transferred to the intermediate ICU with a diagnosis of presumed sepsis. He required dopamine for only 5 h. An ECG performed for a complaint of chest pain that morning showed ST-segment elevation in leads 1, aVL, V5, and V6, with a smaller degree of elevation in lead 2. Cardiology consultation was obtained. Subsequent ECGs performed over the following days showed a return of the ST segments to baseline, with nonspecific ST- and T-wave changes. Serial cardiac enzymes were as follows:

Based on the elevated creatinine kinase (CK) and ECG changes, the patient was transferred to the ICU with the diagnosis of acute myocardial infarction. He was started on heparin and nitrates. Clinical examination was consistent with congestive heart failure, and captopril was added for afterload reduction. An echocardiogram showed a mildly dilated left ventricle, diffuse LV hypokinesis, and a moderately depressed LV ejection fraction. The right ventricle was poorly seen, but mild dilation and depressed function was suspected. A left pleural effusion was identified as well. On the eighth hospital day, a cardiac catheterization was performed and was normal. The patient was discharged after 10 days in the hospital in stable condition, to be followed up by cardiology as an outpatient.

This patient would appear to conform quite closely to the clinical and laboratory picture which characterized the patients in the report by Blum et al.\(^1\) The only departure in this particular case is the initial presence of global, rather than regional wall motion abnormalities and depressed LV (and perhaps RV) function on echocardiogram. This may be explained by the fact that our patient appeared to be truly septic before the onset of the cardiac event. Therefore, sepsis-associated myocardial depression may have been superimposed on dysfunction due to ischemia/infarction.

Our patient did quite well with conservative management and never received thrombolytic therapy. We agree that it would be most useful to obtain angiographic data prospectively in future patients who appear to have infective myocardial infarction, before the institution of thrombolysis, and we would suggest that in young patients without obvious coronary risk factors who present with a similar picture, that approach be taken unless clinically unfeasible at the time. It would also seem reasonable to perform endomyocardial biopsy at the same time to rule out the possibility of myocarditis, especially if no evidence of thrombosis is apparent on the angiogram.

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REFERENCE

To the Editor:

The patient presented here had a global ischemia, perhaps reflecting true classical myocarditis with left ventricular (LV) depression. At a later date, when he developed the acute myocardial infarction pattern, an echocardiogram was not performed and may be in this stage, the myocardium would have a regional contraction defect as our patients. The enigma is to differentiate between true classical myocarditis and acute myocardial infarction when there is a global contraction defect. Therefore, the case presented here is not quite typical of that “syndrome.”

It is well-known that infection and especially sepsis cause stimulation of the immunologic system by monocyte-liberating cytokines such as tumor necrosis factor (TNF), interleukin (IL)-1B, IL-6, and IL-8. The IL-1, for example, directly and indirectly activates platelets and causes them to be in “a hypercoagulable state,” to secrete platelet-aggregating factor and other growth factors that activate the clotting proteins and the “clotting cascade” through Von Willebrand’s factor, fibrinogen, fibrinectin, and other adhesive molecules.\(^1\)\(^2\)

The global, rather than regional wall motion abnormality and depressed LV function, can be caused by cytokines that are secreted by monocytes and lymphocytes during sepsis and cause myocardial depression, for example, TNF and IL-6 are secreted from monocytes and macrophages, and IL-2 is secreted from T-lymphocytes and natural killer cells.\(^3\)

REFERENCES

Pulmonary Vascular Lesions in Chronic Thromboembolic Pulmonary Hypertension

To the Editor:

In the March issue of Chest, Moser and Bloor claim that plexogenic (sic) lesions occur in the small pulmonary arteries of patients with chronic thromboembolic pulmonary hypertension.\(^1\) Figures 4 and 5 in their article illustrate, however, organized and recanalized thrombi rather than plexiform lesions as stated in the respective legends.

Plexiform lesions occur in lateral branches of muscular pulmonary arteries measuring between 100- to 150-μm diameter, shortly after their origin from larger muscular pulmonary arteries. They are slightly more common in supernumerary arteries than in symmetrical regular dichotomous branches.\(^2\) The typical plexiform lesion has a striking appearance and has two basic elements. The first consists of a circumferential dilatation of a pulmonary artery branch close to its origin from a parent artery. In this dilated segment, there is destruction of the arterial wall with pronounced thinning of the media, loss of smooth muscle cells, disappearance of the internal elastic lamina, and sometimes also the external elastic lamina.\(^3\) The second element is a plexus of narrow slit-like channels within the dilated segment. These channels which are lined by cells with hyperchromatic nuclei resemble a vascular plexus. The distal part of the plexiform lesion

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