Alveolar Hemorrhage Following Tirofiban Treatment. A Misleading Diagnosis

To the Editor,

We read with great interest the recent case report by Agnelli et al related with alveolar hemorrhage following tirofiban treatment. Although large clinical trials have demonstrated the clinical effectiveness of therapy with glycoprotein IIb-IIIa inhibitors in patients with ischemic heart disease, combination therapy with aspirin, clopidogrel, and heparin can be associated with bleeding complications. Although major and minor bleeding complications are the most frequent adverse events associated with the use of glycoprotein IIb-IIIa inhibitors, alveolar hemorrhage, as a potentially fatal complication, is often under-recognized. On the basis of the low number of cases reported in the literature, we can conclude that clinical suspicion is required to diagnose this life-threatening complication. The number of patients in whom alveolar hemorrhage might have been misdiagnosed is unknown. Clinical findings in addition to a sudden fall in hemoglobin with hemoptysis following administration of glycoprotein IIb-IIIa inhibitors should alert physicians to the likelihood of this severe complication. In the literature, Ali et al were the first to report a case of pulmonary hemorrhage following tirofiban use and the second case was reported by Yilmaz et al. Although in previous studies, no association was clearly defined between the presence of underlying lung disease and the development of alveolar hemorrhage, Yilmaz
et al. reported that their patient had elevated pulmonary capillary wedge pressure at the time of catheterization. In addition, Sitges et al. reported a case of pulmonary hemorrhage following abciximab use who had radiographic evidence of pulmonary congestion before administration. This probable correlation was also supported by Khanlou et al., who reported 6 patients with pulmonary hemorrhage following abciximab use, 5 of whom had elevated pulmonary capillary wedge pressures. Although the small number precluded any definitive comments, pulmonary congestion or elevated pressures in the pulmonary bed in patients who receive glycoprotein IIb-IIIa inhibitors may predispose patients to alveolar hemorrhage. Since, the radiographic features of diffuse alveolar hemorrhage strikingly resemble cardiogenic pulmonary congestion, we think that before administration of these agents, a radiographic examination may help us to differentiate the high risk group for this life-threatening complication.

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REFERENCES