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BY

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RETINITIS IN BRIGHT'S DISEASE.

THE occasional occurrence of amblyopia in the course of Bright's disease has been observed ever since this malady has been diagnosticated and studied; and although Bright himself, in his first paper on the subject (1827*) gave no eye symptoms beyond œdema of the eyelids, he in 1836† recorded eleven cases, "illustrating some of the more insidious attacks which attend a fatal termination," in four of which failure of vision, coming on at a period varying from six weeks to six days before the fatal termination, was one of the prominent symptoms. In 1840‡ the same author published twenty-four cases, with defective vision in three of them, and in 1843§ Bright and Barlow reported thirty-seven cases of the disease accompanied by uræmic poisoning, with five cases of defective vision among them. It was not, however, till Türck‖ in 1850 demonstrated the occurrence of splotches of fatty degeneration in the retina, that the anatomical changes accompanying it were known; and six years later we find a more exhaustive study of the same subject by Virchow.¶ The first ophthalmoscopic description of these changes was published by Heymann** in 1856—just five years after the discovery of the ophthalmoscope by Helmholtz, and in 1859 Liebreich†† added still further to our

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* Reports of Medical Cases, by Richard Bright, M.D., F.R.S., vol. i, London, 1827.

† Guy's Hospital Reports, 1836, pp. 338–380.

‡ Guy's Hospital Reports, 1840.

§ Guy's Hospital Reports, 1843.

‖ Zeitschrift der Gesellschaft der Wiener Aertze, 1850.

¶ Virchow's Archiv, vol. x, pp. 170–193.

** Archiv f. Ophthalmologie, Bd. ii, part 2, pp. 137–150.

†† Ibid., Bd. v, part 2, pp. 265–268.

knowledge of the subject, illustrating his description with a colored lithographic plate. Since that period the literature of the subject has grown to such proportions that the mere enumeration of the various articles and works on the subject would require of itself many pages, and the reader will find at the end of the chapter a reference to several works where an extended bibliography is given.

Symptoms and Description.

The retinitis of Bright's disease presents very various aspects, not only in different cases but also in different stages of its development in the same case, and distinguishes itself mainly from other forms of inflammation of this nervous sheet by its marked tendency to fatty degeneration. As seen by a specialist or at an eye hospital the disease usually presents a type quite different to that predominating in the wards of a general hospital. In the former the blood-poisoning seems to fall with peculiar intensity on the nervous system, and the patients come complaining of headache, dizziness, and dim vision, these being the only marked symptoms of the malady, while the anæmia, dropsy, and other symptoms are either absent or present in so slight a degree that the patients have not supposed themselves suffering from any constitutional malady or needing advice from their medical attendant. The retinal changes in such cases are usually very extensive, and those in the cerebrum would possibly be found equally developed if we had only as accurate a method of investigating them. In the wards of a general hospital, however, we have a much better opportunity to study the development of the retinitis, and it is there most frequently encountered among those suffering from marked dropsy and cardiac disease, with transparent waxy skins, whose appearance indicates at a glance how seriously their nutrition has been impaired by the ravages of the disease. In these we often see only a few white splotches in the retina, either with or without hæmorrhages, and occasionally only a slight atrophy of the optic disk due to a previous retinitis. When the individual lives and is not markedly relieved by the rest and

treatment adopted, we frequently have an opportunity of seeing the development to a greater or less degree of the typical form of the affection.

Typical Cases.

In typical cases the retinal changes commence with slight œdema of the disk and surrounding retina, with a few irregular white splotches and striated hæmorrhages in the fibre-layer. We see the white patches multiply and extend, mostly within an area of two or three disk diameters from the optic entrance, until in high grades of the affection they coalesce and form a broad zone around the disk, which is itself swollen and prominent, its outlines being hidden by the opaque nerve-fibres which diverge from it. Fresh hæmorrhages occur from time to time, and are striated when in the fibre-layer, and of irregularly rounded outline when invading the deeper portions of the retina. They are usually either entirely absorbed or leave behind them a fatty clot, which adds an additional white splotch to those already existing in the retina. At times they leave spots of black granular pigment as the marks of their previous presence. At the same time irregularly linear or quadrate white splotches are developed, which radiate from the fovea centralis throughout the macular region. These were formerly supposed when present to be absolutely characteristic of the disease, but it is now asserted by several good observers that similar appearances have been seen in the neuro-retinitis caused by brain tumor or by basilar meningitis, where there was no accompanying disease of the kidney. (Gräfe, A. f. O., xii, 2; Schmidt and Wegner, A. f. O., xv, 3; Magnus, Ophth. Atlas, Taf. vi, fig. 2; Leber in Gräfe and Saemisch, Bd. v, p. 581; Carter, Diseases of Eye, p. 382, Am. edit.; H. Eales, Birmingham Med. Review, January, 1880, p. 47.)

