T-CELL LEUKAEMIA IN CHILDREN FROM THE PROVINCE OF NAPLES

Sir,-Several reports1-4 have focused on the relation between some adult T-cell-derived malignancies and HTLV virus in the so-called "endemic" areas—the west coast of Kyushu Island (Japan), and the Caribbean.

Over a 30-month period (August, 1980, to February, 1983) we studied 58 children with acute lymphoblastic leukaemia (ALL). 40, all from the Campania region, were newly diagnosed cases. Lymphoblasts were screened for sIg and E rosette formation, and 25 cases were analysed by a panel of monoclonal antibodies (the OKT series, J-5, B-1, p-24 (BA-2), and OKIal). 16 (40%) of the 40 newly diagnosed cases were T-cell ALL. 7 T-cell ALL children were studied by monoclonal antibodies (table).

While the regional distribution of non-T, non-B ALL cases was heterogeneous and without significant characteristics, 13 of the T-cell ALL cases came from the province of Naples; and 7 were from small towns in the Mount Vesuvius area (Ercolano, Pompei, Castellammare di Stabia), with 4 others from parts of the province with similar geographical characteristics (Campi Flegrei, Ischia, Sorrento). Thus, most of the T-cell ALL cases arose in a small, defined area. Our data suggest that the annual incidence of cases in this area is three times greater than the mean incidence for the rest of Italy.

Patients 4 and 5 came from Castellammare di Stabia; they lived very near each other and went to the same school (a brother of one of them was in the same class as the other). The disease onset in these two cases was only a few days apart. A few months later another little girl with T-cell ALL coming from Ercolano (very near Castellammare) was admitted. She was the only one with a post-tumour lysis syndrome (T-6+, T-4+, T-8+). Two boys with T-cell ALL living in Naples were also admitted almost simultaneously on the same day. All these children were from small towns in the Mount Vesuvius area (Ercolano, Pompei, Castellammare di Stabia), with 4 others from parts of the province with similar geographical characteristics.

The five cases had similar clinical and haematological features. These observations suggest the possibility of horizontal transmission of T-cell ALL in these children. Virological studies were not done.

As stressed in a 1982 Lancet editorial5 a worldwide search for HTLV retrovirus in T-cell malignancies seems advisable. The natural radioactivity of the area around Vesuvius is very high—135HTLV retrovirus in T-cell malignancies seems advisable. The transmission of T-cell ALL in these children. Virological studies were not done.

The first manifestation of the disease. These five cases had similar clinical and haematological features.

**PLATELET MONOAmine OXIDASE ACTIVITY IN A NEVER-TREATED CHRONIC SCHIZOPHRENIC**

Sir,—Lowered platelet monoamine oxidase (MAO) activity has been reported to be a discriminating characteristic of different subgroups of schizophrenic patients, particularly those with paranoid symptoms or auditory hallucinations. However, several groups have suggested that neuroleptic treatment may have contributed significantly to the decreased platelet MAO found in some studies of schizophrenic patients. Typically platelet MAO activity during neuroleptic treatment has been compared with values obtained during a preceding drug-free period, usually of between one and six weeks. It is not clear whether such drug-free periods are long enough to provide a stable baseline for biological indices.

We have seen a 36-year-old man who met both research diagnostic and DSM-III criteria for chronic paranoid schizophrenia, the onset of which was independently confirmed to have occurred 4 years previously. Remarkably, this delusional, hallucinating patient had never received antipsychotic or psychotrophic medication. His parents' involvement had moderated the more severe exacerbations and psychiatric intervention had never been sought.

Before neuroleptic medication began platelet MAO activity was 18.49 nmol/10⁸ platelets/h; the platelet count was 282 000/µl. After the patient had received seventeen daily doses of haloperidol (20 mg for 3 days, 25 mg for 11 more days, then 20 mg for an additional 3 days), platelet MAO activity was 9.77 nmol/10⁸ platelets/h; the platelet count was 195 000/µl. Laboratory personnel doing the platelet MAO assays were "blind" to the patient's diagnosis; blood samples were analysed in duplicate; and the laboratory's intra-assay coefficient of variation is less than 5%.

After 17 days of treatment with haloperidol alone (which was associated with clinical improvement), the patient's platelet MAO activity had fallen to 53% of its level before medication had been administered. This marked decline in platelet MAO activity is striking in view of the reported changes of only 15-20% in platelet MAO activity during neuroleptic treatment. Typically platelet MAO activity that would have been calculated if the platelet count had not been lower at the time of the second blood sample. That we found such a decrease in a never-medicated patient lends support to the assumption that the brevity of the drug-free periods used in earlier studies of the effect of neuroleptics on platelet MAO activity did not confound the similar results obtained.

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