"How to Diagnose Migraine?"

Referring back to the ending of the previous article, in which I explained the lethal nature of migraine, it should be noted that migraine is not always easy to diagnose. I have written that so far, in accordance with the prevailing medical criteria and in social awareness, we have associated migraine with a severe headache. However, migraine disturbances occurring along with a weak or improperly associated headache, and even without such a pain, are sometimes wrongly diagnosed and treated or remain untreated. Migraine can occur in children, which, because of their natural predispositions, can result in the expressiveness of the mechanism of this disease by a rapid occurrence of other symptoms, more dramatic than a headache, such as fainting or loss of consciousness. Patients often impose diagnosing on themselves, having no capabilities (medical knowledge or equipment) to verify the suspicion and do not go with the problem to a doctor, trying to "cure" themselves by alleviating pain. The patients I asked about headaches often denied them, but after a more thorough interview they admitted that they "had never had a headache, but they had pains in their sinuses, ear or eyes very often". For this reason in this article I am going to describe how to take notice of pathologies which, being misinterpreted or underestimated, mask migraine - a disease virulent for the visual system (e.g. open angle and closed angle glaucoma, including normal pressure glaucoma, especially PEX glaucoma, additional block of flow due to qualitative disturbances of the aqueous humour, as well as AION without arteritis, myopia, idiopathic scleritis and episcleritis) and for health and life of the patient (e.g. strokes, cerebral aneurysms and arteriovenous malformations).

The relationship between the symptoms and the severity level of the pathogen in migraine is similar as in case of the lumbar spine degenerative disc disease, where depending on the degree of compression of the intervertebral disc on the nervous structures conditioned by reciprocal spatial relationships, the severity scale of pathological symptoms is dependent on the degree of such a compression and the quantity of its occurrence. In case of foot drop we may be faced with a small disability of the muscle strength or completely impaired dorsiflexion of the foot because of the total palsy of the common fibular nerve.

It appears from my research and observation as well as from the mechanism of migraine developed by me that this is a disease whose essence are various kinds of disorders in the organism that stem from an increase in the intraocular pressure in the posterior chamber and vitreous humour of the eye. By pressing the eye vessels this pressure brings the increase in blood pressure in the cerebral arteries (primarily in the ophthalmic artery and further in the intracranial artery), which in turn leads to their ballooning (widening) and increasing the intracranial pressure. Neurovascular conflicts induced in this way in turn lead to different kinds of systemic complications whose quality and intensity depends on the area and the degree of compression. Therefore, I have defined migraine using an English acronym: **MIGRAINE** - **M**ulti-area Indisposition Generated Rapidly Against IOP (intraocular pressure) increase to Neurological Emergency.

Typical symptoms of migraine are:

- migraine aura (e.g. visual; i.e. visual disturbances, different shapes of flashes and floaters),
- a strong headache (often pulsating, beginning in the eye, around the orbits or in the temples, often one-sided),
- an increase in blood pressure,
- irregular heartbeat (heart palpitations, arrhythmia, syncope).
- gastrointestinal system disorders (pain and compression in the epigastrium, nausea, vomiting, diarrhea),
- a feeling of obstacles in the throat (including "globus hystericus"),
- impaired swallowing and speech (dysphagia and dysarthria),
- photophobia and phonophobia (intolerance to visual perception of light and hypersensitivity to sounds),
- disorders of skeletal striated muscles supplied by cranial nerves: sternocleidomastoid muscle, trapezius muscle, splenius cervicis muscle (various degrees of torticollis and upper limb disability in lifting by more than 90 degrees, neck stiffness),
- impaired cerebrospinal fluid flow (neck stiffness, loss of consciousness),

I have determined that, in many cases, differently intense trigeminal neuralgia ("earaches") and disorders of cranial nerves such as facial, glossopharyngeal, vagus and hypoglossal nerves (impaired swallowing, taste and speech), facial and vagus nerves (stomach ache, nausea, vomiting, arrhythmia), facial and accessory nerve (neck stiffness, mobility disorders of neck and upper limb as well as mimetic muscles disorders) in certain cases manifest the mechanism of migraine. Therefore, they are caused by increased intraocular pressure with the subsequent increased arterial pressure in the internal carotid artery and then the increased intracranial pressure, which in turn leads to neurovascular conflicts (internal carotid artery and cavernous sinus on the Gasser's ganglion - in trigeminal neuralgia, and posterior meningeal artery with the internal cartoid vein (as venous outflow from the cavernous sinus via the sigmoid sinus) on nerves IX, X and XI in the jugular foramen, or the internal cartoid artery and vein on nerves IX, X, XI and XII at the outlet from the skull. Nota bene, it has been supposed so far that bilateral damage to nerve IX carries the risk of hypertensive crisis, however, in the face of my discoveries, it appears that this increase in blood pressure in the cerebral vessels, secondary to an increase in the intraocular pressure in migraine, carries the risk of compression on this nerve, which gives the symptoms of its damage. In trigeminal neuralgia the dominant symptom is a pain that usually starts around the eye and orbit, and then locates around the ear and radiates in accordance with tactile endings of nerve V towards the mandible as well as the zygomatic and frontal area. Most frequently it is one-sided, which in turn is further proof of its migraine origin; half of the head aches in a manner that is actually typical for migraine. However, in my patients I often find eye ache on one side while the headache "migrates" to the other one. Facilitating the aqueous humour outflow from the back to the front in the right eye and then to the veins using cycloplegics and massage of the right eyeball abolishes pain and abducent nerve palsy on the left!!! This is my discovery of the mechanism of this nerve palsy in the cavernous sinus heterolateral in relation to the eve that initiates migraine!!! The proof of the compression on cranial nerves in the jugular foramen are the typical symptoms of acute migraine, among of which impaired swallowing,

