A CASE OF SYMPATHETIC OPHTHALMITIS

BY

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THIS disease is becoming rare and, in my experience in South Africa, extremely rare. I have seen two cases in white women occurring after operation and after the offending eye had been already excised. The last of these was twenty-five years ago. I have never seen a case in a coloured person, or in a native, despite the numbers of severe injuries which these people get, and where the advice to have the eye excised is often refused.

The present case presents some unusual features, among which one must count its remarkably fortunate result.

The patient was a fair haired girl, aged nine years, who was so unlucky as to contract gonorrhoeal conjunctivitis from a girl with whom she was playing, and who proved to be affected with vaginitis. Both eyes were affected, and when I saw her the right had already got a deep marginal ulcer. This perforated next day, March 21, 1926, leaving a large prolapse.

Two weeks elapsed before the eye was quiet enough to have anything attempted, but then under an anaesthetic I made a large conjunctival flap, removed the prolapsed iris, and stitched the flap over the hole. Unfortunately, as the first stitch was put in the lens capsule gave way, and most of the lens escaped. If I had had permission I should then have excised the eye. By the next week, however, it was clear that the flap was going to hold in place and the eye was quiet. I have more than once saved a pretty hopeless eye by this treatment. The eye remained soft, but this was attributed to the fact that probably a large drainage area had been made. The visible iris was normal in colour, there were no precipitates on the cornea, and, presuming on my immunity from sympathetic disease, I was not anxious.

The patient left hospital on April 22, but as she was leaving my Ward Sister said she thought the left eye had looked pink on the previous day. Ordinary examination disclosed nothing wrong, but to be on the safe side I had the patient brought to me next day and examined her with my slit-lamp. I was distressed to find that there were very fine precipitates on the cornea, cells in the aqueous, and a dewy endothelium. The child was sent back to hospital and the right eye excised on April 24.

While under the anaesthetic 200 c.c. of blood were removed for preparing an auto-serum and a dose of salvarsan was given. Atropine and dionine ointment was begun at once and continued all through. The auto-serum was used as recommended by Guirald

but after the sixth injection of 3 c.c. it became slightly septic and caused a temperature and a local erythema. A second supply was destroyed by overheating.

Meanwhile the pupil had dilated well and the precipitates, which at first increased considerably, grew less. The vitreous was very hazy, but one could see that there was congestion of the retinal veins ($V.=6/36$). On May 23, after a consultation, sodium salicylate was administered in large doses, as recommended by Mr. Werner. The salvarsan had been kept up once weekly. In spite of my fears, and a few temporary exacerbations, the eye kept surprisingly quiet, very little watery, and nearly free from pain. The lens was covered, except in the centre, with fine pigment deposited from the aqueous.

On June 9, I was cheered by finding that the patient read 6/9; on July 6, vision had gone up to 6/5. I found the aqueous practically cell-free and the precipitates were beginning to show absorption at their edges. This was eleven weeks after the disease was recognized, and such a result exceeded all our hopes. I saw her again in the beginning of November. There were still plentiful traces of old precipitates, but the pigment on the lens had mostly become absorbed, and there were only a few fine fibrinous deposits, probably marking the site of the early adhesions. Though the brunt of the disease fell on the choroid there were at no time visible spots of choroiditis, nor was the tension much diminished.

Where so many lines of treatment were used one cannot assess their values. Salvarsan was used throughout, but most of the improvement occurred during the time when the auto-serum was administered. The early improvement would, however, be the most noticeable in any case. The worst of the trouble was over before the salicylate was started. There can be no doubt that the most important factor was the recognition by the slit-lamp of the disease in its very early stage and the immediate removal of the exciting eye.

The excised eye was hardened in Bouin's fluid and sectioned in celloidin. The sections were stained with various haematoxylin stains, mainly Heidenhain. Pappenheim's stain was disappointing, possibly owing to the fixative.

Considering the fact that the eye had retained perception of light and had been nearly free from redness, the changes found were surprising. No part was quite unaltered while most parts were profoundly altered.

The retina was everywhere detached and the nerve head so much drawn forwards that at first I thought there was papilloedema. I have since found that in cases of detachment associated with inflammation this is common and may be due either to active

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Sympathetic ophthalmitis. Shows the conjunctival flap, the thickened iris, detachment of retina, and pushing forwards of optic nerve.

Section through optic nerve. Choroid spaced out in deeper layers, infiltrated in vascular layers.

pushing forwards of the retina by exudate or to a negative pressure in front.

The retina was unaltered and stained well and sharply by Heidenhain's method. Most of the vessels near the head of the nerve were surrounded by lymphocytes, but not for any distance up the nerve, nor did the changes reach far from the nerve into the retina.

The choroid showed typical changes. The deeper layers were spaced out through their entire length and the stroma pigment

Section of choroid showing nodules of lymphocytes. $\times 100$.

cells were very distinct. This, I think, was the result of the fixative causing contraction in the inner layers.

