agree that such early onset of the disease is rare and, like you, we think that some cases require in-hospital monitoring at the beginning of amiodarone therapy.

However, we would like to point out that an early variant of amiodarone pulmonary toxicity (albeit not quite as early as in the case we described) can also be seen in patients receiving amiodarone doses as low as 100 mg daily. In these cases, reducing the dose would not prevent the occurrence of this complication.

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Urine Thrombomodulin in Patients With Idiopathic Pulmonary Fibrosis

To the Editor:

Thrombomodulin (TM) is a transmembrane glycoprotein that is released into the blood when vascular endothelial cells are injured. In some collagen vascular diseases, the serum level of TM increases in parallel with their clinical activity.1,2 Idiopathic pulmonary fibrosis (IPF) is characterized by cellular infiltration into the lung interstitium which results in fibrosis of the lungs. These histologic findings of IPF are shared with the pulmonary involvement of collagen diseases. Therefore, in the present study, we measured TM in patients with IPF, and assessed the possibility of using TM as a clinical marker for the disease.

Twenty-two patients with IPF (mean±SD 69.2±8.9 years old) were enrolled in the study, as well as 14 age-matched patients with pulmonary emphysema (68.4±9.0 years old) and 22 normal control subjects (70.5±3.7 years old). Renal function in all the patients was in the normal range. Blood and urine samples were collected between 9 AM and 11 AM. The concentration of TM was measured by a one-step sandwich enzyme immunoassay using a monoclonal antibody, and the value was expressed as U/mL for blood samples and U/mg of creatinine for urine samples according to the methods of Ishii et al.3

There were no differences in serum levels of TM among patients with IPF (21.0±6.8 U/mL), emphysema (21.9±6.5), and normal control subjects (25.2±4.4). However, the urine specimens showed significant elevation of TM in the IPF patients (59.7±21.8 U/mg creatinine) compared with those with emphysema (36.3±12.7) and the normal control subjects (36.5±13.1) (r<0.0001, Wilcoxon rank-sum test, Fig 1).

In a few patients with IPF, TM was repeatedly measured; in one patient whose condition was deteriorating, urinary TM increased from 20.5 (December 12, 1994) to 52.8 (April 19, 1995), then to 75.8 (June 2, 1995), and finally 82.7 U/mg creatinine (June 30, 1995). He died of IPF thereafter. It is noteworthy that his serum TM level was below 20 U/mL throughout the course.

In conclusion, TM was significantly high in urine, but not in serum in patients with IPF. Although the underlying process of the increase of urinary TM should be elucidated in future studies, some kind of concentration of TM in the urine might be postulated.

As the process of IPF is not fully understood, it is not easy to evaluate the clinical activity of the disease, and indicators conventionally used for this purpose, such as erythrocyte sedimentation rate or serum lactate dehydrogenase,4 are unsatisfactory in

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their sensitivity or specificity. Therefore, it might be worthwhile to further assess the possibility of TM as a marker for IPF.

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Figure 1. Concentrations of urinary TM in patients with IPF, pulmonary emphysema, and in normal control subjects. Each perpendicular line reveals the mean and standard deviation.

REFERENCES


Airway Obstruction With Percutaneous Tracheostomy

To the Editor:

I read with interest and some dismay the “Communication to the Editor” by Sakabu and colleagues (May 1997).1 They reported in detail an unusual and dangerous complication of