*Brit. J. Ophthal.* (1971) **55**, 777

# Central retinal vein occlusion complicating iron deficiency anaemia

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Sudden blindness as a consequence of severe blood loss was well known at the turn of the century, when a common cause of blindness in young people was anaemia due to blood loss from the gastrointestinal tract or to abortion (Gowers, 1904). The retinal changes described include pallor of the disc, sometimes with disc oedema, and small numbers of retinal haemorrhages and exudates. Such retinal abnormalities usually develop within 48 hours of haemorrhages and occur typically in patients rendered anaemic by repeated episodes of blood loss rather than a single major haemorrhage. Transient impairment of retinal blood flow during episodes of hypotension was considered to be the most likely cause of the ocular changes in such cases by Pears and Pickering (1960). Though retinal haemorrhages and exudates often occur in severe chronic iron deficiency anaemia (Holt and Gordon-Smith, 1969), the retinal abnormalities are not usually severe and consist, in the main, of "cotton wool" exudates. Profound retinal damage suggesting central retinal vein occlusion would appear to be a most unusual complication of iron deficiency anaemia; such a case is reported here and the pathogenesis of the retinal changes is discussed.

## Case report

**A 44-year-old married woman** (RI 476584) was admitted to hospital complaining of blurred vision in the left eye of 10 days duration. Apart from shortness of breath on exertion and a tendency to bruise easily, she had no other symptoms.

### PHYSICAL EXAMINATION

She was strikingly pale with a few bruises on the right thigh and the feet. There was an aortic ejection murmur radiating into the neck. The brachial blood pressure was 120/70 in each arm. The remainder of the examination gave normal results.

### OPHTHALMIC EXAMINATION

The visual acuity was 6/5 in the right eye and 6/18 in the left. The intraocular pressures were 14 mm.Hg in the right eye and 13 mm.Hg in the left measured by applanation tonometry. The right fundus was normal but the left fundus showed florid features of central retinal vein occlusion with blurring of the disc margin, venous engorgement, haemorrhages, soft exudates, and macular oedema (Fig. 1, overleaf). Ophthalmodynamometry showed the pressure to be 100/45 on each side.

### INVESTIGATIONS

Hb6·2 g./100 ml.; mean corpuscular haemoglobin concentration 30 per cent; reticulocytes 3 per cent.; platelets 280,000/cu. mm.; white blood cells 4,800/cu.mm. (normal differential); blood film—marked hypochromia, anisocytosis, and poikilocytosis; erythrocyte sedimentation rate 4 mm. 1st hr.

Received for publication March 31, 1971
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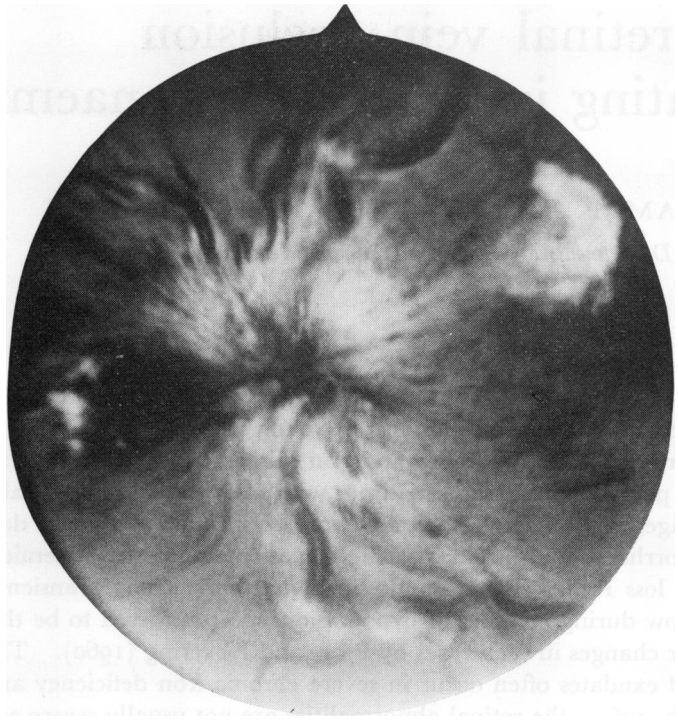

FIGURE *Left fundus, showing features of retinal occlusion*

Serum iron 30 μg./100 ml.; serum folate 3 ng./ml. One stage prothrombin time, kaolin cephalin time, normal. Fibrinogen titre normal.

