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**SHORT COMMUNICATION**

# Multifocal choroiditis - An unusual finding in Crohn's disease

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PURPOSE. *To report a patient with Crohn's disease and acute decreased vision in one eye secondary to multifocal choroiditis and serous retinal detachment.*
METHODS. *A complete ocular examination, including fluorescein angiography, was performed.*
RESULTS. *Fundus biomicroscopy disclosed multifocal, deep, discretely elevated yellowish lesions at the posterior pole of the affected eye. Fluorescein angiographic study of these lesions revealed early hypofluorescence followed by late hyperfluorescence. Subtenonian injection of corticosteroids rapidly induced remission of the choroidal lesions.*
CONCLUSIONS. *Chorioretinal involvement in patients with Crohn's disease may or may not be related to reactivation of this disorder. Therefore, even patients without gastrointestinal symptoms who present with posterior segment inflammation must be informed of this. The chorioretinal inflammatory lesions do seem to respond promptly to periocular injection of corticosteroids. (Eur J Ophthalmol 2004; 14: 345-9)*

KEY WORDS. *Crohn's disease, Choroidal infiltrates, Multifocal choroiditis, Corticosteroids*

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## INTRODUCTION

Crohn's disease (CD) is a chronic inflammatory disorder of the intestinal tract (1). Extraintestinal complications of CD are common and include arthritis, erythema nodosum, stomatitis, ankilosing spondilitis, hepatitis, cholangitis, and pyelonephritis (2).

Iridocyclitis is probably the most common ocular complication seen in CD, occurring in 0.5% to 11.8% of cases (3). Other anterior segment complications include conjunctivitis, episcleritis, scleritis and keratitis (4, 5). Occasionally, posterior segment inflammation has been associated with iridocyclitis, and it generally consists of vitreitis, retinal vasculitis and retinitis (6, 7).

This report presents a patient with CD and acute unilateral decreased vision secondary to multifocal choroidal infiltrates and serous retinal detachment involving the macular region. This ocular picture has rarely been described associated with CD and, in our case, responded promptly to periocular injection of corticosteroids.

## Case report

A 31-year-old male was referred to our department for decreased vision in the left eye (LE). Ocular history revealed a bilateral iridocyclitis ten years earlier, which had been well controlled with periocular injection of cor-

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ticosteroids. CD had been diagnosed by colonoscopy with a specific biopsy at approximately the same time of these ocular problems. The patient subsequently underwent a total abdominal colectomy and iliorectal anastomosis.

The patient presented without any gastrointestinal symptoms of CD. Best corrected visual acuity (VA) was 20/50 in the LE and 20/20 in the right eye (RE). Slit-lamp examination revealed a significant anterior chamber inflammation in the LE (3+ cells/4) and a slight inflammation in the RE (1+cells/4). Fundus biomicroscopy disclosed multifocal, deep, discretely elevated yellowish lesions at the posterior pole of the LE associated with retinal folds, suggesting a serous retinal detachment. A flat, subretinal pigmented lesion was observed temporal to the foveal region. The RE revealed small, round hypochromic spots in the macular region. The optic disc and vessels were normal in both eyes.

Upon fluorescein angiography (FA), the active lesions in the LE showed early hypofluorescence; the late phases showed leakage with faint, fuzzy, hyperfluorescent staining (Fig. 1). In the RE, small, round lesions with

