Short reports

Granulocytic sarcoma with expression of CD30

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Abstract

A case of a young man with a spinal epidural tumour, initially diagnosed as large cell anaplastic malignant lymphoma, is reported. The tumour consisted of poorly differentiated cells showing immunoreactivity with antibodies directed against CD30 and CD45. Ten months later the patient developed acute myeloid leukaemia. The histological slides of the epidural tumour were reviewed, including additional enzyochemical and immunohistochemical stains. As the tumour showed immunoreactivity for myeloperoxidase and chloroacetate esterase, it was reclassified as a granulocytic sarcoma.


Keywords: granulocytic sarcoma, acute myeloid leukaemia, CD30 immunoreactivity.

The Ki-1 monoclonal antibody was originally raised in 1981 against the Hodgkin’s disease derived cell line L428. The antibody showed a modest affinity for the Ki-1 antigen (later renamed CD30 antigen, a membrane bound 105 kilodalton molecule) on aceton fixed cryostat sections but was unreactive in routinely processed histological material. In 1989 a new monoclonal antibody, Ber-H2, was prepared by Schwarting et al against a different, formaldehyde resistant epitope of the same antigen. A group of CD30 positive, large cell anaplastic lymphomas (LCAL) have been recognised as a separate entity among non-Hodgkin’s lymphomas. Most of these lymphomas are of T cell origin, while the remaining are either of B cell origin or fail to express any lineage markers (null cell type). Epithelial membrane antigen (EMA), typically a marker for epithelial cells, is expressed in over 50% of these tumours. Later, it became clear that Ki-1 is not a cell lineage specific antigen but that it also occurred in non-lymphocytic cell lines, such as the K562 and HL-60 cell lines derived from chronic myelogenous leukaemia (CML) and acute myeloid leukaemia (AML), respectively, and even in some non-haemopoietic cells and tumours—for example, embryonal carcinoma.

Case report

A 28 year old man was admitted to hospital because of back pain, sensory loss in both legs and disturbances of gait. General examination revealed obesity. Enlargement of lymph nodes was not present. Peripheral blood counts were normal.

Magnetic resonance imaging (MRI) showed an epidural tumour at T9. Neurosurgical decompression of the spinal cord was performed. Histological examination of a biopsy specimen showed a poorly differentiated CD45 + CD30 + tumour (Ber-H2; Dako, Glostrup, Denmark) (fig 1). Tumour cells did not express EMA, or T or B cell markers (CD3, CD20). Electron microscopy was not performed. The tumour was classified as a Ki-1 positive LCAL, null cell type. A computed tomography scan of the chest and the abdomen did not reveal the presence of tumour metastases. There was no tumour infiltration of the bone marrow. However, tumour cells were detected in the cerebrospinal fluid (CSF).

Chemotherapeutic treatment was initiated with CHOP (cyclophosphamide, doxorubicin, vincristine, prednisolone) and intrathecal administration of methotrexate. The tumour cells disappeared from the CSF, but both the erythrocyte sedimentation rate (ESR) and the lactate dehydrogenase (LDH) concentration increased. After switching CHOP to DHAP (dexamethasone, high-dose Ara-C, platinum) chemotherapy, the ESR and LDH concentra-
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Figure 2 Spinal tumour stained with antibody directed against MPO (immunoperoxidase technique; polyclonal; original magnification ×250).

Nevertheless, these authors claim that the growth pattern, the nuclear morphological features and often the cytoplasmic features in granulocytic sarcoma are different from those of large cell lymphoma. Granulocytic sarcoma infiltrates tissue with preservation of the tissue architecture, whereas LCAL is often associated with tissue destruction and coagulation necrosis. Cells in granulocytic sarcoma have round to oval, reniform, or many lobed nuclei with rather small nucleoli, a thin nuclear membrane and a delicate chromatin pattern. Cells in large cell lymphomas have large nucleoli, a thick nuclear membrane and coarsely clumped chromatin. In more mature granulocytic sarcoma tumour cells there is a moderate amount of eosinophilic and granular cytoplasm, in contrast to the lesser amount of amphophilic cytoplasm seen in LCAL. However, the morphological heterogeneity (including nuclear characteristics) in LCAL is emphasised in a study by Penny et al. who found that only eight of 24 cases of LCAL had prototypic cytological features.

A new finding in the present case was the fact that tumour cells of granulocytic sarcoma and AML can express CD30 in vivo. In an immunohistochemical study of 28 cases of extramedullary myeloid cell tumours by Traweek et al., none of the formalin fixed tumours showed CD30 (Ber-H2; Dako) reactivity. CD30 immunoreactivity has, however, been detected in cell lines derived from CML and AML. In a pilot study on archival material we found that tumour cells from three of nine patients with AML expressed CD30.

The present case report again stresses the fact that CD30 is not a cell line specific marker. In all cases of LCAL, especially of the so-called null cell type and in all atypical lymphoid cell infiltrates of granulocytic sarcoma or leukaemic infiltration should be excluded by appropriate stains, including anti-MPO, a lineage specific marker for myeloid cells.

Discussion
Granulocytic sarcoma (chloroma, extramedullary myeloid cell tumour or EMT) has been defined as a “localized tumour mass composed of immature cells of the granulocytic series” that may occur in association with CML and AML or precede AML. Involvement of the spine, as in the present case, has been described previously. In the series reported by Meis et al. 12 (75%) of 16 cases were misdiagnosed initially, mostly frequently as large cell lymphoma, as in the present case. The poorly differentiated (predominantly myeloblastic) cases were all misdiagnosed initially.

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