malignant disorders. Our experience shows that although there were minor problems in using the catheter in children, there were no complications resulting in major morbidity. In no patient was a catheter removed because of sepsis and this problem has been reported to be only 3% in larger groups of older patients.6

The reasons for our success in using catheters are the motivation and thorough training of the families and the precautions we have introduced to reduce the risks attending the use of catheters in young children. Education of our families consists of a video tape on catheter care, practical teaching sessions in the clinic, and a visiting nurse until the families can manage independently. The length of training time varies from 1 week to 1 month. Catheters are kept dressed at all times with either Op-site Spray Dressing or gauze, and for further security the catheter is taped to the chest wall. Smaller children wear vests or T-shirts to discourage them from playing with the catheter. Luer lock stop cocks are used for intravenous treatment to avoid accidental disconnection, and trained staff only have access to the catheter during hospital admissions and clinic visits.

We observed several benefits from the use of these catheters in children with malignant disorders—readily available venous access; easy administration of intravenous chemotherapy without risk of extravasation; decreased time spent in clinic; less emotional trauma for patients, parents, and staff; and less anticipatory vomiting. The latter benefit was observed in 4 children who were refractory to antiemetics and behaviour modification and whose anticipatory vomiting resolved immediately when venepunctures were no longer necessary.

In our experience the long term use of tunneled central venous catheters in children with malignant disorders is associated with acceptable morbidity and results in many benefits in the provision of specific and supportive care.

We thank Dr R Barr.

References


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T cell lymphocytosis with neutropenia

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SUMMARY A gross T cell lymphocytosis developed in an otherwise healthy 5 year old girl and has persisted for nearly 3 years. It is associated with neutropenia and seems to be typical of a rare adult disorder that may be a type of T cell chronic lymphocytic leukaemia.

‘T cell lymphocytosis with neutropenia’ is the cautious name given to a rare syndrome that occurs in adults and there is doubt whether it is a clonal neoplasm or merely a reactive pleocytosis.1 It has never been described in a child, and we report its occurrence in a young girl.

Case report

A 4 year old girl presented in 1979 with a moderate normochronic anaemia. Her father and 5 other family members had had splenectomy performed for hereditary spherocytosis and the same diagnosis was confirmed in this child. Splenectomy was
eventually performed when she was 5 years old and the clinical response was good. A blood count 9 months later showed a surprisingly marked lymphocytosis (absolute count $37.6 \times 10^9/\text{l}$). In retrospect, a modest lymphocytosis (absolute count $9.35 \times 10^9/\text{l}$) had been evident 6 months before splenectomy, but several earlier peripheral blood profiles had been normal. Clinical examination findings were unremarkable with no lymphadenopathy or hepatomegaly. The patient was well. Marker analysis of the peripheral blood lymphocytes showed them to be predominantly T cells with a particular increase in those with a cytotoxic suppressor phenotype (OKT8 positive). Neutropenia (absolute count $0.40 \times 10^9/\text{l}$) and polyclonal hypergammaglobulinaemia were also present. Her monospot test was repeatedly negative and serological screening gave no evidence of any other viral infection. Bone marrow was difficult to aspirate and both smears and trephine biopsy specimens were hypercellular with a dense infiltrate (92%) of the same mature lymphocytes seen in the blood. These were negative for α-naphthyl acetate esterase but strongly positive for acid phosphatase. Her stimulated peripheral blood karyotype was normal and her haemoglobin concentration and platelet count were normal and have remained so throughout.

The lymphocytosis and neutropenia have persisted, and serial marker studies have shown an increasing proportion of OKT8 positive (suppressor) cells with a corresponding decline in OKT4 positive (helper-inducer) cells (Table). In September 1982 (at the age of 7 years) she had her first serious infective episode with severe mouth ulceration and a swinging fever that responded to parenteral antibiotics. At the time of writing, 35 months after the discovery of her abnormality, she was well, and her growth was following the 25th centile.

### Table Peripheral blood marker studies

<table>
<thead>
<tr>
<th></th>
<th>November 1981</th>
<th>July 1982</th>
<th>February 1983</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total white cell count ($\times 10^9/\text{l}$)</td>
<td>61</td>
<td>54</td>
<td>70</td>
</tr>
<tr>
<td>Neutrophil count ($\times 10^9/\text{l}$)</td>
<td>0.8</td>
<td>0.5</td>
<td>0.7</td>
</tr>
<tr>
<td>Lymphocyte count ($\times 10^9/\text{l}$)</td>
<td>58</td>
<td>53</td>
<td>68</td>
</tr>
<tr>
<td>E rosettes (%)</td>
<td>42</td>
<td>40</td>
<td>84</td>
</tr>
<tr>
<td>*OKT3 (%)</td>
<td>85</td>
<td>95</td>
<td>95</td>
</tr>
<tr>
<td>*OKT4 (%)</td>
<td>36</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>*OKT8 (%)</td>
<td>60</td>
<td>83</td>
<td>82</td>
</tr>
<tr>
<td>Mouse rosettes (%)</td>
<td>11</td>
<td></td>
<td>&lt;1</td>
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<tr>
<td>B cells (SMIg) (%)</td>
<td>12</td>
<td></td>
<td>2</td>
</tr>
</tbody>
</table>

*OKT antisera (Ortho Diagnostic Systems Limited).

Conversion: SI to traditional units—total white cell count; neutrophils; lymphocytes $10^9/\text{l} = 10^{-3}/\text{cu mm}$.

### Discussion

Chronic lymphocytic leukaemia is a clonal lymphoproliferative disorder commonly seen in adults, but there is doubt about whether it occurs in children. Most patients have B cell neoplasms and only 2% show T cell characteristics, but there is a rare and possibly related syndrome where a T cell lymphocytosis is associated with neutropenia. In this disorder no evidence of clonality is found and although some patients have recurrent infections, the clinical course is more benign than the clearly neoplastic T cell chronic lymphocytic leukaemia. The lymphocytes show a cytotoxic suppressor phenotype (OKT8 positive) while the helper-inducer cells (OKT4 positive) are decreased. There is no anaemia, no lymphadenopathy (though moderate splenomegaly is common), chromosomes are normal, and the disorder may remain stable for many years. Our patient seems to have this disorder, although to our knowledge she is the first case described in childhood. She still has a very high peripheral blood lymphocyte count and sharp increases in the count after splenectomy have been noted in this disorder. It is too early to say whether recurrent infection will be a problem but so far she seems to have had less trouble than might have been expected. The long-term outlook is unknown, and at present she is being followed without specific treatment other than penicillin (post splenectomy).

### References


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