The histopathological findings in our patient showed an infiltrate of chronic inflammatory cells and granulomatous changes in the dermis. Granulomatous changes are also seen in *Candida* infection, such as chronic mucocutaneous candidiasis, cheilocandidosis, and juvenile juxtavermilion candidosis.[6]

It is well known that lingua plicata can be involved in the aetiology of oral candidiasis, in the form of median rhomboid glossitis.[7] Our patient also showed furring, containing *Candida*, around the fissure on the tongue. Both the granulomatous cheilitis and the oral candidiasis in this case resolved after treatment with the anti-*Candida* preparation amphotericin B syrup, and both recurred when the treatment was stopped. The granulomatous histology in our patient was similar to that seen in the above-mentioned forms of *Candida* cheilitis, and the response to treatment suggests that secondary oral candidiasis arose from the lingua plicata and was responsible for the granulomatous cheilitis.

Despite an extensive literature search we have been unable to find any report mentioning a relationship between oral candidiasis due to lingua plicata and MRS. The findings in our patient suggest the possibility that chronic oral candidiasis due to lingua plicata could be a cause of granulomatous cheilitis in MRS. Oral candidiasis should be considered in cases of MRS with lingua plicata.

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Polymyositis and malignant melanoma

SIR, Polymyositis and dermatomyositis are inflammatory myopathies characterized by subacute symmetrical weakness of proximal limb muscles and trunk muscles. Dermatomyositis is distinguished from polymyositis by the presence of a rash.[1,2] Their aetiology is unknown, but immunological mechanisms are thought to be involved in the pathogenesis of both conditions.[3,4] Although the first case of dermatomyositis associated with an underlying malignancy was reported almost 80 years ago,[5] debate still continues about the link between dermatomyositis or polymyositis and malignancy.[6] Dermatomyositis has only rarely been described in association with malignant melanoma, and we describe what is, to our knowledge, the first reported case of polymyositis occurring in association with malignant melanoma.

A 71-year-old woman presented in February 1994 with a 3-week history of progressive proximal muscle weakness. She had difficulty in rising from a chair, climbing stairs, and reaching to take objects from shelves. She also had dysphagia for solids, with occasional regurgitation, and suffered from exertional dyspnoea. A 1·79-mm-thick, Clark level 4 nodular malignant melanoma had been excised from her left calf in 1990. There was no evidence of local or systemic spread until November 1993, when lymph nodes containing metastatic tumour were excised from the left inguinal region.

Examination revealed induration of the skin in the left groin and on the left thigh, and mild lymphoedema of the left leg. There was symmetrical proximal weakness in the legs (MRC grade 3/5), and less marked weakness of the trunk and neck muscles (MRC grade 4/5). Neurological examination was normal. There was no rash.

Haematological indices, plasma viscosity, urea, creatinine, sodium, potassium, calcium, phosphate and thyroid stimulating hormone were all normal. The aspartate transaminase was 135 U/l (normal range 6–35 U/l) and creatinine kinase was 2795 U/l (normal range 24–170 U/l). Nerve conduction studies were normal. Electromyography showed fibrillation potentials in the quadriceps muscles, and there were low amplitude units, many of which were polyphasic, particularly in the biceps and quadriceps, and to a lesser extent in the triceps. Histology of a muscle biopsy showed numerous necrotic and regenerating fibres, with a scattered mononuclear cell infiltrate, but no evidence of vasculitis. The cryostat sections did not demonstrate cytoplasmic inclusions or ragged red fibres. Nicotinamide adenine dinucleotide hydride (NADH) immunohistochemical preparations showed marked disruption of myofibrillary architecture, and adenosine triphosphatase preparations showed atrophy of both type I and type II fibres.

A chest X-ray was normal, as was a magnetic resonance imaging (MRI) scan of the head. However, abdominal and pelvic MRI scans showed extensive, enlarged lymph nodes in the left inguinal, internal and common iliac chains, and also in the paraaortic, paracaval and retrocrural areas. There was infiltration of the tissues of the left groin and left pelvic wall, due either to post-surgical lymphoedema or malignant lymphatic infiltration, and there was extensive invasion of the left iliacus muscle by tumour. The liver was normal.

The patient's condition deteriorated in spite of therapy with prednisolone 120 mg daily, and she was therefore given weekly intravenous cyclophosphamide 500 mg. At the patient's and her relatives' request, intravenous treatment was stopped after 3 weeks, and she went home for palliative care. Her muscle strength remained stable after discharge (MRC grade 2–3/5), but she developed pulmonary metastases, and died 14 weeks later.

Our patient had polymyositis associated with disseminated metastatic malignant melanoma. The diagnosis of polymyositis is secure, in that the patient satisfied all the diagnostic criteria of Bohan and Peter, i.e. symmetrical proximal muscle weakness, elevated serum muscle enzymes, electromyographic features of myopathy, and muscle biopsy evidence of an inflammatory myopathy.[7]

Ten cases of dermatomyositis occurring in association with malignant melanoma have been described in the world literature,[8–11] but we are not aware of any reports of polymyositis associated with melanoma. In our patient, the close temporal relationship between the progression of the metastatic malignant melanoma and the development of polymyositis suggests that the malignancy may have been aetiologically relevant to the development of the muscle inflammation.

The association between dermatomyositis or polymyositis and malignancy remains unclear because of the lack of large multicentre case-controlled studies with strict criteria for the diagnosis of dermatomyositis or polymyositis.[6] There is stronger evidence for a link between dermatomyositis and malignancy than for polymyositis, although Sigurgeirsson *et al.* have recently provided support for the latter.[6,12] The association appears to be more frequent in older patients, which may simply reflect the increase in incidence of malignancy with age in the general population.[6,10] The types of neoplasm reported in association with dermatomyositis or polymyositis also seem to parallel those found in the general population.[6] Thus, as might be expected, there are fewer reports of malignant melanoma than of the more common tumours, such as those of lung or breast.

The pathogenesis of dermatomyositis and polymyositis is poorly understood. A number of autoantibodies have been described in association with both conditions, but the pathogenesis of these disorders appears to be different.[3] In polymyositis, muscle fibre damage is thought to be mediated predominantly by direct attack of cytotoxic T lymphocytes.[3,4] In contrast, in dermatomyositis muscle fibre damage appears to be secondary to ischaemia resulting from complement-mediated intramuscular capillary damage.[3,4] The trigger for these processes is unknown, but both genetic and environmental factors may be important, as certain HLA types, and viruses such as coxsackie have been implicated.[3,4] The role of malignancy in the pathogenesis of dermatomyositis or polymyositis remains to be determined, although it may be postulated that a predisposed immune system, following exposure to malignancy-associated antigens, may develop cellular or humoral responses which could cross-react with similar antigenic sequences in muscle.

In view of the increasing incidence of malignant melanoma, an awareness of its possible paraneoplastic associations is important.

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Sneddon–Wilkinson disease in association with rheumatoid arthritis

Sir, Subcorneal pustular dermatosis (Sneddon–Wilkinson disease) is rare, and its pathogenesis is unknown, but the clinical and histological features overlap with those of pustular psoriasis. We describe a patient with subcorneal pustular dermatosis, seropositive rheumatoid arthritis, and an IgM paraproteinaemia. We can find only two previous reports of subcorneal pustular dermatosis occurring in patients with seropositive rheumatoid arthritis.

A 59-year-old woman presented with a 6-month history of a pustular rash which started on the abdomen and spread to the thighs. She had a 7-year history of seropositive rheumatoid arthritis, controlled with ketoprofen (Oruvail®) 200 mg daily. The onset of the pustular rash was associated with a flare of synovitis. Annular, erythematous, crusted lesions and flaccid pustules were present on the trunk and thighs (Fig. 1). There were no mucosal lesions.