
**Tumour Markers in Dermatomyositis: Useful or Useless?**

Sir—The association of dermatomyositis (DM) with malignancy has been recognized for many years, although the frequency of the association is debated to range from 15 to 50% [1, 2]. The value of tumour markers in the detection of cancer is controversial, and recent guidelines [3] suggest that they are not recommended as a screening test for many common tumours. However, there are a number of reports relating particular markers to tumours associated with DM [4–6].

We report the case of a 36-yr-old woman who first presented with classical DM in June 1996, with a facial macular rash, Gottron’s papules, a proximal myopathy, minimally elevated creatinine kinase (CK) at 234 IU/l (normal range 25–170), and typical features on both skin and muscle biopsies. She was a lifelong smoker. At presentation, a routine screen for malignancy was unremarkable, including normal chest X-ray and mammography.

She responded well to combination therapy with prednisolone and methotrexate. Seven months after the initial presentation, she had to increase both drugs because of a relapse of the facial rash. This initially resolved, but she relapsed 4 months later. During that time, the tumour marker carcinoembryonic antigen (CEA; associated primarily with colorectal carcinoma, but also elevated in tumours of lung, breast and pancreas) was being measured by her general practitioner (GP), unbeknown to the rheumatology service. A small elevation 6 months after presentation (8.8 mg/ml, normal <5) was significantly higher 5 months later (57 mg/ml). The trend was confirmed on a further sample 2 months later (167 mg/ml), at which time we were informed (in retrospect, rather too late). Subsequent re-assessment revealed right upper lobe bronchogenic carcinoma with mediastinal nodal involvement.

Those with DM difficult to keep in remission, such as our case, are thought to have a higher incidence of associated malignancy. In addition, a normal CK level (near normal in our patient) has been identified as a poor prognostic sign [7]. While there is broad consensus among oncologists that no tumour marker is sufficiently sensitive or specific for screening purposes, Bayes’s theorem demonstrates that in a population with a higher incidence (prior probability) of a disease (malignancy), the usefulness of a test increases (see nomogram in [8]). Our case highlights the identification of an asymptomatic lesion in a patient at risk, due to monitoring (some would say overenthusiastic!) by her GP. We suggest that tumour markers may be of value in detecting disease at an earlier, treatable stage in such patients, particularly should they relapse. Our case rests.

D. O’Gradaigh, P. Merry
Department of Rheumatology, Norfolk & Norwich Hospital, Norwich NRI 3SR
Accepted 9 April 1998
Correspondence to: D. O’Gradaigh.


**Serious Opportunistic Infection Associated with Gold-induced Panhypogammaglobulinaemia**

Sir—A 59-yr-old woman diagnosed as having seronegative rheumatoid arthritis in 1995 was initially treated with prednisolone 10 mg daily and sulphasalazine 2 g daily. A satisfactory initial clinical response was not maintained and, after 9 months, treatment with i.m. gold (myocrisin) was commenced. After a total dose of 600 mg of gold, at a time when joint symptoms were improving, she developed multiple painful pustules on the fingers and forearms which responded to oral fluclucloxacin 500 mg q.d.s. One week later, she complained of severe breathlessness, right-sided pleuritic chest pain and rigors, and was admitted as an emergency. On examination, she was pyrexial (39°C), tachycardic and cyanosed with fine, late inspiratory crackles audible in both lung fields.

Initial investigations were as follows: haemoglobin...