ALPHA-INTERFERON IN A CASE OF HYPEREOSINOPHILIC SYNDROME

We have read with interest the report of Murphy *et al* (1990) on the use of alpha-interferon (α-IFN) in a case of hypereosinophilic syndrome (HES). Their patient with severe HES showed a dramatic clinical and haematological improvement with the introduction of recombinant α-IFN. However, follow-up was only 3 months.

We also treated a patient with HES using α-IFN (Essex). This led to a good response. However, in a longer follow-up we observed that the response lasted only 6 months.

In a 43-year-old man HES was diagnosed in October 1987 according to the diagnostic criteria of HES as outlined by Chusid *et al* (1975). His initial evaluation revealed a white blood count (WBC) of 113×10^9/l with 90% eosinophils, 1% polymorphonuclear leucocytes, 8% lymphocytes and 1% monocytes. The bone marrow examination showed hypercellular marrow with a marked predominance of eosinophilic precursors (77%). There was no excess of blast cells. The neutrophil alkaline phosphatase score and karyotype were normal. His spleen was enlarged and in the lungs bilateral transient infiltrations were found. Renal function was decreased. An echocardiogram revealed a restrictive cardiomyopathy with pulmonal hypertension and tricuspid-regurgitation. The patient had no HLA-identical sibling donor. He was initially treated with hydroxyurea and prednisone, without further clinical or haematological improvement. As his condition worsened, IFN was commenced in October 1988. He received 5×10^6 units subcutaneously daily. In the first 4 months of IFN therapy the WBC decreased from 120 to $10{\cdot}6 \times 10^9$/l with 90% eosinophils and his condition improved dramatically. The congestive heart failure was controlled by diuretics and afterload reduction. There was stable disease until March 1989. Then IFN was stopped because of progression of the HES. Hydroxyurea was started again, but the WBC rose to 126×10^9/l. He developed progressive congestive heart failure due to thickening of the mural endocardium of the right ventricle and tricuspid valve destruction. Endomyocardial biopsy showed marked fibrosis in the subendocardium. Despite continued activity of the underlying disease, he had cardiac surgery in February 1990. The mitral and tricuspid valves were replaced and thrombotic masses were removed from the right ventricle. His clinical condition improved dramatically after the surgery. Unexpectedly he died of cardiorespiratory failure in April 1990.

HES is an extremely rare disease. Our experience shows that IFN has a place in the treatment of HES and we think it has to be given early in the treatment. However, the haematological response may only be short, as in our patient, although every response may postpone the tissue damage which makes this disease fatal.

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APLASTIC PANCYTOPENIA INSTEAD OF THE MISNOMER APLASTIC ANAEMIA

In his *Blood, Textbook of Hematology* of 1987 Jandl writes on p. 113: 'Pancytopenia arising from inclusive failure of marrow parenchymal elements is customarily termed aplastic anemia or hypoplastic anemia. This confusing convention has generated alternative expressions such as chronic bone marrow failure, but in hematologic parlance aplastic anemia is the accepted and predominant designation for pancytopenia arising from impaired activity of pluripotential stem cells.'

I am sure that most haematologists agree with Jandl that it would have been less confusing if the condition characterized by pancytopenia due to lack of progenitors for each of the three cell lines in the bone marrow had been named aplastic pancytopenia rather than the misnomer aplastic anaemia.

Additional confusion is caused by the International Classification of Diseases which uses pancytopenia as a synonym for aplastic anaemia. This is unfortunate as pancytopenia today in most textbooks and haematological usage means lowered numbers of all three cell lines in the blood, regardless of cause or mechanism.

Although haematologists would welcome a change from aplastic anaemia to the logical term aplastic pancytopenia, most of them doubt that it can be achieved. A decision by the *British Journal of Haematology* to replace the misnomer aplastic anaemia in its columns with aplastic pancytopenia, might give the necessary push to start the changing avalanche. Pancytopenia unqualified could then without risk of confusion be used to describe lowered numbers of all three cell lines in the blood, regardless of cause.

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