

RIBOFLAVIN DEFICIENCY WITH OCULAR SIGNS: REPORT OF A CASE*

BY

C. R. S. JACKSON

From the Nuffield Laboratory of Ophthalmology, Oxford

IN view of its comparative rarity in Great Britain it seems justifiable to report a case which appeared to be one of riboflavin deficiency presenting ocular symptoms.

Case Report

A married woman, 52 years old, first seen September 1, 1948, was complaining of epiphora and a slight irritation of both eyes, particularly the right, with some photophobia. The illness was of some three months' duration, though she said she had had recurrent attacks of "sore eyes" for many years. The present attack seemed to have started after a long train journey.

The visual acuity was right 6/6, left 6/36, the left eye having been known for some time to be amblyopic. Both eyes looked irritable and the conjunctiva was slightly congested. There was no evidence of intra-ocular inflammation but, with the slit-lamp, superficial vessels could be seen extending on to the cornea from the limbal plexus. These vessels were very thin but contained blood and were arranged in a fairly regular manner all round the periphery of the cornea and directed towards the centre. They were mostly straight and anastomosed with each other, forming attenuated arcades. There were no other abnormalities.

The picture of the abnormal corneal vessels was so like that seen in ariboflavinosis as described originally by Sydenstricker and others (1940) and later by Gregory (1943), Ferguson (1944), and Mann (1945), that a provisional diagnosis of riboflavin deficiency was made. The possibility of an infective conjunctivitis was also considered, but bacteriological examination of the conjunctiva showed no pathogenic organisms.

The patient was given no treatment apart from riboflavin 9 mg. by mouth daily. Unfortunately she did not attend again for a month but her non-appearance was apparently due to the improvement in her condition, for she said that her eyes seemed to be entirely normal after about nine days on riboflavin, all grittiness and photophobia disappearing. Improvement had been noticed within a few days of starting to take the vitamin.

When next examined, September 27, 1948, the abnormal corneal vessels had undergone very considerable regression and those still visible contained little blood. There was no congestion of the conjunctiva.

The patient was asked about her diet. She was a hard-working housewife with a husband and son out at work. She admitted that she was not particularly anxious about what she herself ate and liked "to give as much as possible to the men". Cups of tea with bread and butter seemed to figure largely in her diet. When it was put to her that perhaps she was not getting enough to eat, she agreed that, although she usually had something of what she cooked for the men, her portion was never very large.

Twelve months after treatment with riboflavin she wrote, "I have not been troubled with sore eyes since my visit in October last. I have followed your advice about diet."

Discussion

The response to riboflavin was so striking that there seems no doubt that the ocular signs in this case were due to deficiency of the vitamin. It has been pointed out previously (Mann, 1945)

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that a distinction must be drawn between the normal limbal capillary plexus and the appearances of new superficial vessels on the cornea, and that failure to appreciate this distinction may be the cause of the large numbers of cases of this condition in some published reports (Kruse and others, 1940; Frandsen and Lundh, 1941; Tisdall and others, 1943). The present case was examined with this point in mind.

At no time in the course of the illness did the patient show any of the cutaneous manifestations of riboflavin deficiency. She did, however, admit to some feelings of excessive tiredness and lack of energy which may perhaps have been part of the same picture.

It is interesting to note that a recent paper by Das Gupta (1949) suggests that this type of corneal vascularization may not be the only way in which riboflavin deficiency may show itself in the eye. He describes a case of parenchymatous keratitis in which corneal opacity was unaccompanied by vascularization. The condition resolved completely after injections of riboflavin (10 mg. daily for 78 days).

A very similar but much more severe case of the type described here has been reported by Stern (1949). His patient was a man aged 38 years in whom the condition had been precipitated by trauma. Stern suggests that, the metabolic activities of injured corneal epithelium being greater than those of the normal, it can not be provided with sufficient respiratory enzyme in the absence of adequate riboflavin. The picture of corneal vascularization and unhealthy epithelium may therefore be "conditioned" by superficial injury and may develop on top of what has previously been a subclinical deficiency of the vitamin.

Stern suggests that three conditions should be fulfilled before the diagnosis of ariboflavinosis is made. There should be (i) a history of inadequate intake of the vitamin over a long period, (ii) a low riboflavin level in blood or urine, (iii) an obvious response to treatment with riboflavin.

Although the disappearance of photophobia and regression of the abnormal vessels as a result of treatment may be very striking, it seems that the improvement in visual acuity may be disappointing in long-standing cases owing to the persistence of corneal opacities.

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