Neuro-ophthalmological aspects of idiopathic intracranial hypertension

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Idiopathic intracranial hypertension (IIH) is a polygenic syndrome, which is characterized by the following features: intracranial hypertension symptoms (including papilledema), cerebrospinal fluid pressure increase over 200 mm H2O under the absence of focal neurological symptoms (except for the paresis of the VIth pair of cerebral nerves), according to the data of magnetic resonance imaging, the absence of deformation, gastrointestinal tract shifting or obstruction and other cerebrovascular pathology, except for the features of cerebrospinal fluid pressure increase [28, 41, 141].

The synonyms of IIH, which can be seen in the literature, are the following: pseudotumor cerebri, hydromeningitis, otitic hydrocephalus, toxic hydrocephalus. For the first time, the disease was described by N. Quincke as hydromeningitis at 1897 [108]. Patients treated by N. Quincke complained of headaches, defection of acuity vision; papilledema was defined in the fundus of eye. He bound these symptoms with the increase of intracranial pressure and considered them to be caused by the increase of CSF secretion.

In 1904, M. Nonne called this syndrome "pseudotumor cerebri" and provided the cases of clinical implications of cerebral tumor; however, the further follow-up of such patients showed that this diagnosis should be excluded [99]. In 1931, S. Symonds described three cases of intracranial pressure increase of children with middle ear disease. In this work he offered the term "otitic hydrocephalus" [122].

In 1937, D. McAlpine described the cases of external or toxic hydrocephalus, which had the signs of typical syndrome of intracranial hypertension infection and were not bound with middle ear disease [90]. In this year, the neurosurgeon W. Dandy described 22 cases of the increased intracranial pressure without having cerebral tumor and defined first diagnostic criteria of this disease. With the development of neuroradiological methods, the disease was more clearly defined. J. In 1955, Foley introduced a new term "pseudotumor cerebri" to differ this state from other types of intracranial hypertension dangerous for life [40].

Further, some authors [26, 100, 137, 144] registered the cases of severe loss of vision in many patients with IIH and noted that a widely-used term "pseudotumor cerebri" did not comply with the nature of this disease. W. Buchheit and coauthors offered the term "idiopathic intracranial hypertension" and "secondary pseudotumor cerebri" depending on the fact whether the cause of the disease was found or not. [101].

Now the terms "idiopathic intracranial hypertension" and "pseudotumor cerebri" are generally accepted, the pathology was identified as a nosologic unit in accordance with ICD-10 (G 93.2).

Diagnostic criteria

Original criteria of IIH were described by W. Dandy in 1937 [28], changed by T. Smith in 1985, amended by D. Friedman and D. Jacobson in 2002 [41] and are used until the present time as "modified Dandy criteria":

1. Signs and symptoms of the increased intracranial pressure (including papilledema).
2. Absence of focal neurological symptoms (except for abducens nerve palsy).
3. Registered cerebrospinal fluid pressure increase over 200 mm H2O (measured in position on the side).
4. Normal composition of cerebrospinal fluid.
5. Absence of hypertension signs, foreign tissue lesion on magnetic resonance tomography (MRT).
6. No other reason of intracranial hypertension was found.

Epidemiology

IIH is a disease of unknown origin mainly developed in fatty women of childbearing years [16]. According to various authors IIH occurs in 0.03-2.2 cases per 100,000 persons [27, 34, 35, 16, 62, 76]. Frequency of IIH differs worldwide; it is estimated between 1-2 cases per 100 thousand persons [42]. According to the data of prospective research in Livia, incidence rate is 2.2 per 100 thous. [34], in USA 1.07[31], in Japan 0.03 [71], in North Ireland 0.6 [27], in Israel 0.57 [76]. The disease is rarely detected in the countries, where the frequency of obesity as an important factor in the disease development is low, and is often detected in the countries with high quantity of people suffering from obesity among population [31, 71]. So, IIH is relatively infrequent state, but its prevalence is increased up to 13 cases per 100 thousands (women in the age from 20 to 44 years with normal weight) and to 19 cases per 100 thousands (women in the age from 20 to 44 years, which weight is 20 per cent higher than the ideal weight [31]. With the growth of excess weight and obesity prevalence in the world, it is possible that incidence of IIH would increase.

