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Primary ocular toxoplasmosis secondary to venison consumption

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ABSTRACT

Purpose: To describe primary ocular toxoplasmosis infection related to ingestion of undercooked venison.
Observations: This single site, retrospective case series reviewed 4 patients with primary ocular toxoplasmosis that was acquired by ingesting undercooked venison. De-identified data was collected regarding baseline patient characteristics including age, sex, past medical and ocular history, onset of symptoms, visual acuity (VA), response to treatment, and workup. All patients with acquired toxoplasmosis had similar chronology of systemic and ocular symptoms. Exposure occurred in October or November and systemic symptoms developed within 2 weeks, followed by ocular symptoms an average of 2.6 months later. Average age at onset was 56 ± 13 (age ± SD) years old and all were male. Average initial and final VA were 20/50 and 20/50, respectively. Positive anti-toxoplasma IgM and IgG serologies were found in all cases. All patients were treated with trimethoprim/sulfamethoxazole and achieved rapid improvement. Complications occurred in 50% of cases and included epiretinal membrane, cystoid macular edema, vitreoretinal traction, and neovascularization.
Conclusions and importance: Consumption of undercooked venison is a source of primary ocular toxoplasmosis even in immunocompetent hosts and has a clear chronology. A presentation of retinochoroiditis during the winter months should prompt questioning for exposure to wild game.

1. Introduction

Toxoplasmosis gondii is regarded as one of the most common causes of posterior uveitis in the world.[1] Ocular disease is characterized by unilateral, necrotizing retinitis with secondary choroiditis, occurring adjacent to a pigmented retinochoroidal scar and associated with retinal vasculitis and vitritis.[2] In the United States, it is estimated that 22.5% of the population is seropositive for *T. gondii*, while only 2% of those individuals have ocular disease.[3,4] The vast majority of these cases are thought to be related to reactivation of postnatally acquired infections, in which the initial ocular infection either went unnoticed due to minimal symptoms or having occurred in preverbal children.[3] Although past literature presumed the vast majority of ocular toxoplasmosis as reactivation, recent data suggest the prevalence of primary ocular toxoplasmosis may be >10% of cases.[5] Transmission of *T. gondii* can occur by several methods, including ingestion of undercooked meats such as pork, sheep, cattle, exposure to contaminated water or soil, or working with waste.[1] Reports of transmission by ingestion of undercooked venison are limited, but there is growing evidence to suggest a relationship.[6–10] The white-tailed deer (WTD) is considered a major wildlife reservoir for *T. gondii*, and seropositivity increases with the age of the deer.[11] The seroprevalence of *T. gondii* in WTD varies in the United States, with reports of 22.5%–32.2% in Minnesota, 53.5% and 74.4% in the neighboring states of Iowa and Ohio, respectfully.[11–13] WTD hunting season begins during the fall months every year and, consequently, this time period lends itself to isolated outbreaks. Herein, the authors report four cases of primary systemic toxoplasmosis followed by primary ocular toxoplasmosis infection shortly after ingesting undercooked venison in Minnesota.

2. Findings

Verbal informed consent was obtained from all patients, including permission for publication of all photographs and images included herein. Institutional Review Board (IRB) approval was obtained, and the study was performed in accordance with the tenets of the Declaration of Helsinki.

Case 1. A 56-year-old man went on a hunting trip for WTD with 12 other individuals in northern Minnesota in early November 2017, during

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which they processed several WTD and ate undercooked venison. Two weeks after the hunting trip, 6 of the 13 individuals developed fevers, night sweats, myalgias, and weight loss. After several emergency department visits and extensive workup for fevers of unknown origin and respiratory symptoms, the patient was treated for an atypical pneumonia with a course of oral doxycycline, followed by ceftriaxone and levofloxacin. Chest computed tomography scan showed small airway disease, nonspecific nodules, and hilar lymphadenopathy. Additional negative workup included testing for Ehrlichiosis, Anaplasma, Lyme, human immunodeficiency virus, Legionella, Coccidiosis, Histoplasma, antineutrophil cytoplasmic antibodies, acid-fast bacillus sputum stain, and extensive respiratory panel (coronavirus, metapneumovirus, influenza, parainfluenza, pertussis, Chlamydia pneumoniae, and mycoplasma). In mid-December 2017, he suffered bilateral pulmonary emboli in the setting of lower extremity deep venous thrombosis (DVT), positive lupus anticoagulant, and positive family history of DVTs.

