To the Editor:

I applaud the efforts of Sartori et al in their prospective evaluation of bleomycin vs intrapleural interferon in patients with malignant pleural effusion. It is evident that their preliminary results are in keeping with those reported in the literature. In reviewing multiple treatment modalities for malignant pleural effusion, I have found that one common denominator in successful treatment is complete evacuation of the pleural space before definitive treatment. In that regard I feel that the 100% success I experienced in my limited group of 15 patients is because of the more aggressive thoracic drainage used before the high-dose interferon was instilled. In a personal communication with Dr. Muss in Wake Forest, NC, it was agreed that this may have been in part a cause for the limited success that he found in his excellently conducted phase II trial with intrapleural interferon.

I will also briefly comment on the mechanism of action of bleomycin vs interferon. Although it is true that bleomycin has recently been recommended as the sclerosing drug of choice and also has been recommended for the management of malignant pericardial effusion, it should be pointed out that the primary mechanism of action, as discussed in my paper, is not one of sclerosing of the visceral and parietal surfaces. The effect of interferon appears to be related to a direct antineoplastic effect of active tumor cells within the pleural space.2 It is my impression that because there is no actual sclerosing of the surfaces, it is more imperative to drain maximally and thoroughly the pleural space of the malignant effusion before maximal high-dose interferon administration for it to be effective and achieve maximal benefit. This may in part explain why the results in this preliminary study show bleomycin to have an apparent advantage over interferon for treatment of malignant pleural effusion. One other point that I add to this communication is that in an unpublished report by our group at Columbus Hospital, we have had anecdotal success in treating malignant pericardial effusion by employing a similar method of minimal drainage of a malignant pericardial effusion by high-dose interferon before drain removal.

I add my opinion to that of other authors that widening the study to include a further arm with a dosage and timing similar to that in my study will be of value. I further add that if this prospective study is done, it will be interesting to include patients with malignant pericardial effusions as well.

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REFERENCES

A Case of Systemic Lupus Erythematous or Hepatitis C Virus?

To the Editor:

I read with great interest the case report by Susanto and Peters (June 1997)1 and offer the following comments:

Is it possible that the patient was not affected by systemic lupus erythematosus? The serositis was not confirmed by echocardiogram and CT scan of the chest. The pancytopenia may be associated with chronic hepatitis C virus (HCV) infection (hypoplastic and myelodysplastic syndrome)2 and/or with hypersplenism. The antinuclear antibody test is nonspecific, and the rash may be associated with hepatic dysfunction and chronic HCV infection (porphyria cutanea tarda, lichen planus, and other cutaneous lesions).3

The interstitial lung inflammation may be due to idiopathic pulmonary fibrosis and/or essential mixed cryoglobulinemia associated with HCV infection.4

The hypoxemia may be explained by the presence of hepatopulmonary syndrome associated with HCV-related interstitial inflammation.5

In summary, I believe that the patient’s clinical manifestations can be ascribed to chronic HCV infection and cirrhosis.

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REFERENCES
1 Susanto I, Peters JI. Acute lupus pneumonitis with normal chest radiograph. Chest 1997; 111:1781-83

To the Editor:

We appreciate the opportunity to respond to the comments of Dr. Marvisi regarding our case report.1

Dr. Marvisi suggested our patient may not actually have systemic lupus erythematosus (SLE). The patient had at least four of the American College of Rheumatology criteria for SLE diagnosis (serositis, leukopenia/lymphopenia, antinuclear antibody, malar rash).2 In addition, she also had a moderate titer of anticitrulinin antibody in lieu of a false-positive serologic test for syphilis, reflecting the presence of an immunologic disorder (antineoplastic antibody) as the fifth criteria.

Pleuritic chest pain was one of her chief presenting complaints. Lupus pleuritis may occur without pleural effusion.2,3 She had documented normal leukocyte and lymphocyte counts on various occasions prior to and following the pneumonitis, despite a longstanding diagnosis of uncomplicated cirrhosis. Antinuclear antibody is present in 95% of patients with active and untreated SLE and is often present in high titers (1:2,560 in our patient).4 Our patient had a history of cryoglobulinemia-like malar rash without other skin manifestations. Porphyria cutanea tarda (PCT) is often associated with facial blisters and hypertrichosis, hyperpigmentation, and sclerodermaic changes.5 None of which was noted in our patient. She also had no other clinical findings compatible with PCT. Although there is an association between hepatitis C virus and mixed cryoglobulinemia (MC),6 the cutaneous findings of MC include purpura, hyperpigmentation, infarction, and hemorrhagic crusts and ulcers.7 Our patient had none of these cutaneous findings. We are aware of possible alternative expla-