Hst 03

ANTICOAGULATIVE AND ANTIFIBRINOLYTIC THERAPY OF OVARIAN CARCINOMA

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Malignant tumours possess coagulative properties that cause deposition of fibrin around them. Such fibrin is a necessary matrix for proliferating tumour vessels. As in wound repair, old residual fibrin must afterwards be removed. This is done by the fibrinolytic system which is initiated by plasminogen activators produced by the tumour cells.

The aim of the treatment study was to interfere with the coagulative as well as the fibrinolytic properties of the tumours. To 18 women over 70 years of age, and not suitable for treatment with cytostatics, the anticoagulant Warfarin was given together with the fibrinolytic inhibitor tranexamic acid (Cyklokapron®) in a dosage of 4 g daily. All patients were operated upon and six were macroscopically radical (3 stage I, 2 stage II, 1 stage III). Twelve patients had advanced stage III and IV, with bulky disease after surgery. Four of them had liver metastasis.

The five patients in stage I-II and radically operated are alive with a median survival time of 51+ months (range 21+ - 58+). The median survival of the 13 patients in stage III-IV is eight months (range 2 - 72+). Three of them, including two with liver metastasis, are alive and in a good clinical state. One of the latter has the most prolonged survival time of 72+ months.

A certain time seems necessary for this therapy to exert its effect. By starting treatment of advanced tumours with combined Warfarin/tranexamic acid and cytostatics, the often good initial effect of cytostatics should enable Warfarin/tranexamic acid to exert its prolonged effect.

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Lym 01

FAMILIAL MALIGNANT LYMPHOMA - DUNCAN'S SYNDROME

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In 1975, Purtilo described a family in which 6 out of 18 male members had died from lymphoproliferative diseases. All had in common an infection with Epstein-Barr-Virus (EBV) and an immune deficiency of variable degree. Further virological and serological research led to the description of the so-called "X-linked Lymphoproliferative Syndrome" or "Duncan's Disease" after the firstly described family. As only male family members were affected, an X-chromosomal mode of inheritance was proposed. The following immunopathogenetic mechanisms have been discussed: EB-Viruses infect B-lymphocytes, leading to a polyclonal proliferation of EBV-infected cells. Due to an immune defect, there is insufficient production of EBV-antibodies and killer-T-lymphocytes which eliminate EBV-infected B-cells.

We present a family in which two sons have got lymphogranulomatosis. They died at the age of 12 and 20 years from stage IV B Hodgkin's disease. The third son, 19 years old, was followed up for one year for suspected Hodgkin's disease, his moderately elevated EBV-titers being ascribed to this disease. Ad the same time he had a constant deficiency of immunoglobulins. Upon clinical progression another biopsy led to the diagnosis of a polymorphic lymphoplasmacytoid immunocytoma. In this lymph node EBV DNA was demonstrated. Together with the family history this leads to the diagnosis of Duncan's Syndrome. There was no evidence of HLA-linkage of the lymphoproliferative disorder. Althoug there was no numerical aberration of T and B lymphocytes and the subpopulations a functional defect of cellular immunity (areactivity by MLC-testing) could be demonstrated. This can be taken as evidence of a functional T cell defect which favours the development of a lymphoproliferative disease.

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Lym 02

RELATION BETWEEN MYCOSIS FUNGOIDES (MF) AND HODGKIN'S DISEASE (HD)

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There is agreement that tumor cells of Mycosis fungoides (MF) and Hodgkin's disease (HD) are not related to each other since they are of different origin. Here we report two cases, which led to the diagnosis of either Mycosis fungoides and Hodgkin's disease during the course of the disease as judged by various biopsies taken in intervalls although the primary diagnosis was Mycosis fungoides. The immunohistological analysis of these biopsies demonstrated that large tumor cells in the lesions diagnosed as Mycosis fungoides as well as tumor cells from the specimens classified as Hodgkin's disease expressed the Reed-Sternberg-cell associated antigens Ki-1, Ki-24 (STEIN et al., 1982) and in addition the Interleukin-2-receptor (STEIN et al., 1985; HSU et al., 1985).

Since it is well established, that Reed-Sternberg-cell associated antigens Ki-1 and Ki-24 as well as Interleukin-2-receptor are antigens associated with lymphotic activation we conclude

1. Hodgkin's disease (HD) is a neoplasia of activated lymphocytes.
2. Tumor cells of Mycosis fungoides (MF) may differentiate during the course of the disease into "activated" cells whose degree of activation is similar to that of Reed-Sternberg and Hodgkin cells (as measured by the expression of Reed-Sternberg-cell associated antigens Ki-1, Ki-24 and the Interleukin-2-receptor).

This assumption may easily explain the repeatedly observed transformation of Mycosis fungoides in Hodgkin's disease.

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Lym 03

TREATMENT OF ADULT BURKITT-TYPE NON-HODGKIN'S LYMPHOMAS(NHL)

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Non-endemic lymphoblastic NHL of Burkitt-type is a rare disease in adults, accounting for only 2% of all NHL. Prognosis in this age group is poor with a 2-year survival rate of 30%. During recent years, 13 patients with Burkitt lymphoma were treated at this institution. The median age of the patients was 25(range 14-46) years. Chemotherapy consisted of cyclophosphamide, vincristine, methotrexate and prednisone(COMP) in 5 patients. More recently, 4 patients were treated with a combination chemotherapy consisting of medium-dose methotrexate with leucovorin-rescue, cyclophosphamide, teniposide, cytarabin, adriamycin and prednisone and central nervous system prophylaxis with i.th. methotrexate and 24 Gy cranial irradiation.
In the 4 evaluable patients treated with COMP there were 2 complete remissions(CR) who are in continious CR for 38+ and 81+ months respectively. Progressive disease occurred in the remaining 2 patients who died after 4 and 6 months, respectively. By contrast all patients receiving the recently employed intensive chemotherapy reached CR. Of these, 3 patients are in continious CR for 5+ to 17+ months whereas in 1 patient meningeal and bone-marrow relapse occurred after 4 months of CR. From the present albeit limited experience it is concluded that non-endemic Burkitt lymphomas in adults can effectively be treated by combination chemotherapy.

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