By late January 2018, he endorsed progressively worsening blurry vision in the left eye associated with redness and photopsias. Exam was notable for visual acuity (VA) of 20/30 in the right eye and 20/40 in the left eye, 1+ anterior chamber cell in the left eye, and active retinochoroiditis with 2+ vitreous cell in the left eye without an adjacent chorioretinal scar (Fig. 1). Intraocular pressures were 12 mm Hg in each eye. Serologies were obtained for anti-toxoplasma immunoglobulin M (IgM) and immunoglobulin G (IgG) which were both positive (IgG 115 IU/mL, cutoff <12 IU/mL). He was started on a course of double strength trimethoprim/sulfamethoxazole (TMP/SMZ; 160 mg/800 mg) twice daily with excellent response (Fig. 1A–C).

Approximately 3 months after resolution, he developed a visually significant epiretinal membrane (ERM) associated with cystoid macular edema (CME) – see Fig. 1D. These complications were managed successfully with pars plana vitrectomy (PPV) with membrane peel (MP) and topical steroid. Final VA returned to prior baseline, 20/25 in the left eye. One year later, while still taking TMP/SMZ every other day, he developed recurrence of systemic symptoms. The TMP/SMZ was increased to daily dosing with resolution of symptoms. The retinitis did not recur.

Case 2. A 45-year-old, previously healthy man ate fresh undercooked venison in late November 2017 and 1 week later developed headaches, myalgias, malaise, and night sweats. Three months later, he developed new floaters in the right eye. Initial VA was 20/70 in the right eye and 20/25 in the left eye. Intraocular pressure was 10 mm Hg and 11 mm Hg in the right and left eye, respectively. In the right eye, slit lamp exam was notable for 1+ anterior chamber cell, 1+ vitreous cell and haze, and active retinochoroiditis along the distal inferior vascular arcade associated with neovascularization (NV). The left eye anterior and posterior segment exams were normal. Given the patient's clinical presentation and temporal relationship to eating undercooked venison, primary ocular toxoplasmosis was suspected. Serologies for anti-toxoplasma IgM and IgG were positive (IgM 41.7 AU/mL, cutoff <10 AU/mL; IgG 169 IU/mL), and the patient was started on double strength TMP/SMZ twice daily. The area of NV regressed after 4 weeks of treatment; the patient did not require laser photocoagulation or anti-vascular endothelial

