Hypoxic stillbirth due to entangled intrauterine catheter

Uterine pressure is recorded, using polyvinyl intrauterine catheters, in many obstetric units practising active management of labour. It carries only a small risk to mother or fetus. There have been only sporadic reports of uterine perforation or disruption of fetal vessels. A case has been described of umbilical cord entanglement. We report a similar experience.

Case report

The patient, a 31-year-old primigravida, had conceived after 10 years of voluntary infertility. She was admitted at 38 weeks' gestation for rest and observation because of pre-eclampsia. The results of tests for fetal welfare were satisfactory. Labour was induced at term by artificial rupture of the membranes and intravenous oxytocin. A fetal scalp electrode was applied and an intrauterine pressure catheter introduced to 15 cm by the standard technique. The fetal heart rate at that time was normal. Initial progress in labour was slow but acceptable. A lumbar epidural block provided analgesia. After several hours of strong contractions variable decelerations of the fetal heart rate (FHR) were noted (figure) and the progress of labour was unsatisfactory. It was decided to perform lower-segment caesarean section. An attempt to remove the intrauterine catheter met with resistance and coincided with a sharp fall in the FHR, which recovered after several minutes. When the patient was anaesthetised a further attempt to withdraw the catheter was made but failed. A fresh stillborn infant weighing 3560g was delivered. At operation the catheter was found to be acutely kinked and entangled in the umbilical cord, which was compressed.

Comment

Usually intrauterine pressure monitoring using a polyvinyl intrauterine catheter is safe. In our case we did not appreciate that the catheter was compressing the cord during labour, producing variable deceleration of the FHR. Trying to remove it led to intrauterine asphyxia and fetal death. If variable decelerations in FHR occur with an intrauterine catheter in situ umbilical cord compression by the catheter should be considered. When a gentle attempt to remove the catheter meets with resistance or produces further deceleration of the FHR further attempts to remove it should not be made.

Successful treatment of malignant testicular teratoma with brain metastases

The outlook for some patients with metastatic testicular teratoma has improved considerably with recent advances in chemotherapy. The presence of brain metastases, however, is generally accepted as unfavourable and we have found no reports of successful treatment. We report a case in which extensive chemotherapy combined with surgery and radiotherapy seems to have been effective.

Case report

A 25-year-old postgraduate student presented in December 1974 with a swollen right testis. Orchidectomy and histological examination showed a malignant teratoma with trophoblastic features. Lymphangiography showed metastases in iliac nodes. He was treated by pelvic and para-aortic irradiation to a dose of 4000 rads. He remained well until January 1976. He then developed a right lower-lobe pneumonia as well as severe frontal headaches followed by a mild left hemiparesis. Chest x-ray examination confirmed the presence of metastatic tumour in the right lower lobe and also in the left mid-zone. Computerised tomography (CAT) of the brain showed deposits in the right parietal and right frontal regions. His serum gonadotrophin (HCG) concentration was 52 000 IU/l. Alpha fetoprotein (AFP) was indetectable. Lymphangiography showed no evidence of intra-abdominal metastases.

Treatment started in January 1976. Over the next 12 months he received 19 courses of systemic chemotherapy, usually in conjunction with intrathecal methotrexate (figure). By January 1977 there had been a partial response, as judged by: (1) resolution of the hemiparesis after two months; (2) sequential CAT brain scans showing improvement but also a persistent abnormality in the right parietal area; (3) sequential chest radiographs showing partial resolution but isolated shadows in the right and left mid-zones; (4) serial serum HCG concentrations, initially falling to normal after 10 weeks' treatment but later rising, indicating persistent active tumour (figure). Consecutive right and left thoracotomies to remove residual lung metastases were performed in February and March 1977 (Mr A R Makey). A lesion from the right middle lobe contained only necrotic tissue.
Lymphoproliferative disease in two sisters

Several instances of families with several members with chronic lymphatic leukaemia (CLL) have been described.\(^1\) However, few if any such families have been investigated by modern immunological methods that distinguish different varieties of CLL. We report here the cases of two sisters in a family who had lymphocytosis of CLL type one of whom had the characteristics of ordinary CLL and the other of prolymphocytic leukaemia. The two sisters were from a family of eight children, including five sisters. The parents were not known to be related. One sibling had a carcinoma of colon but, apart from the sisters investigated, the others had unremarkable medical histories.

