Disseminated Herpes Simplex Type II Virus Infection Presenting at Birth

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Neonatal herpes simplex virus (HSV) type II infection is usually regarded as a devastating disease and carries a significant morbidity and mortality. Neonatal infection is invariably symptomatic and the clinical presentation is a direct reflection of the site and extent of viral replication(1). HSV II infection manifesting in the newborn period can be acquired *in utero,* postnatally or, most commonly, intrapartum(1). We report herewith a case of neonatal disseminated HSV type II viral infection acquired through an ascending infection associated with prolonged rupture of membranes for more than five days. This case is unusual because of its exfoliative skin lesions secondary to neurotropic spread of the virus and rarity of the development of complete heart block due to the cardiac involvement.

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Case Report

A 31-year-old gravida 8 para 1 (6 terminations of pregnancy and 1 fullterm baby) woman who was a heroin addict presented at 29 weeks gestation with spontaneous rupture of membranes. She was hepatitis B surface antigen positive but tests for other viral and bacterial infections, including HIV, were negative. She and her partner did not have any history suggestive of genital herpes. Dexamethasone was given to facilitate maturation of fetal lungs at the time of her admission. Emergency lower segment cesarean section was performed five days after admission because of cardiotocographic evidence of fetal distress (late decelerations of the fetal heart rate).

The male infant weighed 1000 g and and Apgar scores of 1 and 3 at 1 and 5 minutes, respectively. He required intubation and mechanical ventilation immediately after delivery. At the time of birth, extensive exfoliative lesions were noted on the left side of the baby's face and scalp (*Fig. 1*) and there were multiple vesiculobullous lesions with erythematous bases on the trunk and limbs. The liver was palpable 2 cm and the spleen 1 cm, below the costal margin. There were no obvious congenital malformations. Chest *X*-ray did not reveal pneumonia.

The initial full blood count demonstrated anemia (hemoglobin 11.0 g/dl), thrombocytopenia (60×10^9/l) and neutropenia (0.042×10^9/l) with a marked left shift (immature: total neutrophil ratio = 0.72). Lumbar puncture revealed xanthochromic CSF with 19×10^6 polymorphs/l, 457×10^6 mononuclear cells/l and 124×10^6 unclassifed WBC/l. CSF glucose was normal (5.3 mmol/l) and protein was markedly elevated (7.4 g/l). Liver enzymes were elevated (SGOT = 1690 U/l, SGPT = 350 U/l and GGT = 158 U/l).

On the first postnatal day the baby's heart rate was normal. On the second postnatal day he developed persistent bradycardia, with the heart rate fixed at 60 beats per minute. ECG confirmed the presence of complete heart block and also suggested a small pericardial effusion (*Fig. 2*). On the same day swabs of vesicular fluid were reported to show strongly positive immunofluorescence for HSV type II. HSV type II specific IgM and complement fixation test (CFT) were both positive. The clinical picture was thus consistent with disseminated HSV type II infection, so treatment was commenced with intravenous Acyclovir in the dose of 10 mg/kg/day. There was little change in the clinical condition on day two but on day three the baby developed multifocal tonic convulsions. These were difficult to control in spite of treatment with phenobarbitone, dilantin and clonazepam. Cranial ultrasound revealed intracerebral hemorrhage with ventricular dilatation on the left with massive subdural effusion on the right. There was progressive multiorgan failure over the next 24 hours. The grave prognosis was discussed with both parents and assisted ventilation was withdrawn on day four.

