Misinterpreted Uveitis? A Retrospective Review of Misdiagnosed Cases

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ABSTRACT

Purpose: To assess the frequency, causes, and clinical patterns of uveitis cases initially misdiagnosed as non-inflammatory retinal diseases.

Methods: This retrospective study included 42 patients referred to the retina unit between September 2020 and June 2025 with initial non-uveitic diagnoses. After comprehensive clinical and imaging evaluation, patients who were ultimately diagnosed with uveitis were included. Demographic data, clinical findings, anatomical classification, final diagnoses, time to diagnosis, and imaging modalities used were recorded.

Results: The mean age was 37.3 ± 15 years, and 54.8% of the patients were female. Bilateral involvement was observed in 29 patients, and a total of 71 eyes were analyzed. The most common anatomical classification was panuveitis (57.7% of eyes). The most frequent incorrect referral diagnoses included central serous chorioretinopathy (23.8%), rhegmatogenous retinal detachment (19%), vitreous hemorrhage, and retinal vein occlusion (9.5%). Final diagnoses included Behçet's disease (21.4%), tuberculosis-related uveitis (19%), Vogt-Koyanagi-Harada disease (14.3%), Bartonella neuroretinitis (9.5%), and others. The average time to accurate diagnosis was 3.5 ± 3.9 days. Time to diagnosis was significantly shorter in non-infectious cases ($p = 0.034$).

Conclusion: Uveal diseases can often mimic retinal pathologies, which may delay the diagnosis and result in irreversible visual loss. Detailed anterior and posterior segment examinations, along with appropriate multimodal imaging techniques, are essential for accurate diagnosis. Systemic consultation and advanced imaging further support diagnostic accuracy.

Keywords: Uveitis, Misdiagnosis, Multimodal imaging, Masquerade Syndromes

INTRODUCTION

Uveitis is an uncommon eye disease, with studies reporting an annual incidence ranging from approximately 15 to 50 cases per 100,000 individuals (1-4). Even though it is not a frequent diagnosis in general ophthalmology, its potential to cause serious vision problems makes it a condition that should not be overlooked. The clinical signs of uveitis can vary widely and, in some cases, may resemble more common retinal or optic nerve disorders. This can be particularly challenging for ophthalmologists who have limited experience with inflammatory eye diseases or who rarely encounter such cases in their daily practice. Due to its low prevalence, uveitis can sometimes be missed, and reaching the correct diagnosis may take time (5, 6).

In real-world practice, patients with uveitis are occasionally referred to retina clinics with different initial diagnoses. Conditions such as macular edema, vitreous hemorrhage

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(VH), epiretinal membrane, central serous chorioretinopathy (CSCR) or optic disc swelling are among the most frequent preliminary labels. In these situations, the possibility of uveitis may not be considered from the beginning. This can unfortunately lead to inappropriate treatments like unnecessary intravitreal injections or even surgeries, and in some cases, the correct diagnosis may only be established after a significant delay. Such delays may result in more severe and permanent visual damage (4, 7-9).

Accurate diagnosis of uveitis requires a thorough eye examination, careful assessment of the patient's history, and in many cases, supportive imaging techniques. This approach is especially crucial in referral centers where more complex and atypical cases are typically evaluated. In this study, we aimed to review the characteristics of patients who were initially sent to our tertiary care center with a presumed retinal diagnosis but were eventually diagnosed with uveitis after detailed clinical evaluation. By presenting these cases, we hope to emphasize the challenges in recognizing uveitis in atypical presentations and to increase awareness among ophthalmologists regarding its variable manifestations.

METHODS

This study was designed as a retrospective review and conducted at a tertiary care hospital in Türkiye. The study received full approval from the Ethics Committee's Institutional Review Board. All procedures adhered to the principles of the Declaration of Helsinki.

The medical records of patients who were referred to the retina unit of our clinic between September 2020 and June 2025 were evaluated. Patients who were not suspected to have a uveal disease at the time of referral, but who were ultimately diagnosed with uveitis after comprehensive clinical assessment and additional imaging, were included in this study. Patients with masquerade syndromes, intraocular tumors, or endophthalmitis were excluded from the analysis.

The data collected from patient files included age, gender, systemic comorbidities, laterality, the anatomical classification of the uveitis, the final diagnosis, the time from the onset of symptoms to the final diagnosis, and the diagnostic methods used. The anatomical categorization of uveitis was made based on the Standardization of Uveitