SEVERANCE OF THE OPTIC NERVE AT THE OPTIC FORAMEN CAUSED BY LEAD PELLETS

A report on two cases

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SUMMARY

This is a report on two cases of sudden and complete loss of sight, following upon a laceration of the optic nerve near the optic foramen by means of a single lead pellet. (Such cases are considered to be of interest because they are uncommon and because of the almost identical clinica findings).

Injury to the optic nerve may be indirect or direct. In indirect injury, the nerve damage may be the result of the displacement between the optic nerve and the dural sheath, where it is attached to the bone that is in the bony canal. This mechanism may explain (1) the optic nerve atrophy following a head injury most often subsequent to a fall from a bicycle and (2) the optic atrophy which appears after an eyelid injury in children. The optic nerve may also be involved in a fracture of the orbital roof, implicating the superior orbital fissure and the optic canal.

It is well known that radiographic evidence of damage to the orbital roof and apex of the orbit is often difficult because of the complicated architecture of the bones and their obscuration by dense surroundings. Injury to the optic nerve was found in 30 cases out of 750 (4%) basal fractures (Birsch Hirschfeld 1930).

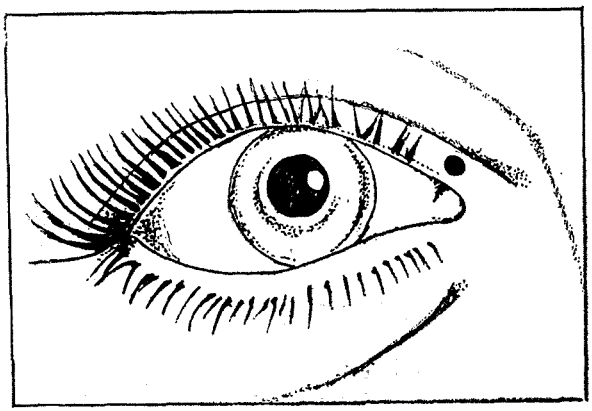

Fig. 1.

The optic nerve may suffer direct injury in penetrating wounds of the orbit by a great variety of sharp objects or by missiles. If the nerve is severed in front of the entrance of the retinal vessels into the nerve, examination of the fundi shows a picture resembling that of an embolism of the central retinal artery. When the nerve is cut behind the entry of the retinal vessels, optic atrophy appears within 3 to 6 weeks.

The report on two cases of sudden permanent loss of vision following upon injury of the optic nerve at the optic foramen by means of a lead pellet from a shot gun is

of interest because it is uncommon. The almost identical clinical findings provide another source of interest.

G.F. aged 41 years, early in the morning of 19.4.72 was struck in the right eye by a lead pellet. He noticed a sudden and complete loss of sight.

The upper lid showed a small perforating wound near the inner canthus. There was total ophthalmoplegia, ptosis and some degree of proptosis. Light perception was absent. There was no direct pupillary reflex. Reflex pupillary reaction was present (superior sphenoidal fissure syndrome).

Fundus examination showed a large subretinal haemorrhage on the middle nasal quadrant. Tension of the eyeball was normal. Radiological examination revealed a lead pellet lodged very near to the optic foramen. There was no evidence of bony orbital damage (fig. 2).

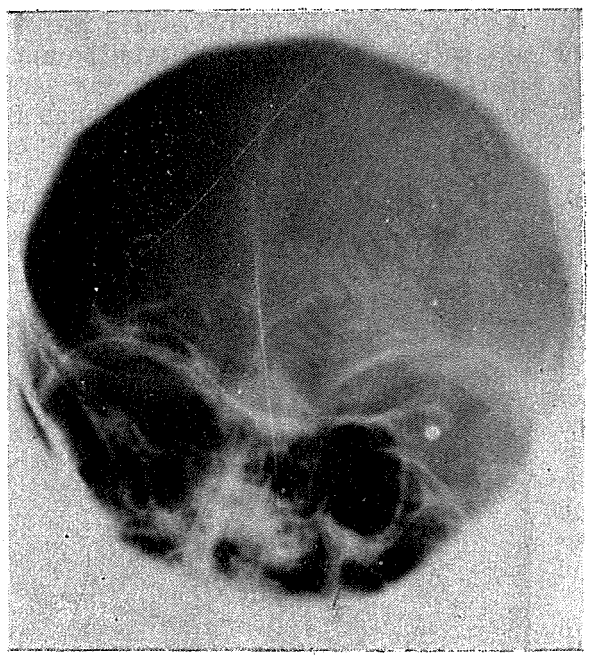

Fig. 2.

A gradual restoration of the ocular motility took place during a period of three weeks. However, the pupil remained fixed and dilated. Reflex pupillary reaction was present all the time. There was no return of light perception.

Examinations of the fundus carried out on the 20.11.73 showed a large atrophic choroidal patch along with extensive pigmentary disturbance at the level of the 30c meridian. There was complete optic atrophy.

An X-Ray examination showed that the lead pellet remained in the same site opposite the optic foramen.

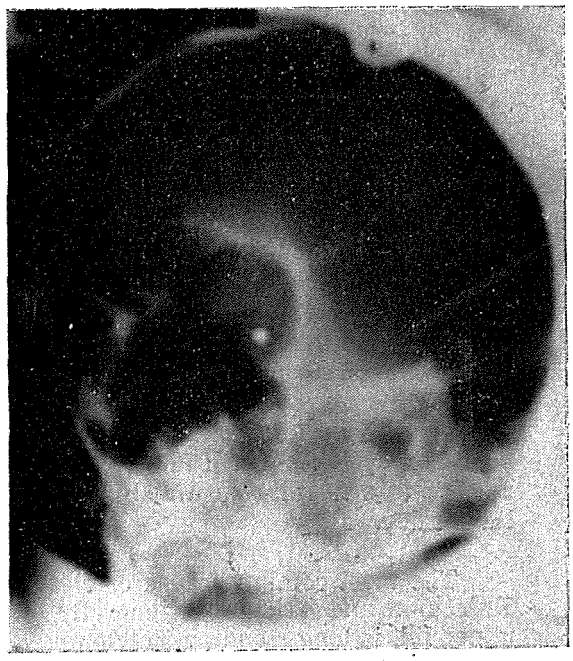

Fig. 3.

Case no. 2: C.C. aged 27, on 26.10.73 was hit by a lead pellet in the right upper lid. He complained of sudden and complete loss of vision.

Examination showed a small perforating wound of the upper lid near the inner canthus. There was a moderate degree of ptosis. Otherwise, the movements of the ocular muscles were normal. The direct pupillary light reflex was completely absent. The consensual reflex was present. The tension of the eyeball was normal. Fundus examination showed a yellowish line in the choroid running in an antero-posterior direction at the level of the 30c meridian. Radiological examination showed a lead pellet very near the optic foramen (fig. 3). There was no movement of the foreign body on up and down views.

Examined after a month, the optic disc is already showing a marked pallor. There was no return of light perception.

The changes in the fundi, seen in these two cases, must have been caused either by a tangential grazing of the sclera by the pellet, or by the concussion waves travelling through the semi-fluid orbital contents.

References

BIRSCH HIRSCHFELD, (1930), Kurzes HB.d. Ophth., *3*, 107.