Prechiasmal Infarction Associated with Intrachiasmal and Suprasellar Tumors*

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There is a little heralded lesion of the chiasmal region that occurs occasionally and has not, as yet, received sufficient attention, namely, the blue, dome-shaped, or "blueberry" infarct of the prechiasmal or anterolateral portion of the chiasm. It is apparently due to simultaneous impairment of the superior and inferior vascular supply to the optic nerve and chiasm. We are reporting two cases of this lesion: an optic nerve glioma, and an infarction of a suprasellar pituitary tumor. Both of these patients underwent a craniotomy with surgical incision of the lesion in the prechiasmal area, and both required intrachiasmal aspiration with a tiny sucker to remove the clotted blood and necrotic tissue. There was partial improvement of the vision in one patient and none in the other. The two cases are presented to emphasize the importance of early diagnosis and treatment and to propose an anatomical concept of how these lesions may occur secondary to impairment of the vascular supply to the optic nerve and chiasm.

Case Reports

Case 1. This child was first seen in 1946 when she was 1 year old and had a cord-like mass in the left cheek and the supraorbital notch, causing limitation of facial movement and partial occlusion of the nares. She had left-sided proptosis, a doughy resistance on palpation of the left globe, and limitation of motion of the left superior rectus muscle. A diagnosis of lymphangioma or neurofibroma was made. The skull x-ray films revealed an enlargement of the left orbit without distortion of the optic foramina or any other bony abnormality. A few "bursts" of radiation therapy had been given at another hospital.

Examination. On July 3, 1956, the patient, now 10 years old, was admitted to the University Hospital with a diagnosis of influenza following 2 weeks of severe frontal headaches, nausea, and vomiting. For 7 days she had had complete blindness of the left eye. Examination showed she had total loss of vision in the left eye and a right temporal hemianopsia (Fig. 1). Skull films showed optic foramina of normal size with a definitely enlarged left superior orbital fissure (Fig. 2).

First operation. A left frontal osteoplastic craniotomy was performed 10 days after admission. Upon retraction of the left frontal lobe, the left optic nerve was found to be three times normal size due to an infiltrating tumor. A blue, dome-shaped ("blueberry") infarct was seen at the junction of the left optic nerve and the chiasm (Fig. 3). When all attempts to aspirate the area of infarction with a No. 22 spinal needle were unsuccessful, a very small incision was made superficially paralleling the fibers at the most distal portion of the swelling, and a tiny suction tip was used to evacuate the lesion. Consideration had been given to the location of the crossing fibers of the chiasm and the best site selected. A minute biopsy was taken of the tumor, which was reported to be a spongioblastoma polare.

First postoperative course. Within 12 days there was light perception in the peripheral portion of the nasal and temporal fields; a tangent screen field was recorded 22 days postoperatively (Fig. 4). Subsequent alterations of the visual field were seen in the left eye 7 months later, demonstrating bizarre patterns (Fig. 5). Four years postoperatively the visual field in the left eye remained improved (Fig. 6).
Second operation. In 1964, because of inability to count fingers with the left eye and bilateral optic atrophy, a left frontal osteoplastic craniotomy was performed. A multiloculated cystic lesion of the orbit was found and drained. The large optic nerve infiltrated with tumor was cut anterior to the chiasm and posterior to the globe and removed.

Second postoperative course. The visual fields were essentially the same as on July 3, 1956, showing a complete loss of vision in the left eye and a right temporal hemianopsia.

Comment. In retrospect, the patient's slow infarction of the tumor at the junction of the chiasm and the left optic nerve probably began 2 weeks prior to her 1956 admission. Had the rapid change of vision been known to the ophthalmologist or neurosurgeon, it should have suggested the urgency of surgical intervention. The case illustrates how infarction may occur within the chiasm with dissection of the optic nerve and the chiasmal fibers causing compression rather than destruction of them. This condition is analogous to an intracerebral hemorrhage in the depths of the brain. The fact that the infarct could not be aspirated and required incision and drainage indicated it was not of recent origin but might very well have been of a week's duration.

It is remarkable that the patient retained good recovery of her vision for 8 years after the operation on the chiasm.

Case 2. This 57-year-old man had a headache in association with an upper respiratory
infection which had caused him to consult his physician in March, 1965. Examination showed bitemporal hemianopsia (Fig. 7), and skull films revealed expansion and erosion of the sella turcica. Corrected vision was 20/30−2 OD and 20/25−2 OS. The diagnosis of chromophobe adenoma was made, and radiation therapy totaling 3100 R to the sella was completed April 19, 1965.

Because of progressive loss of visual acuity to 20/40 − 1 OD and 20/40 + 1 OS and reduction in visual fields as shown by a 2 mm and 18 mm white test object (Figs. 8 and 9), the patient was admitted to the University of Michigan Medical Center on July 9, 1965.

