Arch. Soc. Oftal. Optom. (1983)-17-123

BENIGN CHOROIDAL FOLDS: A DIFFICULT AETHIOLOGICAL PROBLEM TO ASSESS

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SUMMARY

Benign choroidal folds present a controversial aethiological problem.

The authors refer about 6 patients with choroidal folds of unknown origin that failed to improve after treatment. Particular emphasis is given to the ultrasonographic findings, expecially in relation to the differential diagnosis of secondary or idiopathic choroidal folds.

The authors accept the possibility that idiopathic choroidal folds, not improved with treatment, have to be related to a subclinic posterior sclereritis.

INTRODUCTION

Nettleship[1] was the first to recognize, in 1884, the involvement of Bruch's membrane and choriocapillaris in what had previously been considered to represent atrophy secondary to papillitis.

The clinical picture was one of retinal striae, with light and dark lines radially disposed from the optic disc towards the macula. Various authors have subsequently reported choroidal folds.

Walsh (1947)[2], Reese (1953)[3] included illustration of choroidal folds in their publications, without comprehensive discussion of their significance. In 1959, Hedge and Leopold[4], presented 5 cases, derived from a search of the existing

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literature, and a further 2 cases which they had observed among their own patients.

Prior to 1960, choroidal folds were considered characteristic though not invariable indicators of expanding orbital lesions. It was not until 1969 that the matter of choroidal folds received due attention and became subject of full discussion.

Norton (1969)[5], Kroll and Norton (1970)[6] described 10 cases of choroidal folds of which only 4 were associated with tumors.

In 1972 Gass[7] described choroidal folds secondary to hipotony.

In 1973 Newall[8] reported 16 cases of choroidal folds of various aethiologies: 8 due to intra orbital tumors and 8 due to the following: Graves disease, post extraction hipotony, unilateral papilloedema, senile macular degeneration, hypermetropia and uveitis.

In 1979 Bullock and Egbert[9] reported 11 cases and described experimental findings, including histopathology in animals. In this series, only 7 were associated with intra orbital tumors, the others with hipotony, retinal detachment and papilloedema.

In 1977 Cappaert et al.[10] described 6 cases of which 2 were ascribed to acute posterior scleritis but no apparent cause was found in the remaining 4.

The aim of this paper is to describe 6 cases of choroidal folds, wich failed to improve after treatment, out of the 10 we observed in 1982.

Particular emphasis is given to the ultrasonographic findings; the role of ultrasonography in the differential diagnosis with respect to folds secondary to intra orbital tumors and those thought to be idiopathic will be discussed.

AETHIOLOGICAL THEORIES

Many authors have discussed the aethiology of choroidal folds, but no single theory has emerged.

Some causes have been eliminated, including abnormal scleral rigidity, previous choriocapillaris disease, exudative detachment of choroid, pigment epithelium or sensory retina, inner limiting membrane or vitreous.

The theory currently favoured now suggest there is an abnormal adhesion of Bruch's membrane to the underlying choriocapillaris with a relative reduction of its normal elastic properties. This explains why choroidal folds are associated with such a diversity of ocular pathologies.

Any condition which can cause thickening of the choroid or the underlying sclera can cause choroidal folding in the presence of abnormal adhesion between Bruch's membrane and choriocapillaris.

The pigment epithelium passively follows the new contours of Bruch's membrane while the choroid becomes more or less thickened corresponding with peaks and valleys to the folds.

In the absence of these abnormal adhesions Bruch's membrane and the pigment epithelium slide easily over the choriocapillaris as determined by their elasticity, thus, choroidal folds are not seen in association with choroidal congestion or scleral thickening of whatever cause.

Choroidal folds have been described in the following conditions: primary or secondary orbital tumors, orbital pseudo tumors, orbital cellulitis, dysthyroid exophthalmos, posterior scleritis, choroidal malignant melanoma, metastatic choroidal tumors, endophthalmitis, uveitis, phthisis bulbi following scleral laceration or longstanding detachment, severe post operative hipotony, papilloedema, extreme hypermetropia and disciform degeneration of the macula.

