

# CASE REPORT

## Pupil-Sparing Third Nerve Palsy Due to Diabetes Mellitus:

Gordon E. Searles\*

### Abstract

*The symptoms and diagnosis of diabetic oculomotor ophthalmoplegia are examined in a case study. The patient had a right ptosis with diplopia and paresis of all muscles subserved by the oculomotor nerve. Tests for diabetes were positive and patient was placed on therapy to control diabetes, with resolution of palsy three months after initiation of treatment.*

*Diabetic ophthalmoplegia is a form of ischemic neuropathy that could be elicited from metabolic or vascular causes. Differential diagnosis is essential to rule out aneurysms and myasthenia gravis. The cardinal sign of diabetic palsy is the sparing of pupillary function. The case points out the importance of a careful case history to determine the cause of the condition.*

### Abrégé

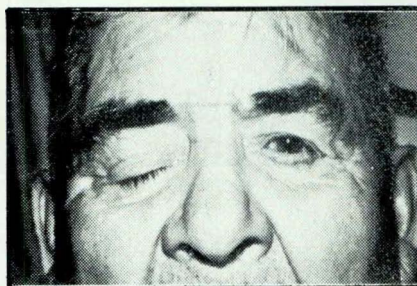
*Les symptômes et le diagnostic d'une paralysie du nerf oculomoteur causée par une diabète, sont misent de l'avant par l'entremise d'une histoire de cas. Le patient manifestait un ptose de l'ocil droit, une diplopie et une paralysie de tous les muscles par le nerf oculomoteur. Une diagnostique de diabète fût faite et le traitement prescrit — trois mois plus tard la paralysie avait disparu.*

Ophthalmoplegia of the extraocular muscles can be caused by a variety of factors. Inflammatory disease, tumour, thrombosis, arteriovenous fistula, diabetes, and an aneurysm at the cavernous sinus can all elicit an ophthalmoplegia.<sup>1</sup> Of these, diabetes and aneurysm are the predomi-

nant causes of most ophthalmoplegias. Due to the great number of diabetics in the population, the eye care practitioner may observe a number of cases of diabetic ophthalmoplegia, and the recognition of the cardinal signs is necessary for performing a differential diagnosis.

### Case History

An 85 year old North American Indian male was examined and indicated a right full ptosis. Upon raising the lid, a lateral squint was realized in the primary position (figure 1). The patient described a di-



**Figure 1:** 85 year old male exhibiting a full ptosis of the right eyelid.

ptopia immediately preceding the onset of the ptosis. A test of ocular motility demonstrated an inability to adduct, elevate, depress, and a slight intorsion was observed when the patient looked to the inferior left (figure 2). It was apparent that the left lateral rectus and superior oblique muscles were relatively normal in function. Pupillary examination showed normal responses and he had a pupil-sparing third nerve palsy. Tests for diabetes were performed by an ophthalmologist, and the glucose tolerance test indicated 420 mg/100 ml. The normal limits for a patient of this age would be 160 mg/100 ml.<sup>2</sup> The patient was then

put on a carefully regulated diet for the control of the blood glucose level. Immediate improvement of the ophthalmoplegia was observed within 48 hours after start of treatment in the hospital. After 3 months, the effects of the ophthalmoplegia were completely resolved.

### Discussion

Diabetic ophthalmoplegia is a form of peripheral diabetic neuropathy of the extraocular muscles subserved by one of the cranial nerves. Specifically, the mechanism of diabetic neuropathy involves segmental injury to nerves, associated with focal demyelination of Schwann cell degeneration with minimal axonal degeneration. The types of neurons affected can be sensory, motor, or those of the autonomic nervous system. The most common ocular involvement includes the abducens nerve, with the oculomotor cranial nerve to a lesser extent. The specific symptoms are pain to the involved eye and area, sensory loss and motor weakness or loss of function of the motor and sympathetic innervation.<sup>3</sup>

The lesion is caused by the ischemia of the neurons when the intraneural arteries in the region of the intracavernous or subarachnoid segment are affected by the diabetic complications.<sup>1</sup> The effect may be metabolic, via the sorbitol pathway, or vascular.<sup>4</sup> In the case of diabetic ophthalmoplegia, the prognosis is usually good, with the resolution and normal function within 3 months, as the axonal regeneration occurs.<sup>1</sup> Damage to the neurons by metabolic changes usually has the better prognosis, since it is potentially reversible, whereas the effects of vascular change are not reversible. Also, the condition may be recurrent, affecting the same or other nerves.<sup>1</sup>

\* Optometry Student, Third Professional Year, School of Optometry, University of Waterloo, Waterloo, Ontario





**Figure 2:** (centre) Patient with lid retracted in the primary position. Surrounding views of eye positions while directed into the various positions of gaze. Note the absence of sursumduction, adduction, and deorsumduction of the right eye. Although not obvious in the views, a slight intorsion of the right eye was observed when the view was directed to the inferior left.

