Bilateral cavernous sinus metastasis and ophthalmoplegia

Case report

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A 59-year-old man developed bilateral total ophthalmoplegia with trigeminal hypesthesia immediately after the placement of burr holes for drainage of a subdural hematoma. Metastatic lung carcinoma to both cavernous sinuses was suspected, eventually proven, and confirmed postmortem.

KEY WORDS - cavernous sinus - ophthalmoplegia - trigeminal nerve - metastatic tumor - oculomotor nerve - brain tumor

The sudden disappearance of all eye movement in a patient following neurosurgical intervention prompted repeated diagnostic investigation which remained fruitless for 2 months. Just before the patient died, metastasis of lung cancer to both cavernous sinuses was discovered and later confirmed at postmortem examination.

Case Report

This 59-year-old man underwent coronary artery bypass grafting 3 months before admission. A right carotid arteriogram (Fig. 1) was obtained during coronary arteriography. A small coin-shaped lesion was found in the left upper lung field. On admission for bronchoscopy, which confirmed undifferentiated lung carcinoma, the patient complained of mild generalized headache, most noticeable behind his right eye, with some nausea. Neurological evaluation was normal except for equivocal left ptosis. Radioisotope brain scan was normal, but a computerized tomography (CT) scan (Fig. 2A) showed a small left subdural accumulation with the density of cerebrospinal fluid (CSF).

Burr holes were placed. The dura was not tense, and the subdural fluid was light pink in color. Dural biopsy and cytological evaluation of the subdural fluid were negative for tumor cells. Drainage tubes were left in place. On awakening, the patient exhibited total paralysis of voluntary and involuntary horizontal gaze, which within 36 hours progressed to total internal and external ophthalmoplegia. Examination revealed absence of eye movement in response to any stimulus, fixed 4-mm pupils, total bilateral ptosis,...
absent corneal reflexes in both eyes, and mild hype-
thesia of the upper portion of the face. A striking los-
of lacrimation, together with corneal anesthesia, was
thought to have caused moderate conjunctival che-
mosis and punctate keratitis in both eyes. Proptosis,
congestion of orbit or retina, and bruit were absent.
All other cranial nerves were intact, and the remainder
of the neurological examination was normal.

Repeat CT scan (Fig. 2B) was unchanged from
preoperatively. Intracranial pressure was monitored
for 24 hours and was normal. The CSF analysis
including cytocentrifugation was normal. Repeat CT
scanning during a course of chemotherapy (Fig. 2C)
was again unchanged, but a repeat arteriogram was
declined. An exhaustive search for a toxic or metabolic
etiology was unrevealing. The patient remained clin-
ically unchanged for 2 months, then, during the last
2 weeks of life, dysphagia and poor gag reflex devel-
oped, followed by rapid deterioration. At that time, a
CT scan (Fig. 2D) demonstrated an enhancing sellar
and parasellar density.

Postmortem examination showed a left upper lobe
bronchogenic carcinoma with metastasis to hilar
nodes, periaortic nodes, liver, left adrenal, thyroid,
and pituitary glands, sella turcica, and both cavernous
sinuses. Representative sections of the tumor demon-
strated a poorly differentiated carcinoma (non-small-
cell type) composed of sheets of lobules of irregular
hyperchromatic cells. In addition, sections through
both cavernous sinuses (Fig. 3) showed complete ef-
facement of the normal architecture by the neoplasm.
Nerve fascicles, although preserved, were noted to be
atrophic as they passed through the neoplasm. Tumor
cells invaded along the endoneurium and into the
nerve substance in structures, which by their relative
size and position, probably represented the third,
sixth, and sensory fifth cranial nerves. The carotid
arteries were encased by tumor, but invasion was
limited to the outer adventitia and along tiny perfo-
rating vessels into the muscularis. Significant intimal
hyperplasia and calcific plaque were present as stig-
mata of intracavernous atherosclerosis.

