Bulging fontanel during a measles infection

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Abstract

Introduction **Causes of a bulging fontanel in children include viral/bacterial meningitis, encephalitis, hydrocephaly, intracranial bleeding, psedotumor cerebri and central nervous system tumors. A less well-known cause of acutely-developing fontanel bulging is transient intracranial hypertension.**

Case report **A 7-month-old female was brought to our hospital with fever, cough, watery nasal secretion and bulging fontanel for the last 3 days. The cranial ultrasound examination and cerebrospinal fluid examinations were normal. The patient's irritability and fontanel bulging improved the day after admission but a maculopapular rash developed. The measles Ig M was positive. Measles PCR was positive from urine and nasopharyngeal swab samples. The signs of intracranial hypertension of the patient recovered completely the day after admission and did not recur during the follow-up. The patient was discharged on the 5th day of admission.**

Discussion **Transient intracranial hypertension is thought to result from a transient increase in the quantity of the circulating cerebrospinal fluid but the pathogenesis is not clear.**

Conclusions **Based on this case, we report that transient intracranial hypertension could develop during a measles infection.**

Keywords **Transient intracranial hypertension, measles, intracranial hypertension, fontanel bulging, fever, meningitis**

Introduction

Causes of a bulging fontanel in children include viral/bacterial meningitis, encephalitis, hydrocephaly, intracranial bleeding, psedotumor cerebri and central nervous system tumors. A less well-known cause of acutely-developing fontanel bulging is transient intracranial hypertension. Transient intracranial hypertension is a benign cause of increased intracranial pressure and is thought to develop due to a temporary increase in the amount of cerebrospinal fluid (CSF).[1] We report a seven-month-old infant who presented with fever, irritability and a bulging fontanel, and subsequently developed a measles rash.

Case report

A 7-month-old female was brought to our hospital with fever, cough, watery nasal secretion and bulging fontanel for the last 3 days. The patient had no history of an upper respiratory tract infection or contact with a child who had a rash. The patient was irritable with a hyperemic oropharynx and vesicles in the posterior pharynx on admission to our hospital. The fontanel was 2×1 cm in size and markedly bulging when evaluated with the patient calm and in the upright position. The neurological examination results were normal. The patient was admitted with a preliminary diagnosis of meningitis. The laboratory tests revealed the following: hemoglobin level 11.2 g/dL, MCV 82.5 fL, RDW 40.7 fL, white blood cell count 7770/µL

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(neutrophils 45%, lymphocytes 49%, monocytes 5% on peripheral smear), and thrombocyte count 409 000/mm^3; C-reactive protein (CRP) 9.7 mg/L (normal range 0-5 mg/L); normal biochemical parameters. The cranial ultrasound examination demonstrated normal cerebral and cerebellar hemispheres, corpus callosum and ventricular system. Lumbar puncture was performed, but the opening cerebrospinal fluid (CSF) pressure could not be measured. Direct microscopic examination of the CSF revealed no cells. CSF glucose was 66 mg/dL (simultaneous blood sugar 101 mg/dL) and CSF proteins were 17.5 mg/dL, thus excluding meningitis. The patient's irritability and fontanel bulging improved the day after admission without any treatment except for lumbar puncture which addressed intracranial hypertension but a maculopapular rash that started from the face and spread to the whole body developed. Koplik's spots were seen on oral mucosa evaluation. The measles Ig M level was 20.77 U/mL (positive). Measles PCR was positive from urine and nasopharyngeal swab samples. The patient was administered perorally 100 000 IU once a day vitamin A for two consecutive days with a diagnosis of measles. The cough increased on follow-up and the respiratory sounds became coarse. Sulbactam-ampicillin (intravenous 200 mg/kg/day q6 h) was started on the 3rd day of admission, when bilateral interstitial infiltration was observed on the chest X-ray. There was no growth on CSF, blood or urine cultures. The patient was discharged on the 5th day of admission when the rash and the respiratory system findings recovered. The patient continued the sulbactam-ampicillin treatment for five days perorally (50 mg/kg/day). The neurologic examination was normal at the 6th month follow-up.

Discussion

Measles morbillivirus, which is the causative agent of measles, is an RNA virus from the *Paramyxoviridae* family. The measles virus enters the body by inhalation of respiratory droplets followed by the invasion of and multiplication in nasopharyngeal epithelial cells. The next step is spread to the regional lymph nodes and secondary viral replication there. The virus then infects lymphocytes and monocytes to cause viremia and to spread to almost all organs. The skin, conjunctivae and respiratory tract are typically involved, leading to the clinical signs of cough, nasal secretion, conjunctivitis, fever and a rash.[2] In addition to this typical clinical picture, the virus can access many organs and systems through viremia and cause other complications.[3]

The central nervous system (CNS) complications of measles are rare but quite serious and the main complications consist of primary measles encephalitis, measles inclusion body encephalitis, postinfectious encephalomyelitis and subacute sclerosing panencephalitis (SSPE). Primary measles encephalitis and measles inclusion body encephalitis are differentiated from the other forms by their development during acute infection.[4] Measles inclusion body encephalitis is seen in immunocompromised individuals. In primary measles encephalitis, the virus invades the CNS and replicates here. The CNS infection typically develops simultaneously with the rash although it can also rarely develop before the rash.[4,5] CSF evaluation reveals lymphocytic pleocytosis and mildly elevated protein levels.[4,6] The presence of fever, irritability and a bulging fontanel in our patient with no immune deficiency resulted in a preliminary diagnosis of CNS infection. However, the completely normal cranial imaging and CSF findings and the rapid recovery of the irritability excluded this diagnosis.

Transient intracranial hypertension is a benign cause of increased intracranial pressure. It has been defined as consisting of a normal level of consciousness and normal neurological findings, no pathology on cranial imaging, normal CSF findings, the lack of another cause and normal neurological development on follow-up in patients with a bulging fontanel.[7] It has been reported that the irritability of the patient improves and the fontanel bulging disappears after lumbar puncture.[8,9] The normal CSF and cranial imaging results, the immediate improvement in the irritability and fontanel bulging following lumbar puncture, the normal neurological findings, and the normal neurological development at follow-up were

consistent with transient intracranial hypertension in our patient.

Transient intracranial hypertension is thought to result from a transient increase in the quantity of the circulating CSF but the pathogenesis is not clear. One hypothesis is that the proinflammatory cytokines secreted into the circulation during viral infections influence the choroid plexus which has a high degree of vascularity and results in increased CSF production here.[1] Transient intracranial hypertension has been reported to develop following human herpes virus 6 infection, diphtheria-tetanus-pertussis vaccination and vitamin A supplementation.[7,9,10] We could not study the changes in plasma cytokine levels. Temporary fontanel bulging has been reported to develop after measles vaccination, however measles infection-related transient intracranial hypertension has not been previously reported.[8]

Conclusions

Many pediatricians have observed that the irritability of an infant with preliminary diagnosis of meningitis is relieved immediately after performing lumbar puncture. We therefore believe that transient intracranial hypertension could develop without the presence of meningitis during various infections, but the diagnosis could be missed as the condition is not commonly known. By presenting this case, we want to remind clinicians of the transient intracranial hypertension diagnosis and also to report that it could develop during a measles infection.

Consent: Written informed consent was obtained from the patient for publication of this case report and images.
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