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Case report

Pseudotumor cerebri complicating measles: A case report and literature review

Haydar Ali Taşdemir [a], Cengiz Dilber [b,*], Mehmet Totan [c], Arif Onder [d]

[a] *Division of Neurology, Department of Paediatrics, Faculty of Medicine, Ondokuz Mayis University, Samsun, Turkey*
[b] *Department of Paediatrics, Faculty of Medicine, Sutcuimam University, Kahramanmaras, Turkey*
[c] *Department of Paediatrics, Faculty of Medicine, Ondokuz Mayis University, Samsun, Turkey*
[d] *Department of Neurosurgery, Faculty of Medicine, Ondokuz Mayis University, Samsun, Turkey*

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Abstract

A previously healthy 8-year-old girl patient was referred with the complaints of severe headache associated with nausea and vomiting. Three weeks prior to her admission, she had measles manifested with fever and typical skin eruptions. Fundoscopic examination revealed bilateral swollen optic discs with tortuous blood vessels. Other physical examinations were unremarkable, except for photophobia. Lumbar puncture demonstrated a cerebrospinal fluid opening pressure of 30 cm H_2O, no cells, normal levels of glucose and protein. Serum measles immunoglobulin M level was elevated (183 AU). Our further investigations revealed that the patient had pseudotumor cerebri (PTC) following measles infection.

Keywords: Pseudotumor cerebri; Measles; Childhood

1. Introduction

Pseudotumor cerebri (PTC) is a perplexing syndrome with increased intracranial pressure without a space occupying lesion or hydrocephalus. This syndrome may arise due to an identifiable specific cause or may be idiopathic. A specific cause can usually be found in children younger than 6 years, whereas most idiopathic cases occur after age of 11 years [1,2].

There are many conditions associated with PTC, some of them are fairly well substantiated and others appear only in isolated case reports. The possible association between infectious or post infectious conditions and PTC has been mentioned. These include bronchitis, sinusitis, middle ear infections, gastroenteritis, urinary tract infections, HIV infection, Lyme disease and varicella [1–3].

In this case an 8-year-old girl with PTC diagnosed following measles is reported. A search of medical literature through Pub MED showed that this condition has not been reported previously.

2. Case report

An 8-year-old girl was admitted to our clinic with the complaints of severe headache, nausea/vomiting, retro-orbital pain and photophobia. Three weeks prior to her admission she was diagnosed as measles manifested with typical skin eruptions.

On physical examination, she was non-febrile. There were no signs of meningeal irritation, and she was fully oriented with fluent speech and intact memory. Fundoscopic examination revealed bilateral swollen optic discs with tortuous blood vessels.

Laboratory evaluations, including a complete blood count, blood electrolytes, urea, creatinine, liver enzymes, thyroid stimulating hormone, parathormone, cortisol, and thyroxin were normal. Serum varicella immunoglobulin G

Abbreviations PTC, pseudotumor cerebri; CSF, cerebrospinal fluid; MRI, magnetic resonance imaging; ICH, intracranial hypertension; CNS, central nervous system.

* Corresponding author. Address: Kahramanmaras Sütcüimam University, Tip Fakultesi Pediatri Anabilim Dali, 46050 Kahramanmaras-Turkiye. Tel.: +90 344 2212337 366; fax: +90 344 2212371.
E-mail address: cengizd@omu.edu.tr (C. Dilber).

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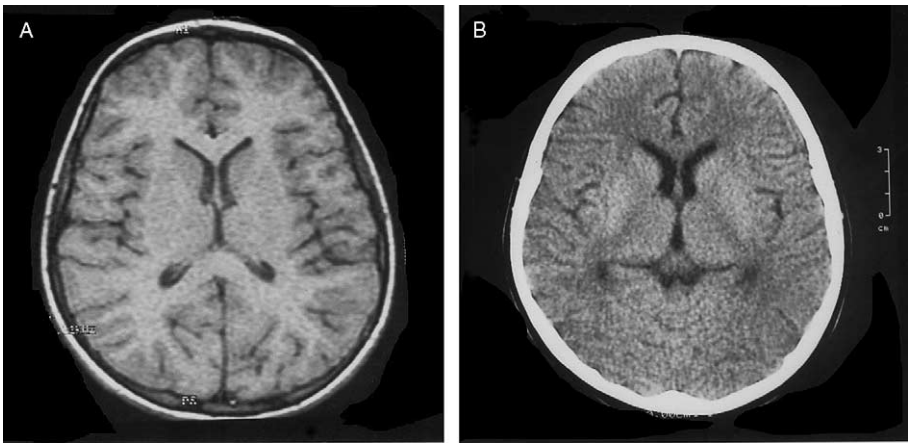

Fig. 1. Magnetic resonance imaging of the patient (A) and cranial CT of the patient (B) performed 7 days after the hospitalization were normal.

and measles immunoglobulin M titers obtained 3 weeks after the active disease were elevated (243 AU and 183 AU, respectively). Serum cytomegalovirus, herpes virus, ebstein barr virus immunoglobulins were negative. Anti nuclear antibodies (ANA), anti DNA and anticardiolipin antibodies were negative. The visual acuity was 20/20 and there was no visual field defect.