Changes in the Color of the Fundus and of the Retinal Blood Columns.

In many cases occurring in the last stages of the disease a remarkably yellowish tint of the fundus is observed, together

with decided alteration in the color of the blood columns in the retinal bloodvessels; the blood in the arteries being too yellow, and that in the veins presenting too little of its usually pronounced red-purple tint; in short, a state of affairs approximating in some degree to that which we find in cases of pernicious anæmia.

Curability.

Where a patient with albuminuric retinitis is admitted to hospital, and under the treatment adopted, the general health for the time improves, we not infrequently see the vision improve with it, and the retinal changes become regressive and partially disappear. I have several times seen patients with a vision of only 20–CC on admission, in a month improve so that they had a vision of 20–L, and could once more read ordinary print and perform for a time their usual duties. I have, however, never witnessed a complete disappearance of all retinal changes except in acute cases, and can well recall a case of albuminuric retinitis occurring during pregnancy, where, three years later, there was no trace of the disease beyond a slight atrophy of the optic disk.

Forms of Kidney Disease in which it may be Developed.

This form of retinitis may be developed (as has been abundantly proved by careful autopsies) during any form of Bright's disease, either with the enlarged mottled kidney of acute parenchymatous nephritis, or with the large white kidney, the amyloid kidney, or the cirrhotic kidney of chronic disease. In the vast majority of cases, however, it is developed in the later stages of the last-named form of disease, and seems to be in some way related to the blood-poisoning caused by it.

Exceptional Forms of Albuminuric Retinitis.

While the foregoing description gives a fair picture of the development of the disease as ordinarily met with, nevertheless we may encounter other varieties. Thus I have seen cases which, at the start, could not be diagnosticated by the

ophthalmoscope from cases of retinal hæmorrhage from other causes (and Magnus has published similar cases), and there are recorded other cases where the only changes seen in the fundus oculi were a pronounced choking of the disk similar to that with which we are familiar in cases of brain tumor. (Magnus, Samelsohn, Gowers.)

Morbid Anatomy.

We find serous swelling of the disk and surrounding retina, especially of the neuroglia; and in the fibre-layer nests of what are usually described as varicose or sclerotic hypertrophy of the nerve-fibres. These swollen fibres are, when slightly developed, spindle-shaped, at other times so dilated that, with their large nuclei, they much resemble ganglion-cells, and they were described by Virchow as sclerosed ganglion-cells. Owing to their position, which is frequently just below the membrana limitans interna, and to the fact that their processes can readily be demonstrated to be continuous with the nerve-fibres of the fibre-layer, they are now classed by most authors as varicose hypertrophies of the nerve-fibres. This affection is, however, by no means peculiar to this disease, but is not infrequently developed in other forms of neuro-retinitis.

We find also fatty splotches, consisting of large numbers of compound granule-corpuscles, usually either in the nerve-fibre layer or in one of the nuclear layers. The radial connective tissue fibres (fibres of Müller) also present numerous minute fat-drops, which, when massed at their upper end, cause the well-known radiating white stripes on the macular region.

The bloodvessels of the retina exhibit dilatation of the veins and capillaries, with fatty degeneration of their walls, often only of their adventitia, and also so-called sclerosis, a transparent thickening of their walls, which resembles amyloid degeneration, but does not respond to the action of iodine. Hæmorrhages are almost invariably present, either striated, and more or less linear in shape between the retinal fibres, or in less regular masses in the outer retinal layers, or in the vitreous humor. The choroid often exhibits changes in its bloodvessels

similar to those described in the retina, especially sclerosis of its capillaries, with fatty degeneration of their endothelium. As has been already mentioned, these changes in the retina are apt to be developed in the later stages of chronic disease of the kidneys, and therefore correspond to the period at which we find marked cardiac hypertrophy, and it has been supposed that this was essential to their development. Thus Traube claimed that the hæmorrhages arose solely from this cause, while he inclined to the belief that the other changes were due to the retention of urea. It is evident that the greater the force of the blood-current the more readily would it rupture the degenerated bloodvessels, and we must therefore assign to hypertrophy of the heart an important rôle in the production of retinal hæmorrhages; but the fact that they are found in acute cases where no cardiac hypertrophy has had time to develop, and that many autopsies are on record where there was no cardiac hypertrophy, proves that it is not an essential factor of their production. The fact also of the abundance of retinal hæmorrhages in cases of pernicious anæmia where there is no increase of the blood-pressure, points to the same conclusion.