The inner layers were changed into a thick mass of lymphocytes arranged in confluent nodules. Among them were the nuclei of epithelioid cells and many small giant cells, with oval nuclei from six to a dozen in number. Both in the choroid and ciliary body one could see that the pigmented cells of the stroma, derived from the mesoblast, were undamaged. Their processes were intact and no pigment could be seen scattered outside them. The pigment of these cells is of course in very fine granules without any recognizable shape even with high magnification, whereas in the hexagonal pigment and its forward extension

over the iris the pigment granules are in two forms, large granules more or less rounded which lie near the bases of the cells and are found through the whole uvea, and fine rods which lie in the part of the cells next the rods and cones, and are found more sparingly beyond the retina. In the present case there has been great

Section through a lymphocyte nodule showing intact stroma pigment cells. $\times 200$.

damage done to the pigmented cells derived from the epiblast, especially in the front part of the eye.

The deposits of lymphocytes thinned off as one traced the uvea forwards, but continued without a break, becoming again much denser and thicker in the ciliary body and iris.

The angle of the anterior chamber was destroyed on one side by the prolapse, but was fairly normal though very narrow on the other.

The iris was surprisingly altered. During life the colour had been but little changed, though the surface was irregular. Sections showed that the tissue was greatly thickened, and that the pigment layers forming the posterior surface were completely

broken up and the pigment scattered. At only one spot near the intact angle of the anterior chamber was there a small continuous layer of pigment. The iris was adherent in part to Descemet's membrane; on the rest of the membrane no precipitates could be seen, agreeing with the naked eye observation. I think this was due to the shallowness of the anterior chamber, *ceteris paribus*, the amount of precipitates being a function of the amount of aqueous. The iris was changed into a mass of lymphocytes, epithelioid cells, and giant cells. Scattered all through were

Section through iris showing giant cells, nuclei of epithelioid cells, plasma cells, and lymphocytes. Pigment granules in giant cells and scattered elsewhere. $\times 300$.

remains of pigment, much of it extra-cellular and in large granules, but some ingested by the epithelioid cells and outlining their bodies. There were also many plasma cells. These never occurred singly but were in groups related to a blood-vessel. This was very evident near the surface of the iris where there were many new vessels, some not much more than a wall of endothelium. With Heidenhain's staining many of the lymphocytes showed an arrangement of their chromatin approaching very closely to that of the plasma cells.

In the ciliary region the damage to the pigment cells was also very great. The pigmented layer was thinned and there was loose pigment visible everywhere. Here again the granules were nearly all of the rounded type and on the whole seemed larger than those which occur normally. The unpigmented epithelium seemed to be

unusually prominent, but as was noted by Dr. McIlroy in her interesting paper (*Roy. Lond. Ophthal. Hosp. Reps.*, Vol. XVII), the cells seemed to be surrounded by an exudate which prevented a sharp view. Nucleoli were usually present, but the clear chromatin-marking seen in the epithelioid cells was either finer or less visible. Nowhere could these cells nor the nuclei of the pigmented epithelium be traced far away from their normal sites. Giant cells were less numerous than in the iris but much larger. They lay for the most part in the ridges of the ciliary

Giant cells from conjunctival flap. $\times 300$.

processes and contained often up to forty nuclei. The cell body was always shrunken from its surroundings, probably by the fixation effect, and pigment could always be found in it and in smaller granules than the pigment outside.

The interior of the ciliary processes contained lymphocytes, plasma cells, and numbers of epithelioid cells, probably derived from the stroma. Epithelioid cells were abundant in the outer part of the ciliary body away from the pigment. Most had elongated nuclei resembling those of the endothelial cells of the blood-vessels, but others had large oval nuclei and these I think came from the stroma cells. My sections did not indicate that epithelioid cells came either from the mesoblastic chromatophores or from the cells belonging to the anterior part of the retina.

Nowhere in any section could I find polymorphonuclear leucocytes, except in the blood-vessels, where their occurrence was curiously variable. Some vessels had their blood contents studded

with leucocytes while in others there were few or none. This is probably not related to the process at work in the particular case.

Another curious and disquieting finding was that not only did my conjunctival flap contain lymphocytes in nodules, but many sections showed large giant cells, so wide-spread had been the effect of the disease.

Since I have perforce been made familiar with the appearances found in sympathetic disease I have discovered similar changes in sections of an eye excised as probably dangerous last year. Unfortunately, there are no records on the case-sheet as to the history, etc., beyond the fact that it referred to a European male. Evidently I was lucky enough to excise the eye in time to prevent disease in the other eye, as no case of sympathetic disease has since been recorded.

It is probable that the reason for the case described being so insidious and free from redness, etc., was that it was a pure case of sympathetic disease without any endophthalmitis in the exciting eye.

CALCIUM DEFICIENCY IN THE BLOOD WITH REFERENCE TO SPRING CATARRH AND MALIGNANT MYOPIA

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FOR a good number of years I have been aware that some ocular conditions are associated with or caused by a deficiency in the amount of calcium in the blood, and I have treated cases empirically with good results, though I had not the means of ascertaining whether such deficiency really existed. I have now for some time been able to have this done through the kindness of Dr. L. Bosman, of the bio-chemistry department of the University of Cape Town, and it has been interesting to find that my expectations have been realised in all the cases where I was able to get the patient at a sufficiently early age.

The first cases to which my attention was drawn were sufferers from spring catarrh, and my early experiments were made with "afenil," a calcium chloride urea made by Knoll. I found that where the patients were young and the disease affected the pericorneal zone a cure could be effected in as little as three weeks by the giving of a 10 c.c. ampoule of afenil intravenously (one grm.) thrice in a week. The thickenings faded away and left only a narrow