



Synthetic Antimalarial Maculopathy: A Case Report

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ABSTRACT

Antimalarial drug-induced retinopathy was first described in the 1950s. Screening for preclinical poisoning prevents evolution to irreversible maculopathy. We discuss, through the case of maculopathy with antimalarial (AM) revealed by progressive bilateral decrease in vision in a patient with lupus, the modalities of monitoring patients treated with AM and the management of a potential intoxication. All authors stress the need for clinical and paraclinical ophthalmological monitoring regularly to detect early signs of impaired retinal function at a reversible stage. Indeed, at a more severe retinal intoxication, impaired visual function remains irreversible and can lead to blindness. A full ophthalmologic assessment is necessary before starting long course treatment with AM, possibly coupled with additional tests (central visual field, colour vision and/or electrophysiological examinations)..

Keywords: Maculopathy, synthetic antimalarials, systemic erythematous lupus

INTRODUCTION

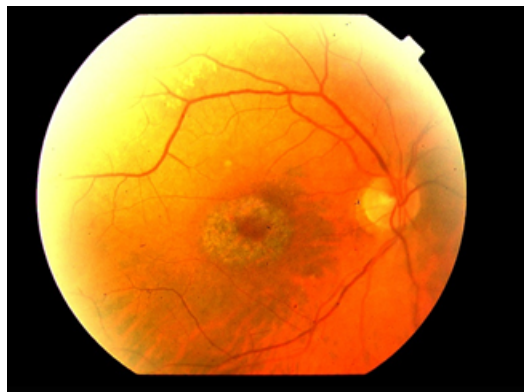
Retinal intoxication with synthetic antimalarials is rare, but remains a possible iatrogenic complication. Screening for preclinical poisoning prevents evolution to irreversible maculopathy. Indeed, at a more severe stage of retinal intoxication, visual impairment remains irreversible and can lead to legal blindness.

We report, through the case of maculopathy with antimalarials (AM) revealed by progressive bilateral decrease in vision in a patient with lupus.

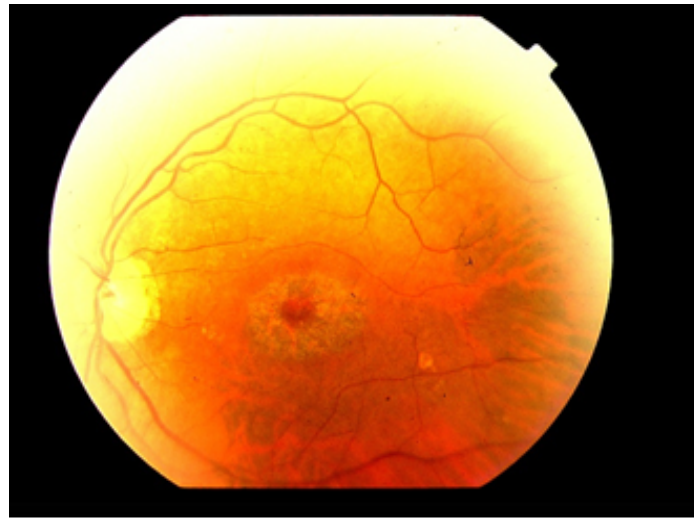
CASE REPORT

We report a case of a 52-year-old patient, followed by systemic lupus erythematosus (SLE) since 1992, treated by Nivaquine (APS) since then at the current dose of 200 mg per day, addressed for a progressive and bilateral decline of vision.

The ophthalmologic examination found a corrected vision at 3/10 right not improvable and 2/10 on the left, an anterior segment of normal appearance to both eyes. The examination of the fundus of eye showed a maculopathic aspect in cockade a in bilateral (Figure 1) allowing to suspect a maculopathy to the synthetic antimalarials.



(a)



(b)

Figures 1 (a) and (b) Fundus of eye showing a maculopathic aspect in cockade in bilateral
Fluorescein angiography showed a bilateral eye-to-bull image (Figure 2).

