Diagnosis of closed neural tube defects by ultrasound in second trimester of pregnancy

By applying the techniques of maternal serum alpha-fetoprotein (AFP) screening and amniotic fluid AFP studies it is now possible to detect over 90% of open fetal neural tube defects^{1 2} early in the second trimester of pregnancy. Campbell³ has reported similar success rates in visualising both anencephaly and open spinal defects by ultrasound at this stage of pregnancy in "at risk" patients. In general, AFP studies do not identify fetuses with closed defects, nor have there been any reports of the ultrasound diagnosis of such cases before 20 weeks. We describe two cases of closed defects diagnosed by ultrasound at 18 weeks.

Case reports

Case 1—A 27-year-old para 2+2 was referred because her first baby, who had died shortly after birth, had had a large open spinal defect. Her second child had been normal. In her current pregnancy an ultrasonic examination at 18 weeks showed appreciable widening of the normal cervical spinal echoes, and a well-defined 2-cm diameter cystic area "attached" to the lower occipital area and the upper cervical spine (figure). The biparietal diameter of the fetal head was within normal limits, and the ventricular echoes appeared normal, as did the remainder of the spine. The amniotic fluid AFP concentration was 8 mg/l, well within the normal range for 18 weeks.

Section A shows a longitudinal scan of the fetus (case 1) along the spinal canal. At its upper end the canal is abnormally wide (Abn Sp). In B a cystic structure, the encephalocele (En), is seen attached to the lower occiput, and C shows the fetus after therapeutic abortion. The encephalocele, which ruptured during delivery, was completely covered by skin, as was the spinal canal.

In view of the conflicting evidence of a neural tube defect, the ultrasonic scan was repeated and the previous findings were confirmed. After detailed counselling, the pregnancy was terminated. Examination of the aborted fetus showed a completely skin-covered occipital encephalocele. The cervical spine was abnormally wide and the laminae were of cartilaginous rather than bony tissue. The lower spine was normal.

Case 2—A 31-year-old para 1+0 was referred at 18 weeks because her previous pregnancy had resulted in a mentally normal but severely physically handicapped child, whose abnormalities had included extensive spina bifida occulta, gross scoliosis, and unilateral absence of ribs. On the basis of several ultrasonic examinations a small low spinal defect and a large exomphalos were provisionally diagnosed. Two amniotic fluid AFP estimates were between the third and fourth SD levels respectively—that is, not in the range normally associated with an open neural tube defect. Because of the ultrasound findings and the existing burden of a handicapped child, the pregnancy was terminated. The presence of an exomphalos was confirmed and its intact sac found to contain the liver. No obvious lesion of the fetal back was seen but dissection showed spina bifida occulta of the lower lumbar and sacral regions.

Comment

Over 10% of neural tube defects are covered by skin, the lesions ranging from small innocuous areas of spina bifida occulta to large myeloceles and encephaloceles, many of which result in major handicap.^{2 4} Since the skin cover precludes the permeation of AFP from the fetal cerebrospinal fluid into the amniotic cavity, AFP studies have been unhelpful in such cases.^{1 2} Thus diagnostic ultrasound currently offers the only practical alternative for their early detection.

The closed defects described here represent both ends of the range. In the first the neural tube abnormality was major, and early diagnosis and selective termination were therefore clearly justified. The second case, however, poses problems for the future application of ultrasound in this type of diagnosis. Although in this case there was an associated exomphalos, we can foresee the problem when a termination of pregnancy might be considered after the ultrasonic demonstration of a clinically insignificant defect. Despite this cautionary note, we think that diagnostic ultrasound has an important part to play in detecting major closed defects, and we would therefore recommend that a careful examination is made in all "at risk" patients before amniocentesis. Where any doubt exists about the nature and severity of the lesion after these procedures, recourse to fetoscopy would be a useful, though invasive, technique.

