INTRODUCTION

This maculopathy is caused by whiplash injury demonstrated three features that are characteristic of this subtle disturbance of the macula: a history of flexion-extension, head and neck trauma; a history of immediate mild reduction of central visual acuity in one or both eyes, and grayish swelling of the foveal zone accompanied by a small (50 to 100-mu) pit or depression in the fovea. In patients with this disturbance, the retinal opacification and the visual disturbance are transient, but the tiny depression in the retina with its whitish border can be permanent.

OBJECTIVE

To report a case of unilateral traumatic retinopathy associated to whiplash injury, and its long-term evolution.

METHODS

A 62-year-old man involved in a minor road traffic accident, with a history of flexion-extension and neck trauma (without any bony injury and without thoracic compression by the seatbelt), developed an immediate mild reduction of central visual acuity in his right eye, which remained unchanged during one year, when he was referred to our hospital. A complete ocular examination, including determination of Visual Acuity (VA), anterior and posterior biomicroscopy, optical coherence tomography (OCT), and fluorescein angiography (FA), was performed.

RESULTS

AT FIRST VISIT (1). VA was 0.3 (decimal notation) in its right eye and 1.0 in its left eye. Anterior segment examination showed no alterations. Funduscopy revealed the presence of small yellowish lesions over the fovea with loss of the normal foveal reflex, but no hemorrhages. Fluorescein angiography showed an area of vessel leakage over the fovea. OCT revealed a disruption of external retinal layers (IS/OS—internal segments and outer segments of photoreceptors, and ELM—external limiting membrane) with a hyperreflective lesion under the ELM. TWENTY MONTHS LATER (2). VA remained unchanged and funduscopy, angiographic and tomographic findings showed similar.

DISCUSSION

So, as discussion, it is interesting to remark that the pathogenesis of the fundal appearances in these cases is discussed. Local microcirculatory disturbances are postulated as the cause of the retinopathy as opposed to those associated with other traumatic retinopathies as Purtischer’s retinopathy (its possibility was eliminated in our case, due to the absence of history of thoracic trauma and long bone fractures, and also due to the absence of some characteristic clinical signs such as wool spots and retinal hemorrhages). On the other hand, it can be also a result of traumatic posterior vitreous detachment at the macula, leading to photoreceptor damage and foveal change.

CONCLUSION

This condition may be underdiagnosed as there may be few abnormal signs on funduscopy. Fluorescein angiography may be very helpful. Increasing awareness may increase diagnosis, which it is important because VA, although may improve, there exists also the possibility that it remains stable (such as occurred in our patient), even that it suffers deterioration over time. Moreover, understanding these conditions, is important not only in patient management but in circumstances of medico-legal significance.