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SENIOR THESIS

RALPH H. FULLER.

1931
THE EYE IN DIABETES MELLITUS
THE EYE IN DIABETES MELLITUS.

The study of the eye and its diseases is one of the oldest and most specialized branches of medicine. Yet it must not be forgotten that the eye is intimately associated with the rest of the body and not to be regarded as an isolated organ (17).

Many systemic diseases result in ocular manifestations. Recognition of the evidences of systemic disease as they appear, often is the vital key to effective treatment.

The invention of the ophthalmoscope by Helmholtz in 1851 was therefore, not only an epoch in ophthalmology, but also an important event in general medicine (17). Since knowledge and practice in the use of this instrument has come to constitute a part of the preparation of every well-trained medical graduate, it has come to be not only a tool of the ophthalmologist but part of the armamentarium of the general practitioner, and especially of the specialist in internal medicine.

Diabetes mellitus is one of the systemic diseases with not infrequent ocular complications. (17) The frequency of ocular disease in diabetes has been variously estimated as from ten to twenty per cent of cases. (24)

This review of the ocular lesions occurring in diabetes is an attempt to study their significance as regards metabolism and pathology in the diabetic individ-
ual, and to draw some conclusions as to their value as diagnostic signs.

Refractive changes are found to occur in fifteen to twenty per cent of diabetic patients. (2)

In untreated cases there may be a sudden or gradual onset of myopia. The use of insulin often results in hyperopia. (3) Sudden or frequent changes in refraction are very suggestive of diabetes. Since diabetes is the only disease causing sudden hyperopia its presence alone is diagnostic. (21)

There have been various theories as to the causation of refractive changes in diabetes. It has been explained on the basis of an index ametropia. In index ametropia the index of refraction of the media as a whole is above or below that of the normal media. It generally is dependent upon disturbed nutrition of the lens. (25)

The theory of Duke Elder has, however, been given the widest acceptance. (10) (21) (3) (11) Duke Elder believes that in untreated diabetes, a raise in the sugar content of the blood results in a loss of sodium chloride from the blood, and an inflow of fluid from the tissues into the blood, so that its osmotic pressure is made abnormally low. As a consequence water passes from the blood into the lens causing it to swell and become myopic. Again, when for any cause the blood sugar is lowered, as when insulin is given, the osmotic pressure of the blood becomes high so that the lens gives up water and becomes
flattened. A lens that is abnormally flat results in hyperopia.

Cataract has been frequently thought of as the foremost ocular complication of diabetes. It probably does not deserve this position. In a comparison of 292 diabetics with 900 non-diabetics between the ages of 56 and 60 a slightly greater incidence of cataract was found in diabetic individuals. Between the ages of 60 and 80 cataract was found more common among non-diabetic individuals. (11)

Cataracts as found in diabetic individuals may be divided into two groups. Those occurring in diabetic patients above 40 years of age are indistinguishable clinically from ordinary senile cataract. (11)

Senile cataract occurring in diabetes is common, but its incidence is little if at all increased in diabetes. (29) On the contrary a case is reported diagnosed as senile peri-nuclear cataract in a man of 56. He later developed diabetes and the lenticular opacity was then found to have disappeared. (27)

What may be called true diabetic cataract is rare. (29) A diabetic cataract develops rapidly; when cataract develops slowly it is more likely a senile cataract. (25) Diabetic cataract may occur at any age, (11) but is more apt to occur in young patients late in the disease and to be bilateral. It is characterized first by fluid vacuoles under the capsules of both lenses with
progression to complete opacity in a period of a few weeks to six months. (11) It is first a central opacity and later becomes per-nuclear. (13) The lens then presents a dirty grey homogeneous appearance (2). Microscopically a diabetic cataract can be distinguished by the fact that the pigment not only in the neighborhood but throughout the entire eye is loose and floats off easily. (13)

One theory of the etiology of true diabetic cataract is that suggested by the observation, that under the influence of light, acetone causes easily soluble albuminous bodies to be changed into poorly soluble globulins. It is pointed out that this may account for, first, sclerosis of the lens, then presbyopia, and later opacity. (13)

Others think that the remarkable changes in refraction occurring in diabetes, are a significant factor in cataract formation. When the sugar content of the blood is raised, its osmotic pressure is lowered and if as a result enough fluid collects in the lens, permanent changes in the form of cataract may develop. (11)

