

BENIGN INTRACRANIAL HYPERTENSION - ETIOLOGY, CLINICAL AND THERAPEUTIC ASPECTS

Tzoukeva Al., N. Deleva, A. Kaprelyan, I. Dimitrov
*I-st Clinic of Neurology, Department of Neurology,
Prof. P. Stoyanov Medical University of Varna*

ABSTRACT

Introduction: BIH is a syndrome defined by increased intracranial pressure, normal CSF composition, absence of ventriculomegaly and intracranial lesion. Review of the literature reveals unknown etiology in majority of patients, various clinical symptoms and different treatment options.

Objective: to study the etiology, clinical and therapeutic features in patients with BIH.

Material and methods: Twelve patients with BIH, admitted to the hospital for the last 5 years were included in the study. The diagnosis was established on the basis of physical and neuro-ophthalmologic examinations, CT or MRI, and CSF evaluation. The major outcome criterion was the regression of papilledema.

Results: All patients were young women. The most frequent clinical manifestations were transient visual obscurations, nausea, and headache. Neuro-ophthalmologic examination showed normal visual acuity, bilateral papilledema, enlarged blind spots, and bilateral abducent nerve paresis in two patients. Obesity, recent increase of weight and menstrual irregularities were potential risk factors. Papilledema resolved over the next 2-3 months after different treatments. In one patient three relapses were registered.

Conclusion: Our own notes suggest that BIH most commonly associates with young age, female sex, and obesity. Neuro-ophthalmologic examination, neuroimaging, and CSF evaluation are the most useful diagnostic tools. The application of diuretics and corticosteroids most often provides a successful control over clinical symptoms and papilledema.

Key words: benign intracranial hypertension, clinical manifestation, risk factors, treatment

INTRODUCTION

Benign intracranial hypertension (BIH) or pseudotumor cerebri is a syndrome that is defined by increased intracranial pressure, absence of ventriculomegaly, no evidence of intracranial extensive lesion and normal cerebrospinal fluid (CSF) composition (1, 4, 6, 14). In the majority of patients with BIH the etiology

remains unknown, but there are several hypotheses regarding the potential risk factors (4, 5, 11, 16). The strongest evidences for associations with BIH exist for young age, female sex, obesity or weight gain, tetracycline or vitamin A use and some endocrine and metabolic dysfunctions (7, 9, 10, 11, 13). According to the literature, the most common presenting symptoms are headache, nausea, and vomiting, transient visual obscurations (1, 5, 14). In general, methods of treatment include weight loss, withdrawal of associated medication, acetazolamide or furosemide therapy, systemic corticosteroids, repeated lumbar punctures, and surgical decompression (2, 3, 4, 8, 12, 15).

Recently, the etiology of BIH remains uncertain and evidence-based guidelines for patients' management are lacking. Therefore, we decided to study the etiology, clinical and therapeutic features in patients with BIH diagnosis.

MATERIAL AND METHODS

We studied retrospectively twelve patients with BIH, hospitalized in Ist University Neurological clinic for the last 5 years (2002-2007). The diagnosis was established on the basis of physical and neuro-ophthalmologic examinations, brain computed tomography (CT) or magnetic resonance imaging (MRI), and CSF examination with pressure measurement. The major outcome criterion was the regression of papilledema.

RESULTS

All twelve patients were women, aged from 28 to 43 years (mean age 34.6 ± 7.4). The most frequent clinical manifestations are transient visual obscuration, nausea, and headache (table 1).

Table 1. Clinical manifestations.

Clinical manifestations	Number of patients
headache	11
nausea	12
vomiting	7
pulsate tinnitus	8
transient visual obscuration	12
bilateral abduction deficit	2

Neuro-ophthalmologic examination showed normal visual acuity, bilateral papilledema, which was asymmetric in ten of all twelve cases, enlarged blind spots at visual field testing in all patients, and bilateral abducent nerve paresis in two patients. Lumbar puncture presented increased intracranial pressure greater than 25 cm water with normal CSF-composition. Cerebroorbital neuroimaging confirmed no evidence of intracranial masses and normal size of ventricles. The most common potential risk factors for BIH are obesity, recent increase of weight, and menstrual irregularities (table 2).

Table 2. Potential risk factors.

