



Figure 1. HCRT Bilateral pulmonary ground glass opacities.

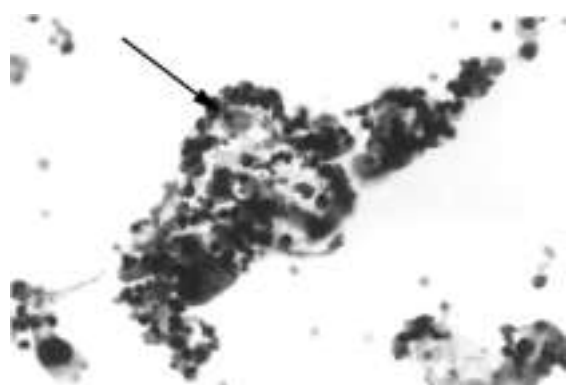


Figure 2. Bronchoalveolar lavage: macrophages, lymphocytes and atypical epithelial cells clustered around cyanophilic amorphous material (arrow).

( $pO_2$  20 mmHg;  $pCO_2$  32.8; sat  $O_2$  32.6; pH 7.37). Patchy alveolar shadows were present in the left lower lobe. HRCT showed areas of alveolar opacification in both lower lobes with an air bronchogram and patchy areas of ground glass appearance. BAL profile: 540,000 cells/mL, macrophages 9%, lymphocytes 3%, neutrophils 87%, eosinophils 1%. Atypical epithelial cells clustered around cyanophilic amorphous material were detected in a background of alveolar hemorrhage (Figure 2). BAL was negative for BK, CMV, adenovirus, syncytial respiratory virus, Herpes simplex type 1, influenza and parainfluenza viruses. The patient did not respond to intravenous trimethoprim-sulfadiazole, fluconazole, ceftazidime or teicoplanin; blood cultures remained negative. The patient rapidly improved only after initiation of prednisolone 60 mg/die.

Pulmonary toxicity of both ARA-C and fludarabine has been occasionally described in the past.<sup>1-6</sup> Based on our data, we want to highlight the occurrence of alveolar hemorrhage in both our patients, not frequently reported in the literature. In Case #1, fludarabine may have played a role in causing a subacute organizing pneumonia-like picture, as previously reported.<sup>6</sup> Steroid therapy ameliorates the lung damage probably by inactivating a cytokine network produced by drug-induced cell toxicity.<sup>7</sup>

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### Key words

Lung toxicity, fludara, Ara-C.

### Funding

The authors thank Ravenna AIL for financial support.

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### Difficulties in the diagnosis of primary cardiac lymphomas

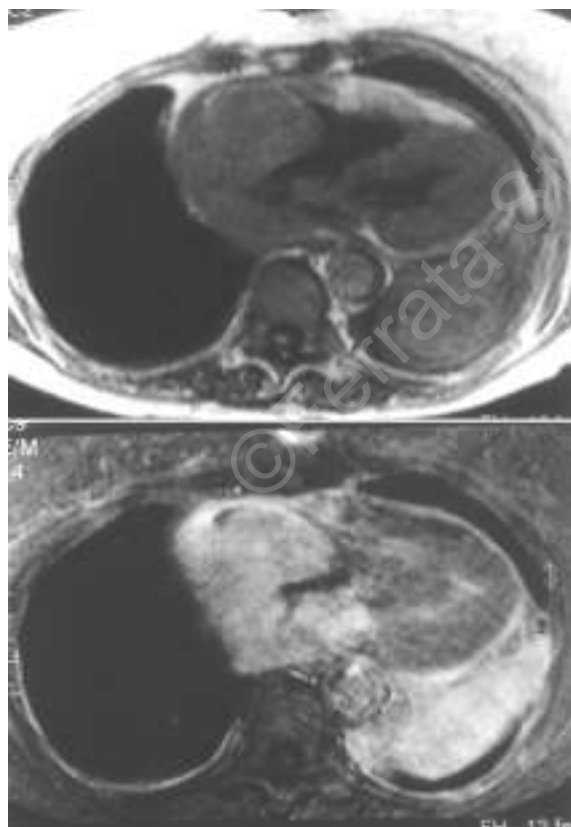
Primary cardiac lymphoma (PCL) is defined as a non-Hodgkin lymphoma involving only the heart and pericardium. Clinical presentations are non-specific for variable involvement of cardiac structures. We describe a case of PCL presenting with left pleural effusion. A cardiac malignancy was suspected by magnetic resonance imaging but pathological diagnosis made only after thoracotomy.

