Wernicke Like Encephalopathy in a Child: A Reversible Cause

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Abstract

Wernicke’s encephalopathy (WE) is the best known neurological complication of thiamine deficiency, characterized by ocular symptoms, mental confusion and ataxia. We report herewith a male child with unusual features, suggestive of WE and with thiamine treatment, the response was dramatic. Participated in the national seminar on Consumerism in DNR College, Bvrm

Discussion

Wernicke described in 1878 four adult patients of encephalopathy with ophthalmoplegia with malnutrition. The autopsy then confirmed the characteristic hemorrhagic lesions in dorsomedial thalamus and periaqueductal/periventricular gray matter. The disease is said to be caused by thiamine deficiency. Beriberi is the systemic counterpart of thiamine deficiency and often manifests in cardiovascular collapse. Thiamine is an important co-enzyme in glucose metabolism and it is converted into its active form, thiamine pyrophosphate. There are three important enzyme systems in the tri-carboxylic acid cycle in the liver, kidney, heart and brain which are dependant upon thiamine. (4) A triad of clinical symptoms, namely, global mental confusion, ocular abnormalities and ataxia are important in the diagnosis of WE. (5) However, most of the patients may not manifest all the above symptoms always; instead a variable combination of presentations is sufficient to diagnose WE. Therefore, a high index of suspicion is necessary in the high risk patients with any of the above myriad of clinical symptoms. (6) Extra neurological
involvement had been also described in WE in children with predominant cardiovascular symptoms namely, chest pain, dyspnoea, tachycardia and heart failure. Confirmation of the diagnosis is possible by reduced red blood cell transketolase levels and high serum pyruvate and lactate but the results could not be obtained immediately. Magnetic resonance imaging of the brain may reveal abnormal T2 bright signals in the dorsomedial thalamic nuclei and putamen with loss of volume of the mamillary bodies. WE is both preventable and treatable condition and treatment should be initiated any patient who presents with any of the above clinical signs and symptoms, especially in the high risk group. The onset of the disease may be acute, sub acute or chronic and symptoms of classical triad may not be evident. Therefore, thiamine therapy is warranted if any component of Wernicke’e encephalopathy triad is present an appropriate clinical setting (7). If untreated, patents may progress to hypotension, hypothermia and respiratory failure and so early treatment could avoid such complications and fatality, provided the awareness of this disease present among physicians. Although known in malnutrition or in patients on parenteral nutrition, our patient was an apparently healthy boy who responded to thiamine with the clinical suspicion and management.
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