A patient with oculomotor ophthalmoplegia may present signs and symptoms of ptosis; a lateral squint due to loss of muscles served by the oculomotor nerve; a normal pupil reflex, direct, consensual and near; and a possible exophthalmos since the extraocular muscles are relaxed.<sup>5</sup> Duke-Elder also suggests the possibility of accommodative deficiency due to a paresis of the ciliaris mus-

cle,<sup>6</sup> amblyopic pupillary paresis,<sup>7</sup> atypical (non-syphilitic) Argyll Robertson pupil,<sup>8</sup> and a tonohaptic reaction.<sup>9</sup> Similarly, a patient with abducens ophthalmoplegia will exhibit an inability to abduct. Diabetic ophthalmoplegia has a very characteristic sign in that associated with the motor weakness, there is no loss of pupillary function. This sparing is believed to be due to the noninvolvement of the circumferential portion of the nerve with the ischemia.<sup>10</sup>

Differential diagnosis of the condition is required to determine correct treatment. The ophthalmoplegia as noted before, can be elicited by many causes, but a close observation of signs and symptoms will provide proper guidance. The cardinal sign of the condition is the degree of pupillary activity. An aneurysm would be indicated if a diminished pupillary reaction was also observed with the motor weakness. Confirmation would be found in the results of a

carotid angiogram.<sup>11</sup> Conversely, the absence of pupillary involvement should be an indication of diabetic involvement. It should be noted that the presence of overt diabetes need not be indicated, and that the ophthalmoplegia may be the primary symptom of the diabetic condition. A 3-hour or 5-hour glucose tolerance test should indicate if diabetes is present. Caird and his co-workers carefully note that if diabetic involvement is at all suspect, then carotid angiography should be deferred until the diabetic suspicion is discounted.<sup>12</sup>

One further possibility in the presence of a pupil-sparing ophthalmoplegia should be made to rule out the presence of myasthenia gravis.<sup>1</sup> A close investigation as to the time and periods of onset of the ophthalmoplegia would be indicative.

cont'd on pg. 111



Treatment of the ophthalmoplegia depends on the cause. Diabetic ophthalmoplegia can be adequately controlled by the proper regulation of the diabetes, depending on the age and condition of the patient. As noted before, the condition normally is self-limiting and resolution normally occurs after three months.

### Conclusion

The important signs to note are the weakness of all structures supplied by the oculomotor nerve with pupillary sparing. Usually in the case of an aneurysm, the pupil reflexes would be involved, but their absence does not rule it out. Also, a blood glucose tolerance test is easier to perform and less dangerous for the patient than carotid angiography. It must be carefully noted in the

history whether the patient has a predilection towards diabetes, since the ophthalmoplegia may be the first indication of the condition. Also, myasthenia gravis should be part of the differential diagnosis if the glucose tolerance test results are inconclusive. Careful examination and scrupulous history taking will aid with the proper diagnosis.

### Acknowledgements

I wish to thank L.S. Landecker, MD. FRCP(C) for his clinical guidance and patience and for offering technical assistance in the manuscript; and A. P. Cullen, OD. PhD. for assisting me in the research aspects.

### References

1. Duane, Thomas D., MD, PhD. "Infranuclear Disorders of Eye Movements", Ch. 12. Clinical Ophthalmology, Vol. 2. Harper and Row. (Hagerstown, 1979). p. 12.
2. Harrison's Principles of Internal Medicine. 7th ed. Ed. Maxwell M. Wintrobe, et al. McGraw-Hill. (New York, 1974). "Diabetes Mellitus" Ch. 88. p. 539.
3. The Merck Manual of Diagnosis and Therapy. 13th ed. Ed. Robert Berkow, et al. Merck & Co. (Rahway, 1978). Sect. 12, Ch. 7. p. 1290.
4. Harrison's Principles of Internal Medicine. Ch. 88. p. 548.
5. Gray's Anatomy. from the 15th English edition. Ed. T. Pickering Pick. Bounty. (New York, 1977) p. 722.
6. Duke-Elder, Stewart. System of Ophthalmology. Vol. V. Kimpton. (London, 1970). p. 462.
7. Duke-Elder, Stewart. System of Ophthalmology. Vol. XII. Kimpton. (London, 1971). p. 655.
8. Duke-Elder, Stewart. System of Ophthalmology. Vol XII. p. 664.
9. Duke-Elder, Stewart. System of Ophthalmology. Vol. XII. p. 688.
10. Goldstein, Jack E., and David G. Cogan. "Diabetic Ophthalmoplegia with Special Reference to the Pupil". Arch. Ophthalmol. Vol. 64:592-600. 1960.
11. The Merck Manual. Sect. 19, Ch. 18. p. 1712.
12. Mausolf, Frederick A. The Eye and Systemic Disease. Mosby. (Saint Louis, 1975). p. 196.

## ENSURE OPTOMETRY IS HEARD ON THE HILL

Contact your Member of Parliament

— Press Home the Issue

*Before the House Reconvenes*

Send a letter of concern to the Minister of

1/ the Department of National Defence

re: Use of Ophthalmic Technicians

2/ the Department of External Affairs

re: Passport Guarantor Status

**ACT NOW!**