Chickenpox, without a characteristic vesicular skin rash, can result in a diagnostic dilemma when an associated complication of the infection occurs.

Complication of chickenpox infection without skin rashes

Chickenpox is a highly infectious contagious disease caused by varicella zoster virus (VZV). Clinical diagnosis in children is usually made in the presence of characteristic vesicular skin rash, starting on the head or body. Prodromal symptoms (symptoms indicating disease onset) of nausea, loss of appetite, aching muscles and headache are generally seen in adolescents and adults.

Absence of the typical vesicular rash (in subclinical infections) can create a diagnostic dilemma when an associated complication of the infection is the first presentation. We present a case of cerebellitis (associated complication) following a chickenpox infection without a rash.

Presentation

Seventeen-year-old Jackie Carter* presented with an unsteady gait and headache. She had symptoms for three days before attending accident and emergency. She also reported a history of sore throat, intermittent fever and mouth ulcers (but no cold sores) two weeks earlier, which had been treated with acyclovir cream for a suspected herpes infection (these features can be associated with chickenpox infections).

Ms Carter had difficulty walking due to ataxia but had no history of vesicular rashes or scabs and her systemic observations were stable. A list of differential diagnosis for cerebellar ataxia, including Guillain-Barré syndrome, acute demyelinating encephalomyelopathy, toxins and viral infections, were considered. As she had a history of previous chickenpox infection in early pre-school years, chickenpox cerebellitis was not initially considered.

Owing to the persistent abnormal neurological findings, Ms Carter was admitted. Blood investigations were sent and intravenous ceftriaxone and acyclovir were started. Initial computerised tomography and magnetic resonance imaging brain scans were normal.

Further management

Nursing observations included systemic observations, monitoring of the Glasgow Coma Scale and support with mobility. In view of the persistence of the neurological symptoms, a repeat MRI scan was done five days later, which suggested cerebellitis. A lumbar puncture was performed; the cerebrospinal fluid sample showed 250 white cell count with 95% lymphocytes, suggesting a current or previous viral meningitis or encephalitis.

The varicella serology sample collected on admission showed evidence for recent chickenpox infection; a sample collected 11 days post-admission showed evidence of developing immunity from a previous chickenpox infection. It became retrospectively evident that cerebellar ataxia was due to complication of a recent chickenpox infection. Ms Carter received 14 days of IV acyclovir and gradually recovered.

Discussion

Chickenpox is usually self-limiting, but complications can occur. In healthy children these include secondary bacterial infections of the chickenpox lesions, otitis media, secondary pneumonia, necrotising fasciitis, subclinical hepatitis and cerebellar ataxia (Paul et al, 2013).

Cerebellar ataxia is a known complication after VZV infection presenting with vesicular rash. VZV reactivation, which usually produces herpes zoster (shingles), can cause neurological disease in the absence of a rash (Gilden et al, 2010).

The case described here is perhaps one of the few reported cases of cerebellar ataxia as an associated complication of primary VZV infection presenting without a rash. It highlights the importance of considering VZV cerebellitis in the list of differential diagnosis in a patient presenting with cerebellar ataxia even in the absence of a vesicular rash.

Varicella vaccine is likely to be beneficial to children in the UK and has been proved to decrease mortality and be cost effective in countries where it is currently part of the immunisation schedule (Paul et al 2013). *The patient’s name has been changed.

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References
