COMMUNICATIONS

CENTRAL AREOLAR CHOROIDAL SCLEROSIS*

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IN an earlier study (Sorsby, 1939) attention was drawn to the genetic nature of central choroidal sclerosis, a condition known in the literature as central senile areolar choroidal atrophy. An account was given of the occurrence of this condition in two brothers, aged 59 and 63 years respectively (Figs 1 and 2, p. 135). Both patients gave a history of visual disturbance beginning at about the age of 20, and worsening progressively. As no other members of the family were affected, there was no opportunity of studying the appearances of the disorder in the earlier or later stages, nor could anything definite be said as to the mode of inheritance. Four further families are now reported, considerably extending the range of ophthalmoscopic appearances—supported in one case by histological material—but leaving the mode of inheritance still undecided.

Case Reports

(1) B...n Sibship (Pedigree Chart I; Figs 3 and 4).

Two sisters, the sole survivors of a sibship of nine (whose parents were not related) were seen and found to be affected.

PEDIGREE CHART I.—B...n sibship.

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FIG. 3.—Left fundus of Mrs. Sarah Cl., aged 70 (B II, 7).

FIG. 4.—Left fundus of Miss Elizabeth B., aged 72 (B II, 6), sister of B II, 7 (Fig. 3).

(i) *Mrs. Sarah Cl.* (B II, 7), was first seen in February, 1946, at the age of 70 (Fig. 3).

Visual acuity: 1/60 in the right eye, hand movements in the left.

Her vision had been poor since about the age of 20, and had deteriorated steadily, especially in the last 3 or 4 years. Refraction: emmetropic. The fundi show exposure of the choroidal vessels over the central area, with some evidence of sclerosis (Fig. 3). When the patient was seen again in April, 1948, there was no substantial change.

(ii) *Miss Elizabeth B.* (B II, 6), sister of the above and 2 years older, showed the same type of central lesion (Fig. 4).

Visual acuity: 1/60 in each eye. Refraction: right - 2.0 D. cyl. at 120°, left + 1.0 D. sph. $\ominus$ + 1.50 D. cyl. at 60°.

The fundus lesion was rather smaller than in B II, 7, but evidence of sclerosis was more marked. Here, too, there was a history of failing vision from about the age of 20. When she was 35 she had been seen by the late Reginald E. Bickerton, who found central choroiditis. When she was re-examined in April, 1948, and in April, 1951, no substantial change was seen, except that the choroidal sclerosis was more marked in the area involved, which itself was no larger.

In neither case was there any history of nightblindness. The three children of B II, 7—a daughter aged 48 and two sons aged 45 and 44 (B III, 12, 13, and 14)—were examined and found normal, but the collateral branches of the family proved difficult to follow up. A reputedly affected niece aged 39 (B III, 8) was found to be a high myope with extensive fundus changes difficult to interpret. Her six living sibs (B III, 1–5, and 7), aged 41 to 62, were reputed normal.

(2) C . . . n-C . . . I Sibship (Pedigree Chart II; Figs 5–9).

The affected sibship originally consisted of seven members, one of whom (C III, 6) died when young. The parents were not consanguineous. The mother is recorded to have had bad sight. The six survivors were seen and five found to be affected (C III, 1, 2, 3, 4, and 5). None of the affected members who were aged 58, 56, 54, 52, and 50 respectively complained of nightblindness. The eldest (C III, 1) was unconscious of eye trouble; C III, 2 had been registered as

FIG. 5.

FIG. 7.

FIG. 8.

FIG. 9.

FIG. 13.

FIG. 14.

FIG. 5.—Right fundus of Mrs Bertha C., aged 56 (C III, 2).

FIG. 8.—Right fundus of Miss Esther Co., aged 60 (C III, 1), sister of C III, 2 and 5 (Figs 7 and 5).

FIG. 13.—Left fundus of Mrs. Charlotte Bu., aged 58 (Br II, 3).

FIG. 7.—Right fundus of Robert Co., aged 50 (C III, 5), proband, brother of C III, 2 (Fig. 5).

FIG. 9.—Left fundus of Miss Helena Co., aged 54 (C III, 3), sister of C III, 1, 2, and 5 (Figs 8, 7, and 5).

FIG. 14.—Left fundus of Mrs. Gladys Br., aged 30 (Br III, 3), proband, daughter of Br II, 3. (Fig. 13).

blind for some 2½ years, her sight having begun to fail some few years before; C III, 3 suspected that she might have some trouble; C III, 4 had had eye trouble for some indefinite time; C III, 5 (the proband) had almost full vision in each eye.

Two of the five, therefore, showed the fully developed picture, but vision was severely affected in only one, and three who were in the earlier stages of the affection showed only mild visual disturbances. It should be noted that there is no direct chronological sequence. The appearances in this sibship in order of severity were as follows:

- (i) *Mrs. Bertha C.* (C III, 2) aged 56, certified blind (Figs 5 and 6).

Visual acuity: counting fingers in each eye; myopia of about 8 D.

She first noticed poor sight at the age of 12 (but this probably represents the onset of her myopia), and became conscious of serious deterioration of vision at about the age of 52. The fundi show myopic crescents and a typical central areolar choroidal sclerosis surrounded by fine pigmentary mottling. She died suddenly in December, 1951, from coronary thrombosis, and the *post-mortem* findings are the subject of a separate communication in this issue (Ashton, 1953, p. 140). Her two sons (C IV, 1 and 2) aged 26 and 21, are normal.

- (ii) *Robert Co.* (C III, 5), aged 50, proband (Fig. 7).

Visual acuity (with correction); 6/9 in the right eye and 6/6 pt. in the left. Refraction: -1.50 D. each eye.

FIG. 6.—Left fundus of Mrs. Bertha C. (C III, 2). Her right fundus is shown in colour (Fig. 5). Both fundi in this patient are shown because the two eyes are the subject of a pathological report (Ashton, 1953, p. 140 of this issue of the *B.J.O.*)

He has been conscious of deterioration of vision during the past 8 years. His fundus lesions are not markedly different from those of his sister. When he was seen again 18 months later in April, 1951, his vision with correction had declined to 6/36 in each eye.

(iii) *Miss Esther Co.* (C III, 1), aged 60 is not aware of any eye trouble (Fig. 8).

Visual acuity: 6/12 in each eye.

(iv) *Miss Helena Co.* (C III, 3), aged 54 has recently noticed some failure of the left eye (Fig. 9).

Visual acuity (with correction): 6/9 in the right eye and 6/24 in the left.

Both central areas, of the left more than the right, are somewhat oedematous and show exudative reactions. The retinal vessels are normal.

(v) *Miss Kitty Co.* (C III, 4), aged 52 lives in New York and is reported by Dr. Milton Berliner to show central lesions.

Visual acuity (with correction): 20/70 in the right eye and 20/50 in the left.

As can be seen from Pedigree Chart II, a brother and sister, children of first cousins (C III, 8 and 9)—members of a sibship of five (C III, 8–12) all seen by Dr. Milton Berliner—show mild central changes of a doubtful character; their three children (C IV, 4, 5, and 6) aged 22, 19, and 7 years, are normal. The father (C II, 3) of these doubtfully affected individuals, who is himself a cousin of (C II, 1) the father of the collateral branch, is blind from glaucoma and has no fundus lesion.

(3) H . . . s Sibship (Pedigree Chart III; Figs 10–12).

The parents were first cousins and there was an indefinite history of eye trouble in the father's mother. The sibship consisted of six members. H II, 4 died at the age of 48 from carcinoma of the gall bladder, having entered a blind home at the age of 32 on account of trouble reputed to date from about the age of 15. H II, 5 died from tuberculosis at the age of 30 with no history of eye trouble. The proband (H II, 2) was aged 65, and in her case the diagnosis of central areolar choroidal sclerosis was not made until her younger brother (H II, 3) was found to show a typical lesion. The four survivors are apparently all affected:

(i) *Arthur H.* (H II, 3), aged 63 noticed difficulty in reading at the age of 36, and this has become progressively worse. He has no nightblindness (Fig. 10).

FIG. 10.—Left fundus of Arthur H., aged 64 (H II, 3).

PEDIGREE CHART III.—H . . . s family.

Visual acuity: hand movements in the right eye at 1' with $\bar{c}$ -2.5 sph. $\ominus$ -1.0 cyl. at 110°; in the left eye it is 6/60 with $\bar{c}$ 1.75 sph. $\ominus$ -2.0 cyl. at 100°.

Fields: right not practicable, left to 10/1,000 white shows peripheral constriction marked on the temporal side and joining up with the blind spot, and a central scotoma.

Colour vision: no perception of any colour on Ishihara test.

General health good except for "gastric trouble".

In the left fundus (Fig. 10), the discs are pale with shallow cupping and atrophic crescents, the retinal vessels are attenuated, and an areolar patch of choroidal sclerosis is seen in the central area with sharply defined edges. There is some generalized choroidal sclerosis peripherally with a few streaks of pigment, some in the shape of bone corpuscles.

Two of this patient's four sons (H III, 1 and 3) aged 34 and 30 were found to be normal; the two other sons (H III, 2 and 4) aged 31 and 27 could not be seen but were reputed normal.

(ii) *Miss Edith H.* (H II, 2) aged 65, proband, had had poor vision for many years. She had recently noticed a black spot floating in front of the left eye, but did not complain of nightblindness.

Visual acuity: 3/60 in the right eye with $\bar{c}$ +3.5 sph. $\ominus$ +2.0 cyl. at 125° = 6/60; in the left eye it is 2/60, with $\bar{c}$ +3.5 sph. $\ominus$ +1.75 cyl. at 70° = 6/60.

Fields: full to confrontation.

Dark adaptation (Crookes adaptometer): normal curve.

Colour vision: no perception of any colour on Ishihara test.

Pigmentary changes in the central area of both fundi were seen against a circular background of whitish-grey dots. This was initially diagnosed as a macular dystrophy. Some white dots were also present at the periphery. The choroidal vessels were not exposed (Fig. 11).

(iii) *Mrs. Nellie K.* (H II, 6) aged 55, reported failure of vision from about the age of 37, but no nightblindness.

Visual acuity: 6/60 in the right eye with c +3.5 sph. $\ominus$ +1.0 cyl. at 180°; in the left eye it was 6/60 with $\bar{c}$ +3.5 sph. $\ominus$ +2.5 cyl. at 180°.

Fields: right and left central scotomata.

Colour vision: no perception of any colour on Ishihara test (Plate 1 only was recognized).

Both fundi show central changes consisting of slight general pallor associated with very fine pigmentary stippling (Fig. 12). This patient has no children.

FIG. 11.—Right fundus of Miss Edith H., aged 66 (H II, 2), proband, sister of H II, 3 (Fig. 10).

FIG. 12.—Left fundus of Mrs. Nellie K. (H II, 6), sister of H II, 3 (Fig. 10) and H II, 2 (Fig. 11). Her right eye showed definite outlying choroidal sclerosis near the central area.

(iv) *Miss Emily H.* (H II, 1) aged 71, the eldest sister, is totally blind; her sight began to fail at about the age of 30 and she was blind by the age of 40. She does not remember nightblindness as a feature in her developing troubles. Both eyes show mature cataract, dilated pupils, normal tension, and no perception of light.

(4) B . . . t Family (Pedigree Chart IV; Figs 13 and 14).

Of the three sibs, two are non-identical twins (one twin being the proband). The other two sibs are normal, but the mother is affected. The mother and father are not consanguineous.

(i) *Mrs. Charlotte Bu.* (*née* C . . . k), (Br II, 3) aged 58, mother of proband (Br III, 3).

Visual acuity in each eye is 5/60. Refraction: right +0.75 D., left +1.0 D. Not improved.

Colour vision: no response to Ishihara test.

Ilford anomaloscope reading: 38/25-80.

Her sight failed in the early forties, and appears to have remained stationary since. The left fundus (Fig. 13) shows a sharply circumscribed oval patch of central choroidal sclerosis but not the typical end-stage of conversion into white streaks. There is fine pigmentary mottling around the circular patch.

(ii) *Mrs. Gladys Br.* (Br III, 3) aged 30, proband, presented herself because of failure in sight over the past year and was regarded as a case of possibly dominant macular dystrophy.

Visual acuity: 6/36 in each eye. Refraction: right +1.50 D., left +3.0 D. Not improved.

