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Bilateral Optic Neuropathy Following bite from Brown Recluse Spider (*Loxosceles reclusa*)

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Abstract

A 63-year-old female with history of a resected frontal lobe meningioma presented with bilaterally decreased vision after a bite from a brown recluse spider. The exam was significant for a left relative afferent pupillary defect, bilateral optic nerve pallor, decreased foveal sensitivity in the left eye, and new bilateral visual field defects, despite stability of her meningioma. The findings remained stable at one year follow-up. To our knowledge, this is the first reported case of optic neuropathy secondary to a brown recluse spider bite. Visual field tests performed prior to the bite allowed us to compare and localize changes related to the bite.

Keywords

Brown recluse spider; Optic Neuropathy; toxic; envenomation; *Loxosceles reclusa*; spider bite

Introduction

Reports of ocular sequelae after insect or animal bite (e.g., envenomation retinopathy/optic neuropathy) have become more frequent since their first description in 1960. [1] Such complications are more commonly related to encounters with bees, [2] wasps, [3] and ticks [4], while other causes include mosquitoes [5], scorpions [6] and poisonous snakes [7]. To the best of our knowledge, here we present the first case of optic neuropathy following a brown recluse spider (*Loxosceles reclusa*) bite.

Case Report

The patient was a retired 62 year-old female from Ohio, first referred to our clinic for a baseline examination after frontal lobe meningioma resection (located at the anterior cranial fossa below the falx, the left anterior frontal lobe and near the high convexity more posteriorly along the falx). The post-operative brain Magnetic Resonance Imaging (MRI) was significant for changes related to the surgery. The visual acuity was 20/20 OU while the ophthalmologic examination was significant for 1+ cataracts OU; there was no optic disc

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edema or pallor. A baseline 24-2 Humphrey Visual Field (HVF) was performed during that visit and documented the patient's baseline visual field defects. The same test was repeated in the follow up exam, 17 months later, but was negative for any changes (Fig. 1 A and B)

Eighteen months after the second visit, while in Wyoming, the patient was bitten by a brown recluse spider in the right index finger. Within 24 hours, she experienced excruciating pain and upper extremity edema. She was admitted to a local hospital for necrotizing fasciitis, which required several operations of skin debridement followed by graft placement. The patient also developed fever (up to 102°F), tachycardia (up to 110bpm), tachypnea (up to 40 breaths per minute) and leucopenia (minimum WBCs of 3.0 K), but repeated blood cultures failed to grow any organisms. Thus, she met the criteria for systemic inflammatory response syndrome (SIRS) which did not respond to treatment with empiric antibiotics including ampicillin/sulbactam, piperacillin/tazobactam and clindamycin. Moreover, she developed hypochromic anemia (minimum Hgb of 8.2, MCH of 27, MCHC of 32) and acute tubular necrosis with subsequent acute kidney injury, which were managed conservatively, without transfusion or hemodialysis. Overall, she was an inpatient for a total three months, four weeks of which were in the medical intensive care unit (MICU). From an ophthalmologic standpoint, the patient was unsure exactly when her vision became affected, but recalled that she first noticed a "film" OS>OD as soon as she regained consciousness in the MICU, about three weeks post injury.

One month upon discharge she was referred to Neuro-Ophthalmology for evaluation of persistent vision loss OS>OD. This symptom had been constant since discovery, while she denied other ocular complaints. The examination showed best corrected visual acuity 20/25 OD and 20/40 OS, 1+ relative afferent pupillary defect (RAPD) OS and the anterior segment was quiet with 2+ nuclear sclerosis OU. Dilated examination showed optic disc pallor OU. The 30-2 HVF revealed new visual field defects OU (Fig. 1 C and D) and decreased foveal sensitivity OS, from 37 dB (Fig. 1 B) to 24 dB. (Fig. 1 D). OCT RNFL showed decreased RNFL thickness OU, more pronounced temporally (Fig. 2 C and D). Thiamine, folate and Vitamin B12 levels were normal. Given the concern for visual symptoms secondary to meningioma progression, brain MRI was repeated but showed stability of the lesion.

By the one-year follow-up the patient had uneventful cataract surgery OU, her vision was 20/30 OD and 20/40 OS and the exam was negative for RAPD. Dilated fundus exam was significant for optic disc pallor OU (Fig 2 A and B) while the 30-2 HVF showed stable visual field defects OU (Fig. 1 E and F) with persistently decreased foveal sensitivity OS (Fig. 1 F). A series of 9 brain MRIs, performed in the 7-year period after meningioma resection, was negative for any evidence of tumor progression. Moreover, 3 of these MRIs, performed within 18 months after the spider bite did not show any radiologic evidence of new abnormal process in the brain or the optic nerve (Fig. 2 F).