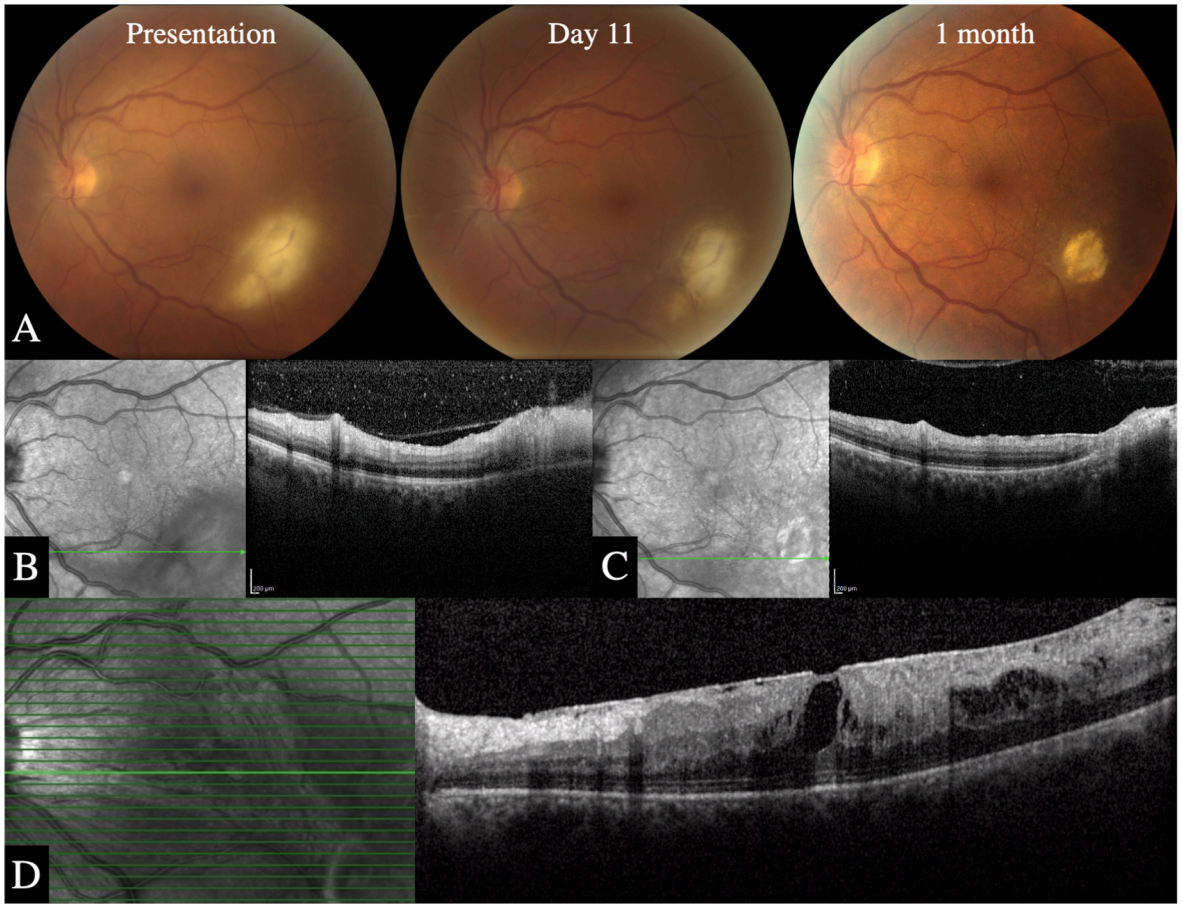

Fig. 1. (A) Fundus photographs of the left eye from Case 1 illustrating a focal area of retinitis in the inferior macula without any adjacent chorioretinal scar. The area of retinitis responded rapidly to initiation of trimethoprim/sulfamethoxazole with reduction in size and vitritis by day 11 and essentially complete resolution by 1 month. (B) Optical coherence tomography of the same patient at presentation showed inner retinal hyper-reflectivity with full-thickness retinal disorganization, retinal thickening, and posterior shadowing. The retinal thickening was greatly improved after 1 month of treatment (C) but an epiretinal membrane developed (D) which was visually significant.

growth factor (anti-VEGF) treatment. However, the regressed NV did result in focal vitreoretinal traction (Fig. 2) which was observed. He required an extended course of TMP/SMZ due to recurrence of systemic symptoms after attempted discontinuation. The retinochoroiditis did not recur and VA had improved to 20/30.

Case 3. A 48-year-old man ate undercooked venison in mid-November 2018 and 1 week later developed a high-grade fever and myalgias. He was treated with oseltamivir for presumed influenza infection, but his symptoms continued to wax and wane. One month later, he developed a scotoma in the left eye. VA was 20/20 in each eye and intraocular pressure was 21 mm Hg and 19 mm Hg in the right and left eye, respectively. Slit-lamp exam of the left eye revealed rare cells in the anterior chamber, vitreous haze, and a white chorioretinal lesion inferior to the optic nerve. He was treated for presumptive primary ocular toxoplasmosis with double strength TMP/SMZ dosed twice daily. This diagnosis was confirmed by positive serologies for anti-toxoplasma IgG of 275 IU/mL, and IgM >160 AU/mL. After months of treatment, oral TMP/SMZ was reduced to once daily. He subsequently developed myalgias and fatigue concerning for recurrent systemic toxoplasmosis, necessitating escalation of the TMP/SMZ dose to twice daily. There was no recurrence of retinochoroiditis, and VA remained stable at 20/20.

Case 4. A 75-year-old man developed myalgias, generalized weakness, fatigue, and recurrent fevers and chills in late October 2018 after eating undercooked venison multiple times throughout the preceding months. A few weeks after onset of systemic symptoms, he developed a "black spot" in the right eye. On initial exam, VA was 20/200 in the right eye and 20/25 in the left eye. IOP was 18- and 16-mm Hg in the right and left eye, respectively. Anterior segment exam showed mild anterior chamber inflammation in the right eye and normal left eye. Dilated fundus exam was remarkable for a central white area of retinitis in the right eye associated with vitritis; the left eye was normal. Serologies for anti-toxoplasma IgM and IgG were positive (IgG 535 IU/mL); Bartonella henselae and quintana, and Treponemal serologies were negative. Treatment was initiated with double strength TMP/SMZ taken twice daily and significant improvement in the retinal lesion was noted after 2 weeks. VA remained poor at 20/400 in the right eye due to chorioretinal scar formation.

3. Discussion

In the present retrospective case series, four immunocompetent patients ingested and handled fresh, undercooked venison processed from Minnesota and later developed primary ocular toxoplasmosis. The diagnosis of primary rather than reactivation is supported by the unilateral necrotizing retinitis in the absence of any adjacent pigmented chorioretinal scar, anti-toxoplasma serologies, chronology of symptom onset after high-risk exposure, and rapid resolution with appropriate therapy. There are a growing number of case reports regarding *T. gondii* infection related to venison consumption,[6–10] but the chronology of primary infection related to venison consumption has not been well established due to small sample size. This case series further demonstrates a clear chronology of systemic symptoms developing within a few weeks of exposure, and ocular symptoms beginning 1–3 months after exposure (Table 1). This chronology is comparable to outbreaks from eating other types of undercooked meat.[14,15]

The diagnosis of ocular toxoplasmosis is clinical but may be supported by ancillary testing.[16] As mentioned previously, the typical exam finding is a site of necrotizing retinochoroiditis associated with vitritis.[2] The diagnosis is more nuanced in primary ocular toxoplasmosis, especially when acquired through uncommon mechanisms. In such cases, various biologic tools exist to substantiate the diagnosis, including serologies for anti-toxoplasma IgM and IgG, direct detection of *T. gondii* by gene amplification from aqueous paracentesis, or detection of anti-toxoplasma antibodies by ELISA from aqueous.[17] The association between serology and active retinochoroiditis for the diagnosis of primary ocular toxoplasmosis has been supported.[18] Each of the patients in this case series had negative workups for alternative etiologies and highly positive *T. gondii* serologies consistent with primary infection.

In an immunocompetent host, the majority of cases of recurrent ocular toxoplasmosis will resolve spontaneously over the course of 1–2 months, but many ophthalmologists still prefer to treat immunocompetent patients with active inflammation.[3] The decision to treat depends on multiple factors, including extent of vitritis, VA decline, and proximity of lesion to the macula or optic nerve.[19] Each of the patients in this study were symptomatic, had lesions involving the posterior pole, and had mild to moderate vitritis. Therefore, treatment was indicated. Many physicians will opt for "classic" therapy which consists of a combination of pyrimethamine, sulfadiazine, and systemic corticosteroid; however, TMP/SMZ is an appealing alternative choice due to its efficacy, low cost, availability, and tolerability.[2]

In this case series, all patients were treated with double strength TMP/SMZ without corticosteroids, achieved rapid resolution of the retinochoroiditis with preservation of VA in all but one case (Case 4), and had no retinitis recurrence. The most efficacious treatment regimen is debated, and trends have changed over time.[19] Factors that contribute to therapy choice include visual outcomes, reduction of lesion size, rates of recurrence, and drug-related toxicity. Although improved visual outcomes or shortened duration of retinitis with treatment are not supported by current evidence, treatment likely reduces the risk of recurrent retinochoroiditis.[20] The absence of retinitis recurrence in any of our patients at 1 year follow up is in concordance with a large prospective randomized interventional clinical trial by Silveira et al.,[21] but risk of longer-term recurrence may still be an issue. Conflicting evidence exists regarding efficacy of lesion size reduction in studies comparing classic therapy to TMP/SMZ with prednisolone.[22,23] Given the potential

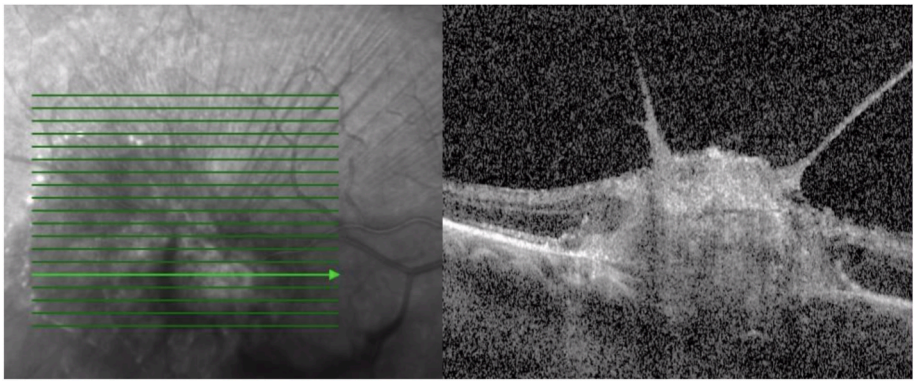

Fig. 2. OCT raster through the retinal lesion showed focal vitreoretinal traction and overlying neovascularization. After treatment with trimethoprim/sulfamethoxazole, the neovascularization resolved, although residual vitreoretinal traction remained and was observed.

Table 1
Clinical course characteristics in each case demonstrating a clear timeline for systemic and ocular symptom onset. Anterior chamber cell was present in most patients but was mild, whereas vitritis was present in all patients. All patients had positive IgM and IgG serologies and were treated with TMP/SMZ with rapid clinical improvement.

