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Pulmonary Embolism and L-Asparaginase Therapy

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In a recent article Barbui et al. [1983] describe 2 patients with acute lymphoblastic leukemia who died from pulmonary embolism following L-asparaginase (L-ASE) treatment. Thromboembolic manifestations have been described following L-ASE therapy with a significant decrease of antithrombin III by other authors [Pitney, 1980; Conard, 1980; Vellenga, 1980; Turn, 1981].

We report a case of a 16-year-old female patient who had acute lymphoblastic leukemia (L3FAB) when she was 13 years old [Majolino, 1983]; CR was obtained by a protocol with L-ASE (40,000 U twice weekly in 4 cycles in September 1980) in consolidation. In July 1983 the patient was admitted to our Hospital with cough, dyspnea, hemoptysis, chest pain and severe right failure. Pulmonary radiography, scintigraphy and blood gas analysis were consistent with pulmonary embolism; the ECG showed a right ventricular hypertrophy (not seen 6 months before). The angiography yielded deep right iliac thrombosis. Bone marrow aspiration still showed CR; coagulation tests including antithrombin III were normal; no family history of thrombosis was present and no use of the pill.

Despite treatment (heparin-urokinase) after a transient recovery, also radiographic, the patient had a fatal relapse of pulmonary embolism.

Critical review of the clinical history showed in 1981 and in 1982 two acute pulmonary episodes, explained as infectious, of which – a posteriori – we cannot exclude the thromboembolic genesis. We wonder if, in the absence of obvious thrombogenic factors in our patient, the L-ASE besides acute thromboembolic incidence might be responsible for persistent deep venous thrombosis; the cause of repeated embolic episodes.

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