

## *C. burnetii* Chorioretinitis: A Case Report

**Paolo Amato\*, Matteo Forlini and Alessandro Mularoni**

Ophthalmology Department, I.S.S. Istituto Per La Sicurezza Sociale, Repubblica di San Marino, Via La Toscana, San Marino

\*Corresponding Author: Paolo Amato, Ophthalmology Department, I.S.S. Istituto Per La Sicurezza Sociale, Repubblica di San Marino, Via La Toscana, San Marino.

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### Abstract

Q fever chorioretinitis

The Q-Fever is a disease caused by *Coxiella burnetii*, infrequent zoonosis which rarely involve the ocular structures. *C. burnetii* is vehiculated by a organic/inorganic particles dispersed in the air (droplets). For this reason the primary site of infections is the respiratory tract. Clinically the Q-Fever may be misdiagnosed for a common flu also because of its self-limiting nature. The acute stage may be followed by the chronic phase in 5% of cases in which can be involved the cardiac and hepatic district [1,2]. The early diagnosis can permit the reduction of chronicity index and therefore the tissue damage and related organ failure, thanks to the use of doxycycline as first line therapy.

A great variety of chorioretinal findings was described related to infections of *R. conorii*, but very poor data was reported in literature concerning ocular findings in *C. burnetii* infection [3,4].

**Keywords:** *C. burnetii*; Fever; Chorioretinitis

### Case Presentation

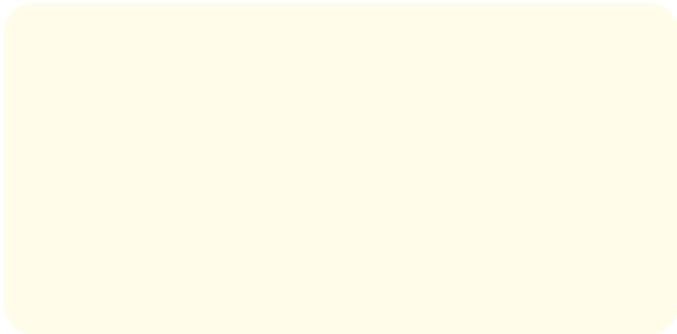
The present case concerns a 62-year-old man who referred at our eye clinic after a diagnosis of Q-Fever. The patients experienced a sudden and progressive vision loss few days after diagnosis was made. IgM titre against *C. burnetii* was 1:1253. The visual impairment corresponding to acute phase of the infections. Doxycycline 100 mg one per day treatment was started.

At the ophthalmological examinations we reported that:

- His Best Corrected Visual Acuity (BCVA) was 0.2 (logMAR) in both eyes.
- The anterior segment was normal
- The fundus exam revealed a wide peripapillary chorioretinal atrophy which in turn involved the macular region with small similar lesions that surrounded the widest. Any vitritis sign was found.
- The Electroretinogram (ERG) showed deep reductions in the first order responses in each ring with a major impairment in the right eye (RE) and a central residual activity in the left eye (LE), such data was accompanied by the evidence of a macular bipolar cells disfunction.
- The VF 24-2 displayed a large area of hyposensitivity in both eyes. The OCT showed wide area of Thinning and atrophy of the outer retina/outer nuclear layer (ONL).
- Autofluorescence revealed Ipo-Autofluorescence at the posterior pole centered to peripapillary region with a spearing in peripheral retinal regions figure 1.
- OCT shows reduction of outer retinal layers with atrophic appearance figure 2.
- A deep impairment of contrast sensitivity was present.
- A DNA test in aqueous humor was performed, but no presence of *C. burnetii* genetic material was found.



**Figure 1:** Chorioretinal atrophy located to posterior pole. Granular area “salt and pepper” mixed to patchy wide atrophy.



**Figure 2:** OCT shows external retinal layers atrophy and a thin choroid.

According to meningoencephalitis Q-fever related, where *C. burnetii* has been rarely detected in the cerebrospinal fluid [6]. These data support the evidence that *C. burnetii* doesn't play an active role to damage the tissues, but in turn is the inflammatory response of the guest that should be incriminated.

One-year follow up showed a clinical stability, but any significant amelioration in clinical and instrumental parameters was reported.

### Discussion and Conclusion

Posterior segment involvement in infectious disease is very frequent. In the family of Rickettsia (*R. conorii* first) of which family *C. burnetii* is part, the ocular manifestations in far infrequent. Non granulomatous anterior and/or posterior uveitis is the common presentations of *C. burnetii* infections. In the case of *R. conorii* which cause the Mediterranean Spotted Fever (MSF) of 30 patients studied, 25 (83.3%) had unilateral posterior segment involvement related to MSF, despite the major part of patients doesn't reported visual impairments [5]. In Q-Fever ocular findings may be misdiagnosed and confused for any others entities, like a Vogt-Koyanagi-Harada syndrome (VKH) with a multiple serous PED and serous retinal detachment [3]. Optic neuritis was also described in farmers as a zoonosis conveyed by the cows [7]. During Q Fever a choroidal neovascularization was found as a epiphenomenon of inflammatory stimulus [8]. Despite the evidence in which this bacteria is rarely find in the ocular structures involved by a pathological process, the uveitis of unknown etiology should be investigated also for *C. burnetii* infections to permit an early diagnosis aimed at preventing the systemic involvement [6]. In this case we noted how the ocular response to treatment has not been fully achieved despite the preservation of BCVA. Anatomical (OCT,

Autofluorescence) and functional (ERG, CVC, CS) outcomes gained stability after the first month of treatment and remained stable to this day.

These data shows the rationale for investigate the presence of intracellular bacteria as *Coxiella* and *Rickettsiae* in all the cases of uveitis of unknown etiology, especially if related to systemic symptoms.

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