LETTERS TO THE EDITOR

Stillbirths and non-lethal abnormalities—a mechanism of death?

SIR,—While counselling parents who have experienced a stillbirth of either a chromosomally abnormal baby or one with non-lethal congenital malformation, we are often asked 'But why did the baby die?'

Why do fetuses with chromosomal abnormalities or congenital malformations that do not involve major cardiovascular or cerebral defects die in utero? It is scientifically inadequate merely to attribute death to the presence of any congenital malformation as only a select few of such malformations can obviously cause death in utero. The latter would include defects such as cerebral tumours and major cardiovascular abnormalities such as transposition of the great vessels, conduction system abnormalities, large septal defects, etc. Death in utero of fetuses with congenital abnormalities without any major organ defects as is the situation with some sex-chromatin abnormalities, trisomy 18, achondroplasia, spina bifida, duodenal atresia, unilateral pulmonary agenesis, etc is not so easily explained.

Studies have shown that chromosomal abnormalities are 10 times more frequent in perinatal deaths than in unselected liveborn infants.[1 2] This does not, however, explain the mechanism of death in utero as most of the chromosomal abnormalities detected in perinatal deaths are compatible with survival after birth and are not usually of the types seen in early abortions.[2]

In a study of fetal heart rate patterns in fetuses with congenital abnormalities, Garite *et al* showed a significant increase of heart rate abnormalities but no characteristic pattern that would specifically identify major congenital abnormality.[3] Macafee *et al* had found a high incidence of growth retardation (46%) associated with major fetal malformations.[4] This may suggest a form of uteroplacental insuffiency and Garite *et al* postulated that abnormal placentation probably accompanies abnormal fetal development despite lack of histological evidence in many cases.[3] The high early miscarriage rate seen with chromosomally abnormal conceptuses probably supports such a theory. Fetal cardiac and neurologic normalcy are perhaps necessary for coordination of adequate nutritive and respiratory placental exchange. This theory however, is not supported by the finding of symmetrically small for date babies with chromosomal abnormality associated with adequate liquor volume (K H Nicolaides, personal communication).

Amniotic disruptions and limb-body wall disruptions can cause both external and internal malformations that can often be difficult to differentiate from malformations due to a primary genetic cause.[5] This, however, leaves a legion of stillbirths in chromosomally and congenitally malformed fetuses where there is no obvious mechanism of death and counselling is therefore less than accurate.

Why do these babies die?

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Renal candidiasis in the preterm infant

SIR,—We read with interest the case report by Drs Rehan and Davidson of a preterm infant with systemic candidiasis and renal candida bezoar. They state: 'to our knowledge there has been no report of a neonate with candidal septicaemia and renal candidal bezoar who has survived without surgical intervention.'[1] We report a preterm infant with systemic candidiasis and candida renal abscess who not only survived with medical management alone but at the age of 13 months has normal renal function.

Case report

A boy was born at 25 weeks' gestation, weighing 840 g. He required prolonged ventilatory support until day 22 and also inotropes, two exchange transfusions, a long line for parenteral nutrition, and repeated courses of broad spectrum antibiotics for suspected bacterial sepsis. He also had bilateral intraventricular haemorrhages and developed arrested posthaemorrhagic hydrocephalus.

On day 45 there was generalised clinical deterioration with recurrent apnoeas and abdominal distension, requiring reventilation. A full infection screen was performed and antibiotics commenced. Three days later there was a palpable left kidney and *Candida albicans* was grown from a suprapubic urine and blood cultures. The cerebrospinal fluid showed no fungal growth and candida serology was negative.

Renal ultrasound showed a hydronephrotic left kidney containing large amounts of debris and a discrete fungus ball. The left kidney was non-functional on intravenous urogram. Antifungal treatment was commenced immediately using intravenous amphotericin and intravenous, then oral 5-flucytosine. Drug concentrations of the latter were monitored and in the therapeutic range. Perineal and oral nystatin were also given. Treatment was continued for a total of six weeks and was well tolerated. Plasma urea and serum creatinine concentrations remained normal throughout. There was close consultation with the paediatric surgeons—to intervene if the fungal debris persisted and the kidney remained non-functioning—but there was continued clinical, radiological, and functional improvement.

A dimercaptosuccinic acid (DMSA) scan 4·5 months later showed an anatomically normal left kidney contributing 40% of total renal function. Ultrasound revealed a little fullness of the left upper pole collecting system only. Subsequent blood and urine cultures remained sterile. General progress 13 months later remains entirely satisfactory with normal growth and renal function.

Systemic candidiasis is well documented in the very preterm and low birthweight infant with a reported incidence of 3–4% in those under 1500 g[2]—and an associated high morbidity and mortality. Insertion of indwelling intravascular catheters, provision of total parenteral nutrition, and administration of broad spectrum antibiotics are associated with an increased risk of systemic disease. In the neonate *C albicans* has a particular predilection for the kidneys and of those reported with renal candidiasis, most have been treated by nephrostomy drainage with both systemic and local antifungal therapy.[3] Outcome has largely been monitored in terms of survival and there is very little documentation of subsequent measurements of renal function in those who survive. Here then is an infant who has been successfully treated with systemic antifungals, without surgical intervention and at follow up has total resolution of his renal disease.

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