speech and taste are exactly the same symptoms as in the Collet-Sicard syndrome, which is a function impairment of nerves IX, X, XI and XII due to venous thrombosis within dural venous sinuses or head injury with a fracture passing through the jugular foramen. Less noticeable symptom may be a feeling of obstacle in the throat. Associating it with the knowledge regarding the mechanism of migraine described by me, in which activation of the sympathetic nervous system plays a significant role in raising the pressure in the arteries and venous system of the brain, I discovered the etiopathogenesis of "globus hystericus" occurring in somatic neurosis, which manifestly seems to correspond to differently intensive conflicts between arteries and veins and cranial nerves V, VII, IX, X, XI and XII, which all provide sensation and motion to the throat and tongue. The proof of the migraine nature of the neurovascular conflict in the superior orbital fissure is the ophthalmoplegic migraine with symptoms typical of Rochon-Duvigneaud's syndrome (superior orbital fissure syndrome), in which an orbit injury with a fracture passing through this fissure leads to symptoms similar to those in the ophthalmoplegic migraine.

While observing symptoms in patients treated by me, I found that the selected cases of tension headaches are less intensive migraine-mediated aches within the meaning of the mechanism described by me. Similarly, in a less intensive migraine pathology patients show less severe symptoms of the above mentioned cranial nerve neuralgias which are manifested by pains around the ear, or impaired swallowing, speech and sensation in the throat.

Therefore, we can reach the conclusion that, in certain situations related to a weaker expression of migraine, its symptoms will be less expressed, but their occurrence will prove migraine to be a complex and individual pathological mechanism beginning always with the compression of the increased intraocular pressure in the posterior chamber and vitreous humour of the eye on its vessels. In these situations we need to look for symptoms of this serious disease, because their minor severity does not constitute a greater safety for the eye or the circulatory system and the brain. It will be so in the situations when a patient ophthalmically predisposed to migraine, for example because of anatomical conditions of the skull which adverse migraine. Increased pressure in cerebral arteries will easier lead to acute intracranial pressure and its symptoms in people with small braincases, and so small anatomical isthmi, which contributes to hemodynamic disturbances in the brain and nervovascular conflicts.

Therefore, we conclude that a "perfect", that is fully expressed, migraine will be developed in a perfect "migrainer", i.e. a human who meets the following criteria:

- ophthalmic predisposition (reciprocal iris-lens relationship that fosters the increase in the relative pupillary block or total pupillary block, e.g. in posterior iris synechiae and total block of aqueous humour outflow from the eye, e.g. in acute angle closure),
- low blood pressure,
- a personality type determining tension in the vegetative system (a sensitive person who is easily excitable and influenced by peer pressure or stress and who restricts emotions),

• a small braincase or small size of a selected anatomical isthmus in the head (e.g. tight superior orbital fissure and/or cavernous sinus - e.g. ophthalmoplegic migraine and/or trigeminal neuralgia),

When we also add low cerebrospinal fluid pressure to the characteristics of a "perfect migrainer", the model fulfilling "super ideal" conditions will be a child, in which both a small skull and standard for this age arterial pressure of 90/60 mmHg and CSF at 70mm of H2O as well as biometrics of anterior segment of the eye, which is disadvantageous in terms of pupillary block, are standard. So a child may develop migraine so easily in predisposing situations that the symptoms may occur suddenly and because of syncope or loss of consciousness may be initially diagnosed as meningitis, which typically is not confirmed in further diagnostics, or idiopathic epilepsy, which has been diagnosed so far in the absence of a known cause. Associating non-standard migraine in children with an increased relative pupillary block, I described the "EYE – CHILD" Nogal Syndrome (EYE complicating Cardiac Head/ache Indigestion Languish Disorders), which will be discussed in detail during my speech at the 4th Forum of Ophthalmic Surgery in Katowice, organized by Ophthalmology Clinic of the Medical University in Katowice.