The majority of the studies showed the age of the disease start between 11 and 58 years, in average 30 years [31, 34, 142]. IIH is the rarity for men, a ratio between women and men amounts from 4.3:1 to 8:1 [29, 46, 51, 55, 92]. In children and adolescents the disease tends to be rare [49, 120]. However, there are the cases of incidence described in children and newborn infants [22, 50, 64, 70, 80, 138]. According to the data of some authors, boys and girls suffer from this illness with the same incidence [50, 120, 146], as the others; girls are more likely to suffer from illness [49].

According to studies carried, ethnic origin does not influence to the rate of IIH incidence [46, 109, 142]. The data of genetic disposition to IIH are found in literature [25, 38, 66].

Aetiopathogenesis

The women at the age of 18-50 years often suffer from obesity. Researcher inform about the increase in body mass index (BMI) in patients from 38% to 91% [31, 34, 76]. Gaining weight before 12 months to the first symptoms of disease is particularly important.

The origin of process pathogenesis is unknown yet, but various modern theories can explain the increase of intracranial pressure, including the following follows: CSF hyper production, resorption breach, venous drainage violation [2, 136, 139]. To explain the connection between obesity and intracranial hypertension there is generally accepted mechanical theory: the increased intracranial pressure causes a rise of intrathoracic pressure followed by the enhance of cerebral intravenous tension and results in higher strength of arachnoidal granulations [46, 139, 142]. At the same time, other authors consider endocrine diseases to be the main reason of the increase of intracranial pressure under obesity [112], but it is difficult to explain why IIH occurs quite rarely, although obesity is characterized with a high prevalence, and primarily in women.

There are no other proved reasons of IIH except for female sex and obesity. The origin of this pathology is bound to a range of pathological states and the list keeps growing. Among endocrine factors it is worth mentioning menstrual disorder [31, 40, 72, 78], pregnancy [51, 148, 149], polycystic ovarian syndrome [69], using contraceptives [31, 72, 123], thyroid body and butterfly adrenal diseases, hypoparathyreosis [31, 142]. IIH can be bound as well with administration of following drugs: vitamin A [110, 115], vitamin B12 [103], tetracycline [126], monocycline [23], doxycycline [82], nitrafurans, lithium, nalidixic acid, long-termed corticosteroid therapy or its termination [97], psychotropic medication [2, 29, 53]. The disease can be bound with systemic disorders (chronic uremia, systemic lupus erythematosus, somnipathy, cardiopulmonary decompensation, Behcet's syndrome), blood diseases (anemia, leucosis, hemophilia, idiopathic thrombocytopenic purpura), infectious factors (Lyme disease, brucellosis, lues, Guillain-Barre syndrome, middle ear inflammatory diseases), heavy metal poisoning, craniocerebral injury [31, 46, 65, 72, 105, 124, 125, 135, 142].

The list of IIH etiologic factors keeps growing, and it is not proved whether these factors caused a clinical syndrome or this connection is occasional. The mechanisms causing the increase of intracranial pressure are entirely unknown, and possibly vary depending on the etiologic factors [82].

Recently, intracranial venous hypertension connected with various stenoses of venous sinuses that results in CSF absorption disorder is increasingly seen as possible mechanism of IIH development.

Clinical diagnostic peculiarities

IIH exhibits signs and symptoms of the increased intracranial pressure. In 68-98% patients with IIH, the most frequent symptom is headache [34, 63, 76, 142], which may have generalized, constant or periodic character, more often occurs in the morning [94, 111, 141] and can grew higher with coughing, tension, Valsalva's manoeuvre [137].

Vision disorder is the second the most developed symptom of IIH. The disease can manifest only vision defection observed in 19.5% according to the data of J. Galvin [46], exhibits vision defection in 35-70% patients [104, 123]. 57-72% complain on periodic visual blur attacks, which are manifested in ablepsia or nearly ablepsia for about some seconds followed by complete recovery of vision [34, 76, 142]. Visual blur attacks may start spontaneously but more regularly in changing body position. There is no definite explain to this phenomenon but the most authors of bind it with temporary disturbed circulation in visual nerves and its compression by edema [57, 114]. Less than 5% patients complain on diplopia, photopsia, flashing lights [109].

