TRANSIENT TRAUMATIC MYOPIA WITH HYPOTENSION*

BY

MAURICE H. LUNTZ

St. Mary's Hospital (Western Ophthamlic Hospital), London

It is well recognized that myopia is the commonest refractive change after trauma; however, the clinical syndrome in which myopia after trauma is associated with anterior chamber loss and hypotension in the absence of any other ocular pathology, although recognized, is rare. A few cases have been reported in the literature between 1870 and 1931, but none appears to have been described in Great Britain, apart from two cases published by Morgan (1940). In both Morgan's cases the condition persisted for over a year and there was associated ocular damage (choroidal tears) so that they do not fall into the category defined above. It was felt that it would be of interest to publish this case report and at the same time to present a brief review and discussion of the literature, taking into consideration modern techniques and ideas.

Case Report

A married woman, 32 years of age, was seen in the out-patients department of this hospital on January 27, 1958, giving a history of having knocked her right eye on the handle of a door 2 days previously. Since then the eye had become swollen, movement was painful, and she had some binocular diplopia with diminution of right visual acuity, which was less than 6/60 with her own glasses (+0.5 D sph., +0.25 D cyl., axis 90°). She was quite sure that she had seen normally with this eye before the accident. This was given added credence by the fact that her left eye was amblyopic (6/24) and had been so since childhood. The visual acuity was not improved with a mydriatic. There was, in addition, traumatic iritis with mydriasis, but this cleared within a few days. The lens was not dislocated nor oedematous nor altered in shape, yet the anterior chamber was very shallow. A careful search with the slit lamp did not reveal any corneal or scleral wound or aqueous leak. There was no abnormality in the fundus, and x-ray examination of the orbit showed no abnormality.

She was refracted under a mydriatic and the visual acuity of the eye was improved to 6/12 with −3 D sph. The tension in the right eye was 11 mm. Hg (Schiotz) and in the left 20 mm. Hg.

The patient was admitted to hospital for observation; 2 days later the ocular tension in the right eye was still 11 mm. Hg (Schiotz) but the visual acuity had improved to 6/18 unaided and 6/6 with −1.5 D sph. The anterior chamber had noticeably deepened. After 2 weeks the ocular tension was 20 mm. Hg in both eyes, the right anterior chamber had returned to normal, and the refraction of the right eye was 6/6 with +0.5 D sph., +0.75 D cyl., axis 120°.

When she was seen again in June, 1958, there was no change.

---

* Received for publication October, 20, 1958.

566
Discussion

Interest in traumatic myopia as such was first aroused when Kugel (1870) reported the condition in two soldiers. Schiess-Gemuseus (1881) and Schmidt-Rimpler (1883) described cases which healed spontaneously.

Knapp (1883) of New York described a case in a 29-year-old man with associated hypotension. Contusion to the eye had resulted in loss of the anterior chamber, hypotension, and myopia. The fundus was normal. The myopia took 8 days to recover and this was accompanied by re-formation of the anterior chamber. On the tenth day, an attack of acute glaucoma supervened, but responded to conservative treatment.

Schiess-Gemuseus (1881) had assumed that the myopia was due to forward dislocation of the lens caused by stretching of the zonule as a result of the blow. Knapp disagreed and felt that in his case there was no question of the lens being dislocated. He postulated that the loss of the anterior chamber was due either to a very small scleral or limbal wound or to depression of the ciliary body secretion. He considered the myopia to be due to the loss of the anterior chamber which resulted in the lens moving forward.

Fromaget (1911) described a similar case in a young woman. Here the myopia was 3·5 D sph. and the ocular tension soft (recorded as −1·5). The lens was not dislocated and the fundus was normal. The anterior chamber was very shallow. A year later she had normal ocular tension, a normal anterior chamber, hypermetropia of 1·5 D sph., and paralysis of accommodation. Fromaget pointed out that the myopia was due neither to dislocation of the lens nor to spasm of the accommodation as this was paralysed. The accommodation was also paralysed in the cases reported by Schmidt-Rimpler (1883) and Bouchart (1901). Fromaget calculated mathematically (according to von Helmholtz’s theory) that a myopia of up to 4·6 D sph. is possible when the lens moves forward 3 mm. Here it is of interest that Bailey (1921) reported a case in which complete loss of the anterior chamber produced a myopia of 8 D sph. which disappeared as the chamber re-formed. Fromaget made a plea for accurate measurement of the refraction, the depth of the anterior chamber, and the corneal curvatures in future cases. He considered the aetiology of these cases to be “lack of formation of aqueous”, and this theory was also supported by Grandclément (1922) who reported a similar case.