Studies of cataract extractions in diabetic patients show that diabetes, and especially low sugar tolerance, prolong the period of convalescence. (25) But it seems to be the general consensus of opinion that if acetone is not found in the urine and if the blood sugar is lowered to a normal minimum cataract extractions may be done with rela-
lative safety. (13) Insulin administration seems to tend to cause hemorrhage, and should be withheld for a period of about four weeks following the cataract extraction. (2)

Lipaemia retinalis was first described by Heyl in 1880. (15) Other descriptive terms have been intracellular lipaemia, and lipaemia angio-retinalis. (19) (26) It occurs in no disease except diabetes uncomplicated by treatment. (2) (6) (22) It is said by some to be the only lesion of the fundus which is pathognomonic of diabetes. (5) The condition is of rare occurrence; only about forty-six cases have been reported in the literature. (19) (26) It is confined largely to young diabetic individuals, probably because of their relative inefficiency in fat metabolism (6) (22), and usually occurs late in the disease. (2) In almost every case, the patient is seriously ill, either in a comatose or pre-comatose state. (20) It does not occur except in acidosis (22) (6).

Lipaemia is accompanied by few signs and symptoms. Aside from a direct examination of the blood there is little evidence of the disease. (22) (6). The vision remains normal. (2)

The ophthalmoscopic picture, is however, very characteristic and specific. The arteries and veins are very like in color and are indistinguishable in advanced cases. They appear enlarged, flat, and ribbon-like, with an absence of the light streak. The color is variously described as
milky, salmon pink, ivory, or sometimes silvery white.

(2) (6) (22) (19) (26) The retina itself is not abnormal in appearance. Hemorrhages are uncommon. (25)

Lipaemia occurs when the faulty fat metabolism in diabetes allows a collection of lipoid in more than normal concentration in the blood. The normal blood lipoid volume is approximately 0.4 to 0.5% (9) The lipoid content must approach a value of 3.5 per cent before the retinal signs are evident. (6) (22) The lowest levels at which lipaemia retinalis have been reported are, in one instance 3.5 per cent, (29) and in another 4.0 per cent. (22) The highest blood lipoid estimation values, recorded in lipaemia cases, are in one instance 48.0 per cent, (3) and in another 26.25 per cent. (6).

The milky appearance of the retinal vessels is explained by some, as entirely due to the increased lipoid content of the blood. (2) Others believe that due to the lower specific gravity of the plasma lipoid matter it adheres to the walls of the vascular lumens, so that the red cells are forced to the center of the blood stream. (5) Ordinarily the choroid vessels appear flat and diffused, as the light reflected from them passes thru the retina with its pigmented epithelial layer a diffusing medium. The retinal vessels lying on the surface of the retina, light is reflected directly from the central axial stream without having to pass thru any but transparent media. When, however, the central axial stream is surround-
by a plasma cream like in appearance the effect of a ground-glass instead of a transparent envelope is produced. By this the central light streak is lost as well as the normal cylindrical appearance of the vessels.

(20)

A finding of lipaemia retinalis in the era before the discovery and use of insulin necessarily indicated a very poor prognosis. (24) Now, with modern anti-diabetic therapy available, the occurrence of lipaemia retinalis is not seen to alter the prognosis of the diabetic case in which it occurs. (6)

A close association between lipaemia and xanthoma, has been pointed out. (9)

The subject of retinitis, as found occurring in diabetes has occasioned a great deal of controversy. Because of its rare occurrence in individuals less than thirty-five years in age, and since it is often found associated with hypertension and with albumin-urea many have doubted the existence of true diabetic retinitis. (2) Others feel that true diabetic retinitis occurs presenting a typical ophthalmoscopic picture, and as the most frequent ocular manifestation of diabetes. Some report its occurrence in 20 to 30 per cent of cases. (5) (2) (20) (24) (14) (7)

Wagener and Wilder (29) say that retinitis does not occur in uncomplicated diabetes, even if severe. They consider that the retinitis of diabetes is due to arteriosclerotic changes, but that the retinitis resulting from
the arterio-sclerosis of diabetes is so modified as to
present a picture that is different from that due to ar-
terio-sclerosis of other than diabetic origin. In a study
of forty-four cases it was observed that retinitis did not
occur in severe diabetes but always in mild, easily con-
trolled cases. Practically all presented other evidences
of vascular disease, and were within the age group of vas-
cular degeneration.

