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ORIGINAL RESEARCH

Clinical Profile and Outcomes of Patients with Toxoplasmic Retinochoroiditis: A Tertiary Care Center Experience

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Background: This study aims to characterize the demographic profile, clinical features, and treatment outcomes of patients diagnosed with toxoplasmic retinochoroiditis (TRC) at a tertiary care center in Riyadh, Saudi Arabia.

Methods: A retrospective chart review was conducted on patients presenting to the uveitis service at King Khaled Eye Specialist Hospital between 2018 and 2024 with clinical signs consistent with TRC. Demographic data, clinical findings, multimodal imaging results, treatment regimens, and disease complications were collected and analyzed.

Results: Thirty-three eyes of 33 patients met inclusion criteria, with a mean age of 36.5 ± 11.8 years. Recurrent disease was observed in 78.8%, while 21.2% presented with primary TRC. Lesions predominantly involved the macula (41.9%), followed by major vascular arcades (25.8%) and peripapillary regions (22.6%), with fewer cases in mid-peripheral or peripheral retina (9.7%). Optic neuritis and neuroretinitis were observed in a small subset. Ipsilateral inactive scars were common (69.7%), and contralateral scars were present in 42.4%. Complications during follow-up included optic atrophy, macular scarring, retinal tears, epiretinal membrane formation, and choroidal and retinal neovascularization.

Conclusion: Toxoplasmic retinochoroiditis has diverse clinical presentations with a substantial risk of vision-threatening complications. Therefore, early recognition, especially in patients without prior chorioretinal scars, is critical for timely intervention. This study fills a crucial knowledge gap in the regional epidemiology and management of TRC, highlighting the need for larger prospective studies to refine diagnostic criteria, optimize treatment strategies, and improve long-term visual outcomes.

Keywords: ocular toxoplasmosis, toxoplasmic retinochoroiditis, *Toxoplasma gondii*, chorioretinitis

Introduction

Ocular toxoplasmosis (OT) is a disease caused by the infection with *Toxoplasma gondii*, an ubiquitous obligate intracellular parasite acquired congenitally or postnatally. It is estimated that nearly one-third of the world's population is chronically infected, with the prevalence of OT varying geographically depending on climate, dietary habits, and socioeconomic factors.[1,2]

It is estimated that approximately 25–30% of the global population is chronically infected with *T. gondii*, with regional seroprevalence ranging from 10% to 20% in low-prevalence areas to over 60% in tropical regions.[3] In Saudi Arabia, data from tertiary centers indicate that toxoplasmosis accounts for 16% of infectious uveitis cases and nearly half of posterior uveitis etiologies,[4] while in Western Saudi Arabia it represented around 6% of overall uveitis.[5]

After primary infection, recurrence may happen at any time and is thought to be caused by the rupture of intraretinal cysts, which start a rapid intraocular immune reaction.[6] Clinically, OT typically presents as focal necrotizing retinochoroiditis with overlying vitritis (headlight in the fog appearance), although atypical forms such as punctate outer retinal toxoplasmosis, papillitis, or Kyrieleis arteritis have also been described.[7,8]

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Diagnosis is primarily clinical, supported by multimodal imaging when necessary. Laboratory confirmation, including PCR and serology, is reserved for atypical or unclear cases.[9]

Management of OT is individualized, as the disease is often self-limiting. However, treatment is recommended for sight-threatening lesions, recurrent disease, or immunocompromised patients. While several therapeutic regimens exist, the most widely used currently is trimethoprim-sulfamethoxazole (TMP-SMX) with oral corticosteroids.[10]

Despite treatment, recurrences are frequent and vision-threatening complications such as glaucoma, cataract, choroidal neovascularization (CNV), and retinal detachment remain a concern, with recurrence rates reported in up to 79% of patients during long-term follow-up.[11]

The objective of this study is to evaluate the clinical characteristics, multimodal imaging findings, laboratory workup, and treatment outcomes of patients diagnosed with Toxoplasmosis retinochoroiditis (TRC) at a tertiary care center in Riyadh, Saudi Arabia. Given the limited local data, this study provides real-world insights into the disease burden and its complications in the Saudi population.

