Children with microcephaly and non-Hodgkin's lymphoma (NHL)

<table>
<thead>
<tr>
<th>Case No</th>
<th>Sex</th>
<th>Age at diagnosis (years)</th>
<th>Type of NHL</th>
<th>NHL stage</th>
<th>Immunodeficiency</th>
<th>Family history</th>
<th>Mental development</th>
<th>Outcome (survival)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>8</td>
<td>III</td>
<td>IgA decreased</td>
<td>Negative</td>
<td>retarded</td>
<td>Dead (8 months)</td>
<td>(survival)</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>3</td>
<td>II</td>
<td>IgA decreased</td>
<td>Negative</td>
<td>Alive in I remission (38 months)</td>
<td>Alive in II remission (28 months)</td>
<td>Alive in II remission (18 months)</td>
</tr>
<tr>
<td>3</td>
<td>T</td>
<td>15</td>
<td>III</td>
<td>Frequent respiratory infections</td>
<td>Positive for abnormality</td>
<td>IQ 61</td>
<td>Dead (8 years), NED (15 months), pericarditis, Alive in I remission (38 months)</td>
<td>(survival)</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>7</td>
<td>III</td>
<td>Decreased reaction to mitogens</td>
<td>Sister with low IgA, cerebral palsy</td>
<td>IQ 76 (TM)</td>
<td>Alive in I remission (35 months)</td>
<td>Alive in II remission (28 months)</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>6</td>
<td>B</td>
<td>IgA decreased, low B cell, low T cell</td>
<td>Sister with microcephaly</td>
<td>IQ 77</td>
<td>Alive in II remission (28 months)</td>
<td>Alive in I remission (18 months)</td>
</tr>
<tr>
<td>6</td>
<td>T</td>
<td>10</td>
<td>IV</td>
<td>IgA decreased, low B cell, low T cell</td>
<td>Sister with microcephaly</td>
<td>IQ 64 (normal school)</td>
<td>Alive in I remission (18 months)</td>
<td>Alive in I remission (4 months)</td>
</tr>
<tr>
<td>7</td>
<td>T</td>
<td>13</td>
<td>III</td>
<td>IgG, IgA slightly decreased</td>
<td>Negative</td>
<td>IQ 87</td>
<td>Alive in I remission (18 months)</td>
<td>Alive in I remission (18 months)</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>3</td>
<td>B</td>
<td>IgA decreased</td>
<td>IgA slightly decreased</td>
<td>IQ 76 (TM)</td>
<td>Alive in I remission (18 months)</td>
<td>Alive in I remission (18 months)</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>9</td>
<td>T</td>
<td>IgA decreased, low B cell, low B4</td>
<td>Brother with hypochromic blood and foot malformation</td>
<td>IQ 87</td>
<td>Alive in I remission (18 months)</td>
<td>Alive in I remission (4 months)</td>
</tr>
</tbody>
</table>

*Lack of information. IQ measured on Wechsler scale or Terman-Merrill (TM) scale.
*Child from hospital for mentally disabled; lack of precise information. NED no evidence of disease.

The data of our nine patients are shown in the table. Familial microcephaly was seen in two patients. In three families other congenital malformations were found, and immunodeficiency was confirmed in six patients.

The outcome of treatment was not affected by the presence of microcephaly, despite treatment modifications to reduce toxicity (including omission of alkylating agents and/or irradiation of the central nervous system). Unfortunately, we were unable to perform complete immunological and cytogenetical examinations in our patients. However, there are reasons to believe that most of our cases were truly children with Seemanova's syndrome. It will be necessary to study more children to help explain the relation between congenital malformations, immunodeficiency, and malignancy.

A DLUZNIEWSKA
D TREDOWSKA-SKOCZEN
T JARMATA
J TACIK
Department of Pediatric Hematology
Polish American Institute of Pediatrics
Collegium Medicum
Jagiellonian University
265 Widzka Str.,
30-663 Cracow, Poland


Ocular relapse in acute lymphoblastic leukaemia

**EDITOR**—Ocular relapse associated with acute lymphoblastic leukaemia (ALL) is rarely seen nowadays as a result of improved treatment of childhood leukaemia. A 12 year old boy with ALL was treated in accordance with the recently updated UKALL protocol which did not include cranial irradiation. He developed leukaemic infiltration of his left eye while in remission and on maintenance chemotherapy, seven months after the original diagnosis. Lumbar puncture revealed central nervous system (CNS) relapse and he underwent local irradiation to the left orbit with a maximum dose of 800 cGy and subsequently reinduction chemotherapy and cranial irradiation. After initial partial resolution of signs, infiltrates persisted and 10 weeks later he underwent further craniospinal irradiation with 2400 cGy applied to the right and left cranium in 15 fractions with 6 MV x-rays, resulting in a gradual improvement over a three month period and he remained in remission.

By six months only residual pigmentary changes were observed in the fundus and maintenance treatment was stopped due to neutropenia with the CNS remaining clear of blast cells. However, two weeks later, he developed further ocular recurrence which temporarily resolved on treatment, and subsequently further ocular and CNS relapse, and died 11 months after his original ocular presentation.

Ocular involvement in leukaemia is most commonly a consequence of associated haematological abnormalities and usually occurs when the patient is in relapse. Anaemia may precipitate a leukaemic retinochoroidal leukaemia. The last document to give guidance on the management of this area was published by DHSS in 1981, but there have been many changes since then, including the emergence of HIV and other viral infections. A milk banking symposium in March 1993 marking the closure of Sorrento Maternity Hospital, Birmingham (which had been in the forefront of milk banking in the UK) highlighted the need to update UK guidelines. An ad hoc working party was therefore established and new guidelines drawn up similar to those published by the Human Milk Banking Association of North America.

The selection of donor mothers was considered very carefully and the guidelines do not reach the eye, and its effect on tumour cells in the optic nerve is demonstrated as far as the termination of the subdural space posterior to the globe.

**Editors**—Interest in milk banking (the collection, storage, and processing of donor mothers' breast milk), has lately reawakened. The last document to give guidance on this area was published by DHSS in 1981, but there have been many changes since then, including the emergence of HIV and other viral infections. A milk banking symposium in March 1993 marking the closure of Sorrento Maternity Hospital, Birmingham (which had been in the forefront of milk banking in the UK) highlighted the need to update UK guidelines. An ad hoc working party was therefore established and new guidelines drawn up similar to those published by the Human Milk Banking Association of North America.

**Guidelines for the establishment and operation of human milk banks in the UK**

**EDITOR**—Interest in milk banking (the collection, storage, and processing of donor mothers' breast milk), has lately reawakened. The last document to give guidance on this area was published by DHSS in 1981, but there have been many changes since then, including the emergence of HIV and other viral infections. A milk banking symposium in March 1993 marking the closure of Sorrento Maternity Hospital, Birmingham (which had been in the forefront of milk banking in the UK) highlighted the need to update UK guidelines. An ad hoc working party was therefore established and new guidelines drawn up similar to those published by the Human Milk Banking Association of North America.

**Editors**—Interest in milk banking (the collection, storage, and processing of donor mothers' breast milk), has lately reawakened. The last document to give guidance on this area was published by DHSS in 1981, but there have been many changes since then, including the emergence of HIV and other viral infections. A milk banking symposium in March 1993 marking the closure of Sorrento Maternity Hospital, Birmingham (which had been in the forefront of milk banking in the UK) highlighted the need to update UK guidelines. An ad hoc working party was therefore established and new guidelines drawn up similar to those published by the Human Milk Banking Association of North America.