Case	Onset of Symptoms After Exposure		Degree of Intraocular Inflammation		Toxoplasma Serology		Treatment	Complications
	Systemic, wk	Ocular, mo	AC	Vitreous	IgM (AU/mL)[a]	IgG (IU/mL)[b]		
1	2	3	1+ cell	2+ cell	"positive"	115	TMP/SMZ	ERM, CME
2	1	3	1+ cell	1+ cell	41.7	169	TMP/SMZ	Traction, NV
3	1	1	Rare	Haze	>160	275	TMP/SMZ	
4	<4	3–4	0	"vitritis"	"positive"	535	TMP/SMZ	

AC = anterior chamber, CME = cystoid macular edema, ERM = epiretinal membrane, IgG - immunoglobulin G, IgM = immunoglobulin M, mo = month, NV = neovascularization, TMP/SMZ = trimethoprim/sulfamethoxazole, wk = week.
[a] Cutoff <10 AU/mL.
[b] Cutoff <12 IU/mL.

for leukopenia and thrombocytopenia related to classic therapy and incredible cost of pyrimethamine, our preferred treatment remains TMP/SMZ. Moreover, our cases suggest treatment for at least three months with double strength TMP/SMZ dosed twice a day may be needed to prevent recurrent systemic symptoms. We postulated that the recurrent systemic symptoms in our cases may be from higher organism content in the wild venison that was consumed, or perhaps different strains of organism that may be seen in the wild deer population.

Even with rapid diagnosis and treatment initiation, ocular complications are common and can lead to progressive vision loss.[24] ERM is not an uncommon complication of ocular toxoplasmosis and results from the secretion of extracellular matrix and migration of glial cells, Müller cells, and fibroblasts in response to inflammation.[25] With improvements in OCT resolution, ERMs overlying active lesions and involving the macula are becoming more evident.[26] One patient in this case series developed a visually significant ERM (Case 1) and had improved visual outcome after PPV with MP, similar to other reports.[27,28] Therefore, MP is reasonable to consider patients with quiescent ocular toxoplasmosis and visually significant ERM. We recommend peri-operative prophylactic treatment for any patient undergoing intraocular surgery with a history of toxoplasmosis retinochoroiditis. CME can also limit visual acuity during the active phase and may linger long after resolution of the retinochoroiditis.[24] Reported rates of CME vary in larger reviews, from 7.5% to 13% during the active attack.[24,29] In our patient (Case 1), the CME developed early in the active phase of primary ocular toxoplasmosis but persisted after PPV/MP and required a prolonged topical steroid course. Numerous reports have been published on the commonness of recalcitrant CME after PPV/MP[30,31] even in the absence of an inciting inflammatory condition. In the majority of patients with CME secondary to ocular toxoplasmosis, the CME resolves with treatment of the infection, although it can linger for longer.[24] The CME in our case is likely multifactorial, related to both toxoplasmosis and post-surgical inflammation. Finally, a rare complication identified in this case series was the development of NV elsewhere (Case 2) during the active retinochoroiditis. Interestingly, this NV regressed shortly after initiation of TMP/SMZ, did not require anti-VEGF, but did result in focal vitreoretinal traction. Secondary choroidal neovascularization membrane, not NV elsewhere, is a more well described sequela of ocular toxoplasmosis but was not seen in our case series.

All patients in this case series were male which we suspect is not a sex predilection of *T. gondii* but rather a reflection of the skewed hunter population in the United States. WTD hunters can minimize the risk of acquiring primary ocular toxoplasmosis by fully cooking venison to an internal temperature of 64 °C.[32] Low temperature storage and/or freezing fresh cuts of meat before cooking also reduce the risk but are unreliable because tissue cysts remain infectious in refrigerated carcasses for up to 3 weeks, and frozen carcasses for >11 days.[32]

4. Conclusion

Ingestion or handling of undercooked venison is a major risk factor for primary toxoplasmosis infection. In this case series, all patients had a highly suggestive temporal relationship between eating undercooked venison and developing systemic and ocular symptoms. The ocular symptoms tend to occur during the winter months, several months following high-risk exposure during the fall hunting season. Appropriate antibiotics led to prompt treatment response, although relapse of systemic symptoms was common when antibiotics were tapered. Despite resolution of the primary infection, secondary ocular complications, such as epiretinal membrane and cystoid macular edema, are not uncommon. Regardless, good visual outcomes are possible if the chorioretinal lesion does not threaten the macula and/or optic nerve.

Patient consent

Verbal informed consent was obtained from the patient, including permission for publication of all photographs and images included herein.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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