Colour vision: Ishihara test normal.

Anomaloscope reading 38/60.

The left fundus (Fig. 14) shows a number of discreet white dots scattered over the periphery, and some fine pigmentary mottling around the central area, which itself has a washed-out appearance, suggestive of an earlier stage of the lesion seen in Br II, 3.

No other affected person was found amongst the mother's four sibs (Br II, 4-7), nor were her two half-sisters affected—one being on the mother's side (Br II, 1), and the other on the father's side (Br II, 8). The proband's brother and sister (Br III, 1 and 2) were also found to be normal.

PEDIGREE CHART IV.—B . . . t family.

PEDIGREE CHART V.—R . . h family.

(5) Follow-up of R . . h Sibship originally recorded in 1939 (Pedigree Chart V; Figs 1 and 2).

The two affected brothers (R II, 4, and 7; Figs 1 and 2) are now dead. No other surviving member of the sibship is affected. The three surviving descendants of the elder affected brother, (R III, 4, 6, and 7) aged 55, 47, and

43 years, were found to be unaffected. The only son of the younger affected brother, (R III, 11) who could not be traced, had been normal when seen in 1938 at the age of 22 years.

FIG. 1.—Left fundus of George R., aged 59 (R II, 7). See *B.J.O.* (1939), 23, 433, Fig. 2. In this patient, as in all except H II, 6 (Fig. 12), the appearances were almost identical in each eye.

FIG. 2.—Left fundus of Richard R., aged 63 (R II, 4), brother of R II, 7 (Fig. 1). See *B.J.O.* (1939), 23, 433, Fig. 4.

Discussion

(1) Mode of Inheritance.—In the five families available the outstanding genetic facts are these:

(1) The affection has been observed in only one generation in four of the families, and over two generations in one family.

(2) Consanguinity has been noted in only one family, the parents being first cousins.

There is some indefinite evidence of the affection in collateral branches in two families. In the C . . n-C . . . l sibship (Pedigree Chart II) it is possible that two cousins of the affected sibship are also affected, these collateral branches being descended from two sisters (C II, 2 and 4) who married men who were themselves first cousins (C II, 1 and 3), though unrelated to the sisters' family. Furthermore, there is some suggestion of transmission over two generations: C II, 2, the deceased mother of one of the sibships, is reputed to have had bad sight, the nature of which could not be determined; the father of the second sibship (C II, 3) likewise has eye trouble, and has been found to be suffering from glaucoma.

Some suggestion of the affection in a collateral branch is also seen in the B . . n sibship (Pedigree Chart I); B III, 8, a niece of the affected women (B II, 6 and 7), has high myopia with indefinite fundus lesions.

The four families in which the affection has been observed in one generation only present no conclusive evidence regarding the mode of inheritance. The consanguinity in one family is at best suggestive only of recessive inheritance. If the collateral branch in Pedigree Chart II is indeed affected, the evidence for recessive inheritance becomes more convincing. It may be significant that the eight individuals aged more than 30 (six of them were over 40), all offspring of three known affected persons, were all normal. The distribution of the disturbance in the affected generations of these four families (as analysed in the Table) shows, however, a considerable excess over the 25 per cent. affected theoretically expected with recessive inheritance, there being sixteen affected and seventeen unaffected. If the analysis is confined to individuals actually examined, the excess and the departure from expectation becomes still more marked—twelve affected and six unaffected. Whilst autosomal recessive inheritance is possible, the available data do not fit too well. Recessive sex-linkage can obviously be excluded, since the twelve known affected include eight women. Intermediate sex-linkage can hardly be assessed in the absence of observations over two generations. Sex-limitation—or possible intermediate sex-linkage with the carrier state manifested by pathological but milder lesions—is not excluded, for there were in all fifteen individuals in affected sibships on whom full information was available: six men, of whom four were affected, and nine women, of whom eight were affected. The four affected men—by no means the oldest in the series—all showed a severe lesion, to which only one of those in the eight affected women was at all comparable. The theoretical possibilities that the pathogenic gene is carried either on disjointed XX chromosomes, or on the Y chromosome of such women, are excluded by the sex distribution in the affected sibships.