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Anuria in pregnancy

Anuria in late pregnancy is normally associated with hypotension, infection, or severe toxæmia.¹ Cases without any obvious cause other than the gravid uterus are rare.²⁻⁴ It seems to be most prevalent in the seventh month of a first pregnancy.³ Treatment has been by ureteric catheterisation or nephrostomy. When there has been no antecedent lesion the prognosis for mother and baby has been good except in one case⁴ where the fetus was dead on admission. Pressure by the pregnant uterus on the already dilated ureters has been suggested as a possible cause.^{2 3} I report a case of complete anuria lasting 48 hours for no obvious cause. The pregnancy was further complicated by deep vein thrombosis.

Case report

The patient, a primigravida, was admitted as an emergency after 28 weeks of amenorrhoea. She gave a history of bilateral loin pain, vomiting, and anuria for 36 hours. Before that her antenatal record had been normal. Her past medical history was unremarkable apart from epilepsy controlled with phenobarbitone 30 mg daily. She was afebrile, normotensive, and without oedema. She had bilateral loin tenderness but neither kidney was palpable. The uterus contained a 30-week pregnancy with the baby presenting by the head. The bladder contained only 4 ml of blood-stained urine. Haemoglobin was 14.3 g/dl, white blood cell count $14.3 \times 10^9/l$. The results of blood chemistry investigations were: sodium 135 mmol(mEq)/l, potassium 4.5 mmol(mEq)/l, chloride 100 mmol(mEq)/l, bicarbonate 24 mmol(mEq)/l, urea 13 mmol/l (71.7 mg/100 ml), creatinine 582 $\mu\text{mol/l}$ (6.6 mg/100 ml), raised total plasma oestriol at 1699 nmol/l (48.9 $\mu\text{g}/100\text{ ml}$), and normal free or unconjugated plasma oestriol at 29 nmol/l (840 ng/100 ml). An intravenous pyelogram showed normal-sized kidneys with delayed nephrograms and no filling of calices after 10 minutes. A renal ultrasound scan showed dilated collecting systems in both kidneys, suggesting bilateral obstruction.

Frusemide 40 mg intravenously had no effect. A central venous pressure

line was inserted and an arteriovenous shunt created in the left leg. After haemodialysis for eight hours the patient passed urine for the first time in 48 hours. A diuresis occurred over the next two days and the blood chemistry reverted to normal. On the fifth day after admission a left deep vein thrombosis developed, which was treated with intermittent subcutaneous heparin 5000 units eight-hourly. The maternal and fetal conditions remained satisfactory and urinary and plasma oestriol concentrations were within normal limits. After 36 weeks' amenorrhoea spontaneous labour occurred and a healthy male infant weighing 3.6 kg was delivered. The puerperium was uncomplicated. Anticoagulation was continued for six weeks. Urine analysis and blood pressure were normal. Two weeks after delivery an intravenous pyelogram and renal scan showed normal postnatal appearances with slightly dilated collecting systems. At the postnatal visit mother and baby were well.

Comment

The absence of infection, shock, or toxæmia and the complete reversibility of the condition suggests that pressure from the gravid uterus may well have been a contributory factor in this case. The short duration and favourable outcome seem to rule out a serious lesion. In the absence of gross hydronephrosis and with the almost universal availability of dialysis units haemodialysis seems to offer a more rational approach to these unusual and mystifying cases.

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Anergy—a prognostic indicator in early breast cancer

The type IV delayed hypersensitivity skin response to recall antigens may be reduced in early breast cancer.^{1,2} The degree of anergy apparently increases as the disease progresses.² We report a three-year follow-up of 100 patients with early breast cancer who were skin tested preoperatively with three recall antigens.