*Bone marrow* Very cellular, showing active normoblastic erythropoiesis but no stainable iron.

Plasma urea, electrolytes, calcium, phosphate, cholesterol, uric acid, proteins and albumin, alkaline phosphatase, bilirubin, lactic dehydrogenase, glutamic oxaloacetic transaminase levels all normal. Antinuclear factor—negative.

*Urine* Normal; no haemosiderin.

Repeated faecal tests for occult blood were negative.

*Jejunal biopsy* Normal histology.

Ferrous iron absorption 21 per cent. (normally 30 per cent. in iron deficiency).

*Gastrin test meal* Achlorhydria with low intrinsic factor secretion. Intravenous glucose tolerance test—$K_G = 1 \cdot 2$, repeated 6 months later, $K_G = 1 \cdot 1$ (low normal result).

*Radiological examination* Oesophagus, stomach, small and large bowels normal.

## TREATMENT

The patient was transfused with 2 units of packed cells and this was followed by treatment with oral iron.

## RESULT

Subsequently no cause for the profound iron deficiency anaemia was discovered and 3 months later the haematological state was normal apart from slight hypochromia of the red cells (Hb 13.3 g./

100 ml.; mean corpuscular haemoglobin concentration 33 per cent.; reticulocytes 0·3 per cent.). At this time the appearance of the left fundus had returned to normal and the visual acuity had improved to 6/5.

She now acknowledged that she had not been so well for many years and had been easily fatigued since the birth of her second child.

COMMENT

Though no previous measurements of her haemoglobin concentration had been made, it seems likely that the iron deficiency anaemia was of long duration, probably related to childbirth. Its perpetuation was most likely to be due to defective absorption of food iron related to gastric achlorhydria, although the possibility of chronic repeated blood loss from the gastrointestinal tract cannot be overlooked, despite the fact that all investigations were negative.

## Discussion

The haemodynamic circumstances of the ocular circulation are unique in that the terminal branches of the vascular supply to the eye pass from an extraocular low pressure system into an intraocular high pressure system. Thus the relative perfusion pressure in the retinal vascular system is low compared with the extraocular circulatory system. Factors which disturb this critical perfusion gradient may be responsible for the development of central vein occlusion. Much evidence incriminating arterial circulatory changes in the pathogenesis of the retinal vein occlusion has been found clinically in ophthalmodynamometric studies by Paton, Rubinstein, and Smith (1964) and experimentally by Hayreh (1965). Lowered retinal perfusion pressure leading to a fundus picture similar to that of central venous occlusion has been recognized in carotid insufficiency and aortic arch syndromes (Hedges, 1964) because of the reduced arterial pressure and in cases of carotid-cavernous fistula where the venous pressure is elevated while the arterial pressure is reduced (Sanders and Hoyt, 1969). Central vein occlusion is often a presenting sign of chronic simple glaucoma, the elevated venous pressure secondary to the raised intraocular pressure being responsible for a lowered retinal prefusion gradient.

Whatever the cause of the reduced retinal perfusion gradient, the ensuing hypoxia produces metabolic dysfunction of the capillary and venous endothelium. The veins become distended and leakage of blood occurs through the vessel wall. In our patient the intraocular pressures were normal, there was no clinical evidence of arterial disease, and such findings would have been unusual in a female patient under 45 years of age. We suggest that, despite an apparently adequate perfusion pressure, the quality of the perfusing blood must also be considered and that in our patient the relative anaemic hypoxia caused the development of the venous changes. This hypothesis is strengthened by the return to normality of the retina and visual acuity on correction of the anaemia. The prompt correction of the anaemia by improving the retinal circulation allowed rapid recovery of the macular oedema before degenerative cystoid maculopathy appeared.

## Summary

Central retinal vein occlusion developed as a consequence of profound anaemia in a 44-year-old woman. Venous endothelial incompetence as a result of anaemic hypoxia is believed to be responsible for the retinal changes despite the presence of an adequate retinal perfusion pressure.

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