OBSERVATIONS ON GLAUCOMATOCYCLITIC CRISES*

(POSNER-SCHLOSSMAN SYNDROME)

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THE syndrome of glaucomatocyclitic crises occupies a unique and important position among the many and heterogeneous types of glaucoma associated with uveitis. Its ominous onset, often highlighted by very high tension, is belied by the benign course it follows if treated with discretion. If the ophthalmologist is aware of this condition, he will be spared needless worry, and, more important, the patient may be spared needless surgery. In private practice, I have encountered eight cases, but, in spite of its relative frequency, the only paper I know of that adequately describes this clinical entity is that of Posner and Schlossman (1948), who gave the syndrome the apt name "glaucomatocyclitic crises".

CHARACTERISTICS OF THE SYNDROME

The syndrome consists of recurrent attacks of glaucoma associated with minimal evidence of anterior uveitis, and is characterized by a number of unusual features which place it in a special category:

(1) *The elevation of tension is out of proportion to the symptomatology.*—The patient may present himself with a tension as high as 75 mm. Hg (Schiötz) and a relatively white eye. Usually his only complaint is slight blurring of vision. Minor discomfort, rather than pain, is the rule. Unlike acute congestive glaucoma, this condition never produces nausea, vomiting, or marked loss of vision, although halos may occur if the tension is over 45 mm. Hg.

(2) *The pupil of the affected eye is larger than that of the unaffected eye.*

(3) *Even though the picture is not typical of congestive glaucoma, one does not think of uveitis until one examines the patient under the slit lamp.*—Here one always finds, within 48 hrs, cells in the aqueous or a few keratic precipitates. These are generally small, discrete, white, and relatively flat. An aqueous flare, if present, is not outstanding. Should corneal oedema render slit-lamp examination difficult, a drop of glycerine may be instilled, after local anaesthetization, in order to clear the cornea temporarily. The examination should be repeated for the first few days as the picture is changeable and may be entirely negative at first.

(4) *The attacks recur.*—A history of recurrent attacks was obtained in every one of my series of cases. In each patient the same eye was always affected.

(5) *A characteristic course is always encountered.*—The tension may remain elevated for as long as 2 weeks. While the cells in the aqueous are fleeting, it may take as long as a month before the keratic precipitates disappear. In a patient with a characteristic history these precipitates may supply the sole clue to the diagnosis.

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(6) *The prognosis is good despite repeated attacks.*—The vision always returns to normal and no field defects have been encountered. Optic atrophy or glaucomatous cupping has not been noted.

(7) *Strong miotics and mydriatics must be avoided.*—In my experience, weak eserine, or even 2 per cent. pilocarpine has resulted in ciliary spasm with pain. 10 per cent. neo-synephrine or 2 per cent. homatropine are the safest of the mydriatics, but even these may cause significant elevations of tension. The capriciousness of the response to therapy in any individual case is fairly typical of the syndrome.

(8) *Topical cortisone has given the best results.*—This neutral type of treatment, probably by its blocking action, allays ciliary irritability and effects recovery in the shortest period of time.

SPECIAL PLACE OF THE SYNDROME AMONG THE GLAUCOMAS

One cannot consider the cases of this condition as primary glaucoma even though they often respond to miotics, because of the almost invariable association with minimal cyclitic signs in every attack. On the other hand, one cannot dismiss them as cases of secondary glaucoma following uveitis, because of the unique picture they present. It almost appears as if the same aetiological factors are operative in both the glaucoma and the uveitis. Thus, it sometimes happens that in some attacks the glaucoma may precede the uveitis by a day or two, or may occur alone without the uveitis. On the contrary, attacks may occur in which the uveitis predominates with little or no rise in tension. These variable manifestations are matched by the capricious responses to treatment.

In classical secondary glaucoma one always encounters a marked iritis, often with posterior synechiae. In such cases mydriatics usually lower the tension. The mechanism appears to be the control of the iritis along with the breaking of synechiae. In the syndrome under discussion, synechiae are rarely seen and the elevated tension may outlast the cyclitis. Furthermore, in the vast majority of attacks, the pupil of the affected eye is dilated, which is the opposite of what happens in iritis.

Finally, the self-limiting character of the individual attacks, whether treated or not, is quite different from the course of an attack of secondary glaucoma due to cyclitis. These differences, in my opinion, make this syndrome unique, so that it stands in an intermediate, borderline position between true primary glaucoma and what we recognize as secondary glaucoma. We have encountered cases with recurrent attacks of acute primary glaucoma, subsiding without any treatment, cases which might verge in the direction of the syndrome under discussion. All these self-limiting types of glaucoma may have the same aetiology, possibly an allergic one.

Report of a Typical Case

R. B., 39-year-old housewife, first seen on January 26, 1949. Five days previously she had seen halos with the right eye and had consulted an ophthalmologist. He told her that she had glaucoma, prescribed pilocarpine, and said she might require operation if the increased tension did not respond to treatment. At the time of my first examination,

tension in the right eye was 35 mm. Hg (Schiötz) and in the left 26 mm. Hg. Although two keratic precipitates were noted, I did not recognize the true nature of her condition and told her to continue with the use of pilocarpine. The vision was 20/20 and the visual field was normal. Two days later the tension was 26 mm. Hg in each eye.