TABLE
DISTRIBUTION IN THE AFFECTED SIBSHIPS

Sibship	Affected		Possibly affected		Unaffected		Unknown, but probably unaffected		No information	
	M	F	M	F	M	F	M	F	M	F
B . . . n	—	2	—	—	—	—	6	1	—	—
C . . n-C . . . l	1	4	—	—	1	—	—	—	—	—
	—	—	1	1	1	2	—	—	—	—
H . . . s	1	2	1	1	—	—	1	—	—	—
R . . h	2	—	—	—	1	1	3	—	1	—
Total	4	8	2	2	3	3	10	1	1	—

The one family showing an affected mother and daughter—the mother and her husband not being consanguineous—raises the possibility of dominant

inheritance—a possibility that is neither proved nor disproved by the appearance of only one case amongst the three children. The fact that the mother's two half-sisters—one on the father's side and one on the mother's side—are unaffected gives no support for dominant inheritance, but does not exclude it. Nor is dominance excluded by the fact that this mother's four full sibs are also unaffected.

(2) Age of Onset.—It would seem that ophthalmoscopically visible lesions may be present for some years without serious symptoms. This is borne out in the C...n-C...l family, where C III, 1, 3, and 4, all aged over 50, show lesions without marked subjective symptoms. On the other hand, in the B...n sibship and in the R...h sibships, definite and progressive trouble dated back to the early twenties. The C...n-C...l sibship and the H...s sibship both show that within the same family severity of lesion and symptoms is not determined by age. It is therefore difficult to indicate a definite age of onset: a range of ages between 20 and 50 years for the onset of symptoms has been observed, and in contrast objective lesions without serious visual trouble have been noted in patients in the fifties.

(3) Ophthalmoscopic Changes.—The most severe changes are those observed in the R...h sibship previously recorded. In the B...n sibship (Chart I) the changes are distinctly less marked. In the C...n-C...l sibship (Chart II) severe ophthalmoscopic lesions are evident in two patients, but consist of little more than mild central exudative reactions in three others. Likewise in the H...s sibship (Chart III) and in the B...t family (Chart IV) both the full picture and early changes are seen. Taking these five groups together the following considerations emerge:

(i) *Course.*—It would appear that the earliest changes are oedematous-exudative reactions in the macular and paramacular areas (Figs 8 and 9), or an atrophic reaction at the macula with mottling and exudative reactions around it (Figs 11, 12, and 14). These stages show nothing pathognomonic, and might well be mistaken for a macular dystrophy or a central chorio-retinal lesion of environmental origin. Exposure of the choroidal vessels and their conversion into white streaks represent a later stage (Figs 3, 4, and 13) when a definite diagnosis becomes possible. The most marked stage in this series is the showing up of the chorio-capillaries as a white network, with some extension of the choroidal sclerosis beyond the sharply delineated central lesion (Figs 1, 2, 5, 6, 7, and 10).

(ii) *Changes in Relation to Age.*—In two families (R...h and B...n sibships) only the fully developed picture was observed, more marked in the R...h brothers (R II, 7 and 4; Figs 1 and 2), who were aged around 60 years, than in the B...n sisters (B II, 7 and 6; Figs 3 and 4) who were some 10–15 years older.

In the C...n-C...l sibship the fully developed picture was present in two individuals, a sister (C III, 2) aged 56, and a brother (C III, 5) aged 50 (Figs 5, 6, and 7), whilst a non-specific oedematous-exudative reaction was present in three other aged 58, 54, and 52 years (C III, 1, 3, and 4; Figs 8 and 9). Likewise in the H...s sibship the patient who showed the full

lesion (H II, 3; Fig. 10) was aged 63 years, whilst one of his sisters, 2 years older, showed a distinctly earlier and uncharacteristic stage (H II, 2; Fig. 11). That the affection is progressive is shown by the appearances in the mother and daughter in the B . . . t family (Br II, 3 and Br III, 3; Figs 13 and 14), but a strict chronological order is not a feature of the disturbance.

(4) Pathology.—Clinically the lesions suggest occlusion of the choroidal vessels in the central area, and the designation of central choroidal sclerosis seems justified, but the histological study of the eyes of C III, 2, does not appear to bear this out. There was evidence of atrophy of the vessels rather than of angiosclerosis; this is fully discussed in the accompanying study by Ashton (1953).

