Congenital herpes simplex type II infection
The estimated incidence of neonatal herpes infection in the USA is 1 in 1000-5000 deliveries. Most infections (90%) are acquired intrapartum. Congenital infections are very rare; the virus is either spread transplacentally or ascending after rupture of the membranes. Ascending herpes infection through intact membranes has not yet been proven. Depending on the clinical presentation, primary herpes infection is divided into 3 groups: localized infection (skin, eyes, oral cavity), localized CNS infection and disseminated infection (liver, adrenal glands, CNS).

A female infant was delivered vaginally at term. Except for a recurrence of genital herpes in the first trimester, the pregnancy had been uneventful. Birth weight, length and head circumference were between the 50th and 90th percentiles. After birth, the infant was noted to have multiple grouped vesicles on an erythematous base on the upper limbs and face (Fig. 1–3). PCR of vesicular fluid revealed herpes simplex type II. Clinical examination was otherwise completely normal. The baby was transferred to our NICU. Suspecting localized herpes infection of the skin, intravenous therapy with acyclovir (30 mg/kg/d) was started and continued for 3 weeks in order to prevent dissemination of the disease.
Cerebral ultrasound on day of life 6 (Fig. 4) showed extensive bilateral periventricular calcifications and encephaloclastic areas in the left parietal and in both temporal lobes. These findings were later confirmed by MRI (Fig. 5). Ophtalmologic examination revealed central retinal scarring as would be seen following chorioretinitis. Ultrasound of the abdominal organs was normal. Serological testing for other congenital infections was negative.

After the 3 week course of acyclovir, the baby was discharged home in good condition with no neurological symptoms and without any skin lesions. Due to the extensive CNS lesions, long-term neurological sequels such as psychomotor retardation and seizures have to be expected.
Face: typical skin lesion.
Left arm: small vesicles with erythematous base.
Right hand with typical skin lesions.
Cerebral ultrasound examination: bilateral perventricular calcifications.
MRI: perventricular calcifications and encephaloclastic lesions of temporal and parietal lobes.
The diagnosis in this patient is congenital herpes infection of the CNS with recurrent skin lesions at birth. The encephaloclastic lesions indicate that the infection took place at the end of the second trimester after almost complete brain development. This might explain the absence of microcephaly and the normal neurological examinations during the early weeks of life.

We speculate that if the diagnosis of congenital herpes infection had not been made at birth, it would be impossible to later link the expected neurological sequelae with congenital herpes infection. An infant with skin lesions at birth should be investigated for herpes infection by PCR testing of the vesicles. The diagnosis of herpetic skin lesions in a newborn should lead to careful examination of CNS, eyes and abdominal organs.