However, as for the "less than perfect" migrainer, that is the one in whom the mechanism of the disease works with no evident acute or subacute symptoms of migraine so far, we must be aware that it is actually these patients who may comprise inexplicable so far cases of vascular incidents remaining beyond the statistics of risk associated with migraine, since it has not been diagnosed in them. In the research based on a new definition of migraine it may turn out that it is actually those patients without diagnosed migraine symptoms and without severe headaches, or with headaches until now recognized as "sinusitis", "earaches" and other various headaches such as "tension", "cluster", "stress" headaches, Horton's syndrome, "idiopathic stabbing" headaches and the like..., comprise a gap in migraine statistics of victims of ischaemic strokes, aneurysms, vascular malformations and cerebral hemorrhages as well as unexplained arrhythmias and heart attacks. As described by me, like a "perfect migrainer", these people also develop the most important pathological factor for the above disorders, which is the significantly increased arterial and venous pressure in the vascular bed in brain and subsequent angiological and neurological pathologies with no obvious symptom of severe headache actually associated with migraine, that is, without "migraine" emerging in its previous meaning.

Summing up my existing works on migraine I have to highlight that knowing the dangerous nature of this disease, we must remember that "headache" may not occur in migraine or may be minor, both in a child as well as in an adult. Most often, however, due to an increase in intraocular pressure, we are faced with pain around the eyes associated with "eye fatigue" or "sinusitis", conditioned by tactile innervation of the choroid, iris and ciliary body by short ciliary nerves of the ciliary ganglion and long ciliary nerves also connecting to the ophthalmic nerve of the trigeminal nerve through a nasociliary nerve, giving eyeache radiation to the temple and ear, and often represents the first feelings of a patient in trigeminal neuralgia attack.

Children, however, among other things due to greater flexibility of eyeball walls and lower critical intraocular pressure, more frequently develop other clear symptoms of migraine (e.g. faining or stomach ache) without tangible so far organic reasons or in the presence of symptoms which have not been associated with migraine so far, and which, in the face of my observations, treatment and analyses and existing migraine statistics, are clearly associated with migraine (e.g. idiopathic episcleritis and scleritis or striking hand in the head).

In older children, teenagers and adults these may only be single signs of function disability of an organ in which the mechanism of migraine starts, i.e. the eye (e.g. myopia, open angle and closed angle glaucoma or loss of optic nerve in AION without arteritis!!!) and subtle neurovascular conflicts in the skull as well as outside it (e.g. cavernous sinus and jugular foramen as well as neurovascular bundle in the neck; "globus hystericus", a feeling of obstacle in the throat, arrhythmia, nausea, dizziness, hearing your own pulse "deep in your ear", etc.).

In such a situation do we, doctors and patients, have to think that the migraine problem has affected us? All of the symptoms that I mention below should be first subject to a detailed specialist diagnosis. Only when tangible organic reasons which may cause them have been ruled out, should we think about migraine, which is still much more difficult to diagnose than meningioma, cerebral haematoma, or sinusitis:

- headache, dizziness, loss of consciousness, pulsation in the ear, neck stiffness (neurologist and radiologist diagnostics for brain tumor, stroke, aneurysm, meningitis, etc.),
- pain around the sinuses, ear, nose, dysphonia, obstacle in the throat, impaired swallowing and speech, "globus hystericus" (laryngologist, neurologist diagnostics for sinusitis and sinus, larynx, throat tumor, nerve damage, etc.)
- eyeache, flashes, floaters, deterioration of vision, visual migraine aura (ophthalmologist diagnostics for glaucoma, inflammation or splitting retina, embolism, eye and orbit cancer, loss of optic nerve, AMD, vision defects, etc.),
- stomach ache, nausea, vomiting, (gastrointestinal specialist, gynecologist diagnostics for peptic ulcer, food poisoning, gastroesophageal reflux, inflammation, pregnancy, cancer of gastrointestinal and reproductive system, inflammation, etc.),
- arrhythmias and heart palpitations, syncope, hypertension (cardiologist -diagnostics for organically conditioned hemodynamic disorders, arrhythmia, coronary heart disease, circulatory collapse, etc.),
- upper limb striated muscles disorders, neck stiffness (orthopedic surgeon/traumatologist and neurologist diagnostics for damage to the motor apparatus),
- hormonal disorders conditioned by pituitary gland dysfunction, e.g. female infertility in hyperprolactinaemia, both sexes FSH and LH disorders, or thyroid hormone

disorders (gynecologist, endocrinologist, radiologist - diagnostics for the organic causes of disorders).

If organic causes are ruled out in the patient complaining about the symptoms listed above, we always have to think about migraine, whose mechanism explains the aetiology of multiple disorders that has been treated so far as idiopathic. We have to take migraine into account, even when, contrary to still applicable criteria, we do not confirm a headache or it is weak enough to be able to be alleviated using analgesics. We must not be content with guessing that "this must be the sinuses". For a quick assessment of whether or not the problem of pain in the sinuses is associated with a pathological condition in their area, it is sufficient to start with an X-Ray AP of paranasal sinuses and filling the roentgenogram with air that in physiological conditions fills the sinuses, makes us look for other causes of pain in the patient. This may save the patient's eyesight, in respect to avoiding optic nerve loss in glaucoma and AION, and even life, in the case of developing an aneurysm or cerebral hemorrhage from malformations which are vascular complications of the migraine mechanism.

In subsequent publications I will present argumentation on the association of the above disorders with the mechanism of migraine described by me.