Sir,
Acquired ichthyoses can either reflect an underlying severe disorder or may be an atypical expression of skin diseases such as cutaneous T-cell lymphomas (1). Otherwise, lymphomatous infiltrates in the skin can cause heterogeneous cutaneous symptoms and lesions, which may range from invisible features to massive tumour nodules. Mycosis fungoides is an epidermotropic cutaneous T-cell lymphoma, which accounts for almost 50% of all primary cutaneous lymphomas. Subtypes and variants may complete its wide clinicopathological spectrum (2). Ichthyosiform mycosis fungoides is a rare variant of mycosis fungoides observed in 1.8% of cases (3–5). We describe here a case of ichthyosiform mycosis fungoides in a 30-year-old man who presented a widespread ichthyosiform appearance coexisting with limited conventional papular lesions.

CASE REPORT
The patient was referred for ill-defined ichthyosis-like, hypo-hyperpigmented patches of 4 years duration. Pruritus was common and severe and crusted excoriations were present on the extremities. The ichthyosiform lesions were spread on the arms and legs (Fig. 1), trunk, back and lower abdomen. Clinical examination was otherwise unremarkable without any enlarged lymph nodes. Histological features of skin biopsies obtained from different ichthyosiform sites disclosed a picture of acquired ichthyosis (orthokeratotic hyperkeratosis, focal parakeratosis, acanthosis and thinned granular layer) combined with epidermotropic lymphomatous infiltration (lymphocytes exocytosis in the basal layer, small epidermal lymphocytic micro-abscesses and lichenoid infiltrate of atypical cerebriform lymphocytes in the upper dermis) (Fig. 2). Immunostaining showed a T-helper phenotype of the infiltrating cells. They were CD2-, CD3-, CD4-, CD45RO-positive but CD8-, CD20- and CD30-negative. Polymerase chain reaction analysis of the T-cell receptor \( \gamma \)-gene disclosed monoclonal rearrangement in the lesion. Biopsy samples of the papular lesions did not show any difference in the histopathological features. Staging procedures, including routine blood cell counts, biochemical analyses and cervicothoracoabdominal computed tomography, ruled out a systemic involvement of the disease. The diagnosis of mycosis fungoides, stage Ib, was established. A combined treatment with acitretin, 25 mg daily, and interferon \( \alpha \)-2a, 3 million units 3 times a week, is now at 6 months’ duration. To date, absence of disease progression and 50% improvement in the ichthyosiform appearance has been noted. On the other hand, the last control biopsy still showed signs of lymphomatous epidermotropism.
Acquired ichthyoses or ichthyosiform eruptions appear as adherent hyperkeratotic hyper- or hypo-pigmented patches widespread or mostly affecting the extremities in the adult life. The age of onset depends on the associated underlying condition. The aetiologies include autoimmune and endocrinological disorders, infectious diseases, nutritional disorders, renal failure, bone marrow transplantation, side-effects of drugs and malignancies (1). When an acquired ichthyosis is related to neoplasms, mostly Hodgkin’s and non-Hodgkin’s lymphomas, it can behave as a paraneoplastic syndrome or as a specific clinical variant of the lymphoma, the latter showing combined ichthyosiform and lymphomatous features in the same lesion. Ichthyosiform mycosis fungoides is characterized by indolent course, good prognosis and response to non-aggressive therapies such as topical treatment, psoralen and ultraviolet light A (PUVA) therapy or combined treatment with alpha interferon and retinoids. The short period of therapy in our patient does not allow us to validate this observation. After 6 months, both the neoplastic acquired ichthyosis and the conventional papular lesions are controlled by the treatment proposed.

REFERENCES