*Interactive case report***A 66 year old woman with breathlessness: case outcome**

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This is the final part
of a three part case
report, which
describes the
outcome and
summarises the
comments made by
readers during the
case presentation

Five weeks ago (20 March, p 698) we described the case of Mrs Dempsey, who was investigated for suspected heart failure. Her symptoms did not improve after initial treatment, and she had further investigation to determine the cause of her bilateral pleural effusions (27 March, p 758). We then suspected an underlying malignancy, and she had computed tomography to assess her chest and extrapulmonary structures (fig 1). This showed large bilateral effusions associated with widespread mediastinal, axillary, and abdominal lymphadenopathy. The likely diagnosis was now advanced lymphoma.

This right axillary node, which had not been detected on previous examinations, was clearly palpable and a biopsy sample was taken. This confirmed the diagnosis of lymphoma. Immunohistochemistry results were in keeping with a grade III follicular B cell non-Hodgkin's lymphoma. Once the biopsy results were available, Mr and Mrs Dempsey were told about the diagnosis of lymphoma at an outpatient appointment, and she was transferred to the haematology unit.

Her effusions were drained and the fluid again contained abundant mature lymphoid cells. Immunophenotyping showed reactive T cells but 35% CD20 positive clonal B cells, similar to the appearances found in the axillary node tissue. These features were in keeping with lymphoma in the pleura. Because of the extensive nature of her incurable low grade lymphoma and her debilitating symptoms, she accepted chemo-

Fig 2 Repeat chest radiograph

therapy with cyclophosphamide, vincristine, and prednisolone in an attempt to control the progression of the disease and limit the recurrence of pleural fluid.

Despite initial pancytopenia and neutropenic sepsis, she has tolerated three cycles of chemotherapy. Currently she reports feeling well in herself and gets breathless only when walking up slopes. Her last chest radiograph showed resolution of her effusions and re-expansion of her lungs (fig 2).

The prevalence of congestive heart failure in women aged 60-69 is 25 per 1000,¹ compared with an incidence of non-Hodgkin's lymphoma of 140 per million population.² Five year survival is 45% for non-Hodgkin's lymphoma² and 38% for women with congestive heart failure.³

Competing interests: None declared.

- 1 Ho KK, Pinsky JL, Kannel WB, Levy D. The epidemiology of heart failure: the Framingham Heart Study. *J Am Coll Cardiol* 1993;22(suppl A):6-13A.
- 2 National Cancer Intelligence Centre, Office for National Statistics, on request, October 2002.
- 3 Ho KK, Anderson KM, Kannel WB, Grossman W, Levy D. Survival after the onset of congestive heart failure in Framingham Heart Study subjects. *Circulation* 1993;88:107-15.

Fig 1 Computed tomography of thorax showing large bilateral effusions, 10 mm mediastinal node, and 30 mm right axillary node

Commentary: Patient's view

Mrs Dempsey with the support of Douglas Lowdon

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I agreed to my case being used because I hoped that it might assist doctors, and in particular remind them to think of the diagnosis of lymphoma, and so benefit future patients. It's quite humbling to think that doctors from every continent have read about my illness. My only negative comment about the responses is the number of doctors who referred to me as "old" or "elderly." I am only 66 after all.

After weeks of suffering with breathlessness, I finally decided to trouble my doctor when I could not play with the grandchildren. When Dr Ogilvie told me I probably had "fluid on the lungs," I was excited at the prospect of feeling better with water tablets.

I thought I would wait months to be seen at the hospital, but I was seen within days. When I was told by Dr Lowdon that the breathlessness was probably a

consequence of “heart failure” I should have been very frightened, but I had never had any heart pain and lived a healthy lifestyle so I do not think I believed him.