Discussion

Although causing initial concern, the appearance of
total ophthalmoplegia just after neurosurgical inter-
vention was thought to be coincidental. However,
extensive investigation failed to confirm an etiology
till just before death, at which time a CT scan
revealed sellar enhancement. Subsequent postmortem
examination confirmed the presence of a metastatic
carcinoma. The causal relationship, if any, between
the sudden onset of ophthalmoplegia and the burr-

hole evacuation of the subdural collection is conjec-
tural. The close temporal relationship suggests that
either some change in intracranial pressure dynamics
and/or toxicity of the anesthetic agents (pentobarbital,
nitrous oxide, and fentanyl) might have precipitated
the ophthalmoplegia.

In contrast to the more common cavernous sinus
meningiomas and aneurysms, which predictably show
abnormalities on plain skull films or CT scans,9 di-
agnosis of intracavernous metastasis is usually diffi-
cult. The only abnormality suggesting the metastasis
is frequently a subtle distortion of the intracavernous
carotid artery,3,8,11 although the artery itself is remark-
ably resistant to tumor invasion.10 In retrospect, the
right carotid arteriogram of our patient performed 3
months before onset of ophthalmoplegia shows mod-
erate intracavernous irregularity. Because of known
severe arteriosclerotic disease, the irregularity was
dismissed as unimportant. Postmortem examination
documented significant intracavernous atherosclerosis

R. P. Mills, S. J. Insalaco and A. Joseph
Bilateral cavernous sinus metastasis


as well as periarterial tumor cuffing without significant arterial invasion. In this case, the arteriographic findings could have been caused by either or both of the anatomic findings.

Symptoms of metastasis to the cavernous sinus often include continuous painful dysesthesia in a trigeminal distribution, but our patient never had this complaint, in spite of partial preservation of trigeminal function. Infiltration and encasement of the trigeminal nerve fascicles in this case was of equal or greater degree than in reported cases of neurotropic involvement which produced painful dysesthesia. The absence of painful dysesthesia, and of orbital venous congestion, in the presence of massive intrinsic cavernous sinus involvement, bears testimony to the variability of clinical signs of the “cavernous sinus syndrome.”

Lung carcinoma metastatic to both cavernous sinuses has not been previously reported, although cranial nerve infiltration with consequent bilateral ophthalmoplegia was documented by Craig, et al. 4

Acute bilateral total ophthalmoplegia is rare from any cause, but especially from neoplasia. When trigeminal nerve function is spared or involved only late in the course, extrinsic compression of the cavernous sinuses is the likely mechanism. Walsh 5 reported two cases of bilateral ophthalmoplegia developing within 48 hours, which were thought to be caused by acute hemorrhage into pituitary adenomas. His patients failed to show loss of corneal reflexes. Hermans, et al. 6 described a case of bilateral ophthalmoplegia with partial pupillary sparing, developing over 1 month, caused by primitive carcinoma located in the midline at the skull base but largely sparing the cavernous sinuses. Trigeminal involvement appeared considerably later. Extension of nasopharyngeal carcinoma was the cause of bilateral third, fourth, and sixth cranial nerve palsies evolving over 6 weeks, with sparing of trigeminal function, in a case reported by Jayle and Ourgaud. Unfortunately, no pathological proof of extrinsic cavernous sinus compression was available.
Intrinsic bilateral cavernous sinus involvement with tumor usually produces early loss of trigeminal sensation. Most such cases in the literature have shown orbital or optic nerve involvement as well, and the development of clinical signs has been gradual over weeks to months. Only a case described by Bito, et al., approached the rapidity of onset of ophthalmoplegia shown by our patient. In that case, the ophthalmoplegia developed over 1 week, and was associated with absent corneal reflexes and forehead hypesthesia. Despite aggressive investigation, the bilateral intracavernous metastasis of liposarcoma was not discovered antemortem.

References

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