Examination. The lateral laminagram of the skull (Fig. 10 top) showed intrasellar expansion and destruction of the posterior clinoid processes. An internal carotid arteriogram demonstrated elevation of the anterior communicating and both anterior cerebral arteries, the right being higher and more attenuated than the left (Fig. 10 bottom). The elevation of the anterior portion of the basilar vein of Rosenthal suggested parasellar extension of the lesion.

Operation. After preoperative steroid preparation, a right frontal osteoplastic craniotomy was performed. A suprasellar tumor pressed on the inferior surface of the chiasm; elevation of the right optic nerve showed a groove formed by the right anterior cerebral artery as it compressed the optic tract. The capsule of the pituitary tumor was firm; the tumor itself was soft, cystic, and hemorrhagic, probably secondary to radiation therapy. A blue, dome-shaped lesion, 1 cm in diameter, was identified on the superior surface at the junction of the optic nerve and the chiasm (Fig. 11 top). The optic nerves and chiasm were carefully decompressed.
Fig. 4. Case 1. Postoperative visual fields, July 25, 1956. The lightly stippled area of the left visual field indicates light perception (left).

Fig. 5. Case 1. Visual fields, February 1, 1957. Left: The left visual field 7 months after the surgical procedure shows light perception temporally, and a large scotoma. Right: Visual acuity has deteriorated somewhat in the right eye with only slight change in the inferior right temporal field.

Fig. 6. Case 1. Visual fields, October 20, 1960. The large left central scotoma persists with an improved left temporal field and some loss in the left nasal field.
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Fig. 7. Case 2. Visual fields, March 3, 1965. Before x-ray therapy, a partial bitemporal hemianopsia is seen.

Fig. 8. Case 2. Visual fields, July 9, 1965. 11 weeks after x-ray therapy. A 2 mm white test object shows a progressive lesion with partial bitemporal hemianopsia and slight superior nasal quadrant involvement.

Fig. 9. Case 2. Visual fields, July 9, 1965. An 18 mm white test object shows a partial left inferior temporal quadrantic defect suggesting the presence of a dorsal lesion in the chiasm in the prechiasmal region.
hemianopsia and no improvement in the visual acuity.

The visual field studies on July 9, 1965 were significant. The use of a 2 mm white test object demonstrated an incomplete bitemporal hemianopsia, whereas an 18 mm white test object demonstrated only a partial inferior temporal quadrantic defect which should have suggested the presence of a right superior prechiasmal lesion. This observation was only made in retrospect.

**Discussion**

*Normal Vascular Supply.* There is considerable controversy about the normal vascular supply to the optic chiasm. Dawson made a dissection of 230 specimens of the optic chiasm, optic nerves, and infundibular area. He concluded that there were two separate sets of arterial anastomotic systems in the chiasmal area: 1) the circuminfundibular anastomosis that supplies the infundibulum and tuberal area, and 2) the prechiasmal anastomosis that supplies the intracranial portions of the optic nerves and chiasm. The prechiasmal anastomosis is the one most pertinent to this discussion. There are three main sources of vascular supply in this group of vessels: 1) the prechiasmal branches, which are derived from the ophthalmic artery; 2) the superior chiasmal arteries, which originate from the anterior cerebral artery and which supply the dorsal surface of the chiasm; and 3) the anterior superior hypophyseal or inferior chiasmal arteries, which arise from the internal carotid artery between the origins of the ophthalmic and the posterior communicating arteries.

Francois, et al., also agree that there is a consistent large branch of the ophthalmic artery that runs along the medial surface of the optic nerve to join the anastomosis at the prechiasmal area, but Hughes and Steele and Blunt deny the existence of such a vessel. The prechiasmal angle is the region between the medial border of the optic nerve and the midpoint of the anterior part of the optic chiasm (Fig. 12). It is at this site that the prechiasmal branches of the ophthalmic artery and the superior chiasmal branches of the anterior cerebral artery unite. On the inferior surface of the chiasm, and very slightly more posteriorly, is the anterior su-

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**Fig. 10. Case 2.** *Top:* Lateral laminogram of the skull discloses an enlargement of the sella with intrasellar erosion of the posterior clinoid processes. *Bottom:* Right internal carotid arteriogram after contralateral carotid artery compression shows elevation of both anterior cerebral arteries, the right more than left.

Initial attempts to aspirate clotted blood from the prechiasmal lesion were unsuccessful, but incision through the dome of the lesion parallel to the crossing fibers permitted cautious removal from within the chiasm through a very fine suction tip (Fig. 11 *bottom*). This produced a marked diminution of swelling and ecchymosis.

