Oculomotor nerve palsy precipitating acute angle-closure glaucoma

A case report

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Summary

A case of acute angle-closure glaucoma precipitated by oculomotor nerve palsy in a patient with shallow anterior chambers is reported. The different ways in which a palsy of the oculomotor nerve can influence the intra-ocular pressure are discussed.

Several anatomical factors may predispose an eye to primary angle-closure glaucoma. A more anterior location of the lens-iris diaphragm resulting in an overall shallowing of the anterior chamber and a narrow entrance to the chamber angle is the typical configuration. A disproportionately large lens size in relation to the size of the anterior chamber, whether it be a large lens or a small anterior chamber (e.g. hyperopic eyes), can also bring about a shallowing of the anterior chamber and angle entrance. These anatomical factors may be said to predispose to closure of the angle, but other physiological or pharmacological factors must be superimposed to bring about actual closure of the angle.

A case in which angle closure was precipitated by pupillary dilatation secondary to oculomotor nerve palsy is reported.

Case report

A 67-year-old woman was awakened by severe right orbital pain associated with diplopia, ptosis and blurred vision of the right eye 3 days before presentation to hospital. There was no history of any previous eye problems. The patient had been treated for hypertension and congestive cardiac failure for years. She was taking amiloride 5 mg plus hydrochlorothiazide 50 mg twice daily, digoxin 0,125 mg daily and methyldopa 250 mg twice daily. There was no history of diabetes.

On ophthalmological examination her corrected visual acuities were right 1/40, left 6/6. There was a complete right ptosis, right circumcorneal redness and corneal oedema. Both anterior chambers were shallow, the right more so than the left. The right pupil was mid-dilated, irregular and did not respond to light and near. The intra-ocular pressures were right eye 75 mmHg and left eye 17 mmHg (applantation tonometry). The right eye was exotropic (on fixation with the right eye the angle was 90 prism dioptres and 40 prism dioptres when fixating with the left eye). There was also decreased elevation, depression and adduction of the right eye. The right anterior chamber angle was closed over 100% of its circumference while the left angle was narrow but open. The

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right fundus was not visible because of corneal oedema. The left fundus appeared normal.

General neurological examination revealed no other abnormalities. The blood pressure was 140/90 mmHg.

A diagnosis of an acute angle-closure glaucoma precipitated by oculomotor palsy was made.

Laboratory test results were: fasting blood glucose 4,9 mmol/l; erythrocyte sedimentation rate 9 mm/1st h (Westergren); negative serology for syphilis and normal serum protein electrophoresis, collagen screening, liver function tests and cerebrospinal fluid. Computed tomography of the brain was normal. A 4-vessel angiogram of the brain showed no abnormality.

The patient was treated with pilocarpine 2% drops in the right eye every hour for 4 hours and then 4 times daily: acetazolamide 500 mg per mouth 6-hourly for 1 day and then 250 mg 6-hourly and paracetamol tablets for pain. The right pupil constricted well in response to pilocarpine. The next day the intra-ocular pressure was down to 12 mmHg, the pupil was well constricted, the anterior chamber shallow and the eye still red. The discomfort had, however, subsided.

Bilateral YAG-laser iridotomies were done on the same day. Bilateral surgical transcorneal peripheral iridectomies were done under local anaesthetic 3 weeks later (since there was doubt about the long-term patency of the YAG-laser iridotomies). Postoperatively both eyes were treated with local antibiotic/steroid combination drops 4 times daily (Maxitrol; Alcon). The postoperative course was uneventful.

The corrected vision on discharge was 6/6 in both eyes. Both drainage angles were 100% open. The right pupil was still mid-dilated with absent reaction to direct and indirect light and near. The oculomotor palsy improved gradually in the ward, but was still present on discharge.

Discussion

Only a few cases of acute angle-closure glaucoma caused by a 3rd cranial nerve palsy have been described. In the patient described by Wilson and Barmatz[1] the angle-closure attack was caused by total oculomotor nerve palsy because of a bleeding posterior communicating artery aneurysm. In the case described by Zaidi[2] the 3rd nerve palsy was associated with diabetes, but it is difficult to ascertain from the article whether the pupil was in fact involved.