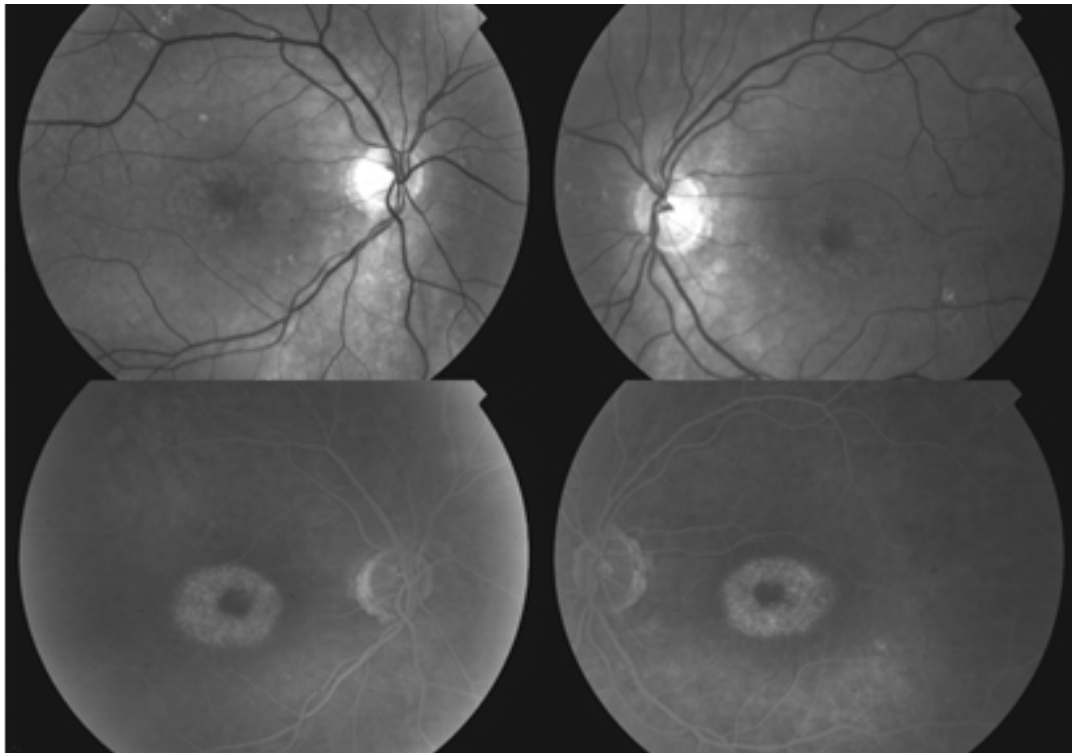
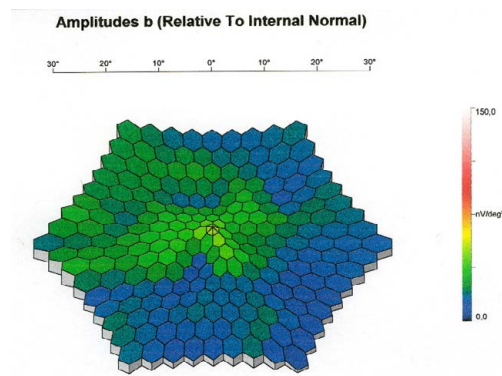
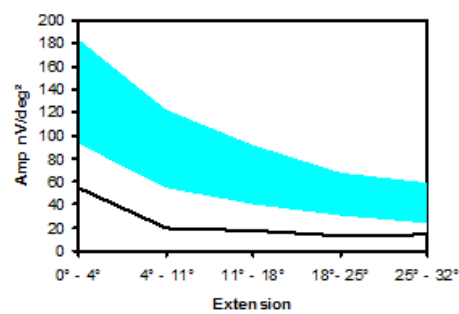


Figure 2 Fluorescein angiography showing a bilateral eye-to-bull image

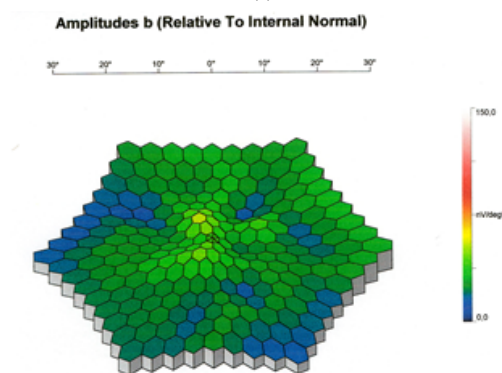
The electroretinogram showed electrical signs of macular bilateral macular retinopathy with photopic ERG with collapsed amplitude, and repercussion on cortical PEVs in accordance with APS maculopathy (Figure 3). The visual field shows a central ring-shaped scotoma (Figure 4).



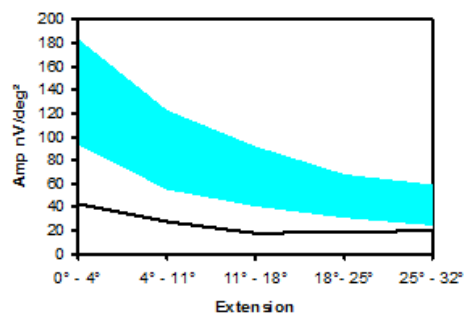
(a)



(b)



(c)



(d)

Figures 3 (a), (b), (c) and (d) Multifocal electroretinogram

It was decided to stop Nivaquine. The evolution was favourable. The vision was increased to 4/10 for the right eye and 5/10 for the left eye.

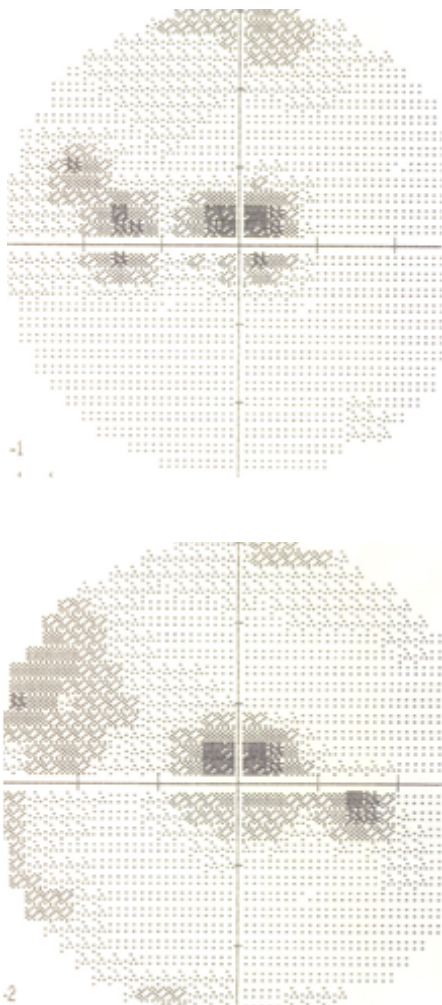


Figure 4 Visual field showing a central scotoma

DISCUSSION

Retinal intoxication with synthetic antimalarials (APS) was first described in the 1950s [1,2]. The prevalence of retinal toxicity is estimated at 4% of long-term APS patients [3].

All authors emphasize the need for regular clinical and paraclinical ophthalmological monitoring in order to detect the first signs of altered retinal function at a still reversible stage [4-6]. All patients undergoing APS should benefit from ophthalmological and complementary examinations of their visual function to detect poisoning at a pre-clinical stage that is still reversible [5].

The rules of prescription are fundamental because there is no curative treatment and maculopathy is irreversible. It is advisable not to exceed 4 mg/kg/d [7] to reduce the risk of retinal intoxication after years of treatment.

A complete clinical ophthalmologic examination is required before APS treatment in the long term, possibly coupled with complementary examinations (central visual field, colour vision and/or electrophysiological examinations). Regular monitoring is then provided.

In the absence of risk factors and abnormalities in the initial assessment, the surveillance is annual, including a clinical assessment combined with a complementary assessment (central visual field, colour vision and possibly

electrophysiological assessment) for subclinical anomalies. However, there are no absolute rules and surveillance must be tailored to each patient according to age, ophthalmological history, pathology and cumulative dose.

Several teams have shown since 2003 [8] that multifocal ERG can detect early and localized decreases in retinal function in long-term APS patients.

For Maturi, et al. [9], the multifocal ERG seems to detect retinal changes earlier than the measurement of visual acuity, colour vision assessment or even the Amsler grid.

Moschos, et al. [10] also emphasized the utility of multifocal ERG in this prevention. They also showed in 3 patients that after 6 months of treatment discontinuation, ERG responses improved.

When retinal intoxication of pre-clinical APS is diagnosed, the treating physician should be advised, depending on the risk-benefit ratio and possible therapeutic alternatives, to discontinue the treatment or at least to revise and adapt the dosage to 'Try to slow down or even stop the evolution.

CONCLUSION

AM treatments still require careful attention to avoid iatrogenic irreversible retinal blinding intoxication. An initial ophthalmologic clinical and paraclinical review must be performed before initiating treatment. Regular monitoring allows the diagnosis of incipient intoxication at a "preclinical" stage.

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