and the actual bicarbonate 16·6 mmol/l. To control the arrhythmia practolol 20 mg was given intravenously over 30 minutes. He was also given 30 units of insulin intravenously and 30 units intramuscularly. Later he became feverish and ampicillin was started. His condition rapidly improved with an immediate fall in the blood pressure to 140/100 mm Hg and the pulse rate to 130/min. The plasma glucose then fell to 10·6 mmol/l (191 mg/100 ml) with an actual bicarbonate concentration of 23 mmol/l.

The patient remained normotensive (blood pressure 130/80 mm Hg) and his diabetes was controlled by Actrapid MC (28 units at 8 am, 24 units at 6 pm). The left hypochondrial mass was shown by intravenous pyelography and abdominal aortography to be a large, left suprarenal tumour. This proved to be a phaeochromocytoma (homovanillylmandelate 238 nmol/24 h). At laparotomy after combined alpha- and beta-blockade,[2] the diabetes being controlled by continuous glucose-insulin infusion, a suprarenal tumour weighing 500 g was removed. Chromaffin staining showed that it was adrenaline secreting. After the operation the patient required no further insulin and the homovanillylmandelate output returned to normal.

Comment

The patient evidently had a chest infection, and this may have triggered off an acute storm, or crisis, due to adrenaline release. The evidence to support an adrenergic crisis is considerable. (1) The blood pressure fell from 190/130 to 130/100 mm Hg after intravenous practolol. (2) A moderate diabetic ketoacidosis was resistant to insulin treatment until after beta-blockade. (3) The inappropriate sweating was not due to fever. (4) The supraventricular tachycardia responded to practolol. (5) The patient's skin was cold, with vasoconstriction and a blue, mottled appearance.

A large rise in circulating adrenaline could explain the hypertension, sweating, tachycardia, and vasoconstriction.[3] This would also decrease circulating insulin and initiate hepatic glucose release.[4] Beta-blockade reduces the metabolic rate, lowers the blood pressure, and decreases the pulse rate in hypermetabolic patients with high urinary catecholamine output.[5] In our patient beta-blockade was undoubtedly life saving. We feel justified in calling this a case of "adrenergic crisis."

We thank Drs J M Fowler and R P L Waldram for their help in preparing this case report.

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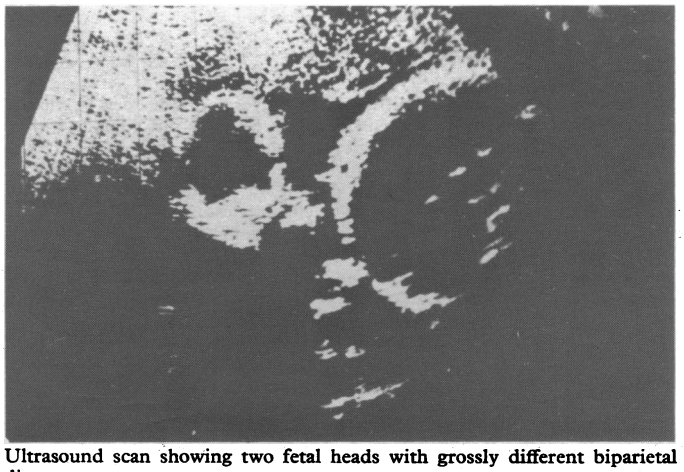
Raised amniotic fluid concentrations of alpha-fetoprotein in a twin pregnancy

Amniotic fluid levels of alpha-fetoprotein (AFP) have been used since 1972 in the diagnosis of neural tube defects of the fetus.[1] At present the normal range for AFP in amniotic fluid is based on tests in single pregnancies because amniocentesis is rarely performed on patients with a multiple pregnancy. We report here a twin pregnancy, initially undiagnosed, in which amniotic fluid AFP levels were raised.

Case report

A 27-year-old primigravida was first examined at 13 weeks' gestation and the uterus noted to be large for dates. Ultrasonic scan showed an apparently single pregnancy at 18 weeks, no abnormalities being detected. At this time plasma AFP was 225 μg/l and a second sample was 560 μg/l at 20 weeks' gestation, both considerably raised concentrations.[2] Clear amniotic fluid was obtained by amniocentesis at 21 weeks and the AFP level was 55 mg/l (upper limit of normal in single pregnancy at 21 weeks 24 mg/l).[3] The cells in the amniotic fluid were karyotyped as normal male.

The patient was admitted to hospital for consideration of termination of pregnancy. A further ultrasonic scan was performed because the uterine size was greater than the period of gestation and showed a twin pregnancy. One infant had a biparietal diameter of 61·5 mm and the other was 31·8 mm, but only one fetal heart movement was observed (see figure). Abdominal *x*-ray examination gave further confirmation. A diagnosis was made of twin pregnancy with death of one fetus. The pregnancy continued to term and a normal baby boy weighing 2·9 kg was delivered as well as a fetus papyraceous.



Ultrasound scan showing two fetal heads with grossly different biparietal diameters.

Discussion

Multiple pregnancy is usually associated with raised concentrations of AFP in maternal plasma. In general obstetric practice, once a multiple pregnancy has been diagnosed, the raised plasma AFP concentration is ignored and amniocentesis is not performed to exclude fetal neural tube defect. Nevertheless, amniotic fluid AFP concentrations may be raised in twin pregnancy in the absence of a fetal abnormality.[4 5] In our patient the raised amniotic fluid AFP concentration may have been due to the multiple pregnancy or to death of one of the fetuses. The amniotic fluid appeared clear and normal, which virtually excludes the possibility that the amniotic sac with the dead fetus was sampled.

To avoid termination of a normal twin pregnancy on the basis of raised maternal plasma concentrations of AFP, leading to amniocentesis, the following criteria should be used. Firstly, a normal range of AFP concentrations in multiple pregnancy should be obtained, but it will take many years before this is available. Secondly, if there is a discrepancy between the gestational age assessed both clinically and by ultrasound, the ultrasound examination should be repeated to exclude a multiple pregnancy.

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