Methods

This is a retrospective descriptive review of all adult patients, 18 years or older, who were diagnosed with OT in the form of TRC at King Khaled Eye Specialist Hospital in Riyadh, Saudi Arabia, from 2018 to 2024.

Exclusion Criteria

1. Pediatric age group.
2. Patients in whom other causes of uveitis could not be ruled out.

Clinical records were reviewed for descriptive analysis. Demographic data were summarized. Best corrected visual acuity (BCVA) before and after treatment was recorded and categorized into five levels for descriptive purposes. Anterior and posterior segment examinations, including evaluation of inflammation according to the Standardization of Uveitis Nomenclature (SUN) grading system, were documented. In addition, multimodal imaging, including intravenous fundus fluorescein angiography (FA), spectral domain optical coherence tomography imaging (OCT; Spectralis OCT, Heidelberg Engineering, Inc, Heidelberg, Germany), and color fundus imaging (Optos PLC, Dunfermline, UK), was reviewed when available. Patient characteristics were summarized as frequencies for categorical variables and as mean or median with standard deviation for continuous variables, as appropriate.

Treatment decisions were individualized based on disease severity and systemic considerations. Patients with mild disease were treated with TMP-SMX alone or in combination with oral corticosteroids. Clindamycin was added in cases of more extensive retinochoroiditis or inadequate response to TMP-SMX. Oral corticosteroids were co-administered when significant intraocular inflammation or vision-threatening lesions were present, with gradual tapering. In patients with a history of tuberculosis or positive screening results, anti-tubercular coverage was added to avoid reactivation during immunosuppression.

Institutional review board approval was obtained for this study. The confidentiality of the patient's data was strictly maintained, and the study was carried out in compliance with the Declaration of Helsinki.

As this is a retrospective descriptive study, no inferential statistical tests were applied.

Results

The study included 33 eyes of 33 patients with a mean age of 36.5 ± 11.8 years. Gender distribution was almost equal (51.5% male, 48.5% female), with the right eye affected in 60.6% and the left eye in 39.4%. Most patients presented with decreased visual acuity (72.7%), followed by floaters (39.4%) and redness (12.1%). Other symptoms such as photophobia, scotoma, or pain were less common, and one case was diagnosed incidentally. Among all cases, 37.5% presented within one week of symptom onset, and 40.6% within one month (Table 1).

Most patients (81.8%) had no pre-existing medical conditions. Among those with a medical history, two patients had diabetes, one had systemic lupus erythematosus (SLE) in remission, and one had gout. The majority of patients were immunocompetent ($n = 32$), with only one patient diagnosed with human immunodeficiency virus (HIV).

Table 1 Characteristics of the Cases (n=33)

Characteristic	Freq	%
Age, years*	36.48 ±11.86	
Gender		
Male	17	51.5
Female	16	48.5
Laterality		
Right eye	20	60.6
Left eye	13	39.4
Complaints†		
Decreased visual acuity	24	72.7
Floaters	13	39.4
Redness	4	12.1
Photophobia	3	9.1
Visual field loss	3	9.1
Pain	1	3.0
Asymptomatic (incidental)	1	3.0

Notes: *mean±SD. †Percentages add up to more 100% (patients could have more than one symptom).

No identifiable risk factor for OT was found in 20 patients (60.6%). The most commonly reported risk factor was contact with animals (cats or sheep), reported by eight patients (24.3%). Less frequently reported risk factors included consumption of undercooked meat (1 patient), HIV infection (1 patient), a maternal history of toxoplasmosis during pregnancy (1 patient), and congenital toxoplasmosis (2 patients).

Clinical Examination

On clinical examination, only eleven patients had a BCVA of 20/40 or better, while fifteen patients had BCVA ranging between 20/50 and 20/400, and seven patients had BCVA worse than 20/400. Intraocular pressure (IOP) was within normal limits in most cases (90.9%). Anterior segment examination revealed diffuse conjunctival injection in all patients. Granulomatous keratic precipitates were present in a minority of cases, and anterior chamber inflammation varied from occasional cells to 3+ cells, with one-third of patients showing a quiet anterior chamber (Table 2).