Hypertension was the presumed cause of the oculomotor nerve palsy in our patient. Hypertension is usually associated with a pupil-sparing palsy, although this is not always the case (this is also true of the palsies associated with diabetes). The persistent pupillary dilatation after pilocarpine was stopped could have been the result of the angle-closure attack as such. The history of a simultaneous onset of a 3rd nerve palsy and the angle-closure attack strongly suggests that the palsy was the precipitating factor, although it is not possible to be certain that pupillary dilatation was the actual cause.

In an eye with a shallow anterior chamber a large area of iris is in contact with the lens. This contrasts with an eye with a

normal anterior chamber where this contact exists only in the immediate vicinity of the pupillary margin. This increased area of contact between iris and lens causes an increased resistance to aqueous humour flow from the posterior chamber (PC) through the pupil to the anterior chamber (AC). This results in a slightly higher pressure in the PC relative to the AC which causes a forward bowing of the iris in the periphery — called relative pupillary block. This is constantly present in normal eyes, but in eyes with increased iris-lens apposition (such as those with shallow anterior chambers) the relative pupillary block is increased. These eyes are thus the most vulnerable to angle closure.

Other factors need to be superimposed to cause closure of the angle. The most important of these is dilatation of the pupil — especially dilatation from parasympathetic blockage whether it is due to topical or systemic anticholinergics or an oculomotor nerve palsy. In such a situation the peripheral iris is relatively flabby and in the presence of a relative pupillary block with a large pressure differential between the PC and AC, the iris is quite easily pushed against the trabecular meshwork. Angle closure is most likely to occur when the pupil is in mid-dilatation since both the relative pupillary block and peripheral laxness are then at the maximum. An oculomotor nerve palsy thus presents the ideal situation for angle closure since the pupil never dilates widely.

With wide pupillary dilatation the area of iris-lens apposition is minimal and the aqueous humour can flow freely into the AC.

The above situation contrasts markedly with pupil dilatation caused by sympathetic stimulation. Although there is increased apposition of the iris to the lens in this instance, there is no peripheral laxness of the iris and the pressure differential needs to be much higher before the iris is pushed against the trabecular meshwork.[3]

It is interesting to consider other possible ways in which an oculomotor nerve palsy can affect intra-ocular pressure. It has been shown that iris sphincter and/or ciliary muscle contraction physically alters the trabecular mesh configuration so as to decrease outflow resistance, while muscle relaxation deforms it so as to increase resistance. It seems virtually certain that this effect is mediated entirely by ciliary muscle contraction and relaxation and not by direct pharmacological effect on the trabecular mesh.[4-8]

The effect of a change in parasympathetic innervation on the production of aqueous humour is not clear. Using a variety of species, conditions and experimental techniques, chlorinergic stimulation has been reported to increase, decrease or not alter the aqueous humour formation rate and to slightly increase the episcleral venous pressure.[9] The results of parasympathetic nerve interruption have been similarly nonspecific.[10,11]

Paralysis of the extra-ocular muscles by an oculomotor nerve palsy would cause a decrease in intra-ocular pressure.[12]

Most sympathetic fibres enter the orbit with nerves other than the oculomotor[13] and are probably not affected by an oculomotor palsy.

The ease with which the patient's pupil was constricted with pilocarpine 2% drops, even after 3 days of angle closure, was interesting. A similar occurrence was reported by Wilson and Barmatz.[1] The functional sensitivity of the iris sphincter has been shown to be inversely correlated with the tissue density of high-affinity muscarinic receptors.[14-16] This receptor density is, in turn, regulated by the level of cholinergic stimulation — the higher the level of stimulation the less the sensitivity and vice versa.[17-19] In the rat iris alteration in sensitivity in either direction begins within hours and is completed after several days.[19]

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