COMMENTARY. The close temporal proximity of the VZV reactivation with 2 varicella vaccinations during the previous 2 to 3 weeks is strong suggestive evidence for a causal association. However, the authors argue in favor of a coincidental association since virology sequencing showed that the VZV infection in the brain was caused by wild-type VZV. Wild-type VZV has been isolated from vesicles in patients with herpes zoster after immunization, indicating that herpes zoster in immunized people also may result from natural varicella infection that occurred before or after immunization [1]. They suggest that the varicella vaccination, rather than having a detrimental effect, may have prevented further inflammation by stimulating a rapid anti-VZV immune response. Furthermore, the surgical excision of the area of inflammation in the temporal lobe may have prevented further extension of viral infection.

Link between VZV reactivation and CNS disease. Neurological complications of VZV reactivation include zoster induced postherpetic neuralgia, myelitis, meningoencephalitis, VZV vasculopathy, and stroke [2]. Stroke as a complication of VZV reactivation is reported in the elderly but is rare in childhood [3]. When these complications occur without rash (zoster sine herpete), VZV-induced disease is diagnosed by detection of VZV DNA or anti-VZV antibody in CSF. Awareness of the expanding spectrum of neurological complications of VZV reactivation leads to earlier diagnosis and antiviral treatment. A case of congenital varicella syndrome (CVS) in a male infant presented with generalized clonic cerebral seizures at age 4 months [4]. An intracerebral viral reactivation following intrauterine VZV infection was suspected and confirmed. Antiviral treatment was aimed at preventing progression of the disease. CVS presents with skin lesions, neurological defects, eye diseases, and limb hypoplasia.

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INTRACRANIAL HYPERTENSION

SYMPTOMS AND ETIOLOGIES OF PSEUDOTUMOR CEREBRI

Investigators from Ankara Pediatrics, Turkey, evaluate the clinical symptoms and etiology in records of 53 patients (32 female) diagnosed with pseudotumor cerebri (PTC) in a child neurology department between 2005 and 2012. Mean age at presentation was 10.9 years (range 3-17) and one half were age 11 years or younger. Prepubertal patients (under 12 years old) were male in >50%, while 74% patients at puberty were girls. Etiology was undetermined or idiopathic in 30 and symptomatic in 23. Obesity rate was 41% for pubertal patients and 31% for prepubertal patients. Obesity was not related to etiology or puberty. In idiopathic cases, headache was the most common symptom (in 88%), nausea and/or vomiting in 30%, diplopia in 28%, and dizziness in 9%. Papilledema was found in 100%, and VI or VII nerve palsy in 11.3%. An etiologic factor for symptomatic PTC was identified in 43% of patients and included cerebral venous sinus

thrombosis in 6 patients, upper respiratory tract infections in 4, iron deficiency anemia in 3, steroid withdrawal in 3 epilepsy patients, risperidone or cyclosporine usage in 3, Brucella infection, post-traumatic and slit ventricle syndrome in one each of the secondary PTC group. Comorbid disorders in the idiopathic group were related to obesity (hypertension, diabetes), and in the symptomatic group, epilepsy, and vitamin D deficiency. Papilledema was lessened by acetazolamide in 72%. (Degerliyurt A, Teber S, Karakaya G, et al. Pseudotumor cerebri/idiopathic intracranial hypertension in children: An experience of a tertiary care hospital. **Brain Dev** 2014 Sep;36(8):690-9).

COMMENTARY. Idiopathic intracranial hypertension (IIH) is defined as intracranial pressure increase with no intracranial pathology and a normal CSF content [1]. The term, "pseudotumor cerebri," is used where an etiology for intracranial hypertension is identified or suspected [2]. Olfactory impairment, an under-recognized complication of idiopathic intracranial hypertension, is studied in relation to astronauts in head-down tilt positions [3]. Many long-duration astronauts develop signs of elevated intracranial pressure and have olfactory threshold dysfunction.

Controversy regarding efficacy of acetazolamide in IIH. The efficacy of acetazolamide in IIH is questioned since a randomized controlled trial failed to show a significant difference in lumbar puncture pressure, headache disability and visual acuity in the acetazolamide vs placebo groups [4]. A prospective cohort study found that weight loss is an effective therapy in IIH [5][6], leading to the proposition that weight loss may be the reason for the small improvement in the acetazolamide cohort in a most recent IIH Treatment Trial [6]. Proponents of a positive effect of acetazolamide in IIH argue that the effect of acetazolamide on visual field function is independent of its effect on weight loss and does not relate to the anorexigenic effect of acetazolamide [7].

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HEADACHE DISORDERS

SYMPTOMS AND ETIOLOGIES OF ALICE IN WONDERLAND SYNDROME

Investigators from Children's Hospital of Philadelphia, PA, conducted a retrospective chart review of 48 children (average age at presentation 8.1 yrs, range 5-14 yrs; 73% male) diagnosed with "Alice in Wonderland" syndrome (AWS) or "Alice in Wonderland"-like syndrome between 1993 and 2013. Micropsia occurred in 69%, teleopsia in 50%, macropsia 25%, metamorphopsia 15%, and pelopsia (objects appear nearer) in 10%. MRI and EEG were normal. Etiology was infection (viral or