Posterior segment examination showed variable vitreous haze, with most patients having grade 1. The location of TRC was most commonly macular, followed by lesions threatening major vessels, and then peripapillary involvement, as illustrated in Figure 1. Optic disc findings and TRC lesion characteristics are summarized in Table 2.

Imaging Findings

Macular OCT and fluorescein angiography, when performed, revealed a spectrum of findings, including subretinal and intraretinal fluid, inner retinal thickening, and hyperfluorescent TRC lesions. FA was not performed in three patients due to fluorescein dye allergy, and OCT was not available for one patient due to a technical error. Detailed multimodal imaging findings are presented in Table 3 and Table 4.

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Table 2 Slit-Lamp Examination Findings at Presentation

Finding		Count	%
Best corrected visual acuity	20/20–20/40	11	33.3
	<20/40–20/100	11	33.3
	<20/100–20/200	1	3.0
	<20/200–20/400	3	9.1
	<20/400	7	21.2
IOP	Normal	30	90.9
	High	3	9.1
Conjunctive	Diffuse/ciliary injections	33	100.0
	Quiet	0	0.0
Cornea	Normal	25	75.8
	Keratic precipitates	8	24.2
AC	Occasional cells	7	21.2
	+1 cells	9	27.3
	+2 cells	5	15.2
	+3 cells	1	3.0
	+4 cells	0	0.0
	Quiet (No cells)	11	33.3
Iris	Iris Nodule	0	0.0
	Iris Atrophy	0	0.0
	Posterior Synechia	0	0.0
	Normal	33	100.0
Lens	Cataract	2	6.1
	Clear lens	31	93.9
Vitreous	+1 vitritis	20	60.6
	+2 vitritis	7	21.2
	+3 vitritis	2	6.1
	+4 vitritis	1	3.0
	Quiet	3	9.1
Optic Disc	Optic disc swelling	7	21.2
	Optic disc hyperemia	9	27.3
	No view to disc details	1	3.0
	Normal	16	48.5

(*Continued*)

Table 2 (Continued).

Finding		Count	%
Vasculitis	Yes	18	54.5
	No	14	42.4
	No view to retinal details	1	3.0
Focal retinochoroiditis	Yes	31	93.9
	No	1	3.0
	No view to retinal details	1	3.0
Number of retinochoroiditis Foci	Single	27	81.8
	Two	3	9.1
	Three	0	0.0
	Four	1	3.0
	NA	2	6.1
Retinochoroiditis location	Peripapillary	7	21.2
	Macular*	13	39.4
	Threatening major vessel	8	24.2
	Midperiphery	1	3.0
	Periphery	2	6.1
	NA	2	6.1
Chorioretinal scar in the affected eye	Yes	23	69.7
	No	9	27.3
	No view to retinal details	1	3.0
Chorioretinal scar in the contralateral eye	Yes	14	42.4
	No	19	57.6

Notes: *One patient with macular involvement had also mid-peripheral lesion.

Laboratory Findings

Almost all patients (97%) underwent a standardized uveitis workup including purified protein derivative (PPD) skin test, QuantiFERON-TB Gold, enzyme immunoassay for syphilis, angiotensin-converting enzyme (ACE) and a high-resolution Computed tomography of the chest. Eighteen patients (54.5%) had toxoplasma IgM/IgG testing; 16 (48.5%) were IgG positive, and one (3%) IgM positive. One patient tested positive for TB.

TRC Classification

TRC was classified into either primary or recurrent. Primary TRC was defined as the presence of creamy-white single or multiple focal retinochoroiditis that is not associated with chorioretinal scars in either eye. While recurrent TRC was defined as the presence of single or multiple focal active retinochoroiditis associated with a chorioretinal scar in the ipsilateral or contralateral eye.[8]

According to this, primary TRC was observed in seven patients (21.2%) and recurrent TRC was observed in twenty-sex patients (78.8%) (Figure 2).