Patients, methods, and results

The patients were part of a unicentric randomised prospective clinical trial comparing simple mastectomy alone with simple mastectomy and radical radiotherapy as treatments for early breast cancer (T1 and T2, N0 and N1, M0). Axillary lymph node biopsy for more accurate staging and a skeletal survey were done preoperatively in all patients but bone scanning was not routine. Ten units purified protein derivative (PPD), 0.02 ml 0.5% *Candida albicans* (Bencard), and 10 units Varidase (streptokinase/streptodornase) were injected intradermally into the forearm opposite the affected breast and the diameter of induration at 72 hours measured: 5 mm or over was called positive.

Seventeen patients failed to react to any of the recall antigens. Ten (59%) of these 17 anergic patients had died or had distant metastases by three years, whereas by then only 23 (28%) of the 83 reactive patients had died or developed metastases. This difference is highly significant (χ^2 with Yates's correction = 6.96, $P = < 0.01$). Treatment did not affect the clinical outcome,³ and five of the 10 anergic patients had had additional radical radiotherapy as a primary treatment. Six (86%) out of seven anergic patients who also had histological evidence of axillary lymph node involvement had died or developed metastases by three years. The mean ($\pm$ SD) age of the anergic patients (62.8 ± 5.9 years) was significantly greater than that of the reactive patients (53.7 ± 8.6 years) ($t = 5.26$, $P = < 0.01$).

Comment

Eilber and Morton⁴ reported that over 90% of unselected cancer patients presenting for surgery who failed to react to dinitrochlor-

benzene (DNCB) were either inoperable or had early recurrence. Our study shows that anergy is also a prognostic indicator in early breast cancer, since over half (59%) of the patients who failed to react to any of three recall antigens had died or had distant metastases by three years. Perhaps anergy to a selection of recall antigens could be used in conjunction with other high risk factors, such as lymph node involvement, in selecting patients for adjuvant therapy. Whether anergic individuals are more prone to develop cancer or cancer induces a state of anergy has yet to be determined. Anergy does become more marked with advancing disease,² however, and failure to react may well be related to this. Our patients who had died or developed distant metastases by three years must have had occult metastases when they were skin tested, which may have affected their responsiveness. The results also confirm the reported⁵ correlation between anergy and advancing age.

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Stercoral perforation: case of drug-induced impaction

Stercoral perforation of the colon, a rare complication of faecal impaction, often results in life-threatening peritonitis. This case report illustrates a potential hazard in giving drugs with anticholinergic side effects to patients with refractory constipation.

Case report

A 74-year-old woman was admitted four hours after sudden onset of severe abdominal pain maximal in the left iliac fossa. Before the onset of pain she had passed a small, hard motion with some bright blood after prolonged straining. She was an active woman prone to severe constipation since childhood. She also had bouts of depression, for which she had taken amitriptyline 10 mg twice daily for four months. During this period her constipation had worsened. Her only other medication was benorylate suspension, which she took occasionally for backache. She looked ill, her facies were ashen, and extremities cyanosed. Her blood pressure was 200/100 mm Hg and pulse rate 100/min. Signs of general peritonitis were present, and rectal examination yielded blood-stained faeces with marked tenderness antero-superiorly. Investigations did not contribute to the diagnosis, which was thought to be a perforated diverticulum.

At laparotomy generalised faecal peritonitis was found associated with a 2-cm circular perforation with bruised ragged edges on the antimesenteric border of the rectosigmoid. There were several scybala in the pelvis and extensive faecal staining of the peritoneum. The entire colon and terminal 90 cm of ileum were solidly impacted with faeces. No other abnormality of the bowel was seen. After scrupulous peritoneal lavage Hartmann's procedure was performed. Intravenous antibiotics and metronidazole were started intraoperatively. Postoperative recovery was complicated by wound infection and deep venous thrombosis which developed after 11 days. She progressed satisfactorily but required regular colostomy enemas for persisting constipation.

Comment

Fewer than 40 cases of stercoral perforation have been reported. Mortality figures are not readily available but faecal peritonitis from all types of colonic perforation treated surgically carries a mortality of about 50%.¹ Perforation occurs most often in the recto- and mid-