Optic hydrops: isolated nerve sheath dilation demonstrated by CT.

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*AJNR Am J Neuroradiol* 1987, 8 (5) 867-870
http://www.ajnr.org/content/8/5/867

This information is current as of October 5, 2023.
Optic Hydrops: Isolated Nerve Sheath Dilation Demonstrated by CT

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This report illustrates the potential of the optic subarachnoid space to dilate selectively with various disease processes involving the orbit, optic canal, sella, and parasellar regions. According to the concept of optic hydrops, isolated papilledema, in the absence of increased intracranial pressure, may be caused by focal disease of these regions.

Certain diseases are known clinically to produce papilledema unilaterally or bilaterally, but still to cause no discernible elevation of intracranial pressure [1–4]. This report elucidates some of the varying acquired states that may lead to this clinical picture and reviews the CT correlates in such cases.

Subjects and Methods

Eleven patients were scanned with a GE-9800 CT apparatus before and after IV contrast administration, traversing the orbit, sella, and suprasellar cistern with 3- and/or 5-mm axial sections (Table 1). None of the subjects in the present study had associated increased intracranial pressure clinically, and therefore this factor did not contribute to the radiologic findings.

Results

Unilateral papilledema was found to be caused by lesions that obstructed the flow of CSF along the optic subarachnoid space. This was seen both in benign-acquired conditions, such as trauma (Fig. 1) and inflammation (Fig. 2), and in neoplasia (Figs. 3 and 4). Certainly, in some cases such as orbital inflammation and sheath tumors, the optic sheath itself is also thickened. However, in each case in this series, a clearly enlarged margin of hypodensity was seen between the sheath and the nerve indicating the dilated state of the peri optic subarachnoid space.

Bilateral optic hydrops was observed in lesions that block normal orbital CSF pathways at the level of the entry of the optic canal into the suprasellar cistern. Tumors, cysts, or arachnoid adhesions in the sellar region might be expected to cause such an obstructive phenomenon (Figs. 5 and 6).

In addition to this dilation of the optic nerve sheath distal to the obstruction, protrusion and edematous thickening of the optic papilla into the posterior aspect of the globe was noted in five patients, thus confirming the active pressure gradient present (Fig. 2B). This swelling of the optic disk is a well-known feature of papilledema.

An associated observation was the absence of hydrops in very benign, slowly progressive conditions such as obstruction of optic CSF flow secondary to osteopetrosis with attendant optic canal constriction [4]. Therefore, the finding of optic hydrops seems to be largely limited to either malignant or aggressive-benign lesions affecting the orbit and parasellar area. To date, none of the patients in this series...
was returned for detailed reevaluation after therapy to assess the possible radiologic resolution of the observed selectively dilated optic nerve sheaths.

TABLE 1: Patients with Optic Sheath Hydrops

<table>
<thead>
<tr>
<th>Type of Finding</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pituitary adenoma</td>
<td>2</td>
</tr>
<tr>
<td>Orbital inflammation</td>
<td>2</td>
</tr>
<tr>
<td>Sellar/suprasellar cyst</td>
<td>1</td>
</tr>
<tr>
<td>Orbital trauma</td>
<td>1</td>
</tr>
<tr>
<td>Optic sheath meningioma</td>
<td>1</td>
</tr>
<tr>
<td>Optic nerve glioma</td>
<td>1</td>
</tr>
<tr>
<td>Orbital retinoblastoma</td>
<td>1</td>
</tr>
<tr>
<td>Orbital nasopharyngeal carcinoma extension</td>
<td>1</td>
</tr>
<tr>
<td>Orbital breast metastasis</td>
<td>1</td>
</tr>
</tbody>
</table>

Discussion

Papilledema is traditionally associated with a number of disease processes of the cranium and orbit. However, the primary condition resulting in this clinical finding in the absence of intracranial hypertension or intrinsic disease of the optic disk/nerve has not been clearly defined. The morphologic findings on the CT examinations in this small group of patients suggest that the basic mechanism causing the papilledema, with or without elevated intracranial pressure, may be the same: that increased pressure within the distal optic sheath subarachnoid space is transmitted to the optic papilla, causing it to bulge and finally to result in true disk edema [6–11].

Why this appears to be seen primarily in aggressive lesions is unknown. Perhaps there is active transudation or exudation of substances in these cases, which in the absence of free CSF flow, causes optic hydrops. This study further points out
Fig. 4.—16-year-old boy with left-sided optic sheath meningioma and ipsilateral papilledema.

A, Postcontrast axial CT scan showing orbital apex tumor with optic sheath hydrops distally (arrow). Dilated distal optic sheath was not histologically involved with tumor and collapsed on incision at surgery.

B, Magnified coronal section through retrobulbar region illustrating distended optic nerve sheath encircling centrally located optic nerve (arrow).

Fig. 5.—25-year-old man with intra/suprasellar arachnoid cyst and bilateral papilledema.

A, Axial CT scan at level of sella turcica illustrating arachnoid cyst filling and expanding bony confines of sella.

B, Magnified section through orbits showing bilateral optic hydrops.

Fig. 6.—29-year-old man with nonfunctioning pituitary adenoma and bilateral papilledema.

A, Postcontrast axial CT scan showing irregularly enhancing intrasellar and left parasellar adenoma.

B, Magnified view of orbits illustrating the symmetrically dilated optic nerve sheaths on both sides, indicating bilateral optic hydrops.
the need for detailed evaluation of the orbit, optic canal, and intra/parasellar regions in patients presenting with unilateral or bilateral papilledema without obvious evidence of intracranial abnormality or cranial hypertension. Optic CSF “block” therefore becomes a significant conceptual factor in the diagnosis and progression of disease of the orbit and anterior optic pathways as well as in the attendant clinical implications.

ACKNOWLEDGMENTS

I thank Mrs. C. Galban for manuscript preparation, Mrs. C. Jinkins for manuscript research, and the Radiology Department of the King Faisal Specialist Hospital for assistance with the illustrations.

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4. Jinkins JR. The optic neurogram: evaluation of CSF “block” caused by compressive lesions at the optic canal. AJNR 1987;8:135–139