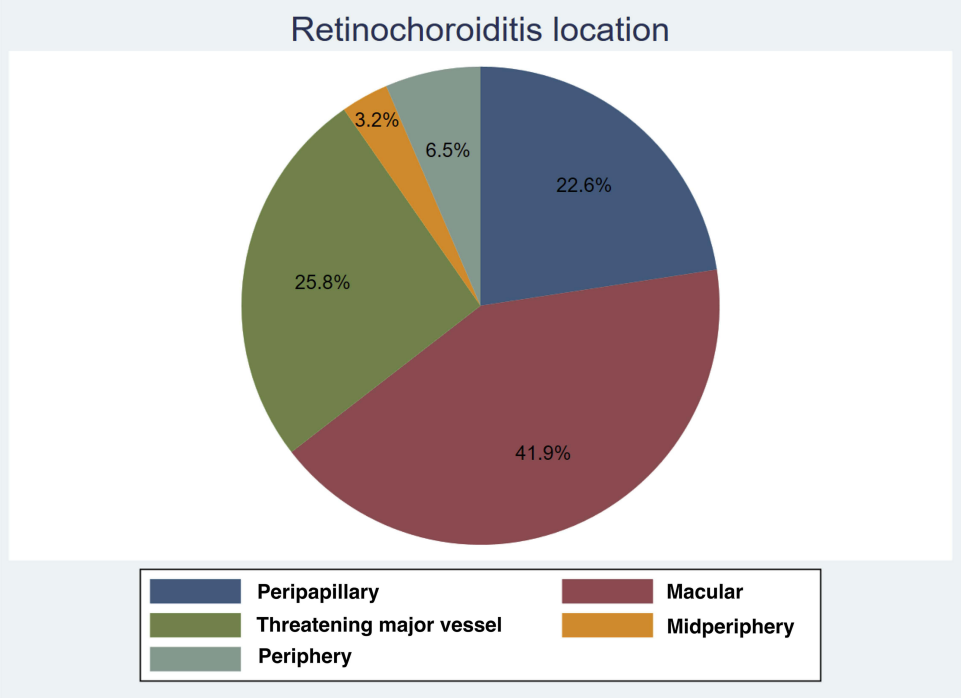

Figure 1 Pie chart illustrating the classification of TRC based on lesion location.

Also, we were able to retrospectively identify congenital toxoplasmosis in two patients.

Treatment Regimens

Most patients received oral prednisolone with combination antimicrobial therapy (TMP-SMX and clindamycin), reflecting the common practice for treating sight-threatening or extensive disease. A smaller number of patients received

Table 3 Multimodal Retinal Imaging Findings: Optical Coherence Tomography (OCT) Findings

Finding		Freq	%
OCT over the macula*	Intraretinal fluid	1	3.0
	Exudative retinal detachment	7	21.2
	Intraretinal fluid with exudative retinal detachment	1	3.0
	Bacillary layer detachment	1	3.0
	Dry	21	63.6
	Hard exudates	1	3.0
	Not done	1	3.0
	Hazy view	2	6.1
OCT over the Retinochoroiditis	Increased reflectivity of the inner retinal layers with thickening	5	15.2
	Increased reflectivity of the inner retinal layers with thickening, and subretinal fluid	7	21.2
	Not done	20	60.6
	No retinitis	1	3.0

Notes: *Percentages add up to more 100% (patients could have more than one finding).

Table 4 Multimodal Retinal Imaging Findings: Fluorescein Angiography (FA) Findings

FA Finding		Count	%
Retinochoroiditis in early FA phase	Early hyperfluorescent lesion with central hypofluorescence	27	81.8
	Early hyperfluorescent lesion	1	3.0
	Window defect	1	3.0
	Hazy view	1	3.0
	Not done	3	9.1
Retinochoroiditis in late FA phase	Increased hyperfluorescence and late leakage	28	84.8
	Pooling around window defect	1	3.0
	Hazy view	1	3.0
	Not done	3	9.1
Retinal vasculature	Focal perivascular leakage	5	15.2
	Diffuse perivascular leakage	3	9.1
	No leakage	21	63.6
	Hazy view	1	3.0
	Not done	3	9.1
Retinochoroiditis double ring sign	Yes	22	66.7
	No	7	21.2
	Hazy view	4	12.1

prednisolone with TMP-SMX alone in cases with less severe involvement, while one patient required additional TB coverage due to latent tuberculosis. Duration of antimicrobial therapy was six weeks in most cases, and oral prednisolone was tapered gradually. Topical corticosteroids and IOP-lowering medications were used as needed (Table 5).