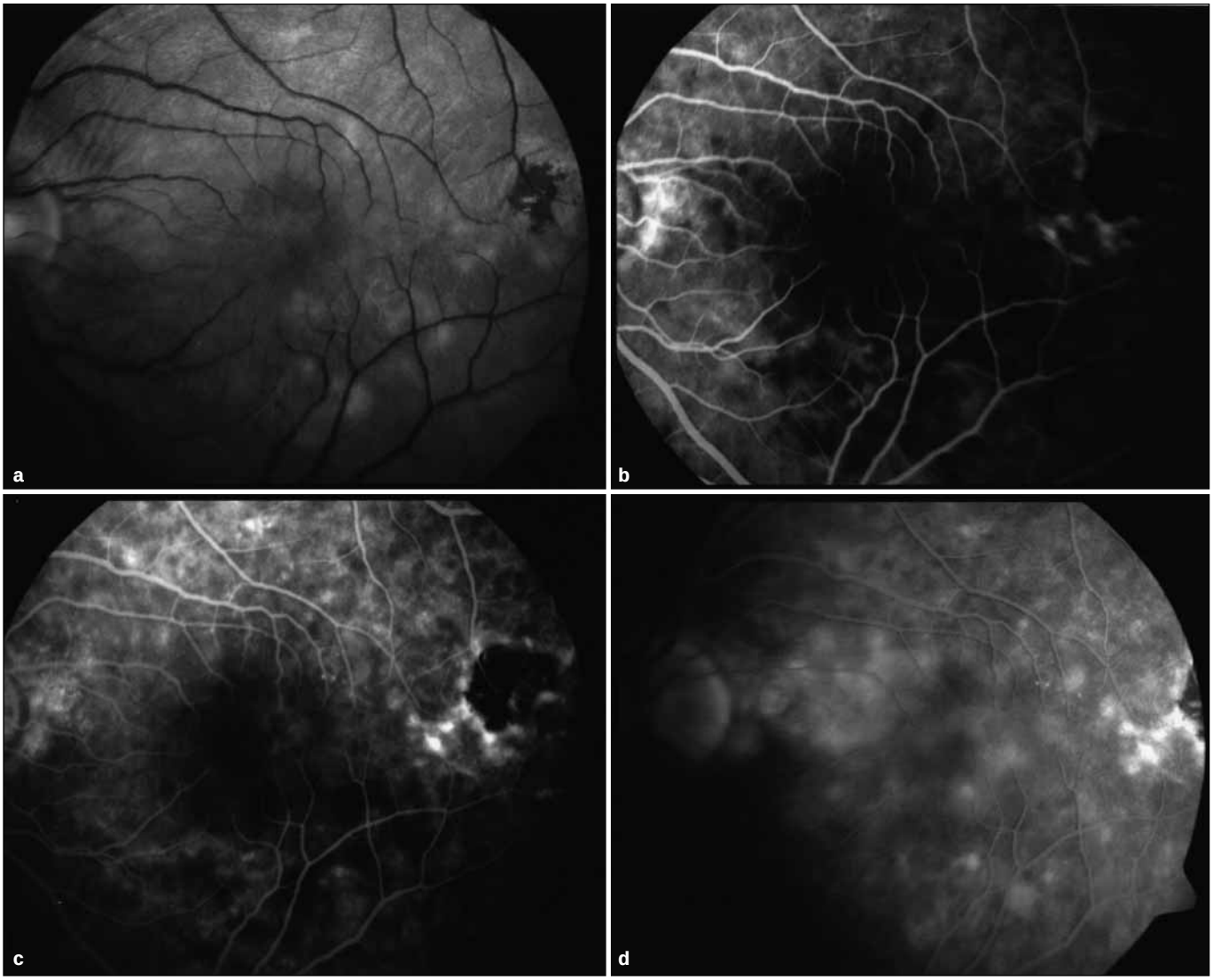


**Fig. 1 - a)** *Red-free fundus photograph of the left eye revealing yellowish multifocal choroidal lesions and retinal folds. Fluorescein angiography showed in* **b)** *early hypofluorescence followed by gradual hyperfluorescence and late leakage* **c, d)** *of the lesions in the subretinal space.*

progressive fluorescence were observed in the macular region and were interpreted as small areas of retinal pigment epithelium (RPE) atrophy (Fig. 2).

Topical medication was prescribed for the left eye with prednisolone 1% 6 times/day and homatropine 1% 2 times/day. After one month the VA was still 20/50 and the ophthalmoscopic appearance of the lesion unchanged. It was then decided to treat the patient's LE with a subtenon injection of triamcinolone acetonide (1 ml/40 mg). Within one month of follow-up the patient had recovered 20/30 of VA and the ophthalmoscopy then revealed RPE pigmentary and atrophic changes in the macular region. The patient refused to do a post-treatment fluorescein angiographic study. As the patient had no gastrointestinal symptoms, he also refused to undergo further investigations to evaluate if CD was active.

After six months of follow-up the VA remained unchanged in both eyes, and the patient continued without gastrointestinal complaints related to CD.