(5) Clinical Features and Course of the Affection.—No final assessment is as yet possible. For the present the following appears to be valid:

(i) *Mode of Inheritance.*—This yet remains to be established.

(ii) *Age of Onset.*—In two families this appears to have been at about 20. In one family there is evidence that subjective symptoms may not develop until about the age of 50, though ophthalmoscopic lesions were already present.

(iii) *Ophthalmoscopic Course.*—The earliest changes appear to be of the nature of a "central choroiditis". The lesion begins as a mottling or an oedematous-exudative reaction of the whole of the central area. This appears to progress only slowly towards exposure of the choroidal vessels and their conversion into white streaks. Severity is not strictly related to age.

(iv) *Subjective Symptoms.*—Central vision deteriorates to 6/60 or less. The peripheral fields remain intact and there is no nightblindness. Colour anomalies appear to set in only when vision is grossly reduced.

(v) *Designation.*—The classical designation "central senile areolar choroidal atrophy" is unwarranted in so far as it stresses senility and atrophy as essential features. It is only in old age that the advanced stage of central areolar choroidal sclerosis has been recognized, but the affection itself is essentially one of early and middle life, with central exudative or mottling reactions rather than atrophy. "Central areolar choroidal sclerosis" would appear to be a more satisfactory designation, even if at the present there is no supporting histological evidence to incriminate sclerosis of the choroidal vessels.

(vi) *Differential Diagnosis.*—The fully developed picture of central areolar choroidal sclerosis is unmistakable, because its three components are clearly defined: the central position, the characteristic changes in the exposed choroidal vessels, and the sharp demarcation of the lesion from the surrounding background.

No difficulty arises in differentiating the lesion from generalized choroidal sclerosis, nor from isolated extra-central patches. Where, however, there is a patch of choroidal sclerosis confined more or less to the central area, difficulty in diagnosis arises. Patches of central choroidal sclerosis are not uncommon in generalized fundus dystrophy, but the families with this latter affection so far reported (Sorsby and Mason, 1949, and Burn, 1950), though they show choroidal sclerosis in the central area, all lack the sharp limitation and areolar stamp seen in the present series. Moreover, the dominant course and the definite age of

onset of generalized fundus dystrophy are useful distinguishing features. No difficulty is likely to arise with choroidal sclerosis consequent on an old inflammatory reaction, or on injury. These are not usually bilateral and central, seldom—if ever—are areolar in type, and occasionally are multiple or sectorial.

In contrast, the early stages of the affection show little that is pathognomonic. Their genetic character may be suspected by the symmetry and similarity of the lesion in the two eyes. The mottling reaction can readily be mistaken for macular dystrophy or the earlier stages of generalized fundus dystrophy, whilst the oedematous-exudative reaction is not unlike that seen typically in the latter affection.

Summary

(1) Four further families showing central areolar choroidal sclerosis are reported, and a follow-up of the sibship originally recorded in 1939 is given.

(2) In four of these families the affection was observed in sibships only. In one family it was seen in a mother and daughter.

(3) The fourteen observed cases show several stages of the affection. The earliest ophthalmoscopic appearances are an exudative-oedematous reaction centrally, simulating the early stages of generalized fundus dystrophy, or a pigmentary mottling of the central area simulating macular dystrophy. The lesion becomes pathognomonic when there is exposure of the choroidal vessels centrally over an areolar area. This progresses to ophthalmoscopically visible obliteration of these exposed choroidal vessels and choriocapillaries. The age of onset seems to vary in different families. In two families it began in the early twenties; in one in the late forties. In a fourth family nothing definite could be determined. The fully established lesion has not been seen in any patient under the age of 50. The lesion appeared to be more severe in all the four affected men than in all but one of the ten affected women, though the affected men were not the oldest in this series.

(4) The available evidence is conclusive as to the genetic character of the affection, but not sufficiently extensive to establish the mode of inheritance.

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We are particularly indebted to Dr. Milton Berliner of New York for kindly following up the collateral branch of the C...n-C....l family resident in the United States.

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