

Bronchial cancer and hypereosinophilia

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ABSTRACT: A 73 yr old man with a marked eosinophilia, associated with generalized bronchial carcinoma was observed. Ten months earlier his blood count was normal. The mechanism of the eosinophilia is discussed.
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Whilst rare, the relationship between cancer and eosinophilia has been established in haematological and lymphatic cancers, but is less frequent in solid cancers. The tumours most often associated with blood eosinophilia are usually glandular-type carcinomas: uterus, breast, thyroid, adrenal, bile canal, pancreas, and especially the colon and rectum [1-2]. Regardless of its histological classification, bronchial cancer is only exceptionally accompanied by blood eosinophilia [5].

Case report

The 73 yr old man, a smoker and retired firefighter, was examined at the end of April 1986 for an unproductive cough associated with weight loss for two months. The clinical examination was normal.

Chest X-ray (fig. 1) and (CT) scan showed a tumorous opacity anteriorly in the right upper lobe and marked right hilar enlargement. Fiberscopy showed compression of the intermediate trunk of the middle lobar bronchus. Aspiration from the anterior segment of the right upper lobe collected squamous bronchial carcinoma cells, with few eosinophils. The abdominal CT scan showed hepatic metastases. Fine needle puncture of a hepatic metastasis confirmed their squamous carcinoma origin.

On April 28, 1986, laboratory tests showed a white blood corpuscle (WBC) count of $27,500 \text{ WBC/mm}^3$, with 35% eosinophils ($10,101/\text{mm}^3$), no anaemia, and a platelet count of $440,000/\text{mm}^3$. No parasitic infestation or drug allergy was found. On May 27, 1986 the WBC count was $51,500 \text{ WBC/mm}^3$ with 42% eosinophils ($21,630/\text{mm}^3$). On May 29, 1986, sternal puncture showed normal marrow, except for a large increase in eosinophil count, raised to 32%. No neoplastic cells were detected.

Ten months earlier, in June 1985, the patient had a cholecystectomy. At this time the eosinophil count was $131/\text{mm}^3$ corresponding to 1% of a WBC count of $10,300 \text{ WBC/mm}^3$.

The patient returned home, at the beginning of June 1986. His condition rapidly worsened, with heart

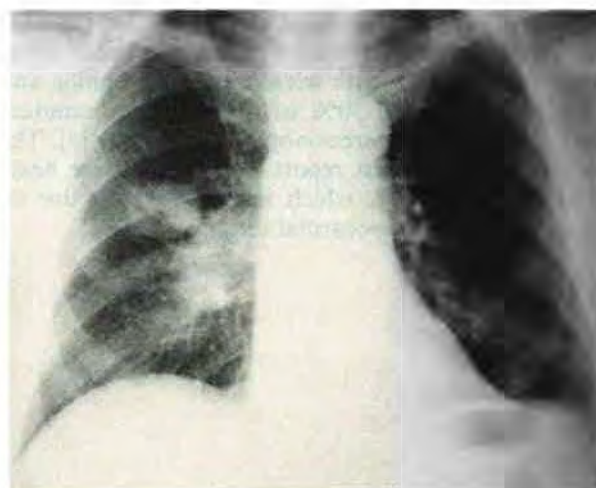


Fig. 1. Chest film showing tumour anteriorly in right upper lobe and marked hilar enlargement.

failure and widespread oedema, resulting in death in early August 1986. Autopsy was not performed.

Discussion

In 1983, SLUNGAARD *et al.* [7] isolated a 45,000 dalton molecular weight glycoprotein from a lung tumour with eosinophilia. This glycoprotein was a powerful eosinophil stimulator in cultured, normal human marrow. It was also detected in the patient's serum, at a lower concentration but with eosinopoietic activity. It seems to be responsible for blood and marrow hypereosinophilia in this syndrome. These authors were not able to isolate the cell type within the tumour responsible for synthesis of the factor which induces eosinophil overproduction in bone marrow. The physical characteristics of this active principle differ from those of the eosinophilopoietin isolated by MAHMOUD *et al.* [4] from mice with induced eosinopenia, which seems to be the mediator of eosinophilic stimulation in parasite infections.

In 1974, WASSERMANN *et al.* [9] extracted, from a large-cell anaplastic carcinoma of the lung, a chemo-

tactic substance for eosinophils, with the same physicochemical action as the eosinophilic chemotactic factor of anaphylaxis (ECFA). Eosinophilia was in both the blood and the tumour.

In most of the cases described, eosinophilia is essentially located in the blood and marrow.

High doses of prednisolone caused a transient decrement in the eosinophil count of eosinophilia associated with lung carcinoma [4-7].

Most of these patients rapidly develop endomyocardial fibrosis which contributes to death as the diseases spread [8].

If eosinophils with their peroxidase and macrophages cooperate in exerting cytotoxicity for tumour cells [6], the endomyocardial effects are due to the toxicity of the mediators liberated by eosinophils, especially the eosinophil cationic protein, and cytotoxic superoxide effects. Moreover, most of the eosinophils are degranulated and vacuolated [3]. This eosinophilic endomyocardial disease has been recognized in parasitosis with widespread eosinophilia and develops in around 30% of patients with tumours that induce this hypereosinophilic syndrome [8]. The patient in the present report developed severe heart failure and oedema, which may have been due to eosinophilic endomyocardial disease.

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RÉSUMÉ: Il s'agit de l'observation d'un patient de 73 ans, qui présente une importante hyperéosinophilie, associée à un cancer bronchique en généralisation. La formule sanguine était normale dix mois auparavant. La pathogénie de cette hyperéosinophilie est discutée.