CASE 1
A 50-year-old man sought care for progressive dyspnea on exertion, abdominal bloating, and bilateral leg edema. He had hypertension that was being treated with atenolol, nifedipine, and enalapril. On examination, his blood pressure was 157/80 mm Hg and his heart rate was 50 beats/min. Jugular venous pressure was grossly elevated with occasional cannon A waves. The patient also had decreased breath sounds in both lower lung zones and moderate pitting edema up to the knees. A chest x-ray showed a small bilateral pleural effusion and no cardiomegaly. An electrocardiogram revealed complete atrioventricular (AV) block with a ventricular response of 50 beats/min. Computed tomography (CT) angiography revealed no evidence of a pulmonary embolus, but did show several enlarged (up to 3.5 cm in diameter) lymph nodes in the upper and middle mediastinum (FIGURE 1). We performed an echocardiogram.

CASE 2
A 79-year-old man with hypertension and diabetes presented to our medical center with acute dyspnea. During the physical examination, we noted bilateral diminished breath sounds with expiratory wheezes and an irregular pulse. Chest x-ray showed mild pulmonary congestion. A chest CT demonstrated bilateral small pleural effusions and multiple enlarged mediastinal lymph nodes with a maximal diameter of 2.4 cm (FIGURE 2A). One week later, the patient’s shortness of breath increased and he was hospitalized. A chest x-ray at that time showed moderate pulmonary congestion, so we performed an echocardiogram.

THE DIAGNOSIS
The echocardiogram for the 50-year-old patient in Case 1 revealed a mildly dilated left ventricle with normal systolic function, diastolic left ventricular (LV) dysfunction, moderate...
tricuspid regurgitation, and mild pulmonary hypertension. Extensive testing for malignancy and tuberculosis was negative.

For the 79-year-old patient in Case 2, echocardiography demonstrated concentric LV hypertrophy, mild dilatation of the left ventricle, normal LV systolic function, LV diastolic dysfunction with elevated LV diastolic filling pressure, and mild-to-moderate pulmonary hypertension.

Based on these results, we diagnosed both patients with diastolic heart failure. The patient in the second case had features of cardiac asthma, as well. Both patients had also developed reversible mediastinal lymphadenopathy (MLN), of which the diastolic heart failure was the only apparent cause. In both cases, radiologists did not note any suspicious findings for malignancy beyond the MLN.

DISCUSSION

Systolic heart failure has been previously recognized as a cause of MLN. Other causes of MLN include sarcoidosis, various malignancies, pulmonary infections, and occupational lung diseases. There are, however, no reports of MLN in patients with diastolic heart failure.

**Heart failure and MLN.** Slanetz et al reported one series of 46 patients who had undergone CT of the chest during periods of congestive heart failure (CHF). There was mediastinal lymph node enlargement in 55% of these patients. In a subset of 17 patients who had elevated capillary wedge pressure, 82% had some degree of lymphadenopathy.

Erly et al retrospectively studied 44 patients who had a thoracic CT performed before cardiac transplantation. Twenty-nine (66%) had at least one enlarged mediastinal lymph node (>1 cm). Eighty-one percent of patients with an ejection fraction <35% had lymphadenopathy, while none of the patients with an ejection fraction >35% had lymphadenopathy. Most enlarged lymph nodes were pretracheal, with a mean short axis diameter of 1.3 cm.

However, Storto et al reported that an association between CHF and MLN was not found in 7 patients undergoing high-resolution CT imaging. There are also cases
of MLN in patients with pulmonary hypertension without systolic dysfunction.\(^4\)

Chabbert et al studied 31 consecutive patients with subacute left heart failure (mean ejection fraction, 39%).\(^5\) Enlarged mediastinal lymph nodes were present in 13 patients (42%). Other radiographic features included blurred contour of the lymph nodes in 5 patients (16%) and hazy mediastinal fat in one patient (3%). Follow-up CT showed a significant decrease in the size of the lymph nodes in 8 of 13 patients (62%) following initiation of treatment.

**Heart failure and malignancy.** A PubMed search with the keywords “diastolic dysfunction” and “lymphoma” found 7 references in the English language. There is a report of 125 survivors of childhood lymphomas treated with mediastinal radiotherapy and anthracyclines,\(^6\) another of 44 children treated for acute lymphoblastic leukemia and Hodgkin’s lymphoma,\(^7\) a report of 294 patients who had received mediastinal irradiation for the treatment of Hodgkin’s disease,\(^8\) and another of 106 survivors of non-Hodgkin’s and Hodgkin’s lymphomas.\(^9\) None of these reports, however, made any mention of mediastinal lymphadenopathy.

**What caused the lymphadenopathy in our patients?**

Our 2 patients had volume overload due to diastolic dysfunction with elevated LV end diastolic pressure. Our first patient also had a loss of AV synchronization—which was reversible upon pacemaker insertion—that probably exacerbated the heart failure.

The mechanism for the lymphadenopathy is not clear, but may be due to cardiogenic pulmonary edema causing distension of the pulmonary lymphatic vessels and pulmonary hypertension. In a study of patients with severe systolic dysfunction undergoing evaluation for cardiac transplant, there was a relationship (albeit weak), between MLN and mitral regurgitation, tricuspid regurgitation, elevated mean pulmonary artery pressure, elevated pulmonary capillary wedge pressure, and elevated right atrial pressure.\(^10\)

**How to accurately detect and treat MLN**

MLN may be detected by chest x-ray, CT, magnetic resonance imaging, or endoscopic ultrasound examinations. The clinical situation will dictate the imaging modality used. Keep in mind that it is difficult to make a comparison between a finding of lymphadenopathy on one modality and another, especially if one is looking for a change in size.

If clinically appropriate, a trial of diuretics, such as intravenous (IV) furosemide 80 mg, should be considered before embarking on invasive procedures such as mediastinal lymph node biopsy.

**Our patients.** The 50-year-old man in Case 1 responded well to 80 mg of IV furosemide after one hour and improved further upon receipt of a pacemaker the next day. A repeat thoracic CT one month later showed complete resolution of the MLN.

The 79-year-old man in Case 2 also received 80 mg of IV furosemide and improved within 3 hours. A month later, a repeat thoracic CT showed a significant reduction in the size of all the enlarged lymph nodes (FIGURE 2B).

**THE TAKEAWAY**

The importance of these 2 cases is that they show that heart failure—even diastolic alone—can produce enlarged mediastinal lymph nodes. In patients with heart failure in whom unexpected MLN is detected, consideration should be given to performing a repeat imaging examination after the administration of diuretics.

**References**