Sir,

A 78-year-old woman was admitted to our hospital with the complaint of dyspnea. She had a well controlled hypertension and suffered from Herpes zoster affecting the gluteal region one month earlier.

Physical examination revealed dullness to percussion at the base of the left lung. There was no jugular

vein distension, hepatosplenomegaly, peripheral edema or lymphadenopathy. Chest X-ray revealed an enlarged heart and left pleural effusion. An ECG was unremarkable. Hematological examination disclosed liver function tests slightly abnormal. Hepatitis screen was positive for antibody to HCV. Erythrocyte sedimentation rate was 106 mm/h. Transthoracic echocardiography showed a small anterior and posterior pericardial effusion. On three occasions, left sided thoracenteses yielded exudates exhibiting no acid fast bacilli or tumor cells on microscopic examination and no growth on culture. Total body contrast-enhanced computed tomography (CT) revealed a large filling defect in the right atrium, a pericardial and left pleural effusion, but no signs of mediastinal or intrabdominal involvement. Transesophageal echocardiography confirmed the presence of a large atrial tumor arising from the free atrial wall. Magnetic resonance imaging (MRI) scans displayed a 6.5x7.0 cm mass in the free wall of the right atrium (Figure 1). Because the nature of the tumor remained unclear, the patient was transferred to the cardiovascular surgery department to secure a tissue diagnosis. Exploratory thoracotomy revealed a firm and fleshy tumor growing out from the right atrium. The tumor,



**Figure 1.A:** Axial T1-weighted magnetic resonance imaging showing a 6.5 x 7.0 cm mass arising from the free wall of the right atrium. The tumor is noted to be isointense to adjacent myocardium. Pericardial effusion is also identified. **B:** Following intravenous gadolinium administration, the mass enhances heterogeneously.

evidently inoperable, was biopsied and the chest closed. The histological and immunohistochemical diagnosis was high grade B-cell lymphoma, Burkitt type.<sup>1</sup> The patient died two days later.

Primary cardiac lymphomas are very rare and represent only about 1% of all non-metastatic cardiac malignancies.<sup>2</sup> Our literature searches revealed fifty case reports between 1949 and 2000. It is possible that acquired immune deficiency disease and immunosuppressive therapy have increased the incidence of PCL in recent years.<sup>3,4</sup>

Non-specific clinical features include chest pain, arrhythmias, congestive heart failure and cardiac tamponade.<sup>5</sup> In our case were no signs or symptoms suggesting the presence of a cardiac tumor. Most of reported PCL arose from the right cardiac chambers, and it has been suggested that mass lesions involving the right heart and its proximate large vessels are difficult to visualize by transthoracic echo.<sup>6</sup> In our case transthoracic echocardiography failed to detect a cardiac tumor, although chest CT scan was suggestive of a filling defect in the right atrium. A mass lesion was however clearly visualized by both transesophageal echocardiography and MRI. In a recent review, MRI and transesophageal echocardiography were superior to computed tomography for the detection of PCL and were estimated to have sensitivities of 100% and 92%, respectively.<sup>5,7</sup> However, the differential diagnosis of cardiac tumors always requires a histologic examination. Detection of cardiac lymphomas by cytological examination of pleural and pericardial fluid or by histologic examination of endomyocardial biopsies may facilitate early diagnosis and institution of effective chemotherapy.<sup>8</sup> Surgical treatment should be considered mainly as a palliative procedure after a positive tissue diagnosis has been obtained.<sup>5</sup>

