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Sir,

Abnormal corneal nerves in a patient with Lyme disease

Lyme disease is a multiorgan disease caused by a spirochete, *Borrelia burgdorferi*. Although numerous ophthalmologic manifestations have been described following Lyme disease,[1,2] this case describes a new feature: a bilateral corneal neuropathy.

Case report

A 69-year-old woman was referred for a suspicion of corneal dystrophy in both eyes that appeared during the last 6 months. Her past medical history was relevant for a tick bite complicated by Lyme disease 2 years ago. Despite oral antibiotic treatment (clarithromycin), she still suffered from peripheral neuropathy in the lower extremities.

She presented with complaints of decrease vision bilaterally. Best corrected visual acuity was 20/25 in both eyes. Slit-lamp examination revealed irregularly enlarged corneal stromal nerves in both eyes (Figure 1). These abnormalities did not reach the central cornea and were predominantly observed in the mid-peripheral area of the right eye. Except this finding, corneal examination was normal in both eyes. No sign of inflammation of the anterior segment was observed. Examination of the posterior segment of both eyes was normal. The slight decrease in visual acuity was explained by a mild bilateral cataract. Using the Cochet–Bonnet aesthesiometer, a marked hypoesthesia was observed in the central cornea of the right eye (3.5 mm), whereas the corneal sensitivity was subnormal in the left eye (5.5 mm). Except the corneas, the sensitivity in the area innervated by the trigeminal nerves was not altered. *In vivo* confocal microscopy (HRT3, Heidelberg Engineering, Heidelberg, Germany) revealed enlarged stromal nerves with tortuous and abnormal branching in both eyes. Some nerve fibers showed a hyper-reflective peri-nerve infiltration (Figure 2).

Discussion

Cranial nerve involvements are well documented in early disseminated Lyme disease,[3] but in the presented case, only the corneal nerves of both eyes showed alterations that were observed 2 years after the infection. In our patient, the association of distal paresthesia, suggesting axonal polyneuropathy and corneal nerves alterations, strongly supported the responsibility of Lyme disease. The mechanisms responsible for peripheral

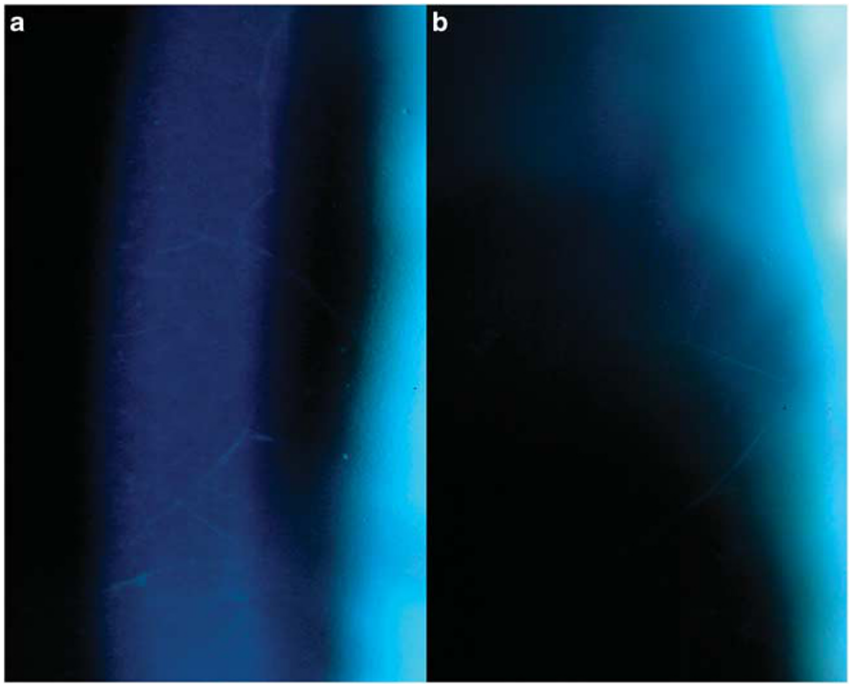

Figure 1 Slit-lamp images (× 20) of irregularly enlarged nerves within the corneal stroma of the right eye (a) and the left eye (b).

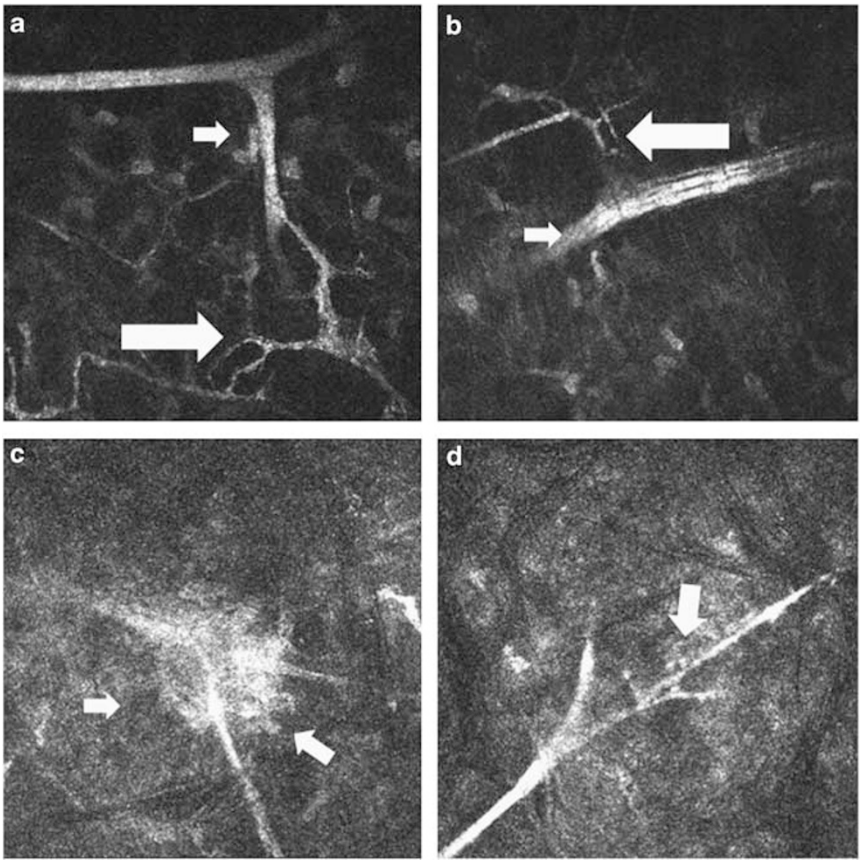

Figure 2 *In vivo* confocal microscopy images (400 μm × 400 μm) of the corneas of both eyes showing corneal nerve alterations. Irregular enlargements of stromal nerves (small arrows) with thin tortuous and abnormal branching (large arrows) were observed in the stroma of the right (a) and the left eye (b). Some nerves demonstrated a peri-nerve hyper-reflective infiltration (small arrows) (c, right eye and d, left eye).

nerve damage in Lyme disease remain unclear. Nevertheless, peripheral sensory symptoms are frequent and could appear months and years after the onset of infection even with an adapted treatment.[3,4] Eye examination is not systematically performed in Lyme disease, the absence of symptoms may explain why this corneal neuropathy remained unobserved. Larger studies evaluating corneal nerves morphology and functionality in Lyme disease are now needed to confirm this finding.

Conflict of interest
The authors declare no conflict of interest.

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