Case Report

A Bullous Purpura Triggered by Warming in a Newborn with Congenital Cytomegalovirus Infection

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ABSTRACT

A newborn with microcephaly presented hemorrhagic bullous purpura triggered by heat in the 1st h of life. Doppler arterial and venous ultrasound excluded vascular complications. Cytomegalovirus was detected in blood, urine, and serum of the lesions. The final diagnosis was cytomegalovirus congenital infection due to reactivation in an immune mother, with associated purpuric rash, confirmed by cerebral magnetic resonance imaging.

KEYWORDS: *Congenital cytomegalovirus infection, newborn, purpuric rash*

INTRODUCTION

Congenital infection by cytomegalovirus (cCMV) is the most common viral infection acquired *in utero*, with a prevalence of 0.5% in live births in Europe. The infection is often undetected because approximately 90% of newborns are asymptomatic at birth in the absence of a screening program, with a risk of neurosensory sequelae of 5%–15%.[1] For diagnosis, the direct detection of virus in a sample obtained before 3 weeks of life is required, and is best performed by polymerase chain reaction on saliva or urine.[1] The occurrence of fetal ultrasound findings consistent with *in utero* CMV infection, brain abnormalities, and also petechiae or purpura at birth are all indications for testing a neonate for CMV.

We report a case of hemorrhagic bullous purpura triggered by warming of extremities in a newborn with known microcephaly at prenatal ultrasound and brain alterations at fetal magnetic resonance imaging (MRI). The purpura was a sign of cCMV due to reactivation in an immune mother, with viral detection in blood, urine, and also serum of bullous lesions, with typical alterations at brain MRI.

CASE REPORT

A term newborn with prenatal finding of microcephaly with MRI alterations and hyperechoic bowel was admitted to neonatal intensive care unit for moderate respiratory distress and need for continuous positive airway pressure.

The mother was a hepatitis B virus (HBV) carrier at low viral loads of HBV DNA and immune to CMV. At prenatal ultrasound checks, fetal cranial circumference resulted below the 2nd percentile for gestational age. Fetal MRI showed reduced biparietal and fronto-occipital diameters and reduced dimensions of the cerebellum and truncus. Encephalic parenchyma appeared thinned, especially in the frontal regions, being consistent with scarcely represented neuronal migration. The anterior horns of the lateral ventricles were wider than normal, with a dysmorphic appearance. After a preliminary genetic evaluation, parents decided to postpone further genetic investigations after birth.

During the 1st h of life, a glove filled with warm water was placed on the right foot of the baby to perform an arterialized capillary blood gas analysis.[2] Immediately after removal, the sudden appearance of a hemorrhagic bullous purpuric rash [Figure 1a] was noticed, probably consequent to warming. Afterward, active and passive immunoprophylaxis against HBV was completed. Platelet count and coagulation parameters were normal

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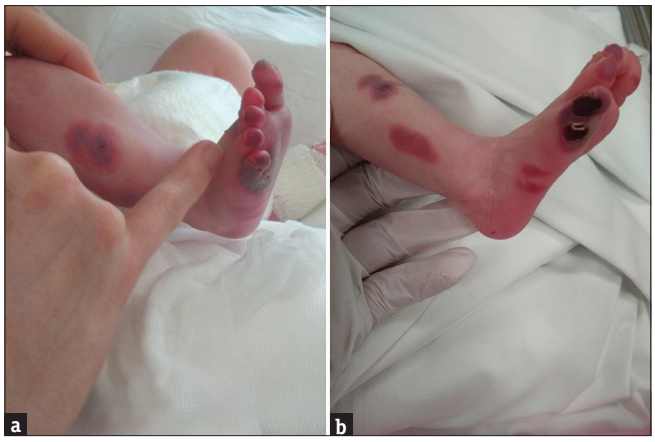

Figure 1: (a) The figure shows the sudden appearance of a hemorrhagic bullous purpuric rash after warming extremities. (b) The figure shows the evolution of hemorrhagic vesicles and bullae into cutaneous necrosis

before and after this episode, and vascular complications were excluded with Doppler arterial and venous ultrasound performed by the angiology consultant. CMV-DNA was detected in urines and blood. The child was diagnosed with cCMV by reactivation in an immune mother. CMV was also detected in the serum of bullous lesions. The hemorrhagic vesicles and bullae evolved into cutaneous necrosis [Figure 1b], with superimposed impetiginization treated with topical antibiotics, ending in skin scarring.

Brain MRI confirmed cCMV encephalopathy (polymicrogyria, temporal cysts, and cerebellar abnormalities), and subsequent ophthalmological examinations showed hemorrhagic target lesions of the macular retina, consistent with the infection.

After the diagnosis, the newborn was treated with oral administration of valganciclovir. After a week, CMV DNA blood levels fell below 1000 copies/ml. Complete blood count and liver enzymes were always normal at subsequent checks. No new skin lesions were ever documented afterward, pointing out that the skin was not exposed to heat again.

The newborn was then regularly discharged home with a follow-up program both for her skin lesions and cCMV infection.

Discussion

Congenitally acquired CMV is a common cause of morbidity in newborns. Only 10%–15% of infants with cCMV are symptomatic at birth, but of those, up to 40%–60% may present permanent sequelae, including sensorineural hearing, visual impairment, and cognitive delay.[1] The risk of neurosensory sequelae in asymptomatic patients is estimated to be 5%–15%.[3] In the case of suspicion at birth, testing should always be performed, in order to confirm the diagnosis, decide whether antiviral therapy is to be started, and to address the newborn as soon as possible to audiological and ophthalmological examination and brain imaging.[3] The best samples to be tested at birth are saliva and urine, as they contain the highest levels of CMV DNA.[4] In the absence of clinical suspicion, the diagnosis can often be missed. Routine screening has not been widely adopted, though it would be reasonably useful and cost-effective. The recommended therapy for moderate and severe infections is oral valganciclovir for 6 weeks–6 months, and neurological and auditory follow-up is to be extended largely beyond the neonatal period, in order to detect delayed sequelae.

In our case, the finding of a bullous purpura, that only appeared after warming and was limited to areas of the skin that had been exposed to heat, helped to raise suspicion and enabled early diagnosis and treatment of cCMV due to reactivation in an immune mother. We first successfully excluded thrombocytopenia and thrombosis and made sure there was no temporal connection with the injection of HBV vaccine and immunoglobulins. When CMV DNA was found in urine and blood and eventually in the serum of the lesions, we had the final confirmation to the fact that the known purpuric rash associated with cCMV infection[5] in our case had been triggered by skin warming, similarly to other purpuric rashes.[6]

Conclusion

On the basis of maternal history and in the case of one or more signs or symptoms suggesting cCMV, the screening on the newborn is not to be delayed. Recommendations for systematic neonatal screening are to be implemented, in order to avoid delays in diagnosis, treatment, and follow-up.

Consent

An informed consent was signed by parents in order to publish images regarding the case.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient's parents have given their consent for images and other clinical information to be reported in the journal. The patient's parents understand that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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