On May 26, 1949, despite pilocarpine therapy, she returned because of "trouble" in the right eye of one week's duration and halos for the past day. Vision in the affected eye was 20/25-3. Although the tension in the right eye was 60 mm. Hg, the eye appeared white. A few cells and fine keratic precipitates were seen. This characteristic picture, coupled with the previous history, made the diagnosis of the syndrome of glaucomatocyclitic crises apparent. The patient was advised to use 1 per cent. pilocarpine every 2 hours. The next day the tension was 37 mm. Hg and cells were still seen. By May 29, the tension in the right eye was 23 mm. Hg and in the left 26 mm. Hg. Fewer cells were noted. On June 1, no cells or keratic precipitates were observed and vision was 20/20.

In October, 1949, a similar, but milder, attack occurred. During the next year she experienced about half a dozen minor attacks which she treated, herself, with pilocarpine.

On November 29, 1950, she had another attack, with a tension of 43 mm. Hg, and many cells and numerous keratic precipitates again were found in the right eye. She used pilocarpine 1 per cent. every 2 hours, but this time she developed a severe ciliary cramp and oedema of the cornea. The vision was reduced to 20/50 and tension rose to 60 mm. Hg. The oedema was cleared with glycerine, and strangely enough, the cells and keratic precipitates were now found to be definitely diminished in number despite the continued elevation of tension. Pilocarpine was discontinued. Ten per cent. neosynephrine was instilled in the consulting room and she was given cortisone drops (5 mg. per ml.) every hour. The pain subsided within 8 hrs. By the next day the tension was 48 mm. Hg and there was no corneal oedema. On December 9, the tension was 28 mm. Hg, and a week later all cells and keratic precipitates had disappeared.

Several minor attacks which followed during the next month were helped by cortisone. At this time, early in 1951, it was discovered that the patient had a blood uric acid content of 7.1 mg. per 100 ml., suggestive of gout. Since she was overweight, she was placed on an obesity diet, and during the next 6 months only one mild attack occurred, followed, a few weeks later, by acute gouty arthritis, associated with slight discomfort about the eye.

COMMENT

The case described above was chosen for detailed presentation because so many of the classic features of the syndrome of glaucomatocyclitic crises were seen. The other cases that I have encountered were, in the main, similar, and do not require individual analysis, but certain special points deserve emphasis.

A possible allergic factor, which may be of aetiological significance, was demonstrated in four cases. Two patients suffered from hay fever. Another had repeated episodes of vasomotor rhinitis which often preceded the ocular attacks. The gout, present in the case reported, may, according to recent concepts, also be considered an allergic manifestation. Furthermore, the fact that the local use of cortisone gave the best therapeutic results, lends additional support to the concept of allergy as an aetiological factor.

All the cases showed unilateral involvement, with a dilated pupil in the affected eye, and tension out of proportion to the mild course of attacks. Blurring of vision was the most common presenting symptom.

The case of R. S., a man of 30 years of age, demonstrates how easy it is,

if slit-lamp examination is not performed, to misdiagnose the condition as acute glaucoma. Impressed only by the high tension, the ophthalmologist whom he first consulted instilled eserine into his eye and planned for an operation as the only alternative should this strong miotic fail to work. When I saw the patient a few hours later, he complained of severe pain, but this soon subsided after cortisone treatment was begun, and the tension then dropped quickly to normal.

Surgical Intervention.—The temptation to perform urgent surgery is a natural one when a high tension persists for more than a few days, even if one is aware of the syndrome and has encountered previous cases. Surgery however, is contraindicated for two reasons:

(1) Patients do not lose visual field or develop permanent visual impairment in this condition. In a case reported by Posner and Schlossman (1949) in which the tension hovered between 36 mm. and 90 mm. Hg for 2 weeks, a return to normal visual function finally occurred.

(2) While carefully performed antiglaucoma surgery may not harm the patient, the attacks in those cases which have come to my attention have continued to recur in the same manner as before surgery. Kornzweig (1951) followed a patient who had had an iridectomy performed in 1941 because of a diagnosis of acute primary glaucoma and records that since that time she had had numerous attacks of glaucomatocyclitic crises. Billet (1952) reports a similar lack of response to surgery. In the belief that the patient was suffering from primary glaucoma, two different ophthalmologists had performed an Elliot trephine and an iridencleisis, respectively. Nevertheless, the patient continued to have recurrent attacks which were later recognized to be typical of the syndrome. Douglas (1951) performed an iridectomy on his patient after the diagnosis of the syndrome was established, in the hope that the iridectomy would at least control the recurrent iritis, but 2 weeks later he had another attack.

SUMMARY

The syndrome of glaucomatocyclitic crises (Posner-Schlossman) should be regarded as a clinical entity, having a special place among the glaucomas. The fact that a condition exists, characterized by recurrent attacks of high tension entirely out of proportion to the associated cyclitis which has an essentially benign course, without resultant visual loss, and does not require intensive treatment or surgery, has not hitherto been emphasized. Once the ophthalmologist is aware of this syndrome the management of this sometimes alarming condition becomes relatively simple. Cortisone and weak solutions of pilocarpine ($\frac{1}{4}$ to 1 per cent.) appear to be the most efficacious medicaments. Surgery should not be attempted.

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