Discussion

Brown recluse spider (*Loxosceles reclusa*) is endemic in 16 states of the central and southern United States and accounts for most episodes of spider envenomation in its native range. [8,9] Commonly reported manifestations are dermatologic, [10] hematologic, [11,12]

nephrologic, [10] musculoskeletal [10] and neurologic [13]. Despite the research performed [14] and the Loxosceles- sensitive ELISA created [15], as of today the diagnosis remains clinical, based on history provided by the patient. The need for treatment is highlighted by the various modalities used so far including dapsone, glucocorticoids, hyperbaric oxygen and surgery. [8] However, currently, there is no consensus for the treatment of such conditions.

During her admission to the hospital, our patient had most of the previously described systemic sequelae of brown recluse spider bite including cellulitis, hemolytic anemia, and acute renal failure [10] [16]. However, to the best of our knowledge this is the first reported case of vision loss secondary to brown recluse spider bite and the second related to any spider bite at all. The previous one was a case of transient unilateral optic neuritis following black widow spider bite in Brazil [17]. Although our patient was bitten in Wyoming, she reported that the spider came out from the luggage she had brought from her garage in Ohio, which is one of the states where this spider population is endemic. [8] Thanks to the visual fields obtained before (series of 2 HVFs) (Fig. 1 A–B) and after (Fig. 1 C–F) the incident (series of 3 HVFs), we were able to make useful comparisons and locate the new changes. Based on the RAPD, the optic nerve pallor and the visual field changes we suspect that the main etiology of this patient's symptoms was asymmetric bilateral optic neuropathy. Similarly, bilateral, OCT-measured, RNFL thinning in this patient without other glaucomatous changes is consistent with this hypothesis (Fig. 2 C and D). However, central visual field depression together with persistently decreased foveal sensitivity OS (Fig 2 B, D and F) cannot rule-out the contribution of an occult maculopathy.

The exact mechanism of optic neuropathy in this patient is unclear since there are no previously reported cases of optic neuropathy after brown recluse spider bite. Thus, we can only make hypotheses about the pathophysiology based on the literature of optic neuropathies after wasp or bee stings. Proximity and laterality of the bite to the affected eyes are some of the previously reported risk factors; [18,19] however our patient had visual changes, more pronounced in the left eye, after a bite in the right index finger. This large distance from the affected target tissue argues against a direct toxicity mechanism in this patient. Other anaphylactic, vasculopathic and autoimmune processes have also been implicated in similar cases. [18,19] Interestingly, fever and post-operative state are some of the non specific factors that may act as adjuvants accelerating the immune response [20] and might have played an important role in our patient with prolonged stay at the MICU. It is also possible that this patient was more susceptible to optic neuropathy due to the history of treated meningioma, with potentially less neurologic reserve. Finally, even though the patient remained normotensive during her hospitalization, according to the official records, it is possible that she also experienced hypotensive episodes, compounded by anemia, which could have contributed to her optic neuropathy. Nevertheless, the visual field defects she developed did not have altitudinal defects consistent with nAION.

Although systemic corticosteroids have been tried successfully for other cases of envenomation optic neuritis treated within hours or days after injury [19,21] we believed that our patient would not benefit from this treatment given the delayed presentation to the

clinic (four months after the bite). Finally, there was no evidence of progression during the six-month or one-year follow up examination (Fig. 2 E–F).

Currently, our poor understanding of the different mechanisms by which insect bites affect vision is a the main cause of limited treatment options. Studying the immunologic and toxicologic process, immediately after the incident, can allow for more focused and efficient treatments.

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Figure 1.

A–F Humphrey Visual Fields (HVF) of both eyes. 24-2 HVF after craniotomy for bifrontal meningioma resection and before brown recluse spider bite showing mild superior hemifield defect and inferonasal mild constriction OD (A) as well as mild non-specific visual field defects OS (B). 30-2 HVF, four months after the spider bite, showing mild general depression on top of superior hemifield defect respecting the midline OD (C) and new general depression on top of an inferior hemifield defect respecting the horizontal midline OS (D). Foveal sensitivity OS (24dB) on the same day was decreased from baseline (37dB). 30-2 HVF during the one year follow up, and after cataract surgery OU showing superior

arcuate defect and peripheral nasal constriction (E) and general depression OS (F). The foveal sensitivity remained low OS (27dB).

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Figure 2.

Fundus photos, during the 1-year follow up exam, showing moderate temporal pallor OU. The optic discs were not suspicious for glaucoma, based on the small cup to disc ratio (A and B). OCT-RNFL with decreased RNFL thickness, after the spider bite, but overall stable in the 6-month and the 1-year follow up OD (66 μ m, 69 μ m and 71 μ m RNFL thickness respectively (C)) and OS (73 μ m, 74 μ m and 71 μ m RNFL thickness respectively - (D)). Pre-operative axial T2 MRI (six years prior to spider bite), showing plenum sphenoidale meningioma, extending up to the right and left orbital apices (E). The last brain MRI, performed 7 years after meningioma resection and 1 year after the spider bite (F) was negative for any evidence of meningioma enlargement.