**Postoperative course.** The patient was free of headache; a visual field test on August 5, 1965, demonstrated persistent bitemporal
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explaining progressive visual field impairment. Normally, the variation in the multiple fine venous plexuses that drain the chiasmatic region is even greater than that of the arterial pattern.

Compression by the Dural Band. Walker and Cushing in 1918\textsuperscript{27} indicated that in suprasellar pituitary tumors a large dural band connecting the anterior clinoid processes might be responsible for compression of the optic nerves. In 1923 Fay and Grant\textsuperscript{6} called attention to such dural bands and pointed out that compression by them of the internal carotid artery, the anterior cerebral artery, and the anterior communicating artery also might produce visual field changes in patients with such lesions. Traquair,\textsuperscript{22} and others have stressed the fact that vascular compression is a cause of the visual field defect and is not due to direct pressure on the nerves. Dawson\textsuperscript{8} believes there may be either a mechanical compression of the optic nerves of the chiasm, or a vascular compression, which may lead to the resulting visual field defects. The fact that recovery from this type of visual impairment may occur after vascular compression has been relieved suggests that these structures recover by Seddon’s neuropraxia. If stretching of the fibers occurred, a slower recovery would be anticipated by axonotomesis.

Compression by Intratumor Hemorrhage. Compression of the optic nerve and chiasm has been reported by numerous observers when sudden hemorrhage has occurred into the pituitary gland either spontaneously\textsuperscript{2,4,6,7,16,15,25,26} or in association with radiation therapy.\textsuperscript{20} List, et al.,\textsuperscript{16} has stressed the fact that the mechanism may be an increased intracapsular pressure in the pituitary adenoma with subsequent ischemia to the tumor and thrombosis; once the infarction has occurred the hemorrhagic mass rapidly increases in size causing compression of the chiasm. He emphasized that there may be irritation to the carotid artery with associated vasospasm. Bleibtreu\textsuperscript{9} in 1905 published the first report of hemorrhage into the pituitary adenoma. Reviewing the literature in 1950, Brougham, et al.,\textsuperscript{4} found only five cases, and reported three of their own. Numerous other reports followed. In 1957,
Fig. 12. Diagram of the vascular supply of the optic chiasm. Dorsal surface of the chiasm with the superior chiasmal arteries arising as small branches from the anterior cerebral vessels forming some collateral channels and branches near the prechiasmal angle. At the junction of the right optic nerve and the chiasm is a dark oval shadow indicating the site of the infarct in Case 2. (After Dawson, B. H. Brain, 1958, 81:207–217, see ref. 6.)

Fig. 13. Diagram of the ventral surface of the chiasm demonstrating the anastomotic arterial blood supply to the chiasm. This supply includes the superior chiasmal arteries from their anterior cerebral artery origins on the dorsal surface of the chiasm, the prechiasmal branch of the ophthalmic artery, the anterior superior hypophyseal artery, the lateral chiasmal artery. (After Dawson, B. H. Brain, 1958, 81:207–217.)
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Uihlein, et al.,25 indicated there were 35 cases of acute hemorrhage into the pituitary in the literature. In reviewing the references cited above, no report could be found of a dome-shaped lesion at the prechiasmal area. There must therefore be an additional factor responsible for the lesion in our two cases other than simply the relatively sudden expansion of the pituitary tumor by hemorrhage or infarction.16

Compression by the Anterior Cerebral Artery. In 1852 Türck23 described a post-mortem examination of a man who had sustained a sudden loss of sight and was found to have a bilateral transverse groove of the optic tract caused by the anterior cerebral artery compressing the optic tract and chiasm. A review of the literature on this topic was made by Fay and Grant2 in 1923. In 1933 Peet18 illustrated this condition. Rucker and Kernohan19 in 1954 reported five such cases and described varying degrees of visual field impairment due to this mechanism. Some of these cases started with an inferior altitudinal visual field loss, indicating compression of the superior fibers of the optic tract by the traversing anterior cerebral artery. The “mystery” of superior nasal quadrant sparing has never been adequately explained in visual field defects with large pituitary tumors. Even with complete loss of other fields, both superior nasal quadrants are often spared until late in the tumor progression.

If we consider purely the anatomical relationship of the optic chiasm to a large pituitary tumor, it is easy to understand that the crossing inferior chiasmal fibers are the first to be impaired, and produce the classical bitemporal field defect. It is more difficult to explain the events that follow. A fact accepted by ophthalmologists is that further loss of visual fields proceeds in a clockwise progression in the right eye and counterclockwise in the left one. Thus, the inferior nasal fields are the next to become impaired. Following the bitemporal loss, the inferior nasal fields become involved, but the superior nasal fields are the last to vanish. A simple anatomical interpretation might indicate that the inferior lateral uncrossed chiasmal fibers (supplying the superior nasal fields) would be compressed by a large pituitary tumor before the superior lateral fibers, which lie farthest away from the tumor. Common clinical experience reveals that this is not true.

