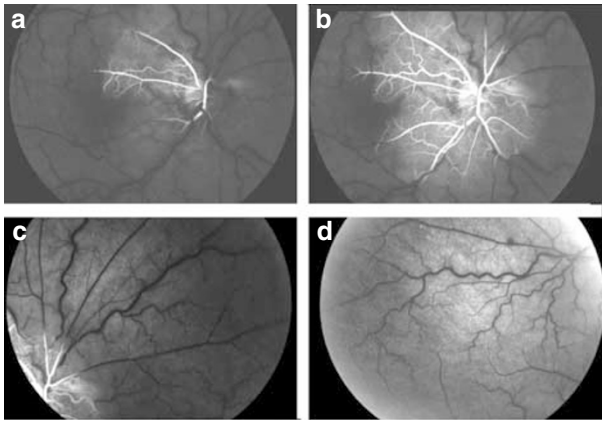

Figure 2 (a,b) Angiograms at 0:21.9 and 1:07.1, respectively, showing shutdown of peripheral vasculature with partial patency of macular circulation. (c) Angiogram at 1:33.6 showing no perfusion of nasal circulation. (d) Angiogram at 2:09.7 showing no perfusion of peripheral temporal circulation.

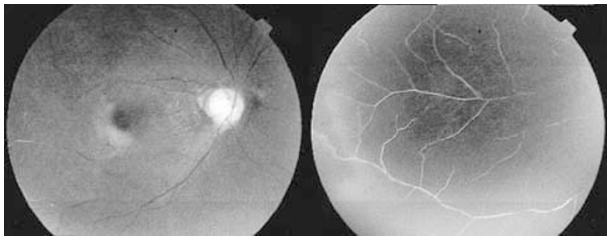

Figure 3 One-month appearance showing disc pallor and sheathed peripheral blood vessels.

a greater effect on blood viscosity and predisposes to vascular occlusion.[5]

There have been case reports of central retinal artery occlusions in association with SS disease.[6,7] This case is unusual because of the superior zone of macular sparing, which is not due to cilioretinal artery sparing. It appears to be due to patency of part of the macular circulation originating from the superotemporal retinal vessels. Vascular obstruction in sickle cell disease occurs due to RBC sickling leading to sluggish blood flow, erythrocyte aggregation, activation of coagulation, and eventual vaso-occlusion.[8] In our patient, we hypothesise that the original insult was dehydration, leading to an increase in plasma viscosity which caused sickling in the central retinal artery. The ensuing panretinal hypoxia led to further sickling downstream of the initial obstruction with sludging and total occlusion of peripheral retinal vasculature. This may have been compounded by the smaller vascular calibre and lower oxygen tensions of the peripheral vessels. The macular sparing was due to a patent macular vessel before the superotemporal vessel became occluded.

Thus, this case illustrates a previously unreported ocular manifestation of SS disease, due to which the patient lost a substantial amount of peripheral vision but retained a narrow field of central vision.

It highlights the prophylactic use of hydroxyurea and the possible therapeutic role of exchange transfusion in managing this problem. These options merit further investigation.

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C Sanghvi, A Laude, A Smith and CL Dodd

Department of Ophthalmology
Manchester Royal Eye Hospital
Oxford Road
Manchester M13 9WH, UK

Correspondence: CA Sanghvi
20, Stablefold
Worsley
Manchester M28 2ED, UK
Tel.: +44 161 288 8659
Fax: +44 161 273 6354
E-mail: casanghvi@yahoo.co.uk

Sir,

Deposition of gold in ocular structures, although known, is rare. A case of ocular chrysiasis in a patient of rheumatoid arthritis on gold treatment is presented