Statistics.

The proportion of cases of Bright's disease in which albuminuric retinitis is developed has been variously stated by different authors. Bright himself has not reported his cases with a view to determining this point, but the clinical record of them is so complete that we can readily do so. Thus we find in the four papers previously referred to records of 95 cases, in 12 of which failure of vision is recorded (12.62 per cent.). In these as in all other preophthalmoscopic data we are fairly entitled to consider the real ratio as far higher than that reported, because marked retinal changes frequently exist without decided impairment of vision, which is only necessarily interfered with when the region of the yellow spot is attacked. Frerichs* gives 10 cases of retinitis in a total of 78 cases, a per-

* Frerichs, quoted by Leber, in Gräfe and Saemisch, vol. v, p. 585.

centage of 12.82. Galezowski* gives 47 out of 154 cases (30.15 per cent.), and quotes Lecorché as giving 62 cases out of 286, or 21.71 per cent. Wagner† gives 12 out of 157 cases, or 7.64 per cent. He rejects, however, six cases of retinal changes, which, in his judgment, were not sufficiently characteristic, and which would, if accepted, raise the percentage to 11.46. Mr. H. Eales,‡ the most recent writer who has examined any considerable number of cases with a view to determining the frequency of retinal disease in them, gives 100 cases, in which 28 had retinal changes, and 3 alterations of the optic disks. I regret that my own researches throw so little light on this subject; but although I have carefully examined a considerable number of cases of albuminuric retinitis, I at first devoted my attention exclusively to the mode of development of the affection, its various forms, and the kind of kidney disease accompanying them, and failed properly to note the negative cases in which there were no retinal changes. For an opportunity for studying these cases, I am much indebted to my friend Dr. J. H. Hutchinson, who has for years placed his wards at my disposal, and also for similar courtesies to Drs. W. Pepper, J. Tyson, and J. M. Da Costa. In the last 41 cases which I have examined, taken as they occurred in the wards of general hospitals, I found 11 cases of decided retinal change, such as œdema with white splotches and hæmorrhages, and have not counted several cases of slight degrees of atrophy of the disk possibly due to foregoing changes. This would give 26.82 per cent; but the number is too small to allow proper conclusions to be drawn from it, and can only be of value as a contribution to the total statistics of the subject.

Treatment.

Rest in bed with moderate purgatives and diaphoretics; in short, the remedies usually prescribed for the constitutional

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* Galezowski, L'Union Médicale, 1873, pp. 924–928.

† Wagner, Virchow's Archiv, Bd. xii, pp. 219–271.

‡ Eales, Birmingham Medical Review, January, 1880, pp. 35–52.

treatment of the disease are the most effective means of clearing up such cloudy retinæ, and I know of no drug or local application to the eye which acts in any degree as a specific.

Uræmic Amaurosis.

Uræmic amaurosis is much more rarely encountered than is albuminuric retinitis in the course of Bright's disease. It is rapid in its development and in its subsidence, is without retinal changes, and the blindness is evidently due to some transient affection of the cerebral centres. It is, however, occasionally developed in cases in which albuminuric retinitis already exists.

Bibliography.

By consulting the following-named books the reader can find a full discussion of the subject and an extended bibliography, while the atlases mentioned will afford good pictorial representations of the various forms of the disease:

Allbutt, Use of the Ophthalmoscope in Diseases of the Nervous System and of the Kidneys. London, 1871.

Leber, in Gräfe and Saemisch's Handbuch der Augenheilkunde, vol. v. Leipzig, 1877.

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Liebreich, Atlas d'Ophthalmoscopie. Paris, 1863.

Jaeger, Ophthalmoscopischer Hand Atlas. Wien, 1869.

Magnus, Die Albuminurie in ihren Ophthalmoscopischen Erscheinungen. Leipzig, 1873.