Case 2—A day old girl was referred, having developed cyanotic episodes not associated with feeding at 12 hours. Catheters were easily passed into the oropharynx through both nostrils. She was prescribed 0·5% ephedrine nose drops for evident nasal congestion, but her condition deteriorated so the drops were withdrawn. Radiological findings were normal, and examination under anaesthesia showed normal airways except for mucosal congestion in the nose. The nasal passages were dilated up to 14 French gauge. Postoperatively regular nasal suction and decongestants were used. Her condition deteriorated again with 0·5% ephedrine drops, which were again withdrawn. Three days postoperatively she still showed intercostal recession but could breathe through her mouth and was feeding well enough to return to the referring hospital. Three months later she was healthy with no airway or feeding problems.

Comment

Nasal obstruction in infants is usually detected soon after birth either because of obvious respiratory distress or by routine insertion of nasal suction catheters. Septal deformities, choanal stenosis and atresia, and nasopharyngeal masses are recognised causes of obstructed nasal breathing, but we believe that the problem of mucosal congestion alone has not previously been recorded. Once radiological and other findings have excluded any disorder of the upper and lower airways an airway should be provided until the child can breathe orally, which is usually within two to three weeks.[1] In one patient we were able to ease breathing by dilating the nasal airways, although we considered inserting a nasopharyngeal airway to relieve laboured breathing. Interestingly, this child's condition deteriorated when 0·5% ephedrine nose drops were given and improved when they were withdrawn. Despite our considerable experience in telling parents how to manage nasal stents, blockage occurred and the stents had to be removed.

We conclude that nasal congestion causing serious airway obstruction may be overcome by simple nasal dilatation after thorough examination of the airways under anaesthesia. If an airway remains blocked after operation a nasopharyngeal airway can be inserted. This allows more comfortable and "natural" breathing than with an oral airway. It is also preferable to nasal stents because a larger bore tube can be placed unilaterally rather than bilaterally owing to the compliance of the nasal septum. As this airway is likely to be in place for some weeks, however, the parents must be given portable suction apparatus and instructions on how and when to clear the airway. Antibiotic cover is required.

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Serologically proved intrauterine infection with parvovirus

It is well known that systemic viral illness in the mother may have an adverse effect on the outcome of pregnancy. Viruses of the TORCH syndrome (*Toxoplasma*, rubella, cytomegalovirus, herpes virus)[1] have received most attention, the clinical features including intrauterine growth retardation, congenital abnormality, mental handicap, and intrauterine death. Hence viral disease in pregnancy is a major problem.[2] We describe a case in which a serologically proved parvovirus infection may have contributed to intrauterine death at term in an otherwise uneventful pregnancy.

Case report

A 35 year old para 0+1 booked at 13 weeks' gestation, confirmed by ultrasound. Her husband had ankylosing spondylitis. Antenatal findings were normal, and maternal serum α fetoprotein concentration was 1·1 multiples of the median at 16 weeks. Detailed ultrasound examination showed no congenital anomaly and amniocentesis was performed at 16 weeks with no complications. Amniotic fluid α fetoprotein and acetylcholinesterase isoenzyme values were normal. Chromosomes showed a normal count with pericentric inversion of one of the number 9 (karyotype 46XX 9qh[inv]), which is not important. Pregnancy proceeded normally to 39 weeks, when the patient reported a flu like illness. This may or may not have been a parvovirus infection, but it did not cause any appreciable maternal problems. Blood pressure remained normal and there was no reduction of fetal activity.

Spontaneous labour began the day after the expected date of delivery. On admission, however, the fetal heart beat was not recordable and real time ultrasound confirmed intrauterine death. Vaginal examination showed the cervix to be dilated 6 cm and amniotomy yielded clear liquor. An epidural was sited for analgesia. Two hours later a stillborn girl was delivered vaginally. The infant was macerated and appeared to have severe ascites but otherwise looked normal. Samples of fetal and maternal blood were taken for viral studies, grouping, and antibody screening.

Necropsy showed a normal stillborn girl weighing 3840 g. Meconium ileus and peritonitis accounted for the ascites, with evidence of obstruction at jejunal level. There was no histological evidence of cystic fibrosis. Subpleural haemorrhages were noted and the cause of death given as intrauterine anoxia.

TORCH screening showed no evidence of recent infection. Nevertheless, both maternal and fetal blood contained parvovirus specific IgM, indicating recent maternal infection and also congenital infection of the fetus in utero. No viral deoxyribonucleic acid was detected in either specimen.

Comment

Parvovirus causes a mild feverish illness with a rash and arthralgia, particularly in adults.[3] At the time of this stillbirth the virus was epidemic in south east England. Despite attempts to identify viral DNA in specimens from spontaneous abortions we had been unable to show any evidence of transplacental infection until this case, which was the sixth stillbirth that we had investigated for parvovirus infection. Finding parvovirus specific IgM in fetal blood is definite evidence of in utero infection. Other viral infections transmitted transplacentally may be associated with poor outcome of pregnancy. In utero infection with parvovirus may therefore be expected to be associated with increased fetal morbidity and mortality.

The meconium ileus and peritonitis may not have been related to the infection in this case. There was no evidence of cystic fibrosis, either histologically or in the family history, but this was the most likely cause of the findings.

This is the first recorded example of in utero infection with parvovirus.[4] There may therefore be a case for testing for this infection in addition to the routine TORCH screen performed on symmetrically growth retarded fetuses and unexplained stillbirths.

1 Nahmias AJ. Perinatal infection associated with toxoplasma, rubella, cytomegalovirus, and herpes virus. *Pediatr Res* 1971;**5**:405.
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[1] International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1982;**284**:1766-70.