

Benign Intracranial Hypertension and its Causes

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Idiopathic intracranial hypertension, also known as benign intracranial hypertension, may be a condition characterized by increased intracranial pressure without a detectable cause. The most symptoms are migraine, vision issues, ringing within the ears with the heartbeat, and shoulder pain [1]. Complications may incorporate vision loss. Risk factors incorporate being overweight or a recent increment in weight. Tetracycline may also trigger the condition.

The diagnosis is based on symptoms and a high intracranial pressure found amid a lumbar puncture with no particular cause found on a brain scan. The common symptom of IIH is migraine, which happens in almost all cases. It is usually worse within the morning, generalized in character and throbbing in nature. It may be related with nausea and vomiting. The migraine can be made worse by any movement that further increases the intracranial pressure, such as coughing and sneezing.

The pain may also be experienced within the neck and shoulders [2]. Many have pulsatile tinnitus, a whooshing sensation in one or both ears, this sound is synchronous with the beat [3]. Different other symptoms, such as numbness of the extremities, generalized weakness, loss of smell, and loss of coordination.

Intracranial hypertension may be a build-up of pressure around the brain. It can happen all of a sudden, as a result of a severe head damage, stroke or brain abscess. Usually known as acute Intracranial hypertension. Benign intracranial hypertension is also characterised by typical cerebrospinal fluid substance. The increased pressure leads to compression and traction of the cranial nerves, a bunch of nerves that arise from the brain stem and supply the face and neck.

This increased pressure cause papilledema, which is swelling of the optic disc, the spot where the optic nerve enters the eyeball. The nerve supplies the muscle that pulls out the eye. Those with 6th nerve paralysis in this manner encounter flat double vision which is worse when looking towards the

affected side. More rarely, the oculomotor nerve and trochlear nerves are influenced; both play a part in eye movements.

The facial nerve is affected sometimes the result is total or partial weakness of the muscles of facial expression on one or both sides of the face. There are various other infections, generally rare conditions, which will lead to intracranial hypertension. In case there's an fundamental cause, the condition is named secondary intracranial hypertension. Common causes of secondary intracranial hypertension incorporate obstructive sleep apnea, systemic lupus erythematosus, chronic kidney illness, and Behçet's disease.

Other neurological abnormalities reported have included facial paresis, neck torment, seizures, hyperreflexia, bruit, hypoglossal nerve paralysis, nystagmus, and choreiform movements. Benign intracranial hypertension could be a headache syndrome characterised by raised cerebrospinal fluid pressure within the absence of an intracranial mass lesion or ventricular dilatation; typical spinal fluid composition.

References

1. Wall M. "Update on Idiopathic Intracranial Hypertension". *Neurologic Clinics*. 35 (2017): 45–57.
2. Binder DK, Horton JC, Lawton MT, McDermott MW. "Idiopathic intracranial hypertension". *Neurosurgery*. 54 (2004): 538–51.
3. Sismanis A. "Pulsatile tinnitus. A 15-year experience". *American Journal of Otolaryngology*. 19 (1998): 472–7.

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