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An asymptomatic 50-year-old white man was referred for corneal evaluation by his optician. His past medical

history was positive for rheumatoid arthritis involving knee, hip, and small joints of the hand (Figure 1a). Control of arthritis required indomethacin (75 mg b.i.d.), maintenance dose of oral prednisolone (5 mg once daily), and more recently methotrexate (7.5 mg/week). In addition, he was receiving intramuscular injections of colloidal gold (50 mg) every alternate week since 1980. He had undergone hip and knee replacement surgery in 1990s because of joint deformity. On external examination, he had slate-grey complexion. Corrected visual acuity was 20/20 OU. On slit-lamp examination similar findings were evident bilaterally. Fine-scattered yellow-brown deposits could be seen on the central corneal epithelium. In the deep central corneal stroma, the deposits were confluent (Figure 1b). The lens was clear. Ophthalmoscopic examination was normal.

Comments

The term chrysiasis is derived from the Greek 'chrysos' referring to effects of gold on various tissues most noticeably recognized in the skin, which may show colour changes ranging from a periorbital mauve/blue to a diffuse slate-grey/blue appearance.[1] Widespread deposit of gold occurs in patients who have received a cumulative dose greater than 1 g.[2] Deposition of gold in ocular structures, particularly in the cornea, is termed ocular chrysiasis.[3] Corneal deposits can be limited to the epithelium[4] or the stroma.[2] Deposition in the lens occurs infrequently.[5] On histopathologic evaluation, gold deposits are present both intracellularly and extracellularly without any inflammatory reaction.[3] Colloidal gold therapy is effective in slowing the progression of rheumatoid arthritis and therefore patients who respond favourably are usually kept on maintenance therapy for many years.[6] Occurrence of ocular chrysiasis does not require discontinuation of gold therapy.

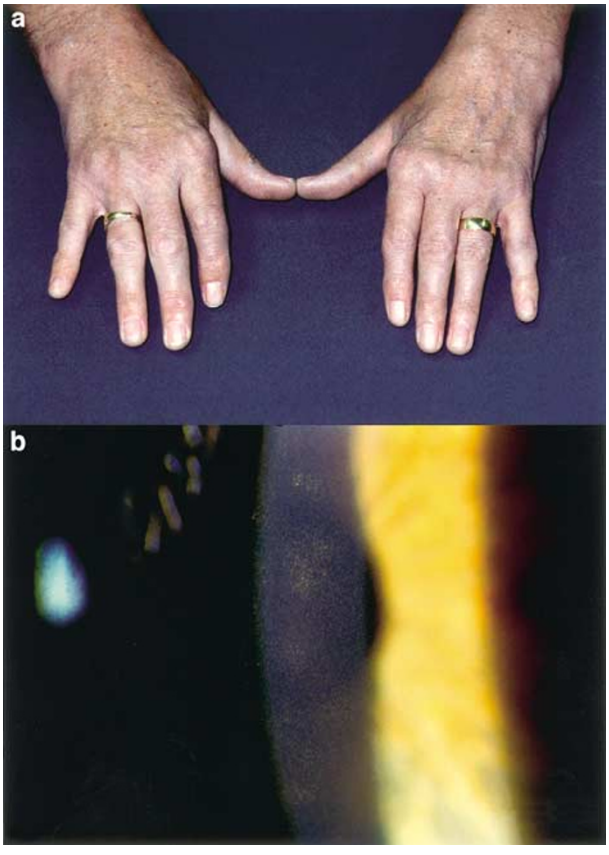

Figure 1 (a) Typical rheumatoid deformity of the hands. (b) Slit-lamp photograph showing yellow-brown confluent deposits in the deep corneal stroma.

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AD Singh[1,2], P Puri[1] and RS Amos[3]

[1]Department of Ophthalmology
Royal Hallamshire Hospital
Glossop Road, Sheffield
S10 2JF, UK

[2]Wills Eye Hospital
Thomas Jefferson University
Philadelphia PA, USA

[3]Department of Rheumatology
Royal Hallamshire Hospital
Sheffield, UK

Correspondence: P Puri
Tel: +44 114 271 2902
Fax: +44 114 276 6382
E-mail: pankajpuri35@hotmail.com