

PERICARDITIS DURING INTERFERON- α THERAPY IN CHRONIC MYELOGENOUS LEUKEMIA

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Sir,
interferon alpha (IFN- α) can induce molecular remission in chronic myelogenous leukemia (CML).¹ This may indicate that it could be opportune to prolong IFN treatment for a long time; however, albeit rarely, this therapeutic strategy can produce unusual complications.² In this regard, we believe it useful to report the development of pericardial effusion in a 28-year-old Ph-positive CML patient during therapy with IFN- α alone. To the best of our knowledge, only 3 such cases have been reported in the literature: 2 were pericardial effusions associated with pleural effusion³ and the third was an isolated pericarditis for which it was necessary to interrupt the IFN- α treatment.⁴

In December 1992 our patient was found to be suffering from stage I CML on the basis of the *synthesis model*.⁵ Therapy with IFN- α (9×10^6 U daily) produced a complete hematological response (CHR) after 3 months and a major cytogenetic response (Ph, 6%) after 10 months.

In March 1994 after 13 months of IFN- α therapy, the patient complained of severe, aching, substernal pain that persisted for two days. Clinical investigations showed an increase in ESR (35 mm at 1st hr) and echocardiographic evidence of modest pericardial effusion. Discontinuation of IFN- α and the subsequent administration of prednisone (50 mg daily) for about 40 days led to rapid regression of the pericarditis, so that in May 1994 IFN- α therapy was restarted at the same dosage as before.

Seven months after resumption of IFN- α therapy, and against a background of CHR, the patient suffered a pericarditis relapse. NSAID therapy, administered for 2 months, led to rapid and complete regression of the pericardial effu-

sion, but IFN- α was withheld for an additional 9 months. During this period, the lack of an HLA donor and the persistence of a major cytogenetic response (Ph, 9%) induced us to explant and freeze a sample of the patient's bone marrow.

Six months ago the patient recommenced IFN- α therapy and at present persists in CHR and in partial cytogenetic response (Ph, 6%).

Despite the presence in the patient's clinical history of allergic asthma since infancy, we believe it improbable that an immune-mediated mechanism is responsible for the pericarditis. This assertion is based on both the rapid regression of the pericarditis and on the persistent negativity of autoimmune disorder indicative testing (even at the subclinical level); of particular note in this respect was a lack of connective tissue disorders.

Our experience demonstrates that life-threatening complications such as pericarditis do not necessarily require definitive interruption of IFN- α therapy.

References

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