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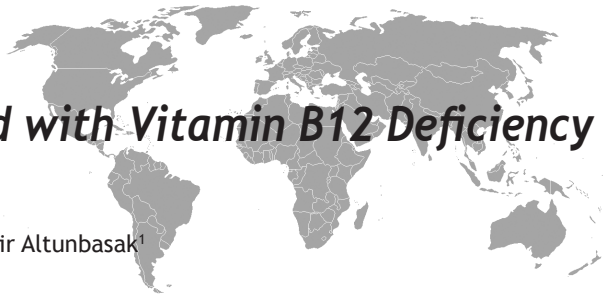
Case Report



Pseudotumor Cerebri Associated with Vitamin B12 Deficiency

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ABSTRACT

Pseudotumor cerebri, also named idiopathic intracranial hypertension, is a condition of raised intracranial pressure in the absence of a mass lesion or cerebral edema. It is rare in childhood and often associated with multiple clinical conditions. In this report, we presented a case with peudotumor cerebri associated with megaloblastic anemia due to vitamin B12 deficiency.

Key words: *Pseudotumor cerebri, vitamin B12 deficiency, children*

Vitamin B12 eksikliği ile İlişkili Psödotümör Serebri

ÖZET

Psödotümör serebri, ayrıca idiyopatik intrakraniyal hipertansiyon olarak da adlandırılan, serebral ödem ve kitle lezyonu olmaksızın intrakraniyal basıncın arttığı bir durumdur. Çocukluk çağında nadirdir ve birden fazla klinik durumlarla ilişkilidir. Bu yazıda, vitamin B12 eksikliğinin neden olduğu megaloblastik anemi ile ilişkili psödotümör serebrili bir olguyu sunduk.

Anahtar kelimeler: *Psödotümör serebri, vitamin B12 eksikliği, çocuklar*

INTRODUCTION

Pseudotumor cerebri (PTC) is a disorder characterized by increased intracranial pressure with no evidence of infection, vascular abnormality or space occupying lesion. Main symptoms are headache, vomiting, diplopia and visual disturbances (1). Pseudotumor cerebri may be either primary or secondary to some conditions. Secondary causes of PTC may include some medications, endocrine abnormalities, autoimmune disorders, cranial venous outflow abnormalities, and anemias (2-4). Pseudotumor cerebri associated with anemia has been reported rarely in the literature (4,5). Here, we reported a case with PTC due to megaloblastic anaemia who recovered rapidly after B12 treatment.

CASE

A previously healthy, 13-year-old boy presented to our hospital with complaints of progressively worsening headaches, vomiting, diplopia, and blurred vision for one month before admission. He had no medical problems and did not take any medication. His family medical history was unremarkable. However, his foods did not containe enough vitamin B 12 for years. On admission, vital signs and anthropometric measures were in normal limits. He was pallor. There was no impairment in his consciousness. Each pupil was normal in size, shape, and reaction. There were no features of neck stiffness and any cranial nerve abnormalities. Biteral papilledema was found on fundus examination. The remainder of the neurological examination was normal. An otolaryngologic examination revealed no abnormalities. There were no ataxia, paresthesia, autonomic dysfunction, and impaired vibration sense in the patient.

His hemoglobin level was 8,4 g/dL, hematocrit 25,2%, mean corpuscular volume 94,2 fL, white blood cell count 6200 cells/mm3, and platelet count 154.000 cells/mm3. Peripheral blood smear revealed anisocyto-

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sis, polikilocytosis, macrocytic erythrocytes and hypersegmented polymorphonuclear granulocytes. Serum vitamin B12, folic acid, and homocysteine were 137 pg/ml (normal:197-866), 12 pg/ml (normal:3.1-17.5), and 9.2 µmol/L (normal: 4.3-9.9) respectively. Other routine biochemical tests were within the normal limits. His thyroid hormone profile was normal. Serological tests were negative for hepatitis A-C viruses, Epstein-Barr virus, Cytomegalovirus, Toxoplasma gondii, Rubella virus, Mycoplasma pneumoniae, and Chlamydia pneumoniae. His lumbar puncture showed an increased opening pressure (430 mm of water). The CSF was clear with normal cell count, protein, and glucose content. CSF culture for bacteria was negative. His cerebral magnetic resonance imaging, and magnetic resonance venography were normal. The patient was diagnosed PTC associated with megaloblastic anemia. He was given acetazolamide orally and hydroxocobalamin intramuscularly. After one-week, the headache, vomiting and visual blurring were improved considerably.

DISCUSSION

Neurological problems resulting from vitamin B12 deficiency have been known for many years. The common characteristics of vitamin B12 deficiency in children include megaloblastic anemia, feeding difficulties, developmental delay, failure to thrive, hypotonia, lethargy, and seizures (6). PTC have also been reported rarely in literature (5). Pseudotumor cerebri is characterized by increased intracranial pressure. This pressure increase can lead to headache, nause, vomiting, associated papilledema and visual disturbance usually blurred vision. PTC may result from several factors including endocrin and metabolic disorders, some medications, intracranial venous thrombosis and anemias (3). PTC cases associated with iron deficiency anemia have been reported in the literature (4). To our knowledge, there are a few PTC cases associated with megaloblastic anaemia (5,7,8,). Murphy et al. reported a 22-year-old man who developed PTC and pernicious anaemia (7). A similar case has been reported by Yetgin et al. (8). Their case was a 14-year-old girl who was diagnosed PTC and vitamin B12 deficiency. The case by van Gelder was a 19-year-old woman with megaloblastic anemia and PTC (5).

With investigation of underlying causes of secondary PTC, we performed several examinations and screening tests for CNS infections, cerebral focal lesion, stroke, cerebral sinus thrombosis, and endocrine abnormalities. We found only megaloblastic anaemia due to vitamin B12 deficiency as etiological reason for increased intracranial pressure. It is unclear that by which mechanisms the anemia can lead to intracranial pressure elevation and papilledema. Several authors are of the opinion that the papilledema will be ascribed to cerebral and optic nerve anoxia, with resultant increased capillary permeability and subsequent cerebral edema followed by increased intracranial pressure (7,9). In conclusion, vitamin B12 deficiency should be kept in mind in differential diagnosis of PTC because of its easy treatment and prevention.

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