At autopsy, excoriation in the distribu-

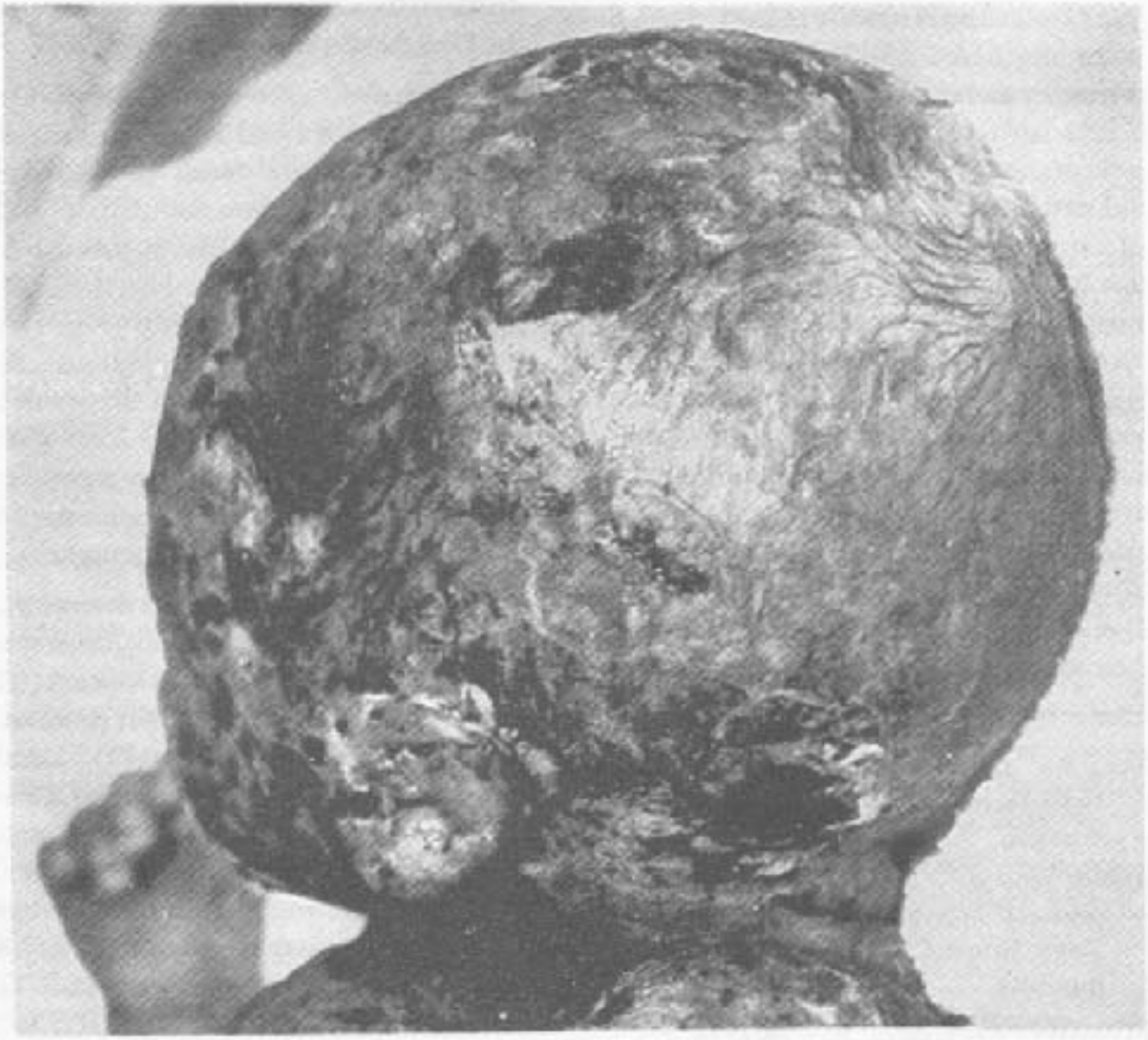

Fig. 1. Exfoliative lesions on left side of baby's face and scalp.

