Figure 1. Initial MR angiogram. A single aneurysm is seen at the bifurcation of the basilar artery (arrow)

Figure 2. Left internal carotid digital subtraction arteriogram 9 days later. A *de novo* saccular aneurysm is seen at the bifurcation of the left middle cerebral artery (arrow)

Figure 3. MR angiogram 16 days after initial subarachnoid haemorrhage. The left middle cerebral artery is completely occluded proximal to the aneurysm, which is no longer visible

Could the middle cerebral artery aneurysm have been missed at the original magnetic resonance angiography? Digital subtraction arteriography is certainly more sensitive; however, a basilar bifurcation aneurysm and the first and

second portion of the left middle cerebral artery are clearly seen in the first magnetic resonance angiogram. Moreover, on examination of the published work⁴⁻⁶ I could find no report of false-negative magnetic resonance angiography for an aneurysm greater than 10 mm in diameter. On this evidence I conclude that this aneurysm did not exist at the time of initial study 9 days earlier.

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Aortic root replacement in pregnancy

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When complex cardiac operations become necessary during pregnancy, timing is crucial to survival of the fetus.

CASE HISTORY

A woman of 31, in the 10th week of her first pregnancy, reported palpitations. She was tall, with fingers and toes unusually long for her family, and had 'double-jointed' thumbs. She was also short-sighted and her hearing was poor. There was no medical or family history of note. The pregnancy had followed 3 years of infertility treatment. Aortic regurgitation was diagnosed clinically, and she met

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the criteria for Marfan's syndrome. On transthoracic and transoesophageal echocardiography the aortic regurgitation was severe, with dilatation of the aortic root and ascending aorta (root 6.9 cm in diameter). There was no dissection. A cardiac surgical opinion was sought, and after discussions between patient, cardiologist, surgeon, cardiac anaesthetist and obstetrician, she was given a date for surgery in the 12th week of pregnancy.

She underwent aortic root replacement with a 27 mm Perimount tissue valve and the ascending aorta was replaced with a 30 mm Gelseal graft. Cardiopulmonary bypass was specifically tailored for this case: pulsatile perfusion was used, with the aim of maintaining mean arterial pressure at 65 mmHg or above, with antegrade cold blood cardioplegia and extensive topical cooling of the heart, although the patient was systemically cooled to 35 °C only; circulatory arrest was avoided entirely. Anaesthesia was maintained with isoflurane, and a glyceryl trinitrate infusion was used to prevent uterine contractions. Fetal heart rate was monitored with a cardiotocograph and no significant decelerations were observed intraoperatively. The patient was weaned easily from cardiopulmonary bypass without inotropic support.

Postoperatively, she was transferred to the intensive care unit where she was extubated three hours later. Fetal monitoring continued for 24 hours and a fetal scan on postoperative day 1 was normal. She was discharged eight days after surgery. Histology of the aorta revealed cystic medial necrosis. At 38 weeks the baby was delivered by planned caesarean section.

COMMENT

When cardiac surgery is performed during pregnancy, fetal mortality is 20–35%, and is highest after closure of intracardiac shunts and aortic valve replacement; maternal mortality is now at the lower end of the reported 3–15%. It is the additional cardiac burden associated with pregnancy that often causes heart disease to show itself at this time. When surgery is needed, timing is of key importance to the welfare of the fetus. During the first trimester, any injury to the fetus, whether due to drugs, hypoxia or changes in blood flow, has a high probability of causing congenital defects and spontaneous abortion. The third trimester is a time of low risk for the fetus, especially beyond 28 weeks, since if labour is precipitated by surgery there is a good chance the baby will survive on a neonatal unit, and the fetus is more resilient in the face of cardiopulmonary bypass at this stage. However, by the third trimester, a mother requiring surgery for a cardiac defect will be in a worse state because of the

extra cardiac output (stroke volume 30% greater, blood volume 30–50% greater, heart rate 10–15% greater) required for a fetus of that size, and the risk of waiting may be too great. In the second trimester the risk for the mother is probably no greater than that for a non-pregnant woman, and the risk of premature labour is also lower at this time.

The changes in fetoplacental perfusion during cardiopulmonary bypass are poorly understood, despite new methods for monitoring flow in the uterine artery, ductus venosus and fetal aorta. It is known that hypothermia can cause fetal hypoxia and that rewarming can likewise cause hypoxia by inducing uterine contractions. There are only a few reports of surgery with circulatory arrest and deep hypothermia, and in all of these the fetus died post-operatively. During cardiopulmonary bypass, haemodilution, lack of pulsatile flow, uterine arterial spasm and particulate microemboli may all alter placental perfusion and contribute to fetal hypoxia. With pulsatile perfusion during cardiopulmonary bypass, the hazard of vasoconstriction in placental vessels, including spiral arteries, is believed to be lessened by release of nitric oxide.² Thus, in pregnancy cardiopulmonary bypass is best conducted with mild hypothermia, pulsatile perfusion, high flow rates and minimal haemodilution.^{1,3}

The conduct of the anaesthesia is also important for the survival of the fetus. Drugs that are known to be safe, or that do not cross the placenta, must be used. Vasoconstrictors should be avoided because of their effect on the uterine spiral arteries. Glyceryl trinitrate infusions may reduce the risk of uterine contractions. In fetal monitoring, heart rate decelerations may indicate hypoxia and distress, although another cause of bradycardia is fetal anaesthesia. Transvaginal ultrasound allows measurement of blood velocity in the uterine arteries, ductus venosus, fetal aorta and cerebral arteries, thus indicating how the fetus is responding to hypoxia; but few cases have been monitored in this way during cardiopulmonary bypass and this remains an experimental tool.

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