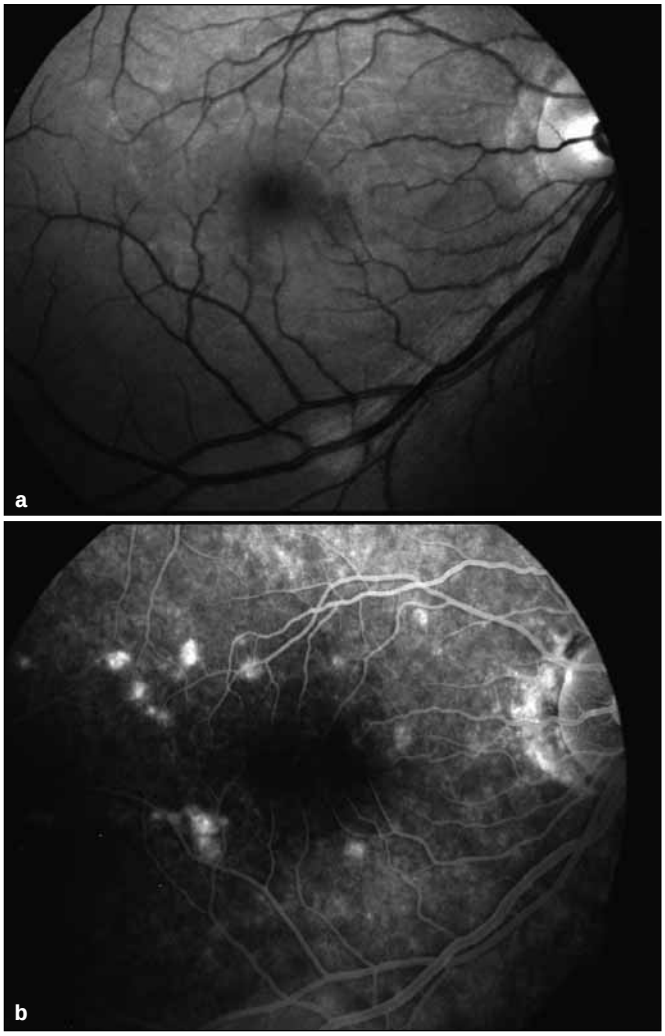


**Fig. 2 - a)** *Red-free fundus photograph of the right eye revealing discrete round hypopigmented lesions surrounding the foveal region.* **b)** *Fluorescein angiography shows small nummular areas of atrophic retinal pigment epithelium, which corresponded to those lesions seen on red-free photograph.*

## DISCUSSION

This paper describes a patient with CD who presented with choroidal infiltrates and overlying serous retinal detachment in one eye, and small, round RPE atrophic lesions in the posterior pole of the fellow eye.

The nature of these choroidal infiltrates is unknown, but histopathologic studies of an eye enucleated with panuveitis from a patient with CD demonstrated choroidal granulomas (8). This might be an explanation for the nature of the choroidal lesions observed in our patient, since granulomas are part of CD (1). In fact, understanding ocular involvement in CD is difficult, since the cause of this disorder is unclear. However, an immune pathogenesis is suggested by histologic and humoral alterations noted in CD patients. Activated CD4+ T cells present in the lamina propria of the bowel mucosa as well as the peripheral blood secrete inflammatory cytokines. Some cytokines directly activate other inflammatory cells (macrophages and B cells) while others act indirectly to recruit other inflammatory leukocytes, and mononuclear cells from the peripheral vasculature into the gut (1). Continuing the inflammatory cascade, neutrophils and macrophages produce prostaglandins and leukotrienes, which cause vasodilation and increase vascular permeability (1).

It is possible that the activation of abnormal intestinal immunity in patients with CD results in circulating antibodies or antigen-antibody complexes that can mediate ocular complications. Thus, we hypothesize that choroidal granulomas, associated with some degree of vasculitis, were responsible for the clinical and fluorescein angiographic picture seen in the LE of our case. The small, round RPE lesions observed in the right eye of our patient might be scars from previous focal serous retinal detachments related to the above mentioned pathophysiology or they may be a sequel

similar to that seen in late-stage idiopathic central serous chorioretinopathy (ICSC). Interestingly, ICSC has also been associated with CD (6).

It has been estimated that less than 1% of patients with CD present posterior segment involvement (4,6). In a survey of 332 patients with CD, 21 patients were found to have ocular changes (4). However, there was only a single patient with chorioretinal involvement, who presented a unilateral macular hemorrhage. Ernst et al (6) reported 8 patients with CD and posterior segment manifestations. From this group, 4 patients (7 eyes) presented choroidal infiltrates associated or not with serous retinal detachment. Further evaluation demonstrated that CD was active in 3 of these 4 patients. Systemic corticosteroid therapy induced remission in the choroidal lesions resulting in atrophic or hyperpigmented scars. In our case, subtenonian injection of corticosteroids rapidly induced remission of the choroidal lesion as well as the serous retinal detachment.

Apart from the ocular features, our patient was systemically asymptomatic when he came to be examined in the eye clinic. No search was done to evaluate a possible reactivation of CD. In order to measure the activity of CD it is usually sufficient to follow the patient's symptoms and signs in response to treatment. Rarely is it necessary to subject the patient to radiographic studies or colonoscopies to ascertain disease activity (9). The patient presented in this paper underwent a total colon resection 9 years previously, but recurrences are commonly seen in CD. Therefore, he was informed that the acute eye problem was possibly related to a reactivation of CD and that we could go further with a gastrointestinal evaluation. However, he refused this option as he was asymptomatic.

The differential diagnosis of the case presented included diseases that have multifocal, scattered, deep yellow-white lesions in the posterior pole, particularly acute posterior multifocal placoid pigment epitheliopathy (APMPPE) and posterior scleritis. The fluorescein angiographic pattern observed in our case simulates that seen in APMIPPE. However, as pointed out by Gass (9) serous retinal detachment is not a finding in APMPPE. Posterior scleritis (PS) has already been described in patients with CD (5). Nevertheless, FA in PS characteristically demonstrates multiple fine pin-point leaks at the level of the RPE which evolve to fill an overlying neurosensory exudative retinal detachment (10). This FA picture was not present in our patient. However, since ocular echography was not done in the case presented, PS can not be definitively ruled out.

In conclusion, we believe that the report of this unusual ocular manifestation of CD is important since the chorioretinal inflammation might be a sign of reactivity of this disorder. Furthermore, the chorioretinal lesions responded promptly to periocular injection of corticosteroids, leading to visual improvement in our patient.

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