and the oedema disappeared within four days. One month later he was again given, by another doctor, nifedipine 10 mg three times daily and three days later he again developed dyspnoea and oedema of his legs. When I saw him one week later he had signs of congestive heart failure. His jugular venous pressure was raised (5 cm at angle 45°), the apex beat was 1 cm to the left of the midclavicular line at the fifth intercostal space, his ventricular rate was regular at 68/min, and he had bilateral fine basal crepitations and moderate pitting oedema of his legs. His resting electrocardiogram was unchanged. He was treated with frusemide, and propranolol and nifedipine were discontinued. After four days he had no signs of heart failure. After one week he was put on metoprolol 300 mg daily. After 30 days' follow-up he had no signs of heart failure.

Comment

Side effects of nifedipine are rare. They are more common at the beginning of treatment. They include headache, facial flush, a sensation of heat, dizziness, nausea, and tiredness. Very rarely, oedema of the lower extremities may occur, as with other vasodilator drugs. The patients reported on here developed heart failure after nifedipine was added to a beta-blocker. When nifedipine was discontinued there were no signs of heart failure, although both patients were kept on the beta-blocker. After the second patient was restarted on nifedipine heart failure recurred.

When nifedipine is given with beta-blocker drugs it should be with caution.

[1] Anonymous. Drug treatment of chronic stable angina pectoris. *Br Med J* 1978;i:462-3.
[2] Gilmer DJ, Kark P. Pulmonary oedema precipitated by nifedipine. *Br Med J* 1980;**280**:1420-1.

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Department of Medicine, Limassol Hospital, Cyprus
C J ANASTASSIADES, MD, physician in charge

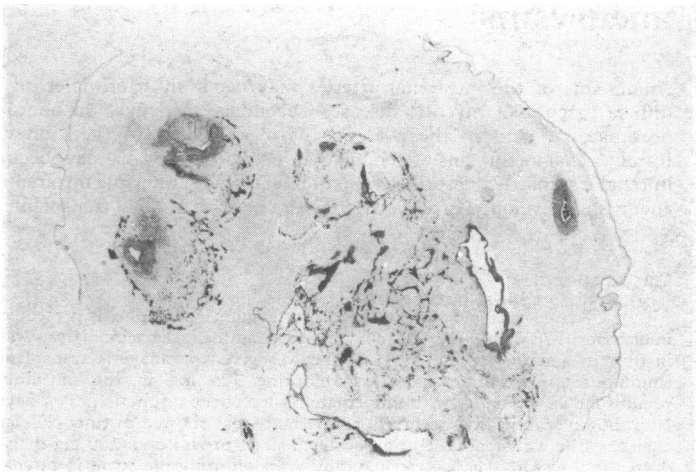
Haemangioma of the cord: further cause of raised maternal serum and liquor alpha-fetoprotein

We report a cause of raised maternal serum and liquor α-fetoprotein concentrations which has not, so far as we know, been previously described.

Case report

The patient was a 28-year-old gravida 2 Iraqi. Her first pregnancy resulted in the delivery of a normal female infant at term, birth weight 3200 g. The marriage was non-consanguineous and there was no family history of fetal malformation. The maternal serum α-fetoprotein concentration at 16 weeks' gestation was raised at 65 μg/ml (2 × median). At 19 weeks' gestation it was 165 μg/ml (3 × median), so an amniocentesis, yielding clear liquor, was performed 24 hours later. The liquor α-fetoprotein concentration of 61 μg/ml was 4·6 × median. (In the UK collaborative study[1] only 0·15% of clear fluids from pregnancies with normal outcomes had amniotic α-fetoprotein concentrations at 19-21 weeks above four times the median and none exceeded 4·1 × median.) The qualitative amniotic fluid acetylcholinesterase test by gel electrophoresis[2] was negative, showing only one band. Ultrasound examination at 19 weeks showed a single, viable fetus with an anterior placenta. The liquor volume was within normal limits and the gestational age was confirmed. Although further ultrasound examination of the fetus showed no abnormality of the neural tube, neck, anterior abdominal wall, or kidneys, the patient and her husband were counselled that the raised liquor α-fetoprotein concentration indicated a high risk of fetal malformation. Both agreed to have the pregnancy terminated, and this was done with extra-amniotic prostaglandin.

A female fetus (karyotype 46 XX) was delivered weighing 362 g and measuring crown-heel 26·4 cm and crown-rump 18·0 cm. No malformation could be detected either externally or on gross dissection of the cranium, spine, thorax, and abdomen. Histological examination of all the major organs, including kidneys, showed no abnormality. The placenta weighed 133 g and was grossly and microscopically normal. The umbilical cord contained three vessels and was approximately 0·7 cm diameter throughout except for the placental extremity, which displayed a rounded swelling 1·5 cm across contiguous with the placental amnion. Histological examination of this showed the presence of a capillary haemangioma within the Wharton's jelly. A minority of the vascular spaces were thrombosed and appeared to communicate with vessels in the placenta rather than with those in the cord (figure).



Cross section of umbilical cord swelling showing numerous irregular vascular spaces within Wharton's jelly × 8·7 (original magnification).

Comment

Vascular hamartomatous tumours of the umbilical cord are rare. Fox[3] reviewed 15 cases of cord haemangioma and Fortune and Ostor[4] recently reviewed 12 cases, preferring the term angiomyxomas of the cord. In common with placental haemangiomata, they are probably often overlooked without a specific search. The present case is important in that the tumour may account for high serum and liquor α-fetoprotein concentrations in an apparently normal pregnancy.

Although amniotic fluid α-fetoprotein concentration is in most cases a reliable indicator of a fetus with an open neural tube defect, several other conditions may also cause raised concentrations—for example, exomphalos, Finnish-type nephrotic syndrome, and Turner's syndrome. Careful examination of the fetus by ultrasound and the acetylcholinesterase gel technique may help differentiate these conditions. Raised α-fetoprotein concentration with normal acetylcholinesterase in a clear fluid indicates a lesion other than neural tube defect in the fetus, but the false-negative rate of the acetylcholinesterase test is uncertain. Nevertheless, while both ultrasound and fetoscopy might theoretically detect an umbilical cord swelling, the diagnosis of haemangioma cannot be made with certainty without histological examination.

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[2] Smith AD, Wald NJ, Cuckle HS, Stirrat GM, Bobrow M, Lagercrantz H. Amniotic fluid acetylcholinesterase as a possible diagnostic test for neural tube defects in early pregnancy. *Lancet* 1979;i:685-8.
[3] Fox H. *Pathology of the placenta*. Philadelphia: W B Saunders, 1978:448-9.
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University Departments of Pathology, Obstetrics, and Medical Genetics, St Mary's Hospital, Manchester M13 0JH
A J BARSON, MD, DCH, consultant paediatric pathologist
P DONNAI, MB, MRCOG, consultant obstetrician
A FERGUSON, MB, DCH, senior house officer in obstetrics
D DONNAI, MRCP, DCH, consultant in medical genetics
A P READ, MA, PHD, lecturer in medical genetics