Neonatal varicella infection: are we being falsely reassured by a maternal history of chickenpox?

Introduction

Neonatal varicella infection can be serious, with up to 25% mortality (Speer, 2012). Babies at greatest risk are those whose mother was exposed to varicella zoster virus 7 days before delivery and developed clinical manifestations of chickenpox within 7 days of delivery (Department of Health, 2006; Health Protection Agency, 2008). Such babies should be given varicella zoster immunoglobulin without antibody testing as the consequences of chickenpox infection can be fatal (Department of Health, 2006; Health Protection Agency, 2008). Varicella zoster immunoglobulin should not be given to infants (more than 7 days of age) when the mother develops chickenpox or in cases where the mother has developed zoster infection before or after delivery as these infants are considered to have protection via maternal antibodies (Health Protection Agency, 2008).

Varicella zoster immunoglobulin does not provide complete protection and parents should be counselled about the signs and symptoms of chickenpox and the need for treatment with aciclovir if the baby develops chickenpox (Reynolds et al, 1999). The same may be true for babies born to mothers with a previous history of chickenpox who are considered to be immune because of the transfer of maternal antibodies (Sauerbrei, 2010). This article describes such a case and discusses the management of these cases.

Discussion

This case demonstrates the need to treat a neonate with aciclovir if chickenpox is suspected and not be falsely reassured by a history of previous maternal chickenpox infection. This baby was not given varicella zoster immunoglobulin as his mother had a positive history of chickenpox in her childhood and maternal antibodies were expected to provide protection to the neonate (Department of Health, 2006; Health Protection Agency, 2008). Neonatal varicella in the context of a previous maternal history of chickenpox is expected to be mild, as in this case (Sauerbrei, 2010). Severe presentations are known to occur but usually in patients with underlying congenital immunodeficiencies.

Although IgM to varicella zoster virus may be detected as early as 3 days after exposure in some cases (Speer, 2012), a negative IgM should not give false reassurance as experience is limited with IgM antibody tests and the actual timing of the IgM response (Lopez et al, 2011). Polymerase chain reaction for varicella zoster virus DNA is currently the most reliable and sensitive method for confirming a varicella zoster virus infection and the clinical specimen should ideally be scabs, vesicular fluid or cells from the base of a lesion (Lopez et al, 2011; Speer, 2012).

Universal varicella vaccination is associated with reduced incidence of both congenital and neonatal varicella infections (Khandaker et al, 2011). Clinicians treating young infants should have a high index of suspicion for diagnosing varicella infections. The authors suggest that samples should be taken for polymerase chain reaction for varicella zoster virus analysis coupled with a proactive approach of early initiation of antivirals and/or varicella zoster immunoglobulin administration (post-exposure) where the index of suspi-

LEARNING POINTS

- Vesicular rash in a neonate should raise suspicion of chickenpox infection.
- A history of previous chickenpox in the mother can be falsely reassuring.
- Polymerase chain reaction of the vesicular fluid is the most reliable method for detecting varicella zoster viral DNA.
- Neonates who develop chickenpox should be treated with aciclovir.
- Introduction of varicella zoster vaccine would be beneficial for children in the UK.

Case Report

A 15-day-old baby, born at term, presented with a 24-hour history of a blister-like rash. His eldersibling had developed chickenpox on the second day after the baby's birth. He was reported to have a mild fever and to be feeding poorly. Initial observations revealed a heart rate of 136/min, temperature of 37.6°C, respiratory rate of 35/min and a central capillary refill time of less than 2 seconds. Systemic examination was normal and a few vesicles were noticed on his trunk. His mother reported having had chickenpox infection while she was in primary school. The rash was thought to be caused by a staphylococcal skin infection, with a differential diagnosis of varicella zoster virus infection considered possible but unlikely in view of the mother's history.

He was admitted for suspected sepsis to an isolation cubicle. His blood inflammatory markers, blood culture and varicella zoster virus IgG and IgM serology were taken as baseline investigations. Vesicular fluid was sent for microscopy and varicella zoster virus and herpes DNA nucleic acid analysis using polymerase chain reaction. Intravenous flucloxacillin and aciclovir were started to cover the possibility of staphylococcal and herpes infection respectively. Blood inflammatory markers were within normal limits and the bacterial skin swab was reported as negative. However, microscopy of the vesicular fluid did not show any viral inclusion bodies. As he remained well and the rash did not spread to other areas of his body, he was discharged home after 48 hours of intravenous antibiotics and aciclovir, with a plan to complete another 5-day course of oral aciclovir. Varicella zoster virus IgG was reported as positive (confirming presence of maternal antibodies) and varicella zoster virus IgM was reported as negative. However, varicella zoster virus DNA was detected by polymerase chain reaction of vesicular fluid (became available 48 hours after sampling), which confirmed a perinatally acquired chickenpox infection. The baby made a full recovery.

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cion is deemed to be high (Sauerbrei, 2010). This would likely reduce morbidity and mortality in serious neonatal varicella infections which often are associated with poor outcomes. BJHM

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Two cases of tuberculous meningitis with stroke sequelae

This article describes two patients who presented to the authors' local stroke unit with preceding tuberculous meningitis complicated with acute stroke.

The first patient was a 68-year-old Filipino woman, resident in the UK, who was unwell with disseminated tuberculosis (renal, miliary and tuberculous meningitis). She had been taking antituberculous treatment for 3 months before developing sudden right-sided weakness and dysphasia. Magnetic resonance imaging of the brain revealed bilateral basal ganglia infarcts, basal meningeal enhancement and tuberculosis-associated secondary hydrocephalus (*Figure 1*). Unfortunately, she had a complicated inpatient stay and died on the unit.

The second patient was a 69-year-old Somalian man who had lived in the UK for 9 years. He had been treated for tuberculous meningitis with cauda equina syndrome for the last 4 months. He had a ventriculoperitoneal shunt in situ for his tuberculosis complicated by hydrocephalus. He developed sudden right-sided weakness and aphasia. Magnetic resonance imaging of the brain confirmed several small recent infarcts in the left frontoparietal lobes and in the right basal ganglia and right cerebellum with signs of basal meningitis. He was eventually discharged.

It was thought that both patients had a stroke as a result of tuberculous meningitis, given the relative paucity of vascular risk factors and the predisposition of the infarcts in the territory of the middle cerebral artery perforating vessels.

Stroke is the most serious complication of tuberculous meningitis, occurring in up to 30% of cases (Anuradha et al, 2010). Patients with tuberculous meningitis-associated hydrocephalus have increased risk of stroke and a worse prognosis (Chan et al, 2003). Other predictors of poor outcome are CSF leucocytosis and meningeal enhancement on brain imaging (Koh et al, 2007). Magnetic resonance imaging is the modality of choice in detecting brain in-farcts, typically revealing multiple or bilateral lesions in the territories of the middle cerebral artery perforating vessels (Lammie et al, 2009). Anti-tuberculous chemotherapy seems relatively ineffective in preventing vascular complications (Lammie et al, 2009). BJHM

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Figure 1. Diffusion weighted magnetic resonance images from the first patient, showing bilateral areas of restricted diffusion within the basal ganglia bilaterally which is consistent with acute infarcts.