Retinal findings in vitamin A deficiency secondary to a liver fibrosis

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Introduction

Vitamin A (retinol) is an essential fat-soluble micronutrient related to proper functioning of the visual system, more specifically in visual phototransduction[1],[2]. The human body needs an adequate intake of this vitamin witch is mainly stored in the liver [2]. Therefore vitamin A deficiency (VAD) is observed in patients with liver disfunction[3]. In retina the deficiency affects the rods and cone dysfunction resulting in night blindness, impairment of daytime vision and visual acuity[2].). In the fundoscopy, is seen multiple punctate dots in the posterior pole (4) At the full-field electroretinography (ERG) exam is observed extinguished scotopic rod-specific function (5) The supplementation restore the retinal function as well as structure, however long-term deficiency can lead to permanent degeneration of photoreceptors (1).

Case report

A 19-year-old female patient with dialytic polycystic kidney disease and liver fibrosis attended the ophthalmological consultation complaining of ocular pain and nyctalopia in both eyes (BE). At examination presented uncorrected visual acuity (UCVA) 20/25 in BE. At fundoscopy (FO), multiple punctate hypopigmented dots were observed in the periphery of the BE. Thus, the hypothesis of VAD causing retinal changes was raised.

Therefore, the following complementary tests were requested: color retinography (RET), autofluorescence (AF), full-field electroretinogram (ERG) and computerized visual perimetry (VF), and vitamin A dosage (figures 1,2 and 3).

Vitamin A dosage showed 0.1 mg/L (adult reference value: 0.3 – 0.7 mg/L), confirming the initial diagnostic hypothesis. It was suggested to start the replacement, but the nephrology team contraindicated it at that time due to the risk of intoxication secondary to renal failure.

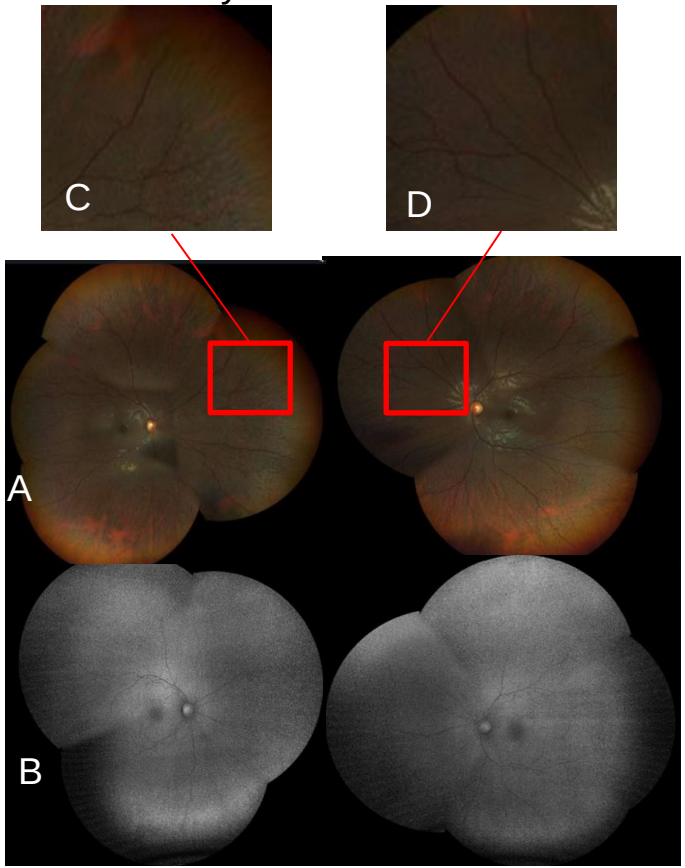

FIGURE 1: RET and AF showing multiple punctate dots in the periphery

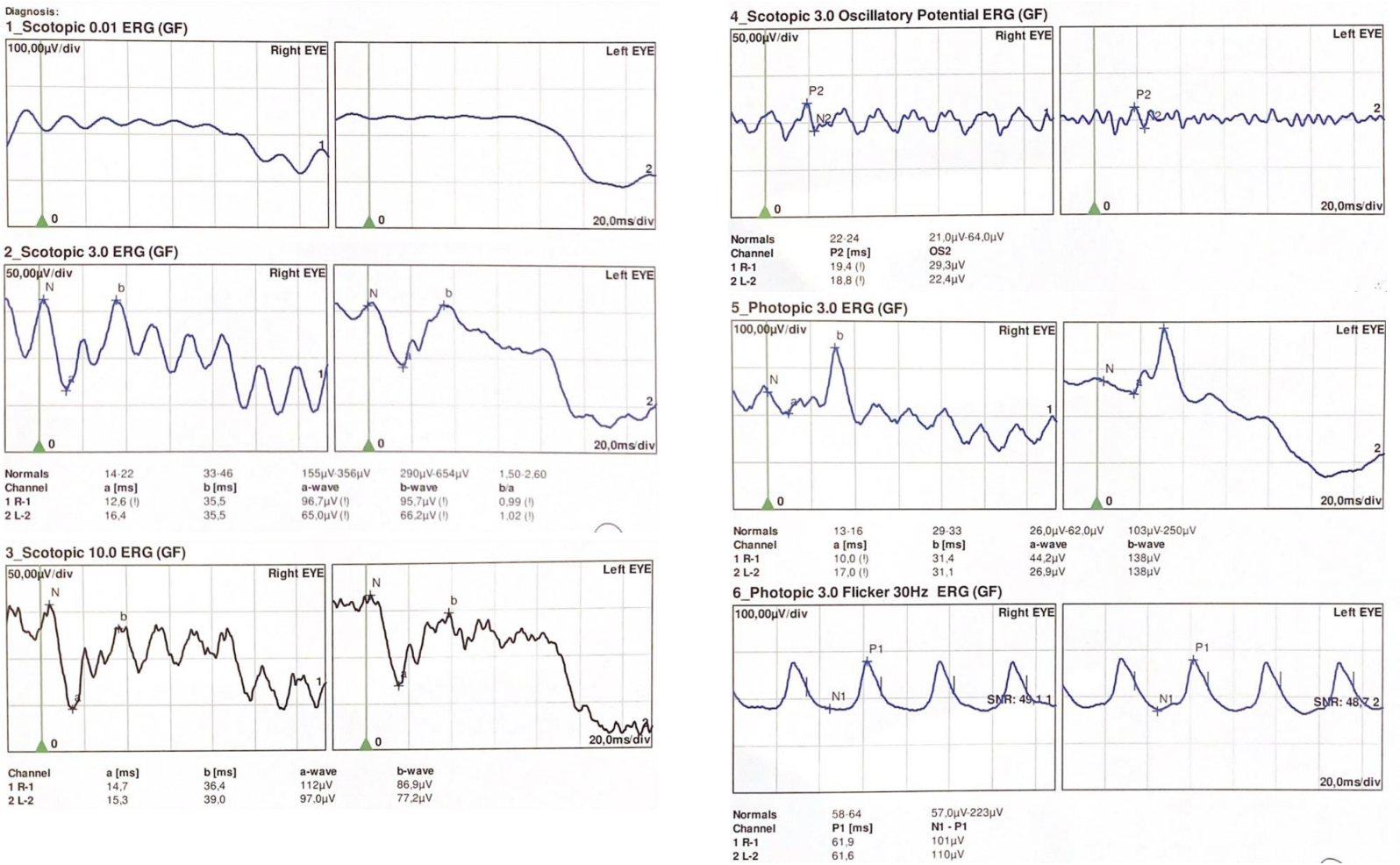

Figure 2: ERG exam manifesting a undetectable rod response in isolated phase and altered b/a ratio.

Discussion

The retinal functions affected by vitamin A deficiency has been a matter of interest from ophthalmologists. Once retinol combines with opsin to form rhodopsin, which is the visual pigment of rods, patients with VAD initially refer night blindness, as reported by the patient in this case, followed by impairment of daytime vision and visual acuity, demonstrating a posterior cone damage (2).

The structural affection can be demonstrated at the ERG exam. Saker at al found moderate reduction in b-wave in dark-adapted 0.01 ERG, while we found a undetectable rod response. Breazzano et al. described extinguished scotopic rod-specific function (5) as shown in this case (figure 2). In the FO, multiple punctate dots are classically described in VAD cases ass seen in our patient (4).

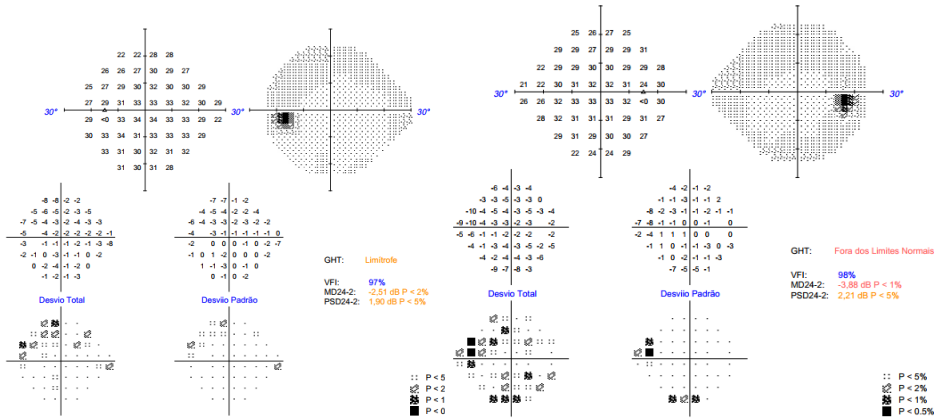

FIGURE 3: VF evidencing a nasal superior scotoma on the right eye (OD) and a temporal superior and inferior scotoma on the left eye (OS)

Visual field contraction is expected in VAD, although in this case we found unspecific visual field scotoma (FIGURE 2), unable to state a correlation with the VAD (7).

All theses retinal modifications are supposedly reversible after vitamin A replacement if its starts in the begging of the case. It is expected that vision will return to normal when supplying all vitamin stores (1).

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