Electroencephalography (EEG), magnetic resonance imaging (MRI) and cranial tomography performed 7 days later were normal (Fig. 1). There was no sinus vein thrombosis on magnetic resonance venography (Fig. 2). Lumbar puncture demonstrated a cerebrospinal fluid (CSF) opening pressure of 30 cm H_2O. Prednisolone, mannitol, acetazolamide and furosemid treatment was prescribed. Within 5 days the intensity of the headache improved significantly, and 14 days later the patient was symptom free. After CSF pressure was normalised (6–12 cm H2O) prednisolone was stopped and the dose of acetazolamide was lowered for 1 week. Since CSF pressure subsequently raised to 24 cm H_2O, the treatment protocol was prescribed again 30 days later. On lumbar puncture performed 2 months later, the opening CSF pressure was 12 cm H_2O. Her repeated fundoscopic evaluations were normal and she is under follow-up by our clinic regularly.

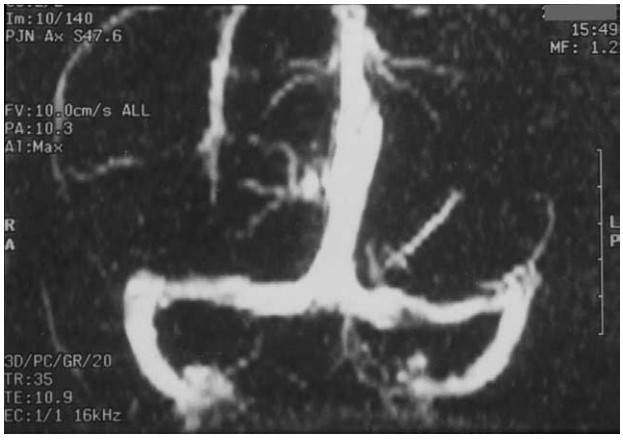

Fig. 2. Magnetic resonance venography performed 27 days after CT and MRI was in normal limits during the second hospitalization period. No sign of sinus vein thrombosis was observed.

3. Discussion

Many reputedly causal factors and associated conditions have been described with PTC [1–7]. These include obstruction to venous drainage, endocrine diseases, drugs, infectious or post infectious conditions, other medical conditions such as antiphospholipid antibody syndrome, occult craniosynostosis, iron deficiency, sarcoidosis, sleep apnea, systemic lupus erythematosus, Turner's syndrome, obstructive nephropathy, atrial septal defect repair and hypervitaminosis A. None of the above mentioned conditions was found in our patient except measles infection.

Three different neurologic complications result from interactions of measles virus with neural tissue: (1) acute post infectious encephalitis, usually appearing 5–14 days after the rash, is thought to be a virus-induced autoimmune disease. (2) Measles inclusion-body encephalitis, which occurs in immune-compromised patients after a latent period of 3–6 months, is believed to be a direct measles virus infection of neural tissue. (3) Subacute sclerosing panencephalitis (SSPE) manifests 2–10 years after primary measles infection as a progressive and fatal chronic neurodegenerative disease caused by persistent defective measles virus in neurons and oligodendrocytes [8]. Furthermore, cerebellar ataxia, retrobulbar neuritis, hemiplegia caused by infarction may be seen after measles [8].

Several theories have been put forward to explain the pathophysiology of PTC. Pathophysiological mechanisms suggested as the cause include increased brain volume caused by increased water content, increased blood volume, increased rate of CSF formation and decreased rate of CSF absorption at the aracnoid villi [9]. A recent study suggested that elevated intracranial venous pressure might be

a universal mechanism in PTC of different etiology. Elevated venous pressure leads to a rise in CSF and intracranial pressure by resisting CSF absorptions [10]. The relationship between PTC and infectious diseases has not been sufficiently defined. In most of the infectious cases, particularly in children, PTC was probably due to thrombosis of one or more of the dural sinuses, specifically the lateral one [2]. However, an autoimmune-mediated post-varicella vasculitis has been speculated to be the responsible cause of PTC in varicella [3]. Furthermore, autoimmune or autoreactive processes as well as direct infectious or inflammatory processes in Lyme disease have also been suggested to be responsible for the pathogenesis of PTC [7].

High incidence of convulsion, personality changes, cerebral edema, and other neurologic deficits may be observed in measles encephalitis. Usually a mononuclear cell pleocytosis of the cerebrospinal fluid and slightly elevated protein levels occur. Although the pathogenesis of measles encephalomyelitis remains uncertain, it does not seem to involve viral replication in the CNS; rather, it more closely resembles the neuropathological findings of experimental allergic encephalomyelitis. Our patient's complaints had begun 11 days after the onset of rash. She had no sign and symptom of clinical involvement of central nervous system during the active course of measles. The diagnostic studies, including EEG, and MRI of the brain were all normal. Because of absence of any other medical conditions, which might explain PTC, her complaints of headache, nausea and vomiting following the illness were assumed to be related to the appearance of PTC as a possible associated complication of measles.

The occurrence of clinical findings in our patient after 11 days of the onset of rash coincided with the initiation of autoimmune neurological complications of measles. Hence, it may be speculated that post measles autoimmune-mediated vasculitis might probably have complicated as PTC. We think that a virus-induced autoimmune vasculitis might be responsible for the development of PTC in measles, and that further research is required to define the relationship between these two conditions.

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