OCULAR MANIFESTATIONS OF INTRACRANIAL SPACE OCCUPying LESIONS (ICSOL): A REVIEW ARTICLE

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ABSTRACT

The ophthalmologist is often the first to detect and place his finger on an intracranial space occupying lesion (ICSOL). The ophthalmologists’ efforts would aid the neurologist due to the extensive distance the optic pathways cover in the brain, and from the fact that 6 of the 12 cranial nerves with their nuclei are associated with the eyes beside vagus and sympathetics. The triad of ICSOL include headache, vomiting and papilloedema. As ophthalmologists, we have to consider the type of headache pupillary reflexes, and the optical system in a certain order, viz. the palpebral fissure, the visual functions, pupillary reflexes, the fundi oculi, the visual fields defects, and endocrine disturbances. X Ray/CT/MRI examinations and angiography are diagnostic modalities.

KEYWORDS

Intracranial Space Occupying Lesions, Ocular, Papilledema, Visual Field Defects, Foster-Kennedy Syndrome.

INTRODUCTION

The association between neurology and ophthalmology is needed never so closely as in ICSOL and often the ophthalmologist is the first to detect and place his finger on the lesion. The ophthalmologists’ efforts would aid the neurologist due to the extensive distance the optic pathways cover in the brain from pole to pole, and from the fact that 6 of the 12 cranial nerves with their nuclei are associated with the eyes beside vagus and sympathetics.[1] Ophthalmologists’ services will be required under the following conditions: headache, visual disturbances, changes in the ocular fundus, and affections of the motor nerves of the eyes, displacement and involvement of the eyeball. The triad of ICSOL include headache, vomiting and papilloedema. Very often it is the type of headache alone that focuses our attention to such a lesion. At times we find papilloedema alone or a visual field-defect as the one and only sign. In other cases we come across signs and symptoms where not one of the triads is present and yet the patient has a fairly large space occupying lesion.

PATHOLOGY OF INTRACRANIAL TUMOURS

Impairment of blood-supply due to a ‘squeezing’ action of a space-occupying lesion causes functional loss due to circulatory failure. Kernohan and Sayre have reassessed the pathology of tumours of the central nervous system. Microscopically, besides the tumour, the phenomenon of oedema is a prominent feature, involving all strata of the cerebrum, with loosening of the ground substance, separation of the myelin sheaths, distension of perivascular spaces and swelling and transformation of astrocytes.

As regards the nomenclature of the various types of cerebral tumours with their gradation, they are named according to the types of neural cells predominantly seen in microscopical sections of the same, like meningioma, medulloblastoma, astrocytoma, oligodendroglioma etc. and combinations of the same.

LOCALISATION

The matter of localising a lesion requires systematic examination. As ophthalmologists, we have to consider the type of headache pupillary reflexes, and the optical system in a certain order, viz. the palpebral fissure, the visual functions, pupillary reflexes, the fundi oculi, muscle paresis, cerebral functions and endocrine disturbances. X Ray/CT/MRI examinations and angiography are diagnostic modalities.

CLINICAL APPROACH

Headache and pain referred to the back of the eyes are symptoms for which the ophthalmologist is commonly visited. These symptoms are not due to increased intracranial pressure, but are due to traction and displacement of venous sinuses, middle meningeal artery, and large arteries at the base of the brain, pressure on the sensory cranial nerves V, IX, X and C1, C2, C3. Papillary oedema pressure and distension changes when ICSOL extends into orbit. Prominent eyes (directly, or indirectly through endocrines) are seen in pituitary tumours, sphenoidal ridge tumours, tumours of optic nerve and chiasm, cavernous sinus infection, hydrocephalus, and cerebral aneurysms.

Peripheral visual fields are affected in pituitary, temporal and occipital lobe tumours. Every case of a space occupying lesion is not accompanied by oedema of the nerve head. On the other hand we may find papilloedema but no increased intracranial tension. When the optic nerve and chiasma are pressed upon by a tumour there is optic atrophy of the primary type, but then we also come across cases of cerebral tumours with normal looking optic discs. Papilloedema is due to axoplasmic stasis in the optic nerve head and secondary vascular changes at disc surface due to increased intracranial pressure.[2]
Incidence of papilloedema as given by several authors is as follows: Hartman and Guillaumat (1938) on a study of 1169 cases, found papilloedema from gliomas in 76%, meningiomas compressing brain 40%, tumours of the posterior fossa 71%. Petroheles and Henderson (1950) reported 59.5% papilloedema out of all cases of cerebral tumours. [3]

FOSTER KENNEDY SYNDROME

Papilloedema on one side with optic atrophy on the other denotes a lesion at the chiasma on the side of the optic atrophy. A frontal or olfactory groove tumor would by its pressure cut off the subarachnoid space round the ipsilateral optic nerve distal to the tumour from the cerebral subarachnoid space and that around the opposite optic nerve and so prevent the effects of increased fluid tension on the affected side whereas pressure atrophy of the nerve will not be prevented.[4,5]

Pseudo-Foster Kennedy Syndrome is described as unilateral optic disc swelling with contralateral optic atrophy in the absence of an intracranial mass causing compression of the optic nerve. This occurs typically due to bilateral sequential optic neuritis or ischaemic optic neuropathy. The third and the sixth nerves are often involved in all kinds of space occupying lesions where the base of the brain is stretched along with the vessels there, so that the third nerve gets compressed between the posterior cerebral and superior cerebellar arteries and the sixth nerve between the internal auditory or the anterior inferior cerebellar artery and the pons.

- Papilloedema, optic atrophy
- Slight bulging of eyeball (ipsilateral)
- If extension into chiasm-Foster-Kennedy syndrome
- Bilateral papilloedema
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- Bilateral papilloedema
- Optic nerve ischemia, then atrophy
- Bitemporal hemianopia
- Colour fields defective
- Pressure on adjacent structures viz 3rd ventricle causing papilloedema; diplopia; progressive vision loss due to pressure on optic tract
- Oculomotor palsies of peripheral type (III, IV, V cranial nerves)
- Homonymous hemianopia
- Papilloedema
- Paralysed retina (congruous)
- Congruous field defects
- Coarse horizontal nystagmus
- Bilateral papilloedema
- Diplopia due to VI nerve palsy (indirect type)
- Paralysis of V, VI, VII cranial nerves, most commonly being V nerve- loss of corneal sensation.
REFERENCES
5. Weinbeyer L. M. Grant F. C. and Adler F.H. (1940) Arch. of ophth, 24, 1197.

Plain CT image of left front parietal region reveals well defined hypodense lesion in left frontal lobe. No calcification or cystic areas within lesion.

**Fig. 1:** Frontal Lobe Gliomas-Computerised Tomography Scan and Fundus Findings

Foster-Kennedy syndrome with optic atrophy on ipsilateral side and papilledema on contralateral side.

**Fig. 2**