Follow-up visits were scheduled in 2 and 4 weeks following initiation of treatment, every 3 months after resolution for a year, and every year thereafter. Subjects were instructed to seek emergency department assistance at any time in case of new symptoms. A minimum of three follow-ups were required to consider the patient followed. Among the 33 patients recruited in the study, two patients have lost follow-up.

Treatment Outcomes

BCVA improved descriptively: at presentation, eleven patients had BCVA ≥20/40, increasing to twenty-one after treatment; five patients had BCVA <20/400 at presentation, decreasing to one patient after treatment (Table 6).

Most patients (96.8%) achieved symptomatic improvement and clinical resolution of TRC, with one delayed due to nonadherence. Chorioretinal scarring occurred in 29 patients (93.5%). Recurrence was observed in four patients (12.1%), all adjacent to previous scars; median time to recurrence was 42 months (range 16–72 months).

Among patients with complete follow-up (n = 31), 22 (71.0%) achieved visual recovery without complications. Nine patients developed complications including macular scars, choroidal neovascular membranes, retinal tears, optic disc atrophy, or macular epiretinal membrane. No patients required cataract surgery.

Discussion

This study describes the clinical and multimodal imaging features of TRC, as well as treatment regimens and outcomes, in patients managed at a tertiary center in Riyadh, Saudi Arabia.

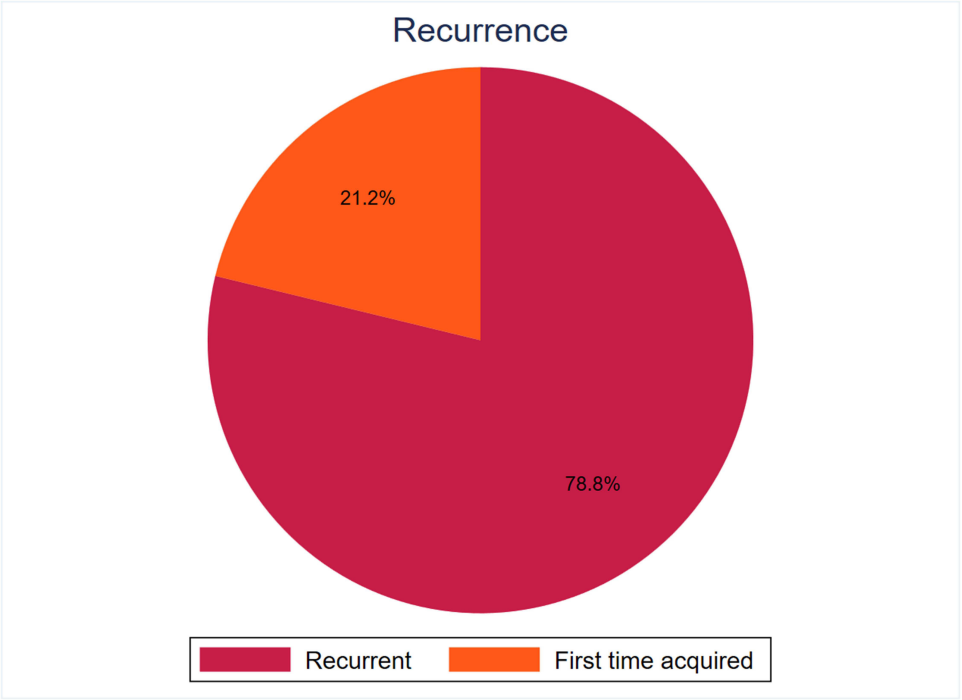

Figure 2 Pie chart depicting the proportion of primary versus recurrent TRC cases.

The mean age of our cohort was 36.5 ± 11.9 years, consistent with previous reports showing OT prevalence peaks between the second and fourth decades of life.[11] Symptoms largely depend on lesion location,[7] with macular lesions causing the most severe visual complaints. In our cohort, decreased BCVA was the most commonly reported symptom,

Table 5 Treatment Regimens Used

Treatment Regimen	Count	%
Trimethoprim-sulfamethoxazole alone	1	3.0
Trimethoprim-sulfamethoxazole and clindamycin	2	6.1
Trimethoprim-sulfamethoxazole and oral prednisolone	6	18.2
Trimethoprim-sulfamethoxazole and clindamycin and oral prednisolone	23	69.7
Trimethoprim-sulfamethoxazole and clindamycin and oral prednisolone coverage for TB (isoniazid and Pyridoxine)	1	3.0

