CORRESPONDENCE

EXPRESSION OF PLATELET GLYCOPROTEIN IIa BY HUMAN OSTEOCLASTS

To the Editor:

In a recent article in Blood, Beckstead et al. reported the immunological localization of a series of membrane and granule proteins by human megakaryocytes in bone marrow biopsy specimens. An unexpected finding in this study was the observation that platelet glycoprotein IIa (GPIIIa), but not GPIIb or GPIIb/IIa complex, was expressed by human osteoclasts. We have previously studied the cell surface expression of a range of hematopoietic antigens by human fetal and tumor-associated osteoclasts. In an extensive survey we found that osteoclasts were HLA-DR negative and failed to express myeloid antigens; the only exception was reactivity with My7-like antibodies, which recognize a 150-kd surface glycoprotein present on the majority of myeloid cells. Further, we have recently raised a series of monoclonal antibodies to human osteoclasts. These fail to react with megakaryocytes in human and animal bone marrow.

We report here our results on the expression of platelet-associated determinants by human osteoclasts isolated from giant cell tumors of the bone (osteoclastomas). Osteoclasts are GPIIIa (C17) positive (Fig. 1) but fail to react with antibodies to GPI (ANS1), GPIIb/IIIa (J15), GPIIb (P256), platelet factor 4, or factor VIII-related antigen, confirming the findings of Beckstead et al.

The coexpression of GPIIIa by megakaryocytes, platelets, and osteoclasts clearly needs further investigation. It would be of particular interest to know if GPIIIa mediated some functional role, such as cellular adhesion, shared among these diverse cell types.

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REFERENCES


FOLLICLE LYSIS IN LYMPH NODES FROM HOMOSEXUAL MEN

To the Editor:

We read with interest the article by Wood et al. about the "follicle lysis" phenomenon, which is due to the disruption of the follicular dendritic cell (FDC) framework in lymph nodes from homosexual men with persistent generalized lymphadenopathy (PGL). Because FDCs express the T4 antigen, the authors suggest that their infection by HTLV III/LAV may play a role in the pathogenesis of follicle lysis.

We agree with them. As is known, viral particles in close contact with the cytoplasmic processes of dendritic reticulum cells (DRC) were seen by Tenner-Racz et al. and Armstrong et al. We were able to demonstrate the strong intracytoplasmic reactivity of the FDC (Fig 1) from PGL and AIDS patients using the CVK monoclonal antibody specific for the P18/LAV protein and modified immunoperoxidase technique. No cells other than FDCs were stained in lymph nodes with CVK, despite the fact that the T4 antigen is demonstrable on other cells in lymph nodes.

These results suggest that FDCs represent the elective target of HTLV III/LAV infection in lymph nodes. However, the progressive disruption of the FDC framework, with follicle lysis, is the main morphological aspect of lymph nodes in PGL patients. We have no certain data about the mechanisms involved in such a phenomenon; perhaps the entrance of T8 cells into the follicles could implicate...
To the Editor:

We read with great interest the letter by Parravicini et al, who report FDC reactivity with a monoclonal antibody directed against p18/LAV protein. As these investigators have noted, their results are consistent with prior immunohistologic and ultrastructural studies suggesting that, in addition to helper T cells, FDC may be a target of HTLV-III/LAV infection. One basis for this hypothesis is that both helper T cells and various histiocytes, including FDC, express the Leu-3/T4 antigen which is an essential component of the cell surface receptor necessary for HTLV-III/LAV infection. Furthermore, it has been shown previously that certain types of histiocytes can be infected by HTLV-III/LAV. Interestingly, Parravicini et al were unable to detect reactivity of other lymph node cells, including helper T cells, with their antibody. This may be related to the fact that less than 0.01% of peripheral blood or lymph node mononuclear cells from individuals infected with HTLV-III contain HTLV-III RNA detectable by an in situ hybridization technique. It is possible that among other Leu-3+/T4+ cells, such as FDC, a larger proportion of cells may contain detectable HTLV-III/LAV. Of course, it will be important to distinguish between active retroviral infection of FDC and their passive acquisition of retroviral antigens, eg, by adsorption or phagocytosis.

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Fig 1. Lymph node biopsy from patient with persistent generalized lymphadenopathy. Two-micron sections from historesine embedded biopsy immunostained with P18/LAV MoAb, showing strong intracytoplasmic reactivity in the follicular dendritic cell.

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