Treatment was started with Benoral Suspension 10 ml twice daily, and steroid treatment was begun two days later at a dose of 30 mg of enteric-coated prednisolone daily. Good clinical control of the polyarthropathy enabled the steroid dosage to be reduced to 5 mg twice daily after 10 days, by which time erythrocyte sedimentation rate was 21 mm in the first hour and the rash had largely cleared. She was discharged taking enteric-coated prednisolone 5 mg twice daily and naproxen 500 mg in the morning with food. Steroids were reduced over the next three months and all medication then stopped; tests for rheumatoid factor remained negative and both the rash and joint symptoms resolved completely.

Comment

We considered that this was a serum sickness type 3 reaction, probably due to the hair straightener. The straightener was later found to contain a total of 14 compounds, of which either hydrolysed animal protein or protamine nucleic acid complex may have been the precipitating factor in this case. Serum sickness due to hair straightener has not previously been reported, and the consumer safety unit at the Department of Trade, the Health and Safety Commission, and the manufacturers of the product are not aware of any similar cases.

I thank Dr M F Grayson for allowing me to report on one of his patients.

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Mumps virus isolated from a fetus

A prospective study[1] confirmed an association between gestational mumps and spontaneous abortion and showed a fetal mortality of 27% in 33 women who developed mumps in the first trimester of pregnancy, compared with a mortality of 13% in uninfected mothers. We report the isolation of mumps virus from a fetus spontaneously aborted four days after the mother developed clinical mumps.

Case report

A 28-year-old woman, gravida 4 para 1, had started her last menstrual period before her fifth pregnancy on 2 May 1981. On 1 August she developed clinical mumps and four days later spontaneously aborted a 10-week fetus. Serum samples taken at an earlier antenatal visit and four weeks later confirmed the clinical diagnosis, the mumps virus complement fixation titres having risen from <8 to 256.

The fetus was examined in the laboratory. Specimens of lung, liver, and brain were pooled and the homogenised suspension inoculated on to tissue cultures for virus isolation. A haemadsorbing virus was detected in primary baboon kidney-cell cultures on the 10th day, and this was shown to be mumps virus by a direct fluorescence antibody technique using a rabbit antimumps serum conjugated with fluorescein. To determine any chromosome abnormalities fibroblast cultures were set up from the fetal material using standard techniques and cultured in Eagles's medium with 20% fetal calf serum. After 19 days chromosome preparations were made and stained with toluidine blue. Of 50 cells examined, only three showed aberrations: one a chromosome break, one a chromatid break, and one an extra fragment. G-banded preparations showed a normal female karyotype.

Comment

We believe this to be the first report of isolation of mumps virus from a human fetus. Little direct evidence has previously existed of fetal infection early in pregnancy. No firm evidence associating maternal mumps with prematurity or congenital malformations appears to exist, and the postulated association with endocardial fibroelastosis is still debatable.[2] Garcia *et al*,[3] however, claimed to have found morphological evidence of mumps infection in three placentas and the adrenal cortex of one fetus obtained from mothers whose pregnancies were terminated because they had had mumps in the second or fifth month.

Experimental infection of rhesus monkeys[4] has shown that mumps virus can cross the placenta. The virus was isolated from fetal tissue for only one week after mothers were infected in the first trimester of pregnancy. The brevity of this period of virus replication in the fetus was thought to be responsible for the lack of mumps antibody in the progeny, who nevertheless showed a delayed hypersensitivity response to intradermal mumps antigen. This "split immunological recognition" has also been observed in man. That mumps virus can cross the placenta late in pregnancy was also suggested by Jones *et al*,[5] who isolated the virus at birth from the pharynx of two out of three infants whose mothers had had clinical mumps four to seven days before delivery.

Studies of the chromosomes of the infected fetus reported on above failed to show any abnormalities suggestive of an underlying genetic cause for the fetal death. Although mumps virus may cause changes in lymphocyte chromosomes similar to those caused by measles—that is, chromosome and chromatid breaks and multiple fragmentation—the few cells (three out of 50) showing this type of damage were within normal limits for cells grown in tissue culture and do not suggest widespread virally induced chromosome damage. Given the presence of the virus, however, some more localised chromosome damage might possibly have been caused in organs not examined.

This case shows conclusively that transplacental mumps infection occurs.

We thank Mr A Williams for referring this patient.

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Chest infection caused by Lactobacillus casei ss rhamnosus

Association of lactobacilli with human infection has rarely been documented,[1] although some cases of septicaemia or endocarditis caused by lactobacilli have been reported.[2-4] Organisms of this genus are generally reported from routine laboratory investigations as "contaminants" or "normal flora" when grown from specimens other than blood. I report a case in which the organism was responsible for chest infection and was isolated from sputum.

Case report

A 61-year-old woman with chronic myeloid leukaemia was admitted complaining of cough with yellow sputum and difficulty in breathing. On examination chest infection was suspected. She was afebrile and there was no evidence of generalised infection. White cell count on admission was 39×10^9/l (neutrophils 32×10^9/l). Oral cephalexin 500 mg six-hourly was prescribed and specimens of sputum and blood taken for culture. Sputum was thick and purulent. Microscopy showed many pus cells and large Gram-positive bacilli. No acid-fast bacilli were present. Culture of sputum produced pure and heavy growth of a diphtheroid organism, which was reported as normal flora. Two days later a second specimen of sputum showed similar microscopic features and produced pure and heavy growth of a diphtheroid as before.

As there was no clinical improvement three days after cephalexin treat-