Patients with IIH may suffer from head noises, tinnitus, sometimes having pulsatory (in 60% of patients) [142]. Complains on nausea and vomiting are possible (in 40% patients) [92]. Unilateral or bilateral paralysis of abducent nerve is possible in 17-33% cases [34, 92, 137]. Lesions of other cerebral nerves were registered very rarely, in single clinical cases [37]. Sometimes the sensation of brain tunic, including nuchal rigidity and photophobia can occur [42]. Some researches inform about hyposmia cases [74]. Sometimes patients complain on impaired concentration, poor memory [118], possible dissociative disorders, depression, and psychic tension [30].

Papilledema is one of the main criteria of IIH and its absence brings this diagnosis into question [87]. Papilledema is diagnosed according to ophthalmoscopic picture that despite of subjectivity is a basis for the majority of classifications, which repeat and complement each other to a large extent. A. Ya. Samoylov (1959), based on the quantitative pupillometry methods he had developed, divided papilledema into three stages: progressive papilledema, maximum papilledema, regressive papilledema [8].

Ye. Zh. Tron (1968) distinguished the following stages of swollen disk: initial, severe, full-blown, papilledema in atrophy stage, chronic optic nerve atrophy. Complicated papilledema is separately distinguished (papilledema together with direct impact of pathological process on visual analyzer) [11].

Hoyt W. F. and Knight C. L. (1973) identify an early stage, maturity stage, chronic edema stage and atrophic edema [59].

Frisen L. (1982) offered to identify the following stages of papilledema development: 0 – normal optic disc, 1 – very early stage, 2 – early, 3 – moderate, 4 – severe, 5 – full-blown [44].

In our opinion, it is advisable to distinguish the following stages of papilledema development: initial, moderately expressed, expressed, a stage of regression, secondary atrophy (N.K. Serova classification) [9]. Division into stages is very conditional, some stages interchange each other being unnoticed, however, it is reasonable and allows characterizing a complex of ophthalmological features by a short term.

The early or initial stage, which is characterized by blurred disk, hyperemia, acentic papilledema (optic disc), congestion, spontaneous venous pulsation absence is more complex for diagnostic. Moreover, each of the listed ophthalmological features, which are distinctive for the early stage of papilledema, can be find in a normal state or other pathology. Inborn, abnormal changes in the optic disc structure can imitate initial papilledema and result in diagnostic mistakes [41]. It may be difficult to subjectively determine optic disc hyperemia. A normal color of the disk depends on arteriolovenular bridge state and can range. In hypermetropia, optic disc tends to hyperemia, under myopia - to blanching.

Hayreh S.S. (1977) carried numerous studies on optic nerve blood supply and pathogenesis of papilledema and reached a conclusion that firstly, the edema appears on the bottom line of the disk, then on the upper one, further, on the nasal, and finally - on the temporal one. Optic disc hyperemia is defined later and results from the capillaries widening on its surface, and only after that, retinal venous dilations appears [56]. Hoyt, W. F. and Knight C. L. (1973) describe the early stage of papilledema as follows: minimal changes in the peripapillary fibers of retina emerge, it looses its line structure properties, and corneal reflex is varied [59].

Some authors consider retina congestion and venous pulsation absence to be very important and early signs of the increased intracranial pressure. However, there is no agreed opinion as for significance of identification of spontaneous venous pulsation. A. K. Golenkov (1992) gives precedence of the disappearance of spontaneous venous pulsation as an early sign of papilledema [3], but Cogan D.G (1972), Miller N., Newman N. (1998) do not consider this symptom to be important [24, 93]. According to the data of Lorentzen S.E. (1970), Levin B. E. (1978) this sign is detected in 80% patients examined [81, 83]. Cogan D.G (1972) considers that venous pulsation can be detected in the increase of intracranial pressure as well and pays this symptom no attention in differential diagnostics of papilledema [24].