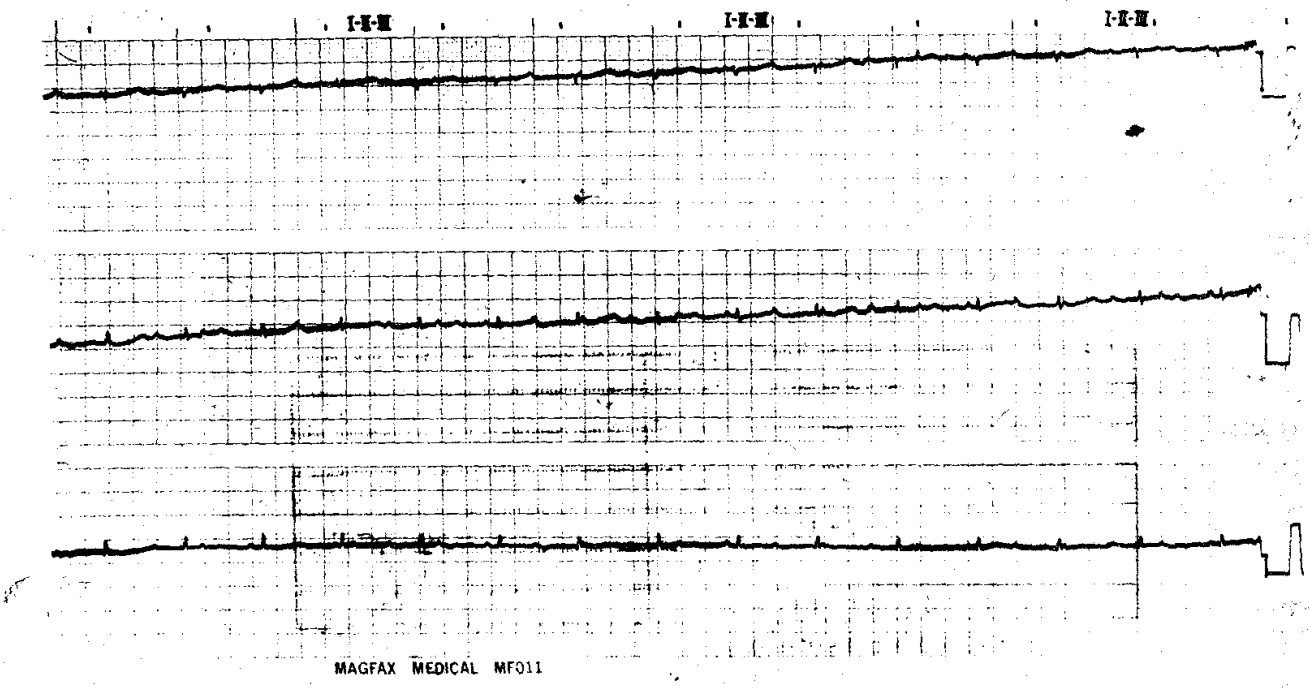

Fig. 2. ECG done on 2nd day revealing complete heart block and low voltage complexes suggestive of pericardial effusion.

tion of the left trigeminal nerve was confirmed. It also revealed extensive brain necrosis with intraventricular hemorrhage with intraparenchymal extension. There was a disseminated necrotizing infective process also involving the heart, liver, lungs, adrenals and kidneys. HSV type II was isolated from the brain and left trigeminal nerve but not from vesicular fluid, blood or CSF. Placental histology revealed the presence of extensive necrotizing chorioamnionitis, and HSV type II infection was confirmed using the immunoperoxidase method.

Discussion

We document herewith a case of disseminated neonatal HSV type II infection with an unusual presentation, in that at the time of birth an exfoliative process was already present in the distribution of the left trigeminal nerve. This suggests neurotropic spread of the virus from the left trigeminal ganglion rather than direct percutaneous infection. Other disorders with similar percutaneous manifestations which need to be considered are congenital syphilis and epidermolysis bullosa(2). There was no history suggestive of epidermolysis bullosa in either parents. Serology for syphilis was negative in both mother and the baby.

The development of complete heart block associated with involvement of the heart in the infective process is unusual and has not been reported. HSV type II virus has previously been reported to cause pericarditis and myocarditis(3), and cardiac arrhythmias have been reported to occur when the conduction system is involved in the process. However, previous reports have not specifically documented the types of arrhythmias seen(4). The widespread involvement of the myocardium in this case is

most likely responsible for the observed arrhythmia.

The maternal infection was most likely a recurrent one, as maternal serology for HSV II was positive by CFT but negative for IgM(4). A maternal cervical swab taken following confirmation of the baby's infection was negative. This does not, however, exclude the presence of such an infection at the time of rupture of membranes, since there was an interval of 8 days between rupture of the membranes and obtaining the maternal swab. Previous studies have shown average durations of viral shedding to be 4 days for recurrent infections and 12 days for primary infections(4).

Most clinicians are aware of the potential risks of ascending bacterial infection associated with prolonged rupture of the membranes, particularly in the premature newborn. The present case highlights the possible additional hazard of viral infection in such situations. If it had been known that the mother was actively shedding HSV at the time of rupture of membranes, it is possible that delivery by cesarean section within 4-6 hours may have avoided this devastating infection(1). The advanced progression of the disease process by the time of delivery in the present case made successful treatment with antiviral therapy very unlikely.

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Maternal Nutritional Status and Neonatal Head Circumference

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Head circumference (HC) is an indirect way of measuring the growth of brain *in utero* as well as after birth(1,2). Maternal malnutrition has been shown to influence the function of the central nervous system in children at a later age(3). We conducted this study with the objective to assess the role of maternal nutritional status over HC of their offsprings.

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