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B-CELL LYMPHOMA LOCALIZED PRIMARY TO THE SUBCUTANEOUS ADIPOSE TISSUE

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Primary lymphoma of the subcutaneous adipose tissue is rare clinicopathological entity. Only a few previously documented cases have been found in a survey of the relevant literature. A case of primary B-cell non-Hodgkin lymphoma of the subcuteneous tissue is presented. In a case of 81-year-old woman the immunohistochemical investigation of subcutaneous adipose tissue pattern showed centrocytic- lymphoma. The patient is currently alive without evidence of lymphoma after chemotherapy /cyclophosphamide, vincristine, doxorubicin and prednisone (CHOP), six courses/.

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JUDICIOUS APPLICATION OF GROWTH FACTORS AND INTERLEUKINS IN MULTIPLE MYELOMA - A NEW APPROACH ?

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Tumor cells in multiple myeloma exert a multiplicity of interactions with other cellular components, such as monocytes and macrophages, T-lymphocytes, polyclonal B-cells, hematopoietic progenitors, and cells involved in bone metabolism. A lot of experimental data and several clinical findings raise strong evidence that cytokines and other soluble factors have a predominant pathophysiological impact in this disease; e.g. paracrine produced interleukin-6 turned out to be the major growth factor for multiple myeloma tumor cells. Findings that cytokines can promote or inhibit tumor growth and may influence tumor complications by direct action on the plasma cells or indirect effects via stimulation of regulator cells suggest the use interleukins, hematopoietic growth factors, and other regulating factors for therapeutical approaches. At present several of these factors have been tested in vitro or are under clinical investigation. Among others, substances such as interferon-α, interleukin-2, interleukin-4, erythropoietin, GM-CSF, G-CSF, and monoclonal antibodies (anti-interleukin-6; anti-CD3) are of special interest. Previous and recent results clearly demonstrate potential impact of these factors. However, just as in other tumors, lots of effort is needed to elaborate the appropriate indications, optimal doses, and schedules for a judicious application of growth factors and interleukins. Multiple myeloma can serve as a suitable model for such an approach.

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Antibody response to pretreatment pneumococcal vaccination and posttreatment revaccination in splenectomized patients with non-Hodgkin lymphomas

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Splenectomized patients undergoing multiagent chemotherapy and irradiation are at highest risk for overwhelming sepsis with Streptococcus pneumoniae (S.pn.). In immunocompetent individuals pneumoccocal vaccines are protective against infections with S.pn. In the present series the antibody response in 11 splenectomized patients with non-Hodgkin lymphomas (NHL) vaccinated with a 23-valent pneumococcal polysaccharide prior to chemo- and/or radiotherapy was studied.

With the bacterial vaccine a 2-5 fold rise of the prevaccination titer against S.pn. (as evaluated by ELISA against an antigen from the 23-valent polysaccharide vaccine) was elicited in 4/11 patients with NHL. Prevaccination antibody concentration in patients with NHL was lower ($p = 0{,}001$) as compared to controls who had undergone splenectomy for other reasons. After vaccination no significant difference in antibody levels against S.pn. was evaluated between both groups ($p = 0{,}082$). NHL patients in remission after having completed chemotherapy received a booster dose of the polysaccharide. Revaccination did not increase the pneumoccocal antibody titer significantly ($p = 0{,}7$).

We conclude that vaccination with pneumoccocal polysaccharides in splenectomized patients with NHL elicits an antibody response in 36% of cases and should therefore be administered. However, a second injection of the bacterial vaccine does not contribute to the protection against invasive pneumococcal infections.

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Follicular dendritic cells in non-Hodgkin lymphoma and neoplastic B-cells display a complementary panel of adhesion molecules

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In non-Hodgkin lymphoma (NHL) with nodular growth pattern, follicular dendritic cells (FDC) form a spherical network which contains neoplastic B-cells. In order to dissect the basis of this close FDC/B-cell association, the antigenic profile of adhesion molecules expressed by individual FDC and NHL-B-lymphocytes was evaluated. FDC isolated from NHL were found to express HLA-ABC (MHC class I antigen), C3bi receptors (CD11b), the very late antigen (VLA) alpha-5- and alpha-6-chain (CDw49e, CDw49f) and the intercellular adhesion molecule-1 (ICAM-1; CD54). Only 50% of the FDC population was positive for the VLA beta-1- and alpha-3-chain (CD29, CDw49c), the vitronectin receptor (CD51) and the vascular cell adhesion molecule-1 (VCAM-1). B-cells obtained from the lymph nodes of patients with centroblastic-centrocytic lymphoma expressed ligands complementary to the adhesion receptors on FDC, *i.e.* LFA-1 alpha- and beta-chain (CD11a, CD18), and ICAM-1 (CD54). Interestingly, monoclonal lymphocytes in the peripheral blood of patients with a leukemic course of this lymphoma entity were devoid of these antigens. These data suggest that neoplastic B-cells without CD11a, CD18, and CD54 surface molecules are unable to associate with FDC and now invade other compartments. Thus, adhesive interactions between FDC and NHL-B-cells may account for the peculiar growth pattern and spread of follicular lymphoma.

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