Case reports

One sister (case 1), aged 82, was seen at Rossendale Hospital in November 1976 complaining of fainting attacks. She had a greatly enlarged liver and spleen but no other lymphadenopathy. The haemoglobin concentration was 7.9 g/dl and white cell count 12.6 × 10\(^9\)/l (12 600/mm\(^3\)) with polymorphs 19%, lymphocytes 78%, and monocytes 3%. The patient was known to have lymphocytosis. The haemoglobin concentration rose to 14.7 g/dl in July 1978 after treatment with iron. The white cell count at that time was 35 × 10\(^9\)/l (35 000/mm\(^3\)) with polymorphs 7%, lymphocytes 92%, and monocytes 1%. Many of the lymphocytes were large, had prominent nucleoli, and appeared to be prolymphocytes. She appeared well and had no lymphadenopathy. She had had three children, one of whom had died of carcinoma of the pancreas. The other sister (case 2) was aged 75. She was investigated for tiredness at Rossendale Hospital in 1976. The haemoglobin was 12.4 g/dl, WBC 69 × 10\(^9\)/l (69 000/mm\(^3\)), with almost all mature lymphocytes, and the bone marrow was reported as showing 80% lymphocytes. Enlarged lymph nodes were found in the submandibular regions and in the right axilla. The liver and spleen were not palpable. She had had four children, one of whom had died of Still's disease and one of carcinoma of the ovary.

Blood from each patient was examined at the Christie Hospital in 1977 and 1978 with the following similar findings on each occasion. Case 1—WBC 25 × 10\(^9\)/l (25 000/mm\(^3\)) with polymorphs 8%, lymphocytes 68%, monocytes 2%, prolymphocytes 15%, and smears 7%. Most of the lymphocytes were found to be negative with the PAS test. Case 2—WBC 296 × 10\(^9\)/l (296 000/mm\(^3\)) with polymorphs 1%, lymphocytes 78%, prolymphocytes 1%, and smears 20%. Most of the lymphocytes had fine PAS-positive granules and were small and of mature appearance. Cytogenetic studies of the peripheral blood cells of both patients by Dr S Muldahl showed no definite abnormality. The results of lymphocyte surface marker tests are shown in the table. The tests were done on the same day and the same antisera, sheep and mouse red cells were used in each case. The results show the characteristics of CLL and prolymphocytic leukaemia, or lymphosarcoma cell leukaemia, respectively.\(^2\) Both sisters show proliferation of a monoclonal of lymphocytes with kappa light chain surface immunoglobulin (SIg). In case 2 there was a small amount of SIg per cell and high mouse red cell rosettes (MRFC), characteristic of CLL. In case 1 there was a large amount of SIg per cell, frequent capping, and low MRFC, characteristic of prolymphocytic and of lymphosarcoma cell leukaemia.

Results of lymphocyte surface marker tests in two sisters with lymphoproliferative disease

<table>
<thead>
<tr>
<th>Case</th>
<th>Poly-Ig</th>
<th>Poly-Ig, fragment of anti-Ig</th>
<th>Anti-CD3</th>
<th>Anti-CD15</th>
<th>Anti-CD20</th>
<th>Anti-CD23</th>
<th>Anti-CD24</th>
<th>Anti-CD34</th>
<th>Mouse ERFC</th>
<th>Sheep ERFC</th>
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<td>+</td>
<td>+</td>
<td>+</td>
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<td></td>
<td></td>
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<td>7%</td>
</tr>
<tr>
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<td>+++</td>
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<td>+</td>
<td></td>
<td></td>
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<td></td>
<td>7%</td>
<td>90%</td>
</tr>
</tbody>
</table>

Comment

The relationship of CLL, prolymphocytic leukaemia, and lymphosarcoma cell leukaemia to one another is not clear. The simultaneous occurrence of two forms of lymphoproliferative disease in two sisters seems more likely to be associated with some inherited instability of the lymphocyte system rather than to be fortuitous. The investigation of further families by modern immunological methods may contribute to our understanding of these conditions.

We thank the late Dr R D Popham, of Bury General Hospital, for first drawing our attention to these patients; Dr Grimshaw, of Rossendale Hospital, and Drs A and D Rushby, of Christie Hospital, for helpful discussion.

\(^1\) Williams, C, Cancer Treatment Reviews, 1977, 4, 275.

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