocardiography, ECG, EMB, immunoglobulin E, and leukocyte with differential count showed no evidence of rejection of, nor recurrence of eosinophilic heart disease in, the transplanted heart. Similar to previous reports, this could be attributed to the immunosuppressant treatment after transplantation. On this particular patient, we conclude that LVAD and heart transplantation is a life-saving and effective treatment of eosinophilic heart disease, though long-term follow-up is still needed.

References

1. Tonnesen P, Teglbjaerg CS. An "unexpected" fatal case of the hypereosinophilic syndrome. *Eur J Respir Dis* 1984;65:389-93.
2. Isaka N, Araki S, Shibata M, et al. Reversal of coronary artery occlusions in allergic granulomatosis and angiitis (Churg-Strauss syndrome). *Am Heart J* 1994;128:609-13.
3. Seshadri S, Narula J, Chopra P. Asymptomatic eosinophilic myocarditis: 2 + 2 = 4 or 5. *Int J Cardiol* 1991;31:348-9.
4. Taliercio CP, Olney BA, Lie JT. Myocarditis related drug hypersensitivity. *Mayo Clin Proc* 1985;60:463-8.
5. Terho EO, Valtia R, Tukiainen H, Iansinies E. Myopericarditis associated with farmer's lung. *BMJ* 1980;281:197.
6. Touze JE, Debonne JM, Scheiner C, et al. Acute necrotizing eosinophilic myocarditis. Favorable clinical course after heart transplantation. *Presse Med* 1992;21:565-8.

Cardiac Involvement in a Case of Acute Lymphoblastic Leukemia

Ashutosh Hardikar, MCh, FRCS, Prem Shekar, MCh, FRCS, John Stubberfield, FRACS, David R. Craddock, FRCS, FRACS, and Leon P. Bignold, FRCPA

Department of Cardiothoracic Surgery, The Royal Adelaide Hospital, and Division of Tissue Pathology, Institute of Medical and Veterinary Science, Adelaide, Australia

We present an extremely rare case of an immunocompromised patient with a T-cell acute lymphocytic leukemia relapse presenting as a right atrial tumor. Problems in diagnosis, vulnerability due to previous immunosuppression and bone marrow transplant, and successful surgical excision are highlighted. Cardiac involvement with hematologic neoplasms should be taken with more than academic interest, as it may be amenable to treatment.

(Ann Thorac Surg 2002;73:1310-2)

© 2002 by The Society of Thoracic Surgeons

Cardiac infiltration by hematologic neoplasms leading to clinically detectable cardiovascular disease is rare. Such manifestations are likely to be more common as these neoplasms are more amenable to therapy than heretofore, and the proportion of immunocompromised population is on the rise [1-4]. Extranodal malignant leukemias or lymphomas involving only the heart without dissemination is extremely rare and very few antemortem cases have been reported. We report an immunocompromised patient with a T-cell acute lymphocytic leukemia (ALL) relapse presenting as a right atrial tumor.

A 31-year-old Caucasian man presented in September 1998 with shotty lymph node enlargements in the neck and raised white cell count with 86% blast cells. Investigations showed him to have ALL, the L₂ subtype. Seventy-six percent of the blasts in the bone marrow showed 11 q 23 rearrangement. Blood tests were negative for human T-cell lymphoma virus, human immunodeficiency virus, or the hepatitis group of viruses. He was treated with leukopheresis and chemotherapy (LaLa [Leucemie Aigue lymphoblastique de l'Adulte]), to which he responded poorly. Heavy contamination of the CD34+ fraction of his autologous stem cells by leukemic cells precluded autologous bone marrow transplant

Accepted for publication Aug 7, 2001.

Address reprint requests to Dr Hardikar, Department of Cardiothoracic Surgery, Royal Adelaide Hospital, Level IV, East Wing, North Terrace, Adelaide 5000, South Australia; e-mail: a_hardikar@hotmail.com.



Fig 1. Gross pathology of the cardiac tumor removed from the right atrium.

(BMT). Hence, matched unrelated donor BMT was performed in December 1998. Post-BMT, the immunosuppression consisted of cyclophosphamide, total body irradiation, and antithymocyte globulin. The postoperative period was complicated by severe mucositis, strepto and enterococcus bacteremia, epistaxis, mild clinical veno-occlusive disease, conjugated hyperbilirubinemia, depression, transient mental confusion, and grade II skin graft versus host disease which responded to intravenous steroids. He later suffered from an episode of left lower lobe pneumonia in 1999.

