Idiopathic intracranial hypertension associated with mild traumatic brain injury in a pediatric patient – Case report

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ABSTRACT

Mild head injury has been described as rare cause of idiopathic intracranial hypertension (IIH). In the presence of IIH, initial treatment is clinical and surgical treatment, such as lumboperitoneal shunt. Most cases have a good prognosis. The patient have 9-year-old male, went to the emergency room with a history of accidental fall, presenting headache, vomiting and blurred vision. Physical examination showed good overall condition. Neurological examination: normal. Fundoscopy: incipient bilateral papilledema. Normal cranial CT. The general picture suggested by exclusion of other causes IIH diagnosis. He underwent symptomatic treatment with acetazolamide, painkillers and rest. Discharged from the hospital on the eighth hospital day with no complaints being referred for outpatient treatment.

KEYWORDS

Intracranial hypertension, craniocerebral trauma, child, papilledema.

RESUMO

Hipertensão intracraniana idiopática associada a trauma cranioencefálico leve em paciente pediátrico – Relato de caso

O traumatismo cranioencefálico leve tem sido uma causa rara de hipertensão intracraniana idiopática (HII). Na presença de HII, o tratamento inicial é clínico e o tratamento cirúrgico é feito por meio da derivação lumboperitoneal. A maioria dos casos cursa com bom prognóstico. Um paciente com 9 anos de idade, masculino, foi admitido na emergência com história de queda acidental, apresentava cefaleia, vômitos e visão turva. Ao exame físico apresentou bom estado geral. Exame neurológico: normal; fundoscopia: papiledema bilateral incipiente; TC do crânio normal. O quadro sugeriu o diagnóstico de HII, por exclusão de outras causas. Foi submetido a tratamento sintomático com acetazolamida, analgésicos e repouso. Recebeu alta médica hospitalar no oitavo dia, sem queixas, sendo encaminhado para acompanhamento ambulatorial.

PALAVRAS-CHAVE

Hipertensão intracraniana, traumatismos craniocerebrais, criança, papiledema.

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Introduction

Idiopathic intracranial hypertension (IIH) is a neurological disorder characterized by increased pressure of cerebrospinal fluid (CSF) in the absence of any expansive processes, with CSF and normal imaging studies.[1] It is most commonly found in young women, obese and of childbearing age.[2] Although rare in childhood, clinical characteristics different of adults may allow develop with significant ocular complications.[3]

The evolution of IIH children is variable, symptoms may appear within hours or evolve progressively and insidious.[4] Headache, diplopia, nausea and vomiting are classic symptoms, but in young children other symptoms are described as photophobia, anorexia and myalgia.[5] The sixth cranial nerve palsy, recently described as one of the signs of IIH is more common in children than in adults.[6] The diagnosis in children is complicated by atypical clinical presentations, such as irritability and drowsiness, and difficulty in diagnosing the decreased visual acuity.[7]

Case report

Child, 9-years-old, male. Admitted to the emergency room with headache, vomiting and blurred vision. The patient presented a mild head trauma, caused by accidental fall twenty-four hours ago. He had a clinical history of bronchial asthma and had sporadic use of corticosteroids. Physical examination showed good general condition. He had height of 1.43 m. and weight of 78 kg and BMI: 38.1. Neurological examination: normal. Fundoscopy: bilateral papilledema. Normal cranial CT: no fracture, brain swelling or bleeding. The general picture suggested by exclusion of other causes IIH diagnosis. He underwent symptomatic treatment with acetazolamide, painkillers and rest. Discharged from the hospital on the eighth hospital day with no complaints being referred for outpatient treatment.

Discussion

The diagnosis of IIH is based on clinical presentation, neurological, ophthalmological, and exclusion of other causes of hypertension intracranial.[8] The pathophysiology has been still unclear. Current theories converge in increased sagittal sinus pressure resulting from extracellular edema.[9] Standridge[10] suggests an increased in cerebral blood volume, decreased CSF absorption, and a possibility of obstruction of cerebrospinal fluid or venous flow.

In children, the association with systemic diseases and medications is more frequent than in adults.[4] Tetracycline, vitamin A and corticosteroids withdrawal are medication often associated with IIH.[11] Among systemic diseases are associated as systemic lupus erythematosus, uraemia and hypothyroidism.[1] Obesity in adult is an important factor associated, about 90% of patients with IIH are obese.[12] Adolescents affected tend to be overweight, but obesity does not to be really risk in patients under 10 years of age.[13]

The mild head trauma previously described in association with IIH, but in most cases further tests showed sinus venous thrombosis or cerebral edema.[4] The CT scan is important for the noninvasive diagnosis to exclude tumor, edema and ventricular dilatation, although their signs are inconsistent and require a more reavaluation.[13]

Magnetic resonance imaging (MRI) has greater sensitivity in detecting isodense tumors, meningeal infiltrations, pathologies of the posterior fossa and other subtle intracranial abnormalities[10] and a set of signals, such as enhancement and protrusion of the prelaminar optic nerve, vertical tortuosity of the orbital optic nerve, distention of the perioptic subarachnoid space and flattening of the posterior sclera, allowing the assistance of the diagnosis of cranial hypertension.[14] The magnetic resonance angiography allow the detection of central venous thrombosis in cases of patients with IIH who have normal CT and MRI, however, the sensitivity and specificity of clinical examination are not satisfactory and the rate of false-positive findings and false-negative further compromises methods.[15] In case of unchanged neuroimaging, lumbar puncture is critical to measure the CSF pressure and to exclude meningitis.[16]

Not all patients with IIH require treatment, asymptomatic patients with no change in vision and minimal papilledema can be monitored to check symptoms progress.[17] In the other patients, the onset of treatment of IIH must first delete the associated factors, such as hypervitaminosis A or tetracyclines use[18] and a controlled diet with low calorie, if obese patient, and with hydrosaline restriction.[19] The treatment is based on monotherapy with carbonic anhydrase inhibitors such as acetazolamide, or their association with systemic corticosteroids such as prednisone or dexamethasone, since unused for a long periods.[20] In refractory cases with progressive loss of visual acuity, are options to lumbar puncture and surgical treatment, such as ventriculoperitoneal or lumboperitoneal shunt, with this infection lower rate.[21] The fenestration of the optic nerve sheath is a surgical option in patients with progressive visual loss who have mild headache or easily control.[18]

The IIH is considered a benign condition compared with other causes of cranial hypertension, but has been reported decreased visual acuity in 25% of cases and possible progression to blindness in not properly treated cases.[22]

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