VISUAL LOSS IN IDIOPATHIC INTRACRANIAL HYPERTENSION DESPITE NORMAL INTRACRANIAL PRESSURE

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Headache, loss of visual acuity and visual field defects are the hallmarks of idiopathic intracranial hypertension (IIH). Raised intracranial pressure (ICP) is thought to cause loss of vision by affecting the axoplasmic flow in the optic nerve (ON). Headache usually subsides after treatment with acetazolamide or cerebrospinal fluid (CSF) shunting procedures. Because CSF is thought to communicate evenly through all CSF spaces, papilledema and visual loss might be expected to improve once ICP is normalized. This is, however, not always the case; indeed, some patients with IIH have persistent papilledema and experience progressive visual loss despite an ICP that has been lowered to normal values (1). Studies assessing lipocalin-like prostaglandin D synthase (L-PGDS) concentration gradients between the intracranial CSF and the CSF in the subarachnoid space surrounding the ON, as well as contrast-aided cisternography demonstrate that the ON in patients with IIH is actually a separate and distinct CSF compartment with the CSF having a different biochemical composition compared with the intracranial CSF (2,3). The concept of a distinct optic nerve sheath compartment provides a pathophysiologic explanation for progressive visual loss despite normal intracranial pressure. 1. Kelman SE et al. Modified optic nerve decompression in patients with functioning lumboperitoneal shunts and progressive visual loss. Ophthalmology 1991;98(9):1449-53. 2. Killer HE et al. Cerebrospinal fluid dynamics between the intracranial and thesubarachnoid space of the optic nerve. Is it always bidirectional? Brain 2007; 129(4): 1027-1030. 3. Killer HE et al. The optic nerve: a new window into cerebrospinal fluidcomposition. Brain 2006; 129: 1027-1030.