To distinguish the initial stage of papilledema you should pay attention for a complex of changes since it may be difficult to subjectively determine optic disc hyperemia, and the blurred disk is a frequent sign of optic nerve congenital anomalies and refraction abnormalities. Indirect and direct ophthalmoscopy are carried out for purposes of diagnostics, the photo of fundus of eye has important meaning for case follow-up.

In some cases, fluorescence angiography (FAG) of optic disc area will be practicable, by which initial papilledema is diagnosed. P. O. Mukhamadiyev (1974), in FAG of the eye fundus in the patients with papilledema, marked extravasate output of fluorescein during its passing on retina's vessels and the increase of optic disc fluorescence, as well as residual fluorescence in 30 minutes after colouring agent is added [5]. This practice is important in differential diagnostics with pseudo papilledema, druses of optic disc. The defects of FAG are invasiveness, possibility of complications and complexity of using for seriously ill patients. Ultrasound investigation (USI) is a recommendable alternative of optic disc, which make the detection of druses possible [7].

USI of the orbital part of ON (optical nerve) is used for the detection of intracranial hypertension by measuring the diameter of ON cover [19, 86, 132, 133]. This method is safe, quick, and noninvasive and has no contraindications for use except for eye fundus injuries [117]. In 1964, S. Hayreh showed that in the experimental study on monkey and people that subarachnoid area, which surround optic nerve, is interrelated with intracranial cavity and the changes in cerebrospinal fluid pressure are passed over ON covers [57]. Many researches proved that the diameter of intrathecal area of ON is increased in patients with intracranial hypertension [117, 121, 128, 130, 132]. In 1996, Helkme K. and Hansen H. showed that there is the correlation between a degree of the increase of intracranial pressure (ICP) and the inhance of the intrathecal area of ON [58].

In some cases it is impossible neither confirm nor deflect the diagnosis of the initial stage of papilledema, and the issue can be solved only using a dynamic follow-up [145] and evaluation of the whole brain symptoms. Optical coherent tomography (OCT) and Heidelberg laser retinotomograph are modern noninvasive methods, which make possible to evaluate steometric parameters of papilledema objectively and allow to dynamically observe its development [48, 60, 96, 106]. In Ie. Karam та T. Hedges (2005) opinion, the complexity of differential diagnostics with pseudo papilledema is a disadvantage of OCT [75]. The studies of D. Mulholland and others (1998), G. Trick and others (1998), C. Tamburrelli and others (2000) showed that HRT method correlates well with other valuation methods of optic disk state. Evaluation difficulties of HRT include the fact that the development of secondary atrophy cannot be distinguished from papilledema regression [96, 106, 132].

Further development of papilledema occured in the increase of fundus, hyperemia, blured disk, outstand of disk in vitreous body. Veins are distensible, sulcated, covered with hydropic tissue; extravasations, transudation focal points appear.

Papilledemas usually develop simultaneously on both eyes and symmetrically enough. However, there are unilateral and dissymmetric cases described in literature [79, 89, 134, 137]. Hayreh S.S., Lepore F. E., Killer H.E. Wall M. and others explain dissymmetry by anatomical features of the structure of optic foramen and intrathecal area of ON [56, 77, 79, 140]. M. Z. Bezsmertny (2000), considering the development of unilateral papilledema, pays great attention to the features of venous blood circulation of optical nerve and brain [1]. In the opinion of Ie. Zh. Tron, unilateral papilledema is an initial stage of disease, and papilledema needs some time to develop on the second eye. Miller N. та Newman N. (1998) think that in unilateral papilledema, minimal signs of the edema can be found under intent study of other eye as well, the development of these changes is shifted in time. [93]. The development of atrophy during the papilledema is accompanied by the disappearance of edema and complete atrophy of optical nerve prevents the development of papilledema. This antipathy between the atrophy and edema can be be explained by the connection between the presence of axonal transport and nerve fibers activity. Loss of the nerve fibers causes the decrease and, in the atrophic process development, the termination of axoplasmatic current in optical nerve. It results in papilledema's antipathy with atrophy that explains a unilateral nature of the process.