It seems true that in diabetes retinitis is seen
that if not a true diabetic retinitis is so far removed
from the type of renal retinitis as to be practically a
distinct entity (14). The ophthalmoscopic examination of
the fundus shows a characteristic inflammation of the
central area of the retina, with small bright spots, (2)
often called white spots or plaques. The plaques are dis-
tinctly outlined and sharply defined. They are arranged
in groups about the macula and optic nerve. (2) (5)
(7) (24) (17) Usually there are punctate hemorrhages.
The characteristic hemorrhages are round, very small and
not easily seen since they occur in the deep layers of the
retina. (8) (20) The optic nerve is not swollen, nor is
there edema of the retina, as in renal retinitis. (2)

While discovery of albuminuric retinitis in a ne-
phretic points to an expectation of life of not longer
than three years and often less than one year — this is
not true of diabetic retinitis; here the prognosis as to
life is relatively good.

Some patients retain good sight for years and even improve as the general diabetic condition is controlled. The typical case of diabetic retinitis is often followed by a series of hemorrhages into the vitreous with a consequent very unfavorable prognosis as to vision -- these patients finally lose their sight even when the general health is good. (7)

Experience has shown retinal lesions in diabetes to occur late in the disease, usually after other serious complications have become manifest. Hence diabetes is not often first diagnosed by the discovery of a typical retinitis. (24)

Recurrences of ecchymosis, or sub-conjunctival hemorrhages, suggest diabetes or other systemic vascular diseases. (25)

Retro-bulbar optic neuritis occurs in about ten to twenty-five per cent of diabetic cases. It may occur in any age individual. (2)

Usually the first complaint is of misty vision or of a dazzling which is worse in bright light and less on cloudy days or in the dusk. There is a gradual loss of central vision and therefore of ability to do fine work or read small print. (25)

No fundus changes can be seen until very late, then some degree of atrophy of the disk may be seen. (3) (24) (17)
The perimeter often reveals a central scotoma for form and color. The scotoma consists of an enlarged blind spot. A blind area that covers both the blind spot and point of fixation is pathognomonic of retro-bulbar optic neuritis. The blind spot is usually horizontally oval.

The pathology is an inflammation of the optic nerve between the eye and the optic chiasms. There is usually an axial neuritis—an involvement of the fibers supplying the macular region. The neuritis is thought to be due to hemorrhages, or to toxic changes in the nervous tissue.

The prognosis of retro-bulbar optic neuritis in diabetes depends upon the course of the disease. Too often the neuritis is accompanied, and sometimes overshadowed by hemorrhages and exudates in the retina. (25)

Several cases have been reported of retinal detachment following the administration of insulin in diabetes. (1) (18) The explanation of this occurrence, like that of diabetic refractive changes, is thought to lie in a disturbance of the electrolytic and osmotic processes in the blood. In hypo-glycemia, with an increased osmotic pressure of the blood, a greater amount of fluid may be thrown from the choroidal vessels into the sub-retinal space, resulting in a detachment of the retina. (1)

Iritis occurs rarely as a complication of diabetes. (25) (4) (17) When it occurs, the iritis is not due to diabetes but to other factors usually focal infections. (4)
Ocular palsies occasionally occur. The commonest is ptosis of the upper lid due to involvement of the third nerve. Disturbance of the fourth nerve may result in an internal strabismus. (2) (17)

Paresis of accommodation may occasionally be found, with the generalized debility of advanced diabetes. (2)

Hypotonia bulbi is held by many observers to be the rule in diabetic coma due to acidosis. (11) (23) (28)

P. Krause reports a total of sixty cases with soft-eye-ball a constant finding and considers it a diagnostic sign of specific value. Other men deny its constancy in coma of diabetic acidosis, and it has been reported in cases of coma other than diabetic coma, and even in the coma due to hypo-glycemia following insulin injection. (30)
SUMMARY

It should be remembered that many people ignore the manifestations of systemic disease for long periods of time. This is especially true of diabetes because it is a disease not productive of characteristic pain or even discomfort. When, however, for any reason there begins to be an interference with vision, people are apt to lose little time in consulting an ophthalmologist or general practitioner.

Unless the physician recognizes the underlying etiology, he is distinctly handicapped in the treatment of the local manifestations.

As ocular signs at least definitely suggestive of diabetes the physician should keep in mind:

(a) frequent changes in refraction especially sudden hyperopia,
(b) rapidly developing bilateral cataracts in young people,
(c) the characteristic picture of lipaema retinalis which is pathognomonic of diabetic acidosis,
(d) the typical and characteristic ophthalmoscopic picture of true diabetic retinitis, and
(e) hypo-tonia bulbi which has been found constant in a large proportion of diabetic comas due to acidosis.
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