Brøns (1931) published another case in a young male, in which there was myopia of 2·5 D sph., hypotony, and shallow anterior chamber, but no subluxation of the lens. The myopia disappeared after 2 months when an attack of acute glaucoma supervened (as in Knapp’s case); the latter responded to conservative treatment.

Cases were described also by Guende (1900) and Bourgeois (1904), but here there was evidence of ocular derangement with grosser damage, and these are therefore excluded from this discussion.
The first real attempt at a classification of the cases of traumatic myopia described in the literature was made by Frenkel (1905), who collected 41 cases and divided them into four aetiological groups. Of these only four had an associated hypotony, in seven the tension was increased, and in the rest it was normal.

(i) Accommodative spasm. These had the best prognosis.
(ii) Relaxation of the lens zonule. Here he postulated that the stretched lens zonule might recover in some cases and the condition resolve. Morgan (1940) maintained that all these cases showed iridodonesis.
(iii) Subluxation or dislocation of the lens. Poor prognosis.
(iv) Lengthening of the anterior-posterior diameter of the eyeball through post-traumatic inflammation of the posterior segment. Poor prognosis and very rare.

This classification is widely accepted and its application to the type of case described here will be discussed later.

Bouchart (1901) had attempted a classification into two groups which is of very limited value, except to point out that the presence of damage to the fundus means a poorer prognosis.

(i) Cases with fundus lesions, of which he found seven in the literature.
(ii) Cases without fundus lesions, of which he found fourteen. He thought eight of these were due to subluxation of the lens or spasm of accommodation, and the remaining six to “benign distension of the zonule”.

Bolotte (1934), Déjean and Guignot (1938), and Sourdille (1908) described cases of traumatic myopia which persisted indefinitely, and they explained this by invoking the theory of accommodation of Hudelo (1930), maintaining that there was paralysis of Brucke’s muscle.

Fox (1942) collected 101 cases of traumatic myopia from the literature, and added two cases of his own, but both of these had gross ocular damage. He pointed out that very few cases had been described of traumatic myopia with hypotension, and he accepted Frenkel’s classification for most of them, except for the type of case described here which does not fit into any of Frenkel’s four groups. Fox considered that these cases were due to “inhibition of aqueous formation”.

The present case belongs to the broad group of traumatic myopia, the degree of severity of the injury representing a rare sub-group not mentioned in any of the classifications so far elaborated. A few cases described in the literature, however, have exactly the same features: trauma in a young patient, loss of anterior chamber, myopia of about 3 D sph., and hypotony, the prognosis for which is almost invariably good. However, Morgan (1940) reported two cases in which the recovery took 2 years, but in these fundal lesions were present (retinal oedema and choroidal tears).

If we accept Frenkel’s classification, which is the best at present available,
these cases would fall into a sub-group between his first and second, with a good prognosis. The aetiology remains unexplained, but speculation is permissible. It seems fairly certain, taking into account the evidence in our case and in those of Knapp, Fromaget, Brons, etc., that we are dealing not with anterior chamber loss caused by forward dislocation of the lens as a direct result of the trauma, but rather with a forward movement of the intact lens and zonule diaphragm secondary to loss of the anterior chamber. The possibility of a stretching of the zonule with subsequent recovery, which was suggested by Frenkel (Group 2 of his classification), Darier (1899), Bouchart (1901), and others, seems unlikely in the absence of iridodonesis or of a change in the shape of the lens on slit-lamp examination and in the absence of a cylindrical element in the refraction. The idea of such a “benign stretching of the zonule” which then recovers its previous “tone” appears unlikely in such a structure, which is so tough in the young and which breaks so easily when degenerate, as in hypermature cataracts. There is no clinical evidence that the zonule is capable of stretch except where it is weakened as in Marfan’s syndrome, and in such cases there is no recovery of “tone”. Finally, Wolff (1954) maintained that the zonule was inextensible and Vail (1957) that it was not elastic. Furthermore, this idea does not explain the hypotension, and thus for many reasons, seems improbable.