When I returned two weeks later I was disappointed to feel no better, but I began to worry only when Dr Lowdon said he didn’t know what was giving me fluid on my lungs. When he told me it was not due to the heart I felt like saying, “I could have told you that.” He wanted me to stay in hospital to get the fluid drained. I said I did not want to take up a bed as there were many elderly patients waiting in the corridor for beds. I felt they were more deserving than I was. I had also seen drains inserted into patients on the television and was scared.

The period when I was waiting for scans and then results was very frustrating because I just wanted answers and to feel better. I felt that life was on hold.

When we were told that I had “cancer of the glands,” the word cancer hit me and I wept for the whole afternoon. I felt “why me” and “I’m on my way out.” My major concern though was how to tell my family. The haematology nurses talked to me and answered all my questions. This made it easier to explain my illness and treatment to my family.

I had repeat computed tomography yesterday after my last scan apparently showed “significant reduction” in the size of my cancerous glands after three cycles of chemotherapy.

At first, I associated cancer with death; I do not now. It has been a long six months, but finally I feel almost back to normal. I never thought that I would feel like this again. At present, I can walk up the hill on the way home from town. In fact, it is my husband’s hip that is holding me up now—we hope the hospital will treat him next.

Commentary: Atypical features make echocardiography mandatory in suspected heart failure

Allan D Struthers

One of the key skills of a good diagnostician is to spot when a patient’s case has enough atypical features for there to be a strong chance that the “obvious” diagnosis might be wrong. This suspicion would lead a good diagnostician to perform extra investigations.

In this case, the normal electrocardiographic appearance virtually excluded the possibility of left ventricular systolic dysfunction (figure). The other less reliable atypical features were the lack of any history of ischaemic heart disease and the lack of any response to furosemide (although the dose might not have been high enough). Of course, these alerting, atypical features had to be balanced against all the positive features suggesting heart failure: hypertension, pansystolic murmur, raised jugular venous pressure, pleural effusions, and possible cardiomegaly.

It was somewhat unusual that an angiotensin converting enzyme inhibitor was given before the result of echocardiography was available. However, the drug could have been justified anyway as an excellent addition to the diuretic to treat hypertension. The only slight risk might have been that the systolic murmur was aortic stenosis; but the pansystolic nature of the murmur and the lack of left ventricular hypertrophy on electrocardiography would make that a risk probably worth taking.

The left ventricular ejection fraction was 44%, neither entirely normal (>50%), nor entirely abnormal (<40%). Even if this finding did represent mild systolic dysfunction, it would be far too mild to produce such large pleural effusions. A good diagnostician also relies on proportionality—that is, is the detected abnormality likely to cause the severity of symptoms or signs found or are the diagnostic test results disproportionate to the clinical picture?

In view of all the atypical features, it was sensible to aspirate pleural fluid to see if it was a transudate or an exudate. Indeed, you could argue that all pleural effusions should be aspirated. Even if the echocardiogram shows left ventricular systolic dysfunction, pleural effusion has many other causes (such as bron-

chial carcinoma, particularly in a smoker) that might coexist with heart failure. I remember being caught out many years ago by a patient with heart failure who eventually turned out also to have a mesothelioma. On the other hand, there is always a small risk of producing a pneumothorax when aspirating pleural fluid, so it should not be done if a good explanation already exists for the effusion. I ask the junior staff to obtain an aspirate only if the fluid is aspirated easily and clearly; they should not poke around for it and increase the chance of a pneumothorax.

This case confirms echocardiography as the standard for diagnosing heart failure. In an ideal world, this would be available quickly. However, in the real world, the lack of availability of echocardiography means that it is often necessary to start treatment before the diagnosis is confirmed. Furosemide is thought of as safe enough to be used even if the eventual diagnosis is different. But it often ruins the quality of life of patients, forcing them to rush to the toilet for a lot of the day. Causing unpleasant urgency in this way seems even more perverse when, as in this case, it adds to their original symptoms and eventually turns out to be unnecessary. This is one of many arguments for increasing the availability of echocardiography in the United Kingdom.

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Patient’s initial 12 lead electrocardiogram

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