Risk factors	Number of patients	
Obesity	over weight - BMI 25-29	1
	obese class I - BMI 30-35	7
	obese class II/III - BMI >35	3
weight gain	8	
secondary hypoparathyroidism	1	
risperidone treatment	1	
doxycycline treatment	1	
menstrual irregularities	5	

Different treatment strategies were applied (table 3). Therapy with acetazolamide (one patient) or furosemide with mannitol (ten patients) was started. In cases where papilledema did not resolve, steroids were added (five patients) or therapeutic lumbar puncture was repeated (one patient). Correction of hypocalcaemia was made in one patient. Papilledema resolved over the next 2-3 months. In one of the BIH patients three relapses were registered.

Table 3. Treatment strategies.

Treatment	Number of patients
weight loss	4
acetazolamide	1
furosemide with mannitol	10

corticosteroids	5
therapeutic lumbar puncture	1
correction of hypocalcaemia	1
cessation of doxycycline	1
cessation of risperidone	1

DISCUSSION

The presented cases reveal that several conditions and risk factors are associated with BIH. In our study, obesity and weight gain were the most commonly recognized risk factors as it is reported in the literature (4, 8). Doxycycline is currently considered a cause for BIH (7). Hypoparathyroidism is probably associated with BIH as well (11). Menstrual irregularities are likely to be a co-morbidity of obesity rather than a cause of BIH itself. Risperidone has not been reported to cause BIH, furthermore weight gain occurs due to risperidone use. That's why it is uncertain which of these agents may cause BIH. Clinical symptoms of BIH in our study were those presented in the literature (1, 4, 5, 6, 15).

There is no clear consensus of BIH therapy. First-line therapeutic intervention is weight loss, diuretics, corticosteroids, withdrawal of medication-associated intracranial hypertension, and repeated lumbar punctures (4, 8, 12, 15). Surgical interventions at later stages are performed in cases of progressive visual loss refractive to medical therapy (2, 3, 6). The treatment with diuretics - corticosteroids, serial lumbar punctures, cessation of doxycycline and risperidone, in combination with diet and weight loss, were effective in our cases - papilledema resolved in three months at the latest. According to the previous studies, treatment of BIH, associated with the most common etiology - obesity or weight gain, is usually successful when conducted with diuretics, diet and corticosteroids (8, 12, 14, 15). The knowledge of different etiological causes of BIH, some of them still uncertain or casuistic, allows the addition of other specific therapy (substitutional), or withdrawal of BIH associated medication (4, 7, 9, 10, 13, 16). The evidences exist that in cases with rapidly deteriorating visual function with persistent headache, surgical treatment is available (1, 2, 6, 14).

CONCLUSION

Our own notes suggest that BIH most commonly associates with young age, female sex, and obesity. Neuro-ophthalmologic examination, neuroimaging, and CSF evaluation are the most useful diagnostic tools. Early diagnosis and application of diuretics or corticosteroids most often guarantee the successful control over clinical symptoms and papilledema.

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Address for correspondence:

Dr. Aleksandra Tzoukeva, PhD,
Department of Neurology,
Prof. Paraskev Stoyanov Medical University of Varna,
55, M. Drinov Str., 9002 Varna, Bulgaria,
E-mail: al_tz@abv.bg

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CRANIAL AND SPINAL NEUROPATHY IN LYME NEUROBORRELIOSIS

N. Deleva, A. Kaprelyan, S. Geneva, Al. Tzoukeva, I. Dimitrov
*1st Clinic of Neurology, Department of Neurology,
Prof. P. Stoyanov Medical University of Varna, Bulgaria*

ABSTRACT

Introduction: Lyme borreliosis is a multisystem inflammatory disease caused by the spirochete *Borrelia burgdorferi*, transmitted to humans by infected ticks. The neurological involvement most frequently presents with various types of neuropathy or may include symptoms and syndromes caused by cerebral and spinal cord damage.

Objective: to study the clinical manifestations and outcome of cranial and spinal neuropathies in patients with Lyme neuroborreliosis.

Material and methods: Twelve patients (6M/6F, between 22 to 57 years of age) with cranial and spinal neuropathy of various types were included in the study. The diagnosis of neuroborreliosis was based on the medical history, neurological examination, EMG, and specific laboratory tests. Up to 3 years patients' follow-up was done.

Results: Two patients had optic neuritis with asymmetrical reduction of vision in both eyes. Two patients presented with involvement of cranial nerves and two with