During long-termed papilledema, ophthalmoscopic image changes. Disc hyperemia changes into blanching that is connected with the process of development of nerve fibers atrophy. In the development of the second post congestive atrophy, a decrease of papilledema is not a sign of intracranial hypertension regression. In this case, to evaluate ICP at an ophthalmoscopic image following OCT and HRT data is impossible. To solve this task it is necessary to develop other methods of the research of optical nerve at ICH.

According to the data of I. Merculov (1979), O. N. Sokolova (2002); Miller N., Newman N. (1998), papilledema regression develops for 30-60 days after ICP normalization. According to the data of Zh. Tron (1968), significant reduction of papilledema signs is noticed in 10-14 days, a complete regression occurs within 3-4 weeks after treatment [4, 10, 11, 93].

In absence of treatment, papilledema proceeds to the second atrophy. The period required for the development of atrophy depends on a lot of factors, including heaviness and uniformity of ICH. Atrophic changes can appear within some weeks or days after the first detection of papilledema, especially in those cases when ICH is quickly increased (peracute or cancerous ICH). Sometimes papilledema is in the stage of a chronic edema for months and even years, and then, for unclear reasons, the second atrophy quickly develops [93].

Normal state of visual functions for a long term is a specific feature of papilledema. The data of modern literature state that the decrement in visual acuity is possible in 25-30 % of patients [34, 112, 137, 142], visual field disorder - in 49-92% patients [100, 137, 142].

According to various authors, visual field research is the most informative for the detection of dysopsia at papilledema. Visual field's disorders are represented with the blind spot enhance, concentric arctation, bottom nasal quadrant arctation, defects in the nose. Arcual, central, cekocekal, paracentral scotomas are less prevalent defects [46, 112, 141, 143]. Some researches generalize them instead of describing various visual field disorders and use the system of group classification of the disorders. Wall M. and George D. (1991) published a classification system for static and kinetic perimetry, which was based on a study of 50 patients with IIH [142]. Subsequently, this classification system has been adopted in some published studies in patients with IIH [67, 112, 131]. Many modern authors emphasize the importance of the automated static perimetry for early detection of the visual field defects [12, 17, 36, 54, 116, 144]. The Wall M. and George D. study (1991), which enrolled 50 patients with IIH revealed changes in the visual field of 92% of patients with automatic perimetry and 87% — in patients with static perimetry by Goldmann, although the visual acuity was reduced only in 22 % [142]. Rowe F. and Sarkies N. detailed study (1998) indicates a greater sensitivity of the automatic static perimetry [112]. In 1983 Levin P. S et al. demonstrated the initial impairment of the periphery axons of the optic nerve while maintaining them in the central part. The same results were obtained by Gu X. et al. in 1995 [15, 102].

Decrease in visual acuity is slow and irreversible at later stages of the disease except for patients with sudden vision loss with peracute IIH. The vision can deteriorate from a slight deterioration to blindness [112, 142]. The deterioration of the visual acuity is not typical in the early stages of the disease, but the eye exam is of great importance. Decrease in visual acuity evidences the significant optic nerve damage and indicates the need for its immediate treatment. At the first place, the patients with early central vision loss require the exclusion of the ocular pathology: optic neuritis, ischemic optic neuropathy.

Loss of contrast sensitivity is defined in patients with IIH regardless of the study methodology. Some researchers recommend using of contrast sensitivity studies for further visual function evaluation [112, 119]. Wall M. and George D. (1991) and Verplanck M. (1988) studies indicate a high sensitivity of this method in the primary visual impairment compared to the visual acuity and even visual field study [33, 142].

It is difficult to predict the development of visual impairment in patients with papilledema at IIH. There are opposite views concerning the factors that can affect the appearance of visual impairment. In the opinion of N. Miller (1998) papilledema rapid development leads to more severe visual impairment [93]. According to J. Orcutt (1984), M. Wall et al. (1998) the more pronounced optic nerve edema leads to a significant loss of vision [100, 140]. This view does not match the data from J. Rush (1980), who believes that the disease duration, severity of papilledema, obnubilation availability is not a risk factor for vision loss [113]. Unfavorable factors for the visual function prediction according to J. Corbett et al. (1982) include hypertension and early visual function impairment [137]. H. Quigley (1982) believes that it is possible to avoid significant visual impairment until 30% of the optic fibers are lost [107].

