Malignant islet-cell tumour of the pancreas presenting with non-bacterial thrombotic endocarditis and eosinophilia

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Summary  
A case of severe persistent eosinophilia and non-bacterial thrombotic endocarditis complicating a malignant islet-cell tumour of the pancreas is documented. Although these are well recognized complications of malignant tumours, they do not appear to have been previously documented from the same patient. These complications probably reflect ectopic synthesis and are unlikely to be causally interrelated.

Introduction  
Non-bacterial thrombotic endocarditis (NBTE) (Leading Article, 1978) and eosinophilia (Isaacson and Rapaport, 1946) have been well documented with a variety of malignant tumours. The authors discuss the simultaneous occurrence of both conditions in a patient with an occult malignant islet cell tumour of the pancreas.

Case report and investigations  
A 56-year-old man was admitted with a 3-week history of vague pains in the right iliac fossa, left subcostal area and epigastrium, associated with weight loss of 3 kg over 2 months. Examination was normal apart from one finger-breadth hepatomegaly. Over the next 12 days he complained of flitting bone pains and lethargy which were unexplained. During the following 6 days he developed signs of severe progressive congestive cardiac failure and renal failure followed by coma associated with bilateral upper motor neuron lesions of his limbs. Despite treatment with therapeutic dosage of digoxin, diuretics and hydrocortisone, he died within 3 weeks of presentation.

Investigation revealed a persistent eosinophilia varying from 41·5·10^4/l to 99·6·10^9/l (total WCC 62·10^9/l–118·6·10^9/l). His Hb was 15·9 g/dl with normal red cells and a platelet count which was initially 300·10^9/l fell to 65·10^9/l with normal fibrinogen degradation products before death. Blood biochemistry was normal apart from an alkaline phosphatase of 258 i.u./l (normal range 35–150 i.u./l). Bone marrow aspiration showed marked eosinophilic hyperplasia with a normal karyotype (46 xy), no Philadelphia chromosome and no evidence of lymphoma. Neutrophil alkaline phosphatase was normal. Liver biopsy revealed a conspicuous number of eosinophils in the lobular sinusoids and portal tracts but no evidence of malignancy. Stool for occult blood, ova and parasites was negative on 3 occasions. Muscle biopsy showed non-specific foci of perimysial inflammation consisting largely of eosinophils with no evidence of muscle degeneration, vascular involvement or parasitic infection. A bone scan and CSF examination were normal. Chest radiography which was normal on admission showed a diffuse alveolar infiltration with a normal heart size and contour before death. Serial ECGs were consistent with an old anterior myocardial infarct. Isotope scanning of the liver was deferred owing to the rapid clinical deterioration.

Post-mortem findings revealed a malignant islet cell tumour of the pancreas, surrounded by an infiltration of eosinophils with multiple hepatic metastases; thrombotic endocarditis with non-infective vegetations on the anterior mitral cusps and circumferential sub-endocardial infarction with organizing mural thrombus almost filling the cavity of both ventricles, minimal non-occlusive atheroma of the coronary arteries and no coronary emboli; multiple areas of infarction in both cerebral hemispheres and the spleen; renal tubular necrosis and uraemic lung.

Discussion  
This case report highlights the recognized difficulty (Studdy and Willoughby, 1976) in diagnosing NBTE which on this occasion typically complicated an occult metastatic carcinoma. In this case NBTE
presented with a cerebral embolus in a patient who was being investigated for the cause of his eosinophilia and hepatomegaly. Eosinophilia when complicating carcinoma is seldom of the order of severity documented in this patient. The clinical progression of cardiac failure and cerebral episodes in a patient with a 'hypereosinophilia' of unknown cause was consistent with a 'hypereosinophilic syndrome' (Parillo et al., 1979).

Eosinophilia has been shown to remit following resection of tumours and has been attributed to ectopically secreted eosinophil chemotactic factor (ECF) (Wasserman et al., 1974). In this context it is of interest that this pancreatic tumour was surrounded by eosinophils rather than the usual polymorph reaction. NBTE has also been documented with secretory tumours, notably mucin secreting adenocarcinomas, and may likewise be improved following tumour resection suggesting the production of a thrombogenic substance analogous to ECF.

As these 2 complications of malignancy do not seem to have been previously documented together it would appear that they are not causally related in this case but are possible markers of ectopic synthesis by the tumour tissue.

References
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