In September 2000, he presented again with swollen abdomen and ankles. He was essentially afebrile and diagnosed to have ascites, with the possibilities of recurrent ALL and inferior vena caval thrombosis in mind. He was investigated with a computed tomographic abdomen and duplex study for the great vessels, which failed to pinpoint anything other than the ascites. Finally, he underwent a diagnostic laparotomy when 1.5 liters of ascitic fluid was found with white cell count of 23,000. No

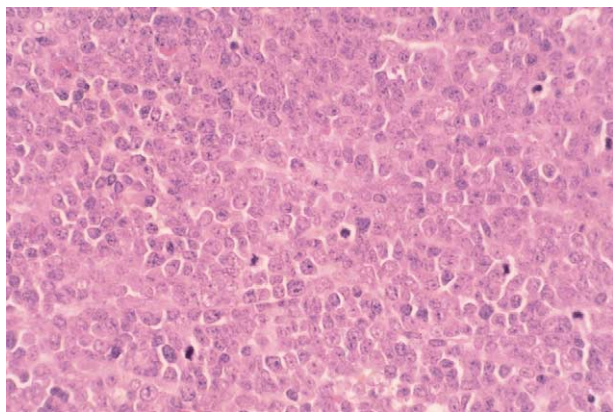


Fig 2. Histology of the atrial tumor showing closely packed lymphoid cells with occasional mitoses. (Hematoxylin and eosin; original magnification $\times 400$.)

organisms were grown on repeated cultures. He was then labeled as a case of spontaneous bacterial peritonitis and the ankle swelling was attributed to isolated right heart failure, for which an echocardiogram was performed. This showed him to have a large right atrial mass involving interatrial septum and partially obstructing the tricuspid orifice.

He was then taken up for surgery on a semi-urgent basis. At surgery, the right atrium was found to be greatly distended due to an intraluminal mass. The pericardium was grossly not involved. Venous cannulation was established using direct superior vena caval and inferior vena caval cannulation. Right atriotomy revealed a $12 \times 11 \times 3$ -cm mass, which was excised along with its attachment to the interatrial septum. The mass weighed 98 gm (Fig 1). Tricuspid valve was intact and the rest of the heart had no focal macroscopic involvement.

Histologic evaluation of the mass showed it to be a uniform tumor composed of small cells having scant cytoplasm and vesicular nuclei (Fig 2). The immunoperoxidase staining showed it to be consistent with T-cell acute ALL relapse. Flow cytometry showed the phenotype as CD1+ CD5+ CD7+ CD4- CD8- CD34+ HLA-DR-, CyCD3+. The margins were found to be containing malignant cells. The patient did well in the postoperative period, except episodes of supraventricular tachycardia, which responded to digoxin. He was discharged uneventfully on Warfarin (as the interatrial septum was denuded). He underwent a high resolution computed tomographic scan of the chest for pulmonary recurrences and a transthoracic echocardiogram, which ruled out focal macroscopic disease. Now at 3 months postoperatively, although he has no cardiac related symptoms, he has presented with testicular and skin recurrence. Because of this, and the fact that the margins of the cardiac mass were not free, he has been on intensive chemotherapy once again.

Comment

Cardiac involvement by metastatic neoplasms is relatively uncommon and usually occurs with widely disseminated disease. Very few cases of hematologic malignancies have been reported, and most have been lymphomas or T-cell leukemia lymphoma [2, 3, 5, 6]. Even primary cardiac lymphomas have been described [2]. In a review of 3,314 consecutive autopsies, 806 (24.3%) had malignancies, and 95 (11.8%) of these had cardiac involvement [1]. The most common malignancies encountered in the order of decreasing frequency were lung, lymphoma, breast, leukemia, stomach, melanoma, liver, and colon. Cardiac involvement has been described in chronic adult T-cell leukemia. It is caused by human T-cell lymphoma virus-1 and is endemic in southwest Japan, the Caribbean, Africa, and South America [5]. We report this rare case of cardiac involvement in a relapse of ALL.

In a review of clinical and autopsy study of cardiac involvement in 45 bone marrow transplantation patients

[4], 15 showed electrocardiogram ST segment or T-wave changes and arrhythmias. Fifteen patients had increased heart weight, with myocardial edema, fibrosis, and cellular hypertrophy predominating. Only 2 had marantic endocarditis of the aortic valve. They found that clinically significant heart involvement affects 5% to 10% of patients undergoing BMT after undergoing pretreatment with cyclophosphamide and total body irradiation. Acute T-cell leukemia/lymphoma involving the myocardium diffusely is known. A patient with double valve replacement for the same was described at autopsy as having widespread cardiac infiltration with malignant cells [3]. One hundred and thirty-one cases with pericardial involvement in advanced malignancy have been described which presented with features of tamponade [7]. Lung is the most common primary, followed by lymphomas and leukemias. In the present case, we see a very large ($12 \times 11 \times 3$ cm) involvement of the right atrium with no obvious involvement of myocardium, pericardium, or valvular tissue.