Table 6 BCVA at Presentation Compared to Post Treatment

N=31		BCVA Post Treatment					Total
		20/20–0/40	<20/40–20/100	<20/100–20/200	<20/200–20/400	<20/400	
BCVA at presentation	20/20-20/40	11	0	0	0	0	11
	<20/40-20/100	7	3	1	0	0	11
	<20/100-20/200	0	1	0	0	0	1
	<20/200-20/400	1	0	1	1	0	3
	<20/400	2	0	2	0	1	5
Total		21	4	4	1	1	31

and macular TRC was the most frequent location, though non-macular TRC also caused decreased BCVA, indicating symptom severity may not always correlate directly with lesion location.

Intraocular pressure (IOP) elevation was observed in 9.1% of cases, lower than previously reported rates of 30–33%,[12,13] possibly due to the predominance of recurrent rather than primary TRC in our cohort. Anterior chamber inflammation was generally mild, with granulomatous inflammation observed in only eight eyes. Vitreous inflammation was typically localized to the area of TRC, and three patients had no detectable vitreous inflammation due to partial treatment elsewhere, exudative retinal detachment, or deep retinal involvement.

Pre-existing chorioretinal scars were present in 78.8% of patients, consistent with prior reports of recurrent TRC.[11] Primary TRC, which is more difficult to diagnose, was observed in seven patients, mostly located in the posterior pole.

OCT findings demonstrated hyperreflectivity and thickening of the inner retinal layers in 36.4% of cases, consistent with previously reported features.[14] ERD was observed in 24.2% of cases involving the fovea and in 21.2% adjacent to TRC. Following resolution, OCT revealed retinal thinning and ellipsoid zone loss in some patients.

Typically, in the active stage of the TRC, FA reveals early hypofluorescence caused by masking effect of the lesion, followed by progressive hyperfluorescence at the edges of the lesion in late phases.[7] This gives the classic appearance of "double ring sign".[15]

In most of our cases, active TRC demonstrated persistent hypofluorescence at the center of the lesion, surrounded by hyperfluorescent edges (81.8% of eyes). This hyperfluorescence increased in later frames and showed leakage. The classic double-ring sign was observed in 66.7% of eyes in this cohort.

It is more common for toxoplasmosis retinal vasculitis to involve the venous vasculature, but arterial vasculature may be involved.[16] Kyrieleis arteritis was described as a segmental arteritis, with frequent association to TRC, among other causes of posterior uveitis. Clinically, it presents as segmental plaques or exudates in a track-like manner within the retinal artery.[16] We identified Kyrieleis arteritis in one case involving a major retinal artery superionasal to optic disc, with an adjacent active TRC in the right eye, and concomitant optic neuritis. This patient also had pre-existing three chorioretinal scars nasally. The FA in his case showed normal filling of the artery with no staining or leakage (Figure 3).

Optic nerve involvement in OT, though atypical, has been well described with a reported prevalence of 5.3%.[17] Eckert et al classified it into five types: juxtapapillary retinochoroiditis (observed in five of our cases), neuroretinitis (one patient), distant lesion with disc edema (one patient), and the rarely reported pure papillitis and mixed lesions, which were not seen in our series.

Optic neuritis secondary to OT is even rarer and has mostly been reported in immunocompromised patients.[18,19] We encountered two such cases: one patient initially presented with peripheral TRC and moderate inflammation but progressed to severe vision loss despite scarring of the lesion and unremarkable MRI. Another patient had profound visual loss with impaired color vision and 1+ RAPD; following treatment, vision fully recovered to 20/20.