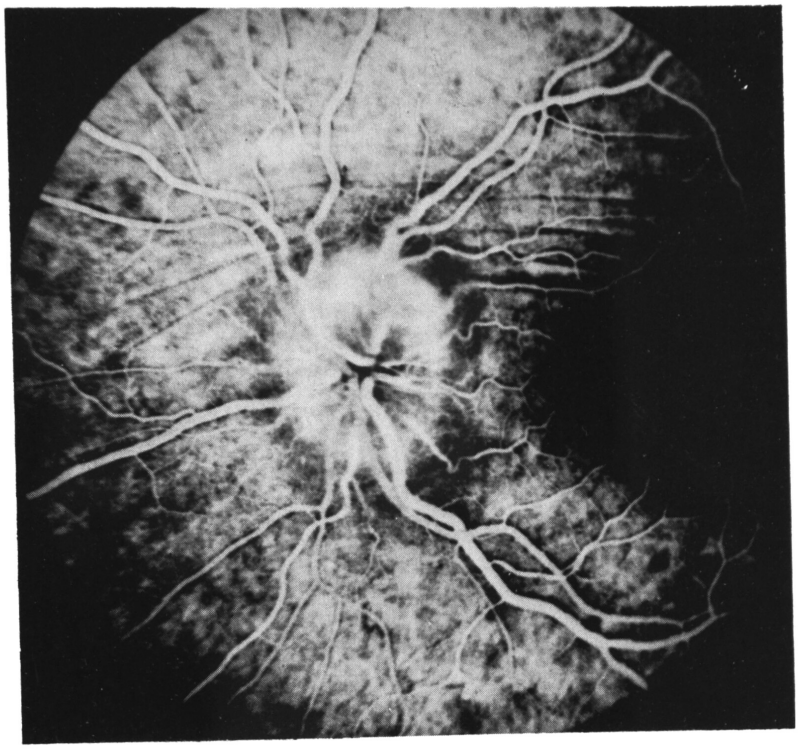
CASE REPORT

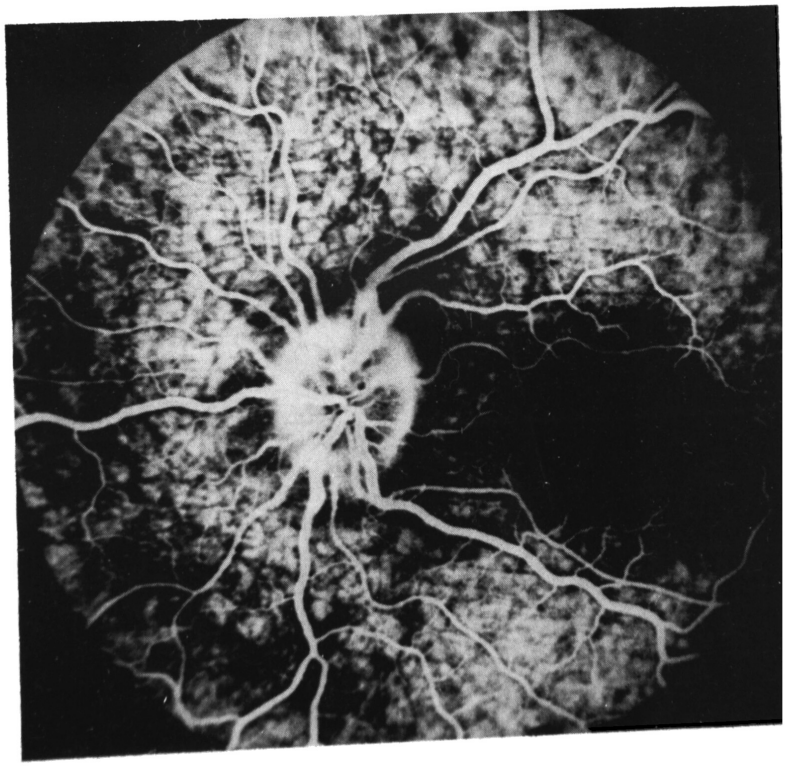
In 1982, 10 patients with choroidal folds were reported to our Department, after a routine eye examination for further testing.

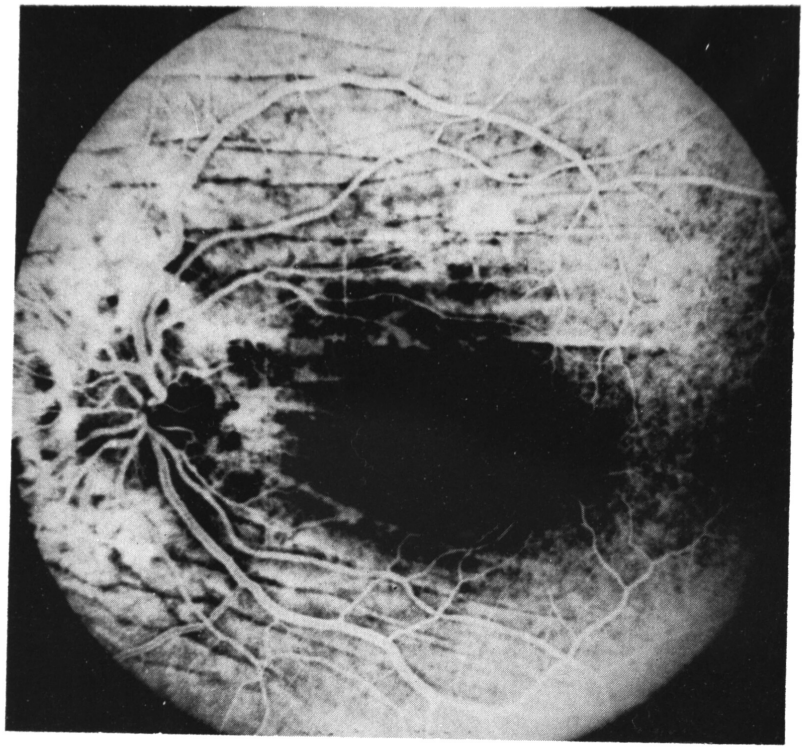
Using the fluorescein angiography the choroidal folds appeared like light and dark lines localized principally to the posterior pole, the lines were virtually parallel but various in lenght and width. (Figs. 1, 2).

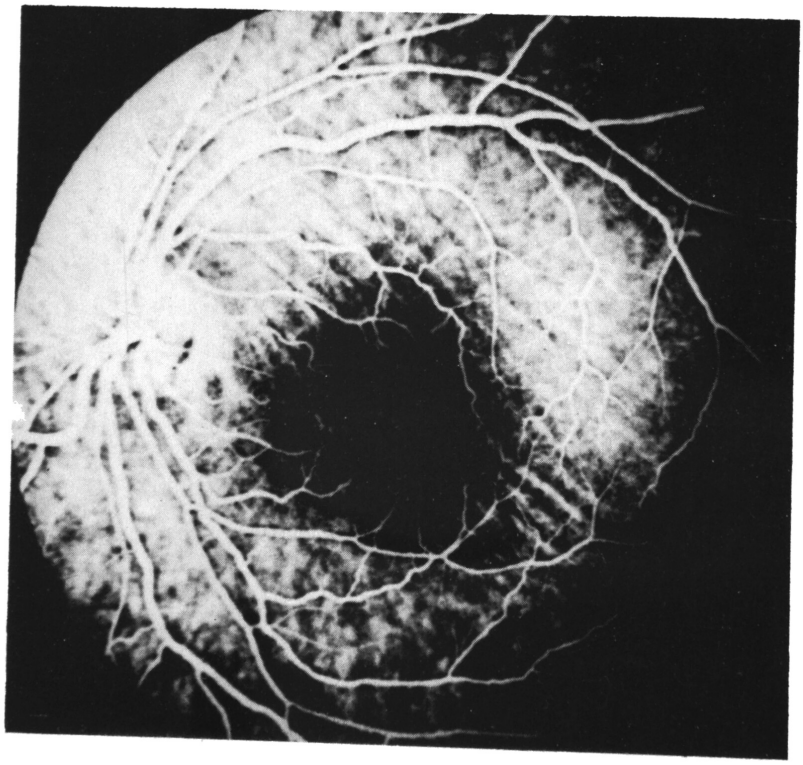
Horizontal, vertical and oblique choroidal folds were present. (Figs. 3, 4, 5). No leakage of contrast medium was present. A rapid B-contact scan was performed allowing resolution of the retino choroido scleral envelope.