The idea of a small scleral or limbal wound as suggested by Knapp is possible but unlikely, as we now have the advantage of the slit lamp with its high magnification and no such wound or leak could be detected. Furthermore, Magitot (1917) showed that, in cases of small scleral rupture or intra-ocular foreign body, the resultant myopia is not due to scleral tears, the eye acting as though it had sustained a concussion injury.

It would seem that Knapp’s alternative suggestion (i.e. a temporary paralysis of the ciliary body secretion), corresponding to Fromaget’s and Grandclément's “lack of formation of the aqueous” and Fox’s “inhibition of aqueous formation”, is probably correct. Duke-Elder (1954) stresses the presence of a vasomotor instability following concussion injury to the globe. It is submitted that this is best explained by oedema of the ciliary body with paralysis of its function as a result of the contusion. Since it is the most vascular structure in the eye, only the framework being of loose connective tissue, oedema would most readily occur here resulting in narrowing of the ciliary ring. This would not only diminish the aqueous secretion and cause hypotension, but would also slacken the suspensory ligament (as in accommodation, according to Fincham, 1939). At the same time the lens would be pushed forward and this would take up the “slack” of the suspensory ligament, so that there would be no alteration in the shape of the lens. As a result, the anterior chamber becomes shallow, and the lens moving forward causes hypotension and myopia. This supposition accords with present-day theory, and does not involve an elastic property for the zonule, which may or may not recover “tone”.
If this theory is applied to Frenkel's classification, it appears that his second group of cases does not exist. This group contains cases in which ciliary body oedema has caused some "slack" in the zonule (but not a primary stretching), allowing the lens-zonule diaphragm to be pushed forward by the increased volume of the ciliary body behind it, the injury not having been severe enough to cause further ocular damage. The prognosis in such cases is good, the myopia and hypotension usually resolving within 2 months. In cases previously included in this group which did not recover (and here there was nearly always considerable ocular damage noted especially in the fundus), rupture of the zonule with subluxation or dislocation of the lens was probably present, so that they should be placed in the third group where the prognosis is poor. Such cases were described before routine slit-lamp examination was available; Bourgeois, for example, classed three cases in Frenkel's Group 2 which did not resolve after a year; yet when iridectomy was performed (because of the shallow anterior chamber) vitreous presented as the chamber was opened.

All this is pure surmise, but it would appear to be a more acceptable explanation than "benign stretching of the zonule", which may or may not recover.

Unfortunately we did not, in this case, measure the curvature of the corneal meridians, nor did we accurately measure the depth of the anterior chamber. If and when another case of this type presents itself it would be of interest to measure these and to correlate the result with the degree of myopia and its rate of recovery. It would also be of interest to measure the facility of aqueous outflow in view of the possibility that acute glaucoma may supervene during the period of recovery. Here it is of interest to note that in one case reported by Bailey (1921) the lesion was the result of a deep corneal foreign body; Bailey felt that the myopia was due to a change in the corneal curvature rather than to anterior chamber loss. Accurate measurements of the anterior radius of curvature of the lens would also be valuable in assessing this theory of aetiology.

Taking into consideration the points discussed above, it is suggested that Frenkel's original classification of cases of traumatic myopia should be modified as follows:

(i) Accommodative spasm. Good prognosis.

(ii) Oedema of the ciliary body, usually associated with hypotony and loss of the anterior chamber. No other ocular damage. Good prognosis.

(iii) Subluxation and dislocation of the lens with or without a perforating wound. There may be anterior chamber loss and hypotony with clinical evidence of dislocation of the lens. Poor prognosis.

(iv) Lengthening of the anterior-posterior diameter of the globe. Poor prognosis.
TRANSIENT TRAUMATIC MYOPIA WITH HYPOTENSION

Summary

A case is described of transient traumatic myopia associated with anterior chamber loss and hypotension with a good prognosis. The literature is briefly reviewed and discussed, and a modification of the accepted classification is suggested.

My thanks are due to Mr. R. McIvor Paton, under whose care this patient is, for granting me permission to use the case notes and for encouraging me to publish this report. I also wish to thank the Board of Governors of St. Mary's Hospital for access to the case notes. Finally my thanks are due for the assistance and co-operation extended to me by the staff of the Institute of Ophthalmology, in particular to Miss Lynch.

REFERENCES