Right atrial tumors, commonly myxomas, presenting with features of right heart failure, with evidence of tricuspid stenosis have been described [8]. Isolated cardiac aspergillosis after bone marrow transplant has also been described [9]. In the present case, the mass went undiagnosed for a long time. He even underwent an exploratory laparotomy, when a timely echocardiogram would have revealed the diagnosis.

To conclude, we point out that cardiac involvement with hematologic neoplasms should be taken with more than academic interest, as it may be diagnosed early and may be amenable to aggressive treatment [6].

References

1. Abraham KP, Reddy V, Gattuso P. Neoplasms metastatic to the heart: review of 3314 consecutive autopsies. *Am J Cardiovasc Pathol* 1990;3:195-8.
2. Margolin DA, Fabian V, Mintz U, Botham MJ. Primary cardiac lymphoma. *Ann Thorac Surg* 1996;61:1000-1.
3. Furihata M, Ido E, Iwata J, et al. Adult T cell leukemia/lymphoma with massive involvement of cardiac muscle and valves. *Pathol Int* 1998;48:221-4.
4. Kupari M, Volin L, Suokas A, Timonen T, Hekali P, Ruutu T. Cardiac involvement in bone marrow transplantation: electrocardiographic changes, arrhythmias, heart failure and autopsy findings. *Bone Marrow Transplant* 1990;5:91-8.
5. Takata J, Taguchi H, Miyoshi I, Doi YL. Cardiac valve invasion in chronic adult T cell leukemia. *Heart* 1998;80:311-2.
6. Wiernik PH, Sutherland JC, Stechmiller BK, Wolff J. Clinically significant cardiac infiltration in acute leukemia, lymphocytic lymphoma, and plasma cell myeloma. *Med Pediatr Oncol* 1976;2:75-85.
7. Islam N, Ahmedani MY. Renal carcinoma presenting as cardiac tamponade: a case report and review of literature. *Int J Cardiol* 1998;64:207-11.
8. Frishman W, Factor S, Jordan A, et al. Right atrial myxoma: unusual clinical presentation and atypical glandular histology. *Circulation* 1979;59:1070-5.
9. Johnson RB, Wing EJ, Miller TR, Rosenfeld CS. Isolated cardiac aspergillosis after bone marrow transplantation. *Arch Intern Med* 1987;147:1942-3.

Myocardial Revascularization With the Posterior Tibial Artery

Vichai Benjacholamas, MD, Sirachai Jindarak, MD, and Wacin Buddhari, MD

Cardiothoracic Unit and Plastic and Reconstruction Unit, Department of Surgery, and Cardiology Unit, Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

A large number of coronary artery bypass grafts are performed in Thailand. Some patients develop restenosed coronary arteries or stenosed graft conduits. Great saphenous veins, internal thoracic arteries, radial arteries, and right gastroepiploic arteries are used for redo coronary artery bypass grafting. But even with many conduits to choose from, sometimes graft conduits are not available. We report a case of redo coronary artery bypass grafting where the posterior tibial artery was harvested for the graft conduit. Clinical outcome and angiographic results are reported at 1 year postoperation.

(Ann Thorac Surg 2002;73:1312-4)

© 2002 by The Society of Thoracic Surgeons

In coronary artery bypass graft surgery, the patency of arterial graft conduits is better than venous graft conduits. The posterior tibial artery has been widely used in plastic and reconstruction surgery with low morbidity but has never been used in coronary artery bypass graft surgery. This graft conduit may be useful when a good graft conduit is lacking. We report here a case of redo CABG with posterior tibial artery graft conduit.

A 64-year-old man had a previous coronary artery bypass graft (CABG) for angina, 8 years prior to presentation. At that time, the left internal thoracic artery was grafted to the left anterior descending artery (LAD). An entire great saphenous vein of the right leg and great saphenous vein of the lower half of the left leg were harvested and used for the graft conduits. He was symptom free for 8 years. During the last 6 months, he developed dyspnea and his New York Heart Association functional class changed from I to III. He underwent a repeat angiography. It showed that the left internal thoracic artery was occluded, the vein graft to the obtuse marginal branch was totally occluded, the vein graft to the posterior descending artery (PDA) was patent, the diagonal and posterolateral branches were stenosed at their origin, and the mid-right coronary artery and proximal PDA were also severely stenosed. The left ventricular function was fair, as the ejection fraction was 0.45.

He was scheduled for redo CABG, and four graft conduits were required. Both radial arteries could not be

Accepted for publication Aug 2, 2001.

Address reprint requests to Dr Benjacholamas, Cardiothoracic Unit, Department of Surgery, Chulalongkorn Hospital, Bangkok 10330, Thailand; e-mail: vichaicu@hotmail.com.