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### Key words

Primary cardiac lymphoma

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### Gallium-67 uptake by cutaneous lesions in a patient with Burkitt-like non-Hodgkin's lymphoma

Sir,

Although Gallium-67 ( $^{67}\text{Ga}$ ) uptake by primary cutaneous B-cell lymphoma has been reported, this technique does not have a defined role in monitoring cutaneous lymphoma lesions.<sup>1</sup> We present a  $^{67}\text{Ga}$  scintigraphy and confirmatory SPECT study (Figures 1 and 2) showing uptake by a secondary skin lymphoma two months before clinical relapse of a Burkitt-like non-Hodgkin's lymphoma with typical diffuse large B-cell lymphoma translocations involving BCL-6 and p53 genes.  $^{67}\text{Ga}$  scanning and SPECT may be useful for monitoring cutaneous lesions in Burkitt-like and diffuse large B-cell lymphomas.

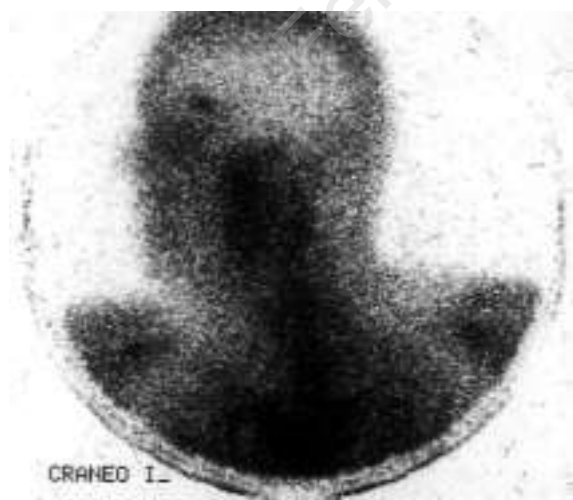


Figure 1. Cranial projection image of a Gallium planar study. A diffuse Ga-67 uptake in the scalp region was noted.

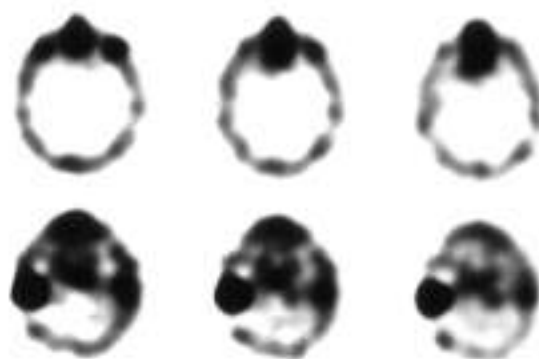


Figure 2. Axial projection of SPECT study showed a pathologic uptake in right parotid gland and homolateral submaxillar region, as well as a visualization of the multiple and nodular scalp lesions.

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#### Key words

Cutaneous lesions, Gallium-67 uptake, Burkitt-like non-Hodgkin's lymphoma

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### Simultaneous occurrence of multiple myeloma and Hodgkin's disease. A case report

We report the case of a 52-year old male diagnosed as having multiple myeloma (MM) and Hodgkin's disease (HD) simultaneously, in the absence of any prior treatment. His plasma cell dyscrasia did not progress autonomously but was associated with HD.

Sir,

Our patient presented in June 1993 with a 3-month history of relapsing-remitting fever. Physical and laboratory findings were normal except for anemia (hemoglobin 9 g/dL), erythrocyte sedimentation rate of 102 mm/h, and presence of an IgG- $\kappa$  monoclonal component with Bence-Jones protein in urine. The bone marrow was infiltrated by plasma cells up to 60%, but a bone ray survey was normal. The patient was diagnosed as having MM and was started on melphalan-prednisolone. Two months later, all findings remained unchanged and he developed left axil-