Clinical and histopathological report of sympathetic ophthalmia after retinal detachment surgery

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SUMMARY A case of sympathetic ophthalmia following retinal detachment surgery is reported. Although previous blunt injury complicated the underlying causes of the sympathetic ophthalmia, we considered that the condition was probably related to a second operation that was done to remove the exposed implant and that resulted in rupture of the globe at the site of necrotic sclera.

Sympathetic ophthalmia is a bilateral granulomatous panuveitis usually caused by penetrating injury presumably involving the uvea. Operative wounds are the second most common cause; more than half of these cases follow cataract operation.1 Sympathetic ophthalmia has only rarely been reported after retinal detachment surgery. Winter2 in 1955 found only one such case in 95 cases of sympathetic ophthalmia following intraocular surgery. A few other cases have been reported.3-5 We present herein a case of sympathetic ophthalmia following retinal detachment surgery with histopathological confirmation of the disease.

Case report

A 16-year-old boy was admitted to hospital in February 1975 because of rapid visual loss in the right eye of one week's duration following retinal detachment surgery in the left eye. Five months previously his left eye had been hit by a basketball. Mild hyphaemia and vitreous haemorrhage resulted, which resorbed over several days with marked visual improvement. Two months after the injury the visual acuity in the left eye decreased again, and a shadow appeared in the supranasal field. An inferotemporal quadrantic retinal dialysis with detachment was diagnosed. Scleral buckle surgery was performed that involved dissection of a scleral bed, diathermy to close the retinal tear, penetrating diathermy to release subretinal fluid, and placement of a Teflon implant. The patient did well in the early postoperative period. Three weeks after the operation the Teflon implant was found to have eroded through the conjunctiva, which caused persistent irritation as well as inflammation. A second operation to remove the Teflon implant was undertaken. At operation the globe was accidentally ruptured in the area of necrotic sclera, with loss of vitreous and exposure of choroid. The wound was closed promptly and no further surgery was performed. Two months after the procedure acute loss of visual acuity was noted in the right eye. A diagnosis was made of uveitis with suspicion of sympathetic ophthalmia. The patient was referred to the Eye, Ear, Nose and Throat Hospital of the Shanghai First Medical College for treatment.

On admission, his visual acuity was no light perception in the left eye and counting fingers in the right eye. The left eye showed slight conjunctival congestion. Fine keratic precipitates and mild aqueous flare were present in the anterior chamber. The iris was thickened and bound down to the anterior lens capsule; the pupil was irregular and about 5 mm in diameter. Behind it a yellow pupillary reflex was noted. The fundus could not be viewed. Intraocular tension was very low. The right eye was also congested. Moderate fine keratic precipitates and slight aqueous flare were present. The pupil was fully dilated. The lens was clear. The fundus looked hazy because of fine vitreous opacity. Shallow retinal detachment was present over the inferior quadrants. No retinal breaks were seen. The left eye was enucleated soon after admission. The right eye was treated with a vigorous regimen that included systemic, local, and periocular injection of steroids and immunosuppressive drugs, but the uveitis persisted with many exacerbations over a 2-year period, resulting in atrophy of the eyeball.
HISTOPATHOLOGICAL EXAMINATION

Gross examination of the enucleated left eye showed that it was deformed by a shallow depression with roughened surface inferotemporally (Fig. 1), corresponding to the healed rupture in the sclera. The globe measured 20 mm vertically, 22 mm axially, and 21 mm transversely. Microscopic examination revealed that the limbus blood vessels were dilated, with exudation of lymphocytes and plasma cells. The corneal epithelium was degenerated and detached from Bowman’s membrane in several places. The anterior chamber was relatively deep in the dependent part, where fibrous tissue was present in a wide, round chamber angle (angle recession). The iris was diffusely infiltrated with plasma cells, lymphocytes, and epithelioid cells, which destroyed the pigment epithelium and extended to the posterior chamber as well as to the ciliary body and was accompanied by membrane formation. The iris adhered totally to the lens capsule. A nest of epithelioid and giant cells appeared in the vascular layer of the ciliary body. The choroid was enormously thickened, especially on the operative side. The granulomatous tissue involved almost the whole choroid with the capillary layer spared in places (Fig. 2), but granulomatous tissue was present in the emissaria. The sclera was severely necrotic and was absent at the operative wound, where the choroid was exposed. The retina appeared thinned and highly degenerated.

The pathological diagnosis of the left eye was: (1) sympathetic ophthalmia, (2) postoperative scleral rupture, (3) retinal detachment, and (4) healed angle recession after blunt trauma.

Discussion

This case demonstrates sympathetic ophthalmia both clinically and histopathologically. The underlying cause of the disease may be traced to multiple injuries. First, the possibility of scleral perforation after blunt injury should be considered. Rupture of the sclera posterior to the insertion of the rectus muscles after contusion is not infrequent. Characteristic features of this entity are subconjunctival haemorrhage with chemosis, deepened anterior chamber, and hyphaema, as well as massive vitreous haemorrhage precluding fundus viewing and soft eye. Our patient did not show these features. Furthermore no evidence of healed scleral scar was identified during the first operation. However, even though multiple sections were examined, minute scleral perforation cannot be ruled out with certainty. In Stafford’s case report, after serial sections and thorough examination, an
initially diagnosed sympathetic ophthalmia after contusion injury was eventually proved to result from scleral perforation associated with uveal tissue incarceration.

Secondly, penetrating diathermy for release of subretinal fluid can theoretically be an aetiological factor in sympathetic ophthalmia by damage to the uveal tissue, but this has seldom been reported and has not been borne out by our clinical experience. We do not think this factor should be seriously considered in our case.

The third factor is involved in our patient's second operation to remove the irritating Teflon implant. The globe ruptured, with loss of vitreous and exposure of choroid. Bilateral granulomatous panuveitis developed 2 months after this operation; sympathetic ophthalmia was suspected and was later confirmed by histopathology. Because of the time interval and the overt perforating injury, we considered that the second operation probably caused the disease.

Samuels and Trowbridge reported some cases of sympathetic ophthalmia in which heavier infiltration occurred near the site of a perforating wound. Greatly thickened choroid with severe inflammatory reaction near the area of ruptured sclera in our case may further implicate the rupture of the eyeball at the second operation in the occurrence of sympathetic ophthalmia.

This case illustrates that, after heavy application of diathermy and resultant necrosis of the sclera, the eyeball may be perforated during a reoperation. The danger of sympathetic ophthalmia should be guarded against in these cases.

References

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