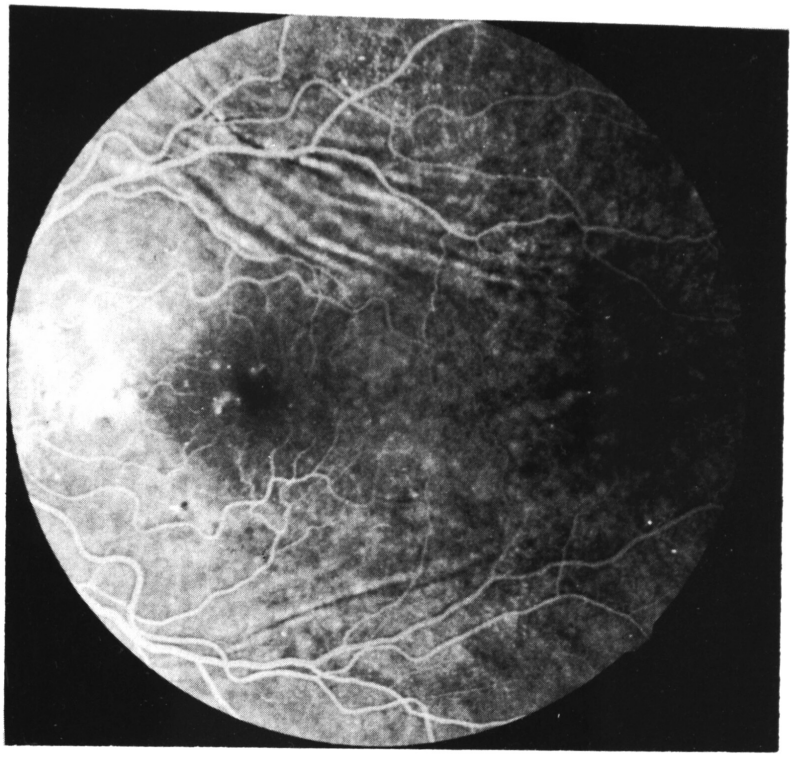
This was distinct from the retrobulbar fat and was of measurable thickness. The posterior pole, in the presence of choroidal folds appeared ultrasonographi-











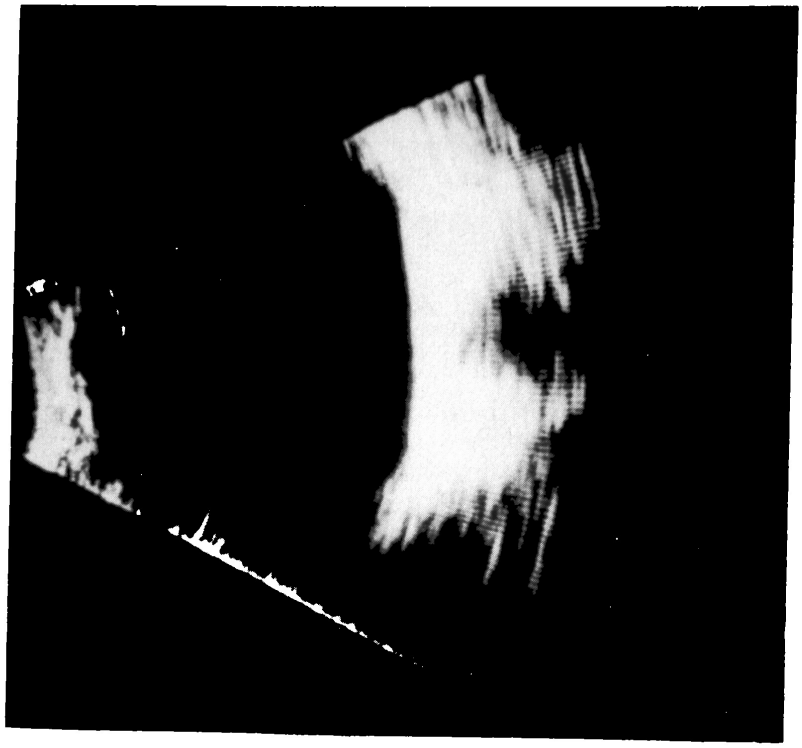
cally to have flatening of the normally curvilinear aspect of the retroequatorial retina. (Figs. 6, 7).