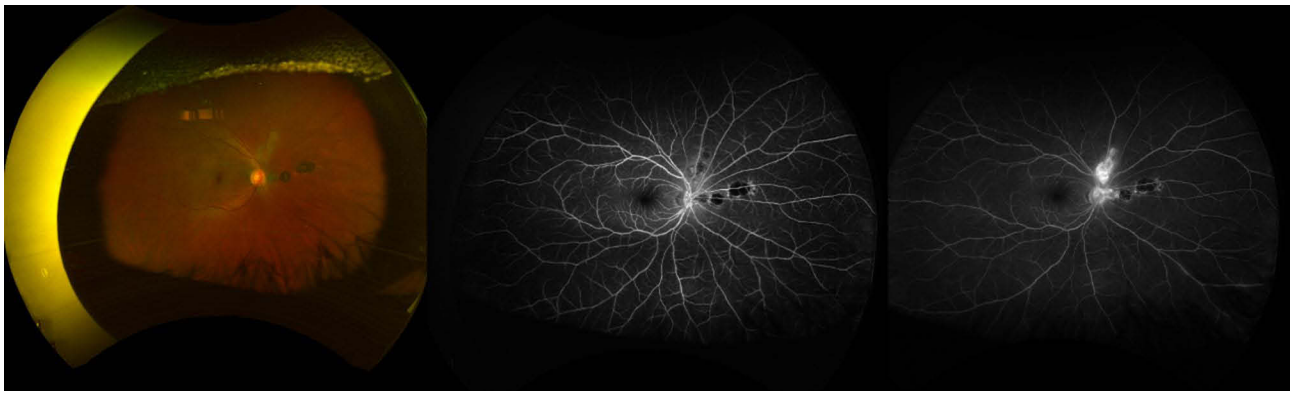

Figure 3 From left to right: pseudocolor fundus photograph of the right eye showing TRC located superior to the optic disc, with segmental Kyrieleis plaques along the superonasal retinal artery and nasal chorioretinal scars. This is followed by wide-field fluorescein angiography images in the arteriovenous and recirculation phases, demonstrating an early hyperfluorescent ring with a hypofluorescent center, and late leakage corresponding to active TRC, along with staining at the edges of nasal chorioretinal scars.

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Serologic testing showed one patient positive for IgM and sixteen positive for IgG, reflecting previous exposure rather than acute infection.[6,20–22] PCR testing was not performed; however, clinical and imaging findings, along with treatment response, were sufficient for diagnosis.

Treatment with trimethoprim-sulfamethoxazole with or without clindamycin, combined with oral prednisolone, led to lesion resolution in nearly all cases. Corticosteroids were initiated 48 hours after antimicrobial therapy and discontinued before cessation of antimicrobials. These results align with prior studies demonstrating effective control of TRC with this regimen.[10,23,24]

The recurrence rate in our study was 12.1%, lower than previously reported rates of up to 79%.[11,25] This difference may reflect early diagnosis, prompt treatment, and structured follow-up. Recurrences occurred adjacent to pre-existing scars. The most common complications were CNV (6.5%) and retinal tears (6.5%), consistent with previously reported prevalence.[26–28]

Limitations of this study include the retrospective design, small sample size, and short follow-up. The use of the SUN classification may introduce observer bias and intraobserver variation. Despite these limitations, this study provides one of the largest descriptive cohorts of OT in Saudi Arabia, contributing valuable epidemiologic and clinical data.

Conclusion

This study provides a comprehensive clinical and multimodal imaging characterization of TRC in a tertiary care center in Riyadh, Saudi Arabia, addressing a significant gap in local data. TRC predominantly affects young adults and presents with a wide spectrum of clinical manifestations, with macular involvement being the most common and associated with decreased visual acuity. The study highlights the variable inflammatory response and the relatively low prevalence of elevated intraocular pressure compared to previous reports. Multimodal imaging proved valuable in identifying subtle manifestations, such as subclinical exudative retinal detachment, aiding in early diagnosis and management. These findings provide a foundation for improving diagnostic accuracy and guiding management strategies in similar settings, and underscore the need for prospective studies with larger cohorts and longer follow-up to better understand disease recurrence and optimize care.

Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding author upon request.

Ethics Approval and Consent to Participate

This study was approved by the internal review board/ethical committee at KKESH, Riyadh, Saudi Arabia, and adhered to the tenets of the Declaration of Helsinki.

Informed consent was not required due to the retrospective strictly observational nature of the study.

Consent for Publication

Informed consent was not required due to the retrospective strictly observational nature of the study. This paper does not contain any personal identifying information.

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Disclosure

The authors declare no competing interests.

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