In an effort to explain this mystery of the superior nasal sparing several theories have been expounded. Rucker and Kernohan19 postulated that the grooving of the superior optic tract fibers by the anterior cerebral artery would account for this inferior nasal field loss and leave the inferior uncrossed chiasmal fibers free (superior nasal fields). Another explanation, based on vascular supply was proposed originally by Dawson6 and advocated by Hughes14. This involves the blood supply to the inferior uncrossed chiasmal fibers by the lateral chiasmal arterial branch of the internal carotid artery. Thus, the blood supply to these fibers would be well protected far laterally, and perhaps they would be the last to be impaired in a medially compressing tumor of the pituitary fossa. The anatomy of the vascular supply to the chiasm therefore, may hold the answer to the common clinical findings seen for so many years with pituitary tumors.

The Mechanism of Prechiasmal Infarction. The prechiasmal area is a most vulnerable one, for it contains the main confluent or collateral arterial supply to the chiasm. The simultaneous interference with the arterial and venous circulation serving this region may lead to infarction of the prechiasmal zone. Both intrinsic and extrinsic mechanism may be involved. In our Case 1, the gradual increase in the size of the optic nerve glioma resulted in intraneural and intrachiasmal (intrinsic) compression of the venous return, with edema within the non-yielding pial sheath followed by intraneural and intrachiasmal ischemia with subsequent infarction. In our Case 2, expansion of the pituitary tumor simultaneously caused external compression of the inferior surface of the chiasm and stasis of the vascular supply to the right optic tract on its superior surface due to pressure from the right anterior cerebral artery. The result of these extrinsic factors was a marked ischemia and ultimate infarction of the prechiasmal area.

The question may well be raised as to
whether such lesions might be iatrogenic. The patients’ histories, the course of the visual loss as demonstrated by the visual field examinations, and the gross appearance of the lesion in both of our cases make an acute lesion unlikely and support the concept of a subacute condition with gradual infarction occurring over several days. In 1939 Henderson reviewed Cushing’s series of 338 pituitary adenomas and found four patients had had progressive impairment of vision postoperatively. Bakay reported on the postoperative results of Olivecrona’s series of 300 pituitary adenomas and noted that two of the patients had sustained visual loss after the operative procedure. Both of these authors indicate that suprasellar blood clots might have been responsible for these sequelae. In one of the four cases reported by Henderson, reoperation with aspiration of intrasellar fluid led to improvement in vision.

Significance of Vascular Supply. Although attention has been paid to the vascular supply of the chiasm by Dawson, Blunt, Steele and Blunt, Francois, et al., and others, clinicians have been slow to recognize its importance. An excellent review of the literature was published in 1962 by Udvarhelyi and Walsh, who also presented 11 cases of sellar and parasellar lesions in which there were postoperative ocular complications related to the surgical procedure. They emphasized Dawson’s work on the vascular supply to the chiasm and stressed that visual loss was probably due to ischemia resulting in localized hypoxia. It was believed that some of the unexpected visual involvements might be due to variations in the vascular supply. Morello and Frera more recently have discussed the importance of vascular disturbances of the optic nerve and chiasm as being responsible for the visual damage incurred after removal of hypophyseal adenomas. In none of these reports were we able to find evidence of prechiasmal infarcts. We therefore believe the lesion must be dependent on very special anatomical and physiological circumstances. However, we predict that there will be more instances of this type of chiasmal lesion reported in the future.

Treatment. Since there is no specific pre-
supply and the impairment of venous drainage may result in subacute prechiasmal infarction.

Two patients have been presented, each of whom had a blue, dome-shaped infarct in the prechiasmal area. One had an optic nerve glioma; here an intrinsic mechanism for the subacute infarct was postulated involving hypoxia and venous impairment associated with marked intraneural and intrachiasmal edema. The lesion was drained and the patient regained some useful vision for 8 years.

In the second patient the prechiasmal infarction was due to an extrinsic mechanism involving simultaneous external compression from an infarcted pituitary tumor infrachiasmally and a non-yielding anterior cerebral artery crossing the dorsal surface of the optic tract. In this instance, evacuation of the prechiasmal infarct did not result in visual improvement.

This type of lesion must be exposed through a subfrontal operation for it would not be visualized through a transsphenoidal approach. We have emphasized the importance of further microneurosurgical studies of the vascular supply of the optic nerves and chiasm to prevent some of the unexplained visual losses that occasionally result from surgical attacks on lesions in this area.

References