An ophthalmoscopic sign of the optic fibers atrophy is the optic disc blanching against its edema, but the visual functions can be slightly affected or not affected. According to N. Miller and N. Newman (1998), the pale optic disc always indicates the unfavorable prognosis for visual function and it is impossible to stop the vision deterioration even in if ICP normalizes immediately [93].

It should be noted that the IIH course may be asymptomatic, changes can be found during routine ophthalmologist's examinations as papilledema.

Neurovisualization studies

IIH is a diagnosis of exclusion. Patients with the IH signs and symptoms are carefully examined to exclude the mass lesion and ventricular system expansion. Prior the development of the modern brain scanning techniques the skull X-ray examination and PEG was utilized. IIH shows no pathognomonic signs on the magnetic resonance tomography (MRI) and computed tomography (CT), but according to Brodsky M. and Vaphiades M. (1998) the "empty" Turkish saddle is often revealed, as well as normal or reduced in size ventricular system, expansion of the basal cisterns, posterior pole sclera flattening, optic nerve intrathecal space expansion, vertical tortuosity of the optic nerve orbit portion, intraocular protrusion of the optic nerve preliminary portion intraocular protrusion [20]. According to the latest diagnostic criteria it is necessary to exclude the pachymeninx sinuses thrombosis, which has a clinical presentation similar to IIH, which requires CT venography or MR venography [41, 84]. There are few optic nerve CT and MRI studies; the data obtained was not matched with the ophthalmologist examination data and data from other study methods. In 1998 Brodsky M. C. and M. Vaphiades conducted study of the papilledema alignment using high-resolution MRI [20]. Gass A. in 1996 and Seitz J. in 2002 measured the diameter of the optic nerve intrathecal space within normal and in case of papilledema. Neurovisualization is also necessary to determine the contraindications for lumbar puncture.

Lumbar puncture

Lumbar puncture (LP) is used to measure the spinal fluid (CSF) pressure and confirm the diagnosis. To exclude false positive results the LP is performed in lateral position. In IIH the pressure increase is observed above 200 mm. CSF analyzes is mandatory to exclude other neurological disorders [28, 41].

Treatment

Treatment of patients with IIH is aimed at reducing the elevated ICP and visual function preservation. General recommendations include the identification of possible factors that contribute to the disease onset and reduce body weight by at least 10-15% of the total body weight [21, 32, 88]. Typically, a combination of medication and surgical treatments is used. Drug therapy includes diuretics (acetazolamide), glucocorticoids, Topiramate [18, 88, 127, 129]. In case of conservative methods failure to prevent the loss of vision the neurosurgical treatment is applied: lumbar puncture, lumbar drainage, CSF bypass, optic nerve decompression, endovascular stenting, which can effectively reduce ICP [21, 26, 73, 84].

A number of issues and provisions relating to this nosology remain, which require clarification and definition.

Such study will allow examining more thoroughly the papilledema clinical course at IIH, analyzing factors that influence the occurrence of visual disorders in IIH , identify the patients risk groups with regard to the secondary optic atrophy development.

The early diagnostics issue is not resolved yet. No IIH diagnostic criteria developed, no diagnostic sensitivity defined for a number of examination methods, which is necessary for disease early diagnostics. The early diagnostics relevance is not a cause of doubt because with adequate and timely treatment it can prevent irreversible changes and stop the pathological process without the optic atrophy development and its consequences.

Intravital study of the optic nerve characteristics in IIH using modern neurovisualization techniques and comparing the results with ophthalmological examination data will help to better understand the papilledema stages, offer new methods of such patients' examination for IIH early detection and timely treatment.

No effective methods developed for follow-up and evaluation of the neurosurgical treatment quality in patients with IIH.

Lack of IIH issue publications in the domestic ophthalmic literature requires an algorithm of examination, treatment, and monitoring of patients with such pathology.

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