Correspondence

Comparative risks of amniocentesis and fetal blood sampling

Sir,

We read with interest the paper by Youroukos *et al.*¹ on porencephalic cysts after amniocentesis. The report adds a central nervous system malformation to the literature regarding injury to the fetus after second trimester amniocentesis for prenatal diagnosis. The authors state that fetal trauma after second trimester amniocentesis is rare, and that the risk of such complications does not outweigh the value of the procedure in the prenatal diagnosis of β -thalassaemia.

In fact, the prenatal procedure used was not amniocentesis (removal of amniotic fluid), but fetal blood sampling. Midtrimester amniocentesis for antenatal detection of chromosomal and some genetic and congenital defects in at-risk populations is an accepted medical procedure. The risk of damage is low (about 0.5 to 1% at experienced centres).²

Fetal blood sampling however, is currently considered to be at an applied research state, and carries a much higher risk for the fetus (about 9.4% at experienced centres).³

It is important that the two techniques should remain separate when assessing the risks of fetal injury. Families who might benefit by amniocentesis might well deny themselves testing because of incorrect information.

References

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Porencephalic cysts as a result of placental damage at amniocentesis?

Sir,

Youroukos *et al.*¹ reported on an infant with porencephalic cysts after midtrimester amniocentesis for exclusion of β -thalassaemia. Obviously, the placenta was punctured without fetoscopy and fetal blood was obtained. There were no problems in obtaining fetal blood nor were there other apparent complications of the procedure. The

authors considered brain damage through the amniocentesis needle the most likely origin of the porencephalic cysts, especially as 2 subcutaneous nodules were overlying the defect.

I should like to suggest another mode of origin for the cysts. The puncture of the placenta, on which occasion 'several specimens of fetal blood were obtained', probably resulted in placental damage. Embolisation of clots of necrotic tissue or of thrombotic material via the umbilical vein might have led to occlusion of a fetal cerebral artery. Consequent brain necrosis could have resulted in a porencephalic cyst. Such events have often been observed in monovular twin gestations after the death of one twin with a common placenta with vascular anastomoses.^{2,3} The most common defects were porencephalic cysts, microcephaly, or hydrocephaly; however, skin lesions similar to the nodules in the patient of Youroukos *et al.*, have been observed too. A Japanese group³ has recently shown by combined angiography and computerised tomography scan that a cerebral artery ended abruptly at the location of a cyst. Hence vascular occlusion was the most likely cause of the cyst.

In the case reported by Youroukos *et al.*¹ several observations leave both possibilities open or might even favour a placentogenic origin of the cysts. Firstly the fact that the fetus (which would have been severely damaged by the needle) was not spontaneously aborted, secondly the uneventful puncture, and thirdly the discrepancy between severe brain damage and discrete 'needle points'.

In this particular case neither direct nor indirect damage of the fetus could be demonstrated; however, when using invasive prenatal measures the possibility of indirect damage of the fetus from embolic events caused by placental damage through a needle puncture should be considered.

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Metoclopramide poisoning in children

Sir,

I read with great interest the paper by Low and Goel¹ and should like to comment on two points: