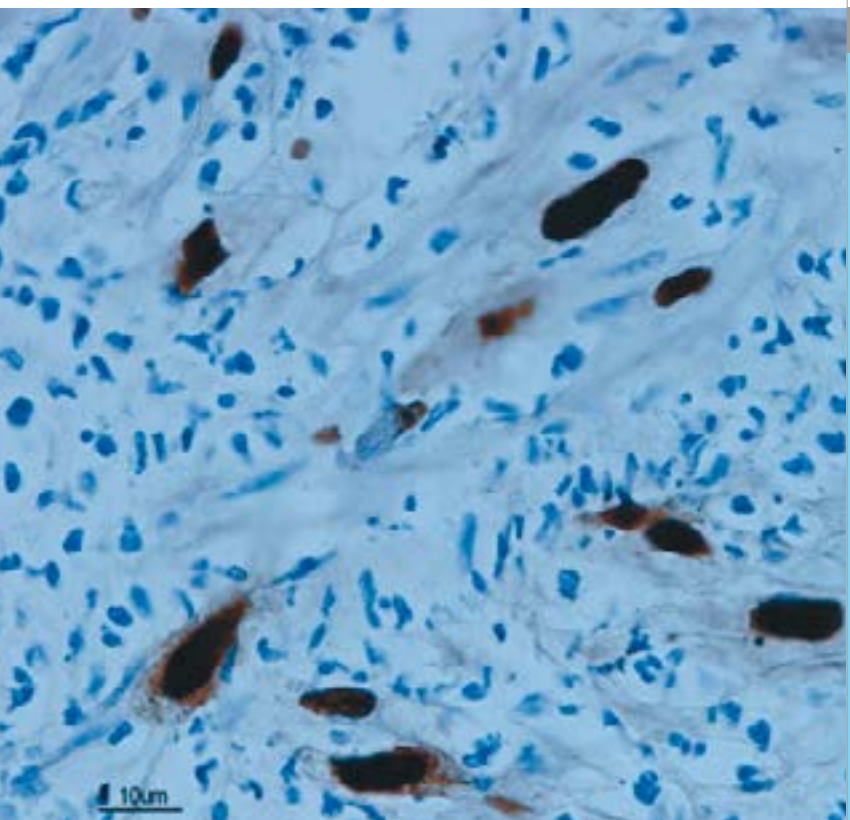
SWISS SOCIETY OF NEONATOLOGY

Severe esophagitis in a newborn associated with postnatally acquired cytomegalovirus infection



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This 3260 g female infant was born to a healthy 27-year-old G1/P1 mother at 40 4/7 weeks of gestation after an uneventful pregnancy. Postnatal adaptation was uncomplicated and the baby was discharged fully breastfed on the fifth day of life with a weight of 3150 g.

On day 19 of life, the infant was readmitted to the hospital because of prolonged crying episodes and frequent vomiting after breastfeeding and failure to thrive. The infant had been breastfed and in addition had received an age adapted formula milk (Aptamil HA) because the mother suspected a shortage of breast milk. The symptoms were observed regardless of the type of milk used. Apart from not having attained the birth weight yet (3220 g), physical examination was unremarkable. Laboratory work-up revealed a normal white and red blood cell count as well as normal electrolytes (potassium, sodium, calcium, chloride). Blood gas analysis and serum lactate were within normal limits. A pyloric stenosis was excluded by ultrasound, and no pathology was found on an upper GI series. The cerebral ultrasound was normal.

When the child continued to vomit and cry after breastfeeding an upper endoscopy was performed. This study revealed an impressive erosive esophagitis (Fig. 1,2). Histopathology showed a severe granulating inflammation and the characteristics of a cytomegalovirus (CMV) infection with viral inclusions

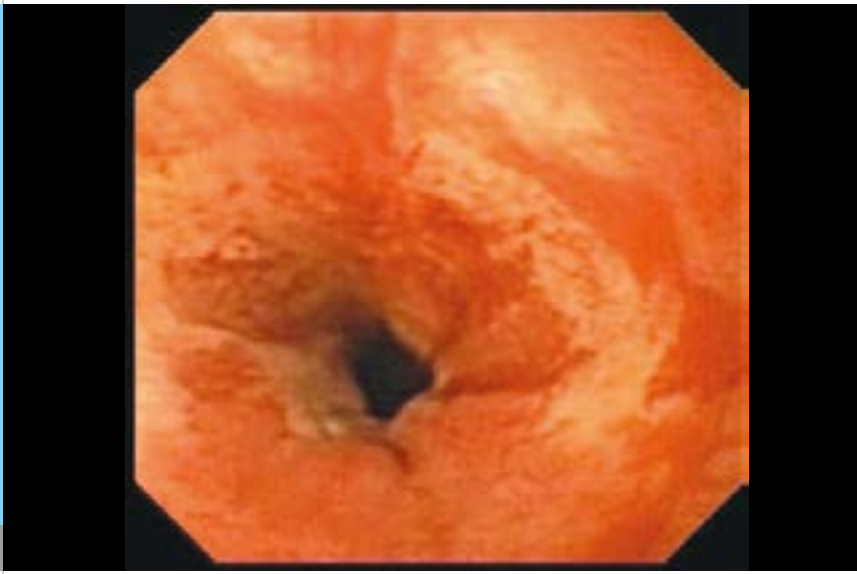

Fig. 1

Endoscopy showing severe erosive esophagitis.

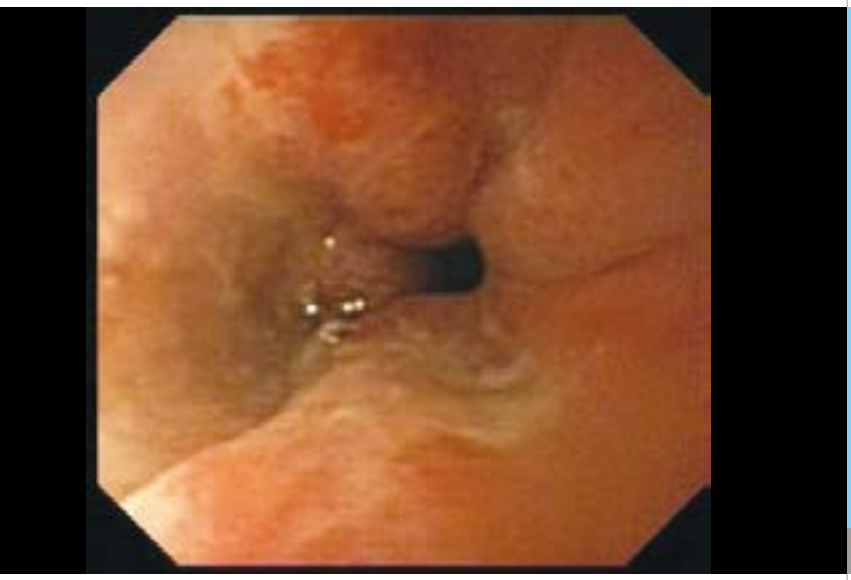

Fig. 2

Endoscopy showing severe erosive esophagitis.

(Fig. 3-5). Further work-up to detect other CMV-associated organ manifestations was negative with the exception of CMV being cultured from urine and a positive serology. Analysis of blood from the newborn screening card (day 4 of life) was negative for CMV. Maternal serology for CMV had been negative in the first trimester, but was positive at the time of delivery (IgM positive, IgG negative) and 23 days postpartum (IgM and IgG positive). Breast milk on the 23rd day after birth was positive for CMV (DNA-amplification). Since there was no evidence of an immunodeficiency no antiviral treatment was started.

Despite treatment with omeprazol, the child kept vomiting and continued to lose weight. Oral feeding was discontinued and the child was put on parenteral nutrition. Vomiting stopped and after five days an amino acid based formula milk (Pregomin AS) was introduced and well tolerated. On the 12th hospital day, breastfeeding was restarted without problems. The girl gained 200 g of weight in seven days and was discharged after 18 days of hospitalisation.

Two months later, at a regular well-baby-visit, she had recovered completely; urine analysis, however, was still positive for CMV.

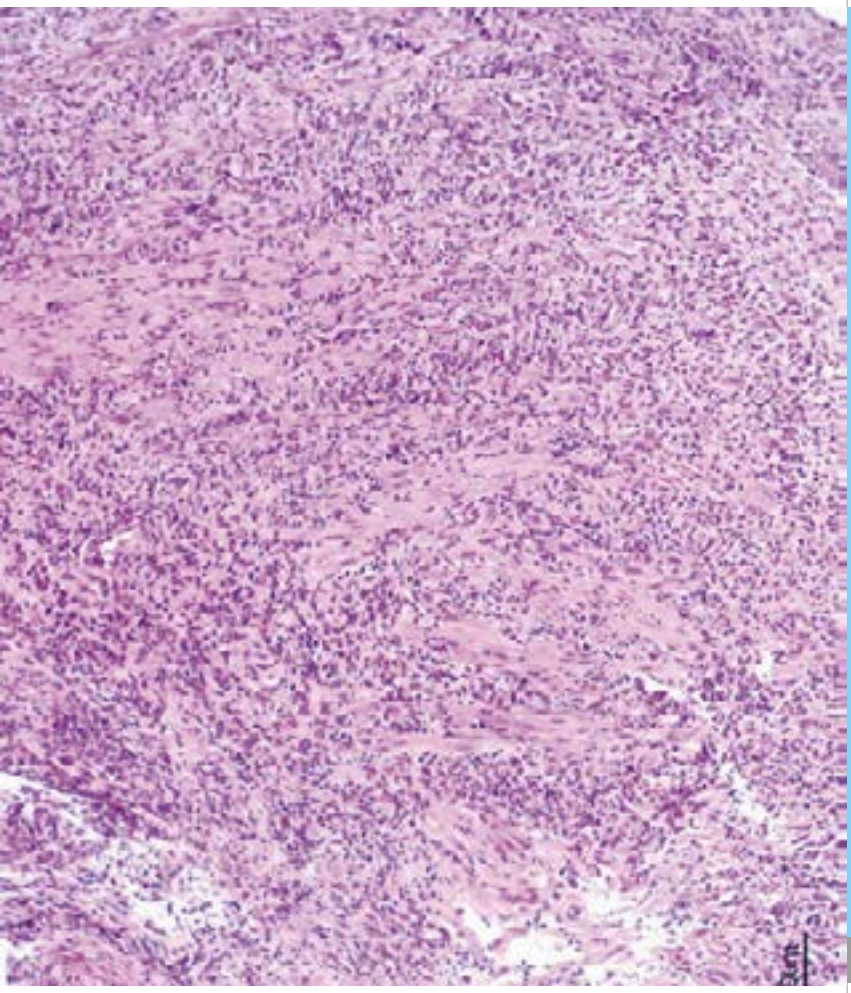

Fig. 3

Histopathology (HE stain): marked granulating inflammation.

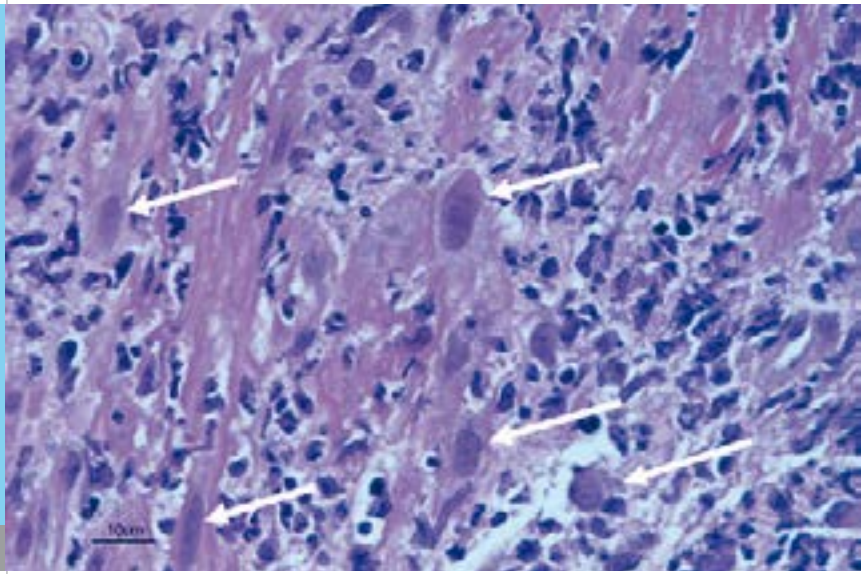

Fig. 4

Histopathology: nuclear virus inclusion bodies in smooth muscle and endothelial cells.

DISCUSSION

The symptomatic CMV infections are rare in the newborn and CMV esophagitis is mostly seen in children with congenital or acquired immunodeficiency. In 1996, Azimi et al. reported an immunocompetent newborn with congenital disseminated CMV infection and severe esophagitis which was successfully treated with ganciclovir (1).

In our patient, the predominant symptoms were recurrent vomiting and failure to thrive. This was felt to be consistent with either gastroesophageal reflux disease and/or cow's milk allergy (2), and the child was treated empirically with omeprazol and on a ami-

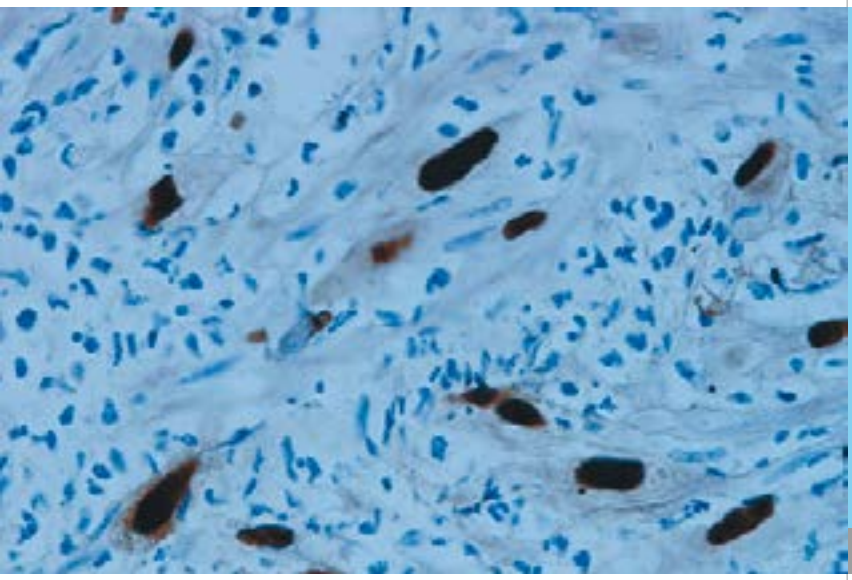

Fig. 5

Histopathology (immunostain, PAP technique): dectection of CMV antigen in the affected nuclei.

no acid based formula. Symptoms resolved only when all oral intake was stopped and total parental nutrition was started. The correct diagnosis was only made after endoscopy and biopsy. Despite the severity of CMV esophagitis and the difficulty to feed the infant, no antiviral agents such as ganciclovir or foscarnet where used. This decision was based on the result of several studies recommending such medication only in life- or sight-threatening CMV infections or in infants with known immunodeficiencies (3, 4).

Our patient was likely infected postnatally through breast milk during the first days of life since blood

tests from the neonatal screening card were negative for CMV. Based on maternal serologies, CMV infection occurred late in pregnancy without transplacental transmission of CMV. The level of immunity among women in the childbearing age is an important factor in determining the incidence and significance of congenital and perinatal CMV infections (3). Seropositivity rates in young women in Western Europe range from less than 50 to more than 85% (3). The newborn can be infected congenitally or perinatally. Congenital infection through transplacental transmission is relatively rare (0.2-2.2% of all newborns). Sources of perinatal and postnatal infections are the infected genital tract or breast milk (3). The vast majority of primary infections in immunocompetent hosts are clinically silent (4). Symptomatic infections commonly involve the central nervous system, the ears, the eyes, the liver, the hematopoietic system, the kidneys, the endocrine glands, the lungs and the gastrointestinal tract. To our knowledge, isolated CMV-associated esophagitis has not yet been reported. Our case illustrates a possibly underappreciated manifestation of CMV infection in an immunocompetent infant.

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