

Case Report

Malnutrition and central retinal vein occlusion in a young man

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ABSTRACT

Can vitamin B₁₂ and folate deficiency cause central retinal vein occlusion? We conducted a literature search to find out whether nutritional deficiency of vitamin B₁₂ and folate can lead to impaired vision. The patient in the article reported in Ophthalmology, department of MMIMSR, Haryana, India, with complain of gradual painless visual loss over six weeks. He was found to have unilateral central retinal vein occlusion with significant anemia and vitamin B₁₂ and folate deficiency. Vitamin B₁₂ and folate deficiency can lead to elevated levels of homocysteine. We found a large amount of published data relating central retinal vein occlusion to elevated homocysteine levels, but there was a lack of conclusive evidence for this association. Patients should be asked about their dietary history where a thrombotic event is suspected or confirmed.

Keywords: Folate deficiency, Malnutrition

INTRODUCTION

The incidence of retinal vein occlusion varies in population based studies from 2 per thousand to 8 per thousand persons.^{1,2} Patients who develop central retinal vein occlusion are typically over 50 years of age and it is a common cause of visual morbidity.³ There is an increased incidence of central retinal vein occlusion in people with conditions such as diabetes mellitus, hypertension, collagen vascular diseases and hyperviscosity syndromes, with smoking and contraceptive pill use being additional risk factors. When young patients develop a central retinal vein occlusion it is important to obtain a detailed nutritional history, as is shown by this case.

CASE REPORT

A 30-year old male patient came to OPD of ophthalmology casualty department by his mother. He had suffered gradual and painless visual loss over the previous six weeks. His visual acuity on a standard Snellen chart was 6/6 in right eye and 4/60 in left eye.

He had no other symptoms other than visual loss, occasional headaches and recently increasing breathlessness on exertion. Previously he had good vision, not requiring correction.

His social history was that he lived at his parents' house; and was a non-smoker and non-alcoholic. Systemic examination was done his BP was 130/90 mmHg and pulse 88/min. No pallor/icterus/clubbing or lymphadenopathy was found.

Fundus examination revealed a hyperemic disc OS, with filled cup and blurred disc margins. Blood vessels overlying the disc were dilated and tortuous with splinter haemorrhages. There was generalized dilatation and tortuosity of the veins with altered arteriolar: venular ratio of 2:4, and multiple dot and blot hemorrhages in the posterior pole and periphery of all four quadrants. Flame shaped hemorrhages and cotton wool spots were noted to be distributed along the inferotemporal quadrant. Intraocular pressures were normal in both eyes.

Fluorescein Angiography (FA) was notable for delayed A-V transit time, more in the inferotemporal vasculature.

Areas of blocked fluorescence were noted, corresponding to haemorrhages and cotton wool spots. Diffuse hyperfluorescence was noted in all four quadrants along the vascular arcades and in the macula. Extensive areas of capillary non-perfusion were seen in the inferior quadrants with late-staining of the venous architecture.



Figure 1: Fundus photograph OS.

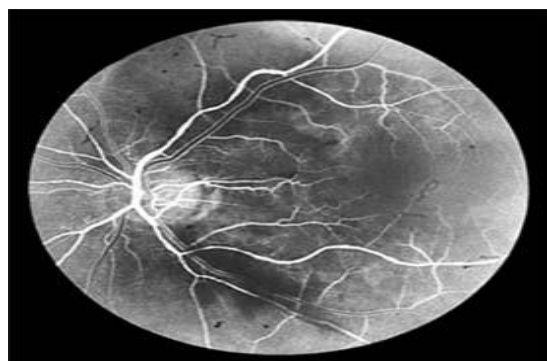


Figure 2: Fundus angiogram OS.

He was admitted and found to have haemoglobin of 5.5 g/dl. Mean cell volume was 125 fl. Platelet count, white cell count and erythrocyte sedimentation rate was normal. Bilirubin was 50 $\mu\text{mol/l}$ with otherwise normal liver function tests. Serum glucose was 5.6 mmol/l and serum lipids were normal.

Blood film showed a megaloblastic anaemia with nucleated red cells, macrocytosis and hypersegmented neutrophils. Absolute reticulocyte count was not raised. Low levels of folate and vitamin B₁₂ were confirmed on serum testing (folate: 1 ng/ml , vitamin B₁₂: 54 ng/l).

Fluorescein angiography confirmed the clinical picture of non-ischaemic central retinal vein occlusion. Protein C, protein S and antithrombin III levels were normal. There was no resistance to activated protein C and lupus anticoagulant and antiphospholipid antibodies were negative.

On further closer questioning it was found that the patient had not eaten vegetables for several years and lived on a diet involving a processed corn snack, chips and fast food

chain meals. He explained that he did not like the taste of vegetables and dairy products.

Malabsorption causes were excluded over the next weeks and he was given folic acid, hydroxycobalamin and iron supplementation. He was referred to a dietitian and advised on a healthier diet. Visual acuity improved to (Snellen chart) 6/12 on the right and 6/12 on the left over the subsequent months. His haemoglobin levels returned to normal over the subsequent months.

DISCUSSION

The case describes a young man with severe anaemia caused by very poor diet. His visual acuity gradually deteriorated over several weeks and it turned out he had a unilateral central retinal vein occlusion. This is a very rare event in a young patient.

When we searched the literature, we found associations with malabsorption disorders and retinopathy for example in patients with pernicious anaemia.⁴ Isolated retinal haemorrhages are a well-recognised complication of severe anaemia and there are case reports describing such presentations with folate and vitamin B₁₂ deficiency.⁵ There also appears to be evidence for a link between vitamin deficiencies and retinal veno-occlusive disease; both low serum folate and vitamin B₁₂ levels can lead to elevated homocysteine levels and in conjunction pose an important theoretical risk factor for the development of central retinal vein occlusion. Moderately elevated levels of homocysteine are already known to be associated with arterial and venous thrombotic events. We found a large amount of published data relating central retinal vein occlusion to elevated homocysteine levels, but there was a lack of conclusive evidence for this association. In young patients a definite link between high homocysteine levels and risk of developing central retinal vein occlusion has not been established.

CONCLUSION

Our initial history-taking had focussed on smoking, alcohol consumption and foreign travel. We tried in vain to tie these facts together to determine aetiology, for this atypical case of unilateral central retinal vein occlusion in a man in this age group. However, it turned out that the important part of the social history was this patient's nutrition and this is a salutary lesson to doctors of the risks of omitting this important detail from history taking. We suggest including a section in each patient's social history asking specifically about dietary habits, whenever a thrombotic event is suspected, in order to identify quickly nutritional extremes.

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