Mycosis Fungoides with Recurrent Hodgkin’s Lymphoma and Diffuse Large B-cell Lymphoma

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Sir,

Mycosis fungoides (MF), a common form of primary cutaneous T-cell lymphoma (CTCL), is a type of non-Hodgkin’s lymphoma (HL). Although the coexistence of multiple distinct lymphoid neoplasms in the same individual is rare, cases of CTCL associated with HL or other types of non-Hodgkin’s lymphoma have been described previously (1–3). We report here a rare case of MF associated with a composite recurrent HL and diffuse large B-cell lymphoma (DLBCL).

CASE REPORT

A 75-year-old Japanese man was referred to our hospital with scaly erythematous plaques distributed over most of his body. The patient had a history of nodular-sclerosing subtype HL diagnosed at the age of 62 years with persistent fever and left axillary lymph node swelling. Biopsy specimens from axillary lymph nodes showed a pleomorphic cellular infiltrate with bands of fibrosis and contained numerous large atypical cells, including Reed-Sternberg cells and lacunar cells (Fig. 1a). Immunohistochemistry and in situ hybridization showed that the Reed-Sternberg cells were positive for CD30 and Epstein-Barr virus-encoded RNA (EBER) and negative for CD20. In addition, T-cell markers were negative. These features confirmed the diagnosis of nodular-sclerosis subtype of HL. He was treated with polychemotherapy (cyclophosphamide, vincristine, procarbazine and prednisolone) therapy, and a complete remission of the relapsed HL and DLBCL was achieved. In addition, the skin lesions of MF improved partially during the chemotherapy. After the chemotherapy, however, his MF began to deteriorate gradually with extensive patches and plaques and the development of nodules. Although the HL and DLBCL were still in remission, the patient died of MF at the age of 81 years (3 years after the diagnosis of a composite lymphoma with the relapse of HL and DLBCL).

DISCUSSION

Patients with CTCL are known to have an increased risk of developing second malignancy, especially lymphoid neoplasms of different lineages (1–3). On
the other hand, it is well known that patients with HL are at increased risk of developing second malignant neoplasms including non-Hodgkin’s lymphoma (4–6). Occurrence of MF in patients with previous HL has also been reported occasionally (7).

Various explanations for the coexistence of different lymphomas in an individual have been proposed (4, 8), such as a genetic predisposition for malignancies (9). In our case, the patient’s father had died of gastric cancer. It has been suggested that underlying viral infection may result in multiple lymphoproliferative disorders (10, 11). While our patient was negative for HTLV-1 and HIV antibodies, tumour cells of HL were positive for EBER. Furthermore, the mutagenic effects of cytostatic drugs have been assumed to be involved in the pathogenesis (12). In our patient, MF occurred after polychemotherapy combined with radiation therapy for HL. Patients with HL, advanced MF and Sézary syndrome are reported to have impaired immunity with an altered cytokine profile (13, 14). It was also possible that HL and CTCL were derived from the same pluripotent stem cells, which had a dual capacity for evolution of T and B cells. Although immunohistochemical staining for stem cell markers was not performed in our case, some reports have described a common clonal origin of CTCL and other types of lymphoma (15).

REFERENCES