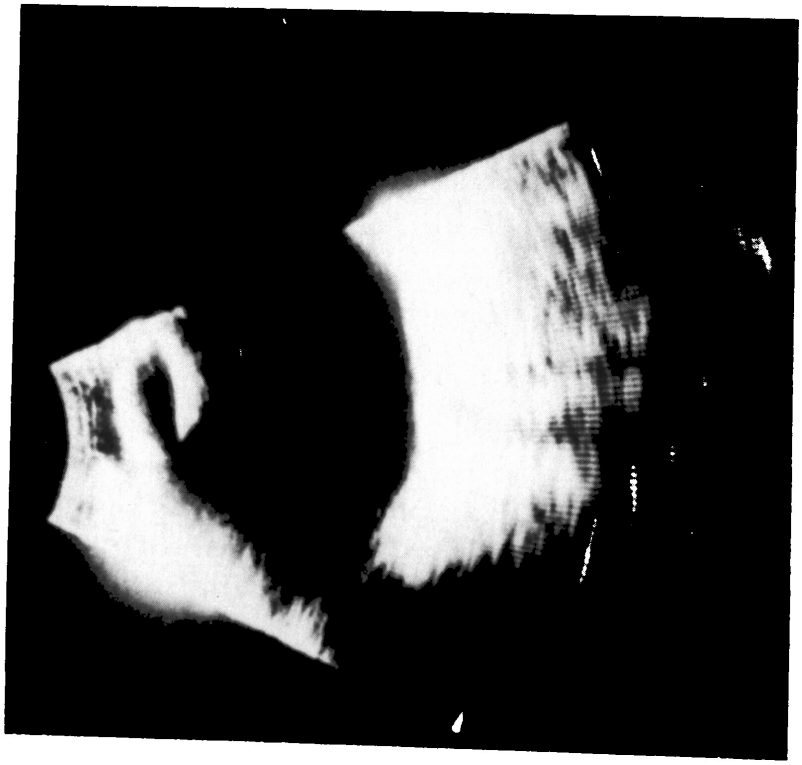
In addition, there was a concomittant thickening of the choroido sclera. It was generally impossible to specify whether the observable thickening seen echographically was due to the choroid, sclera or to both structures together.

It was, however possible to differentiate through the B-scan examination the appearance of localised orbital inflammation, as with acute posterior scleritis, or more diffuse inflammation, as with pseudo-tumor, or the presence of a concomittant orbital tumor.

In 6 of the 10 patients it was impossible to evidence any of these alterations; they were treated with sistemic steroids according to the usual schemes, but no improvement were noticed.

Of the 4 other cases of choroidal folds we treated, one was associated with an orbital tumor that was removed, and the remaining 3 involved a papilloedema and improved after treatment.





CONCLUSION

Choroidal folds are often missed at routine ophthalmological examination as signs of ocular pathology.

Accurate fundus examination using the binocular indirect opthalmoscope or the Goldmann lens with Slit-lamp, should reveal choroidal folds if present. Where there is doubt, fluorescein angiography can demonstrate the typical light and dark lines which run horizontally or vertically in the retroequatorial retina.

It is thus possible to identify choroidal folds using the fore mentioned techniques.

Having diagnosed choroidal folds, the concomittant pathology should be determined as it is only in rare cases that no ocular or orbital cause is recognised. To this end, the use of the contact B-scan is of great value not only demonstrating and localising choroidal folds, but also to exclude or confirm the presence of a solid mass or inflammatory process of globe or orbit.

Our findings support the concept of scleral shrinkage causing benign choroidal folds, when no causal pathology is recognised, as first proposed by Norton 1969[5].

Ultrasonic evidence supports the hypothesis that acute phase of scleritis can cause choroidal folds associated with retro scleral inflammation signs.

Chronic, mild, asymptomatic, inflammation of posterior sclera, clinically undetected, can, on the other hand, cause choroidal folds without any other associated finding. In this occasion, any treatment fails in changing opthalmoscopic, angiographic and echographic appearance as it is happened in our patients.

For this reason we tend to accept the possibility, proposed by Cappaert et al. (1977)[10], that, excluding any causal aethiology and having failed any treatment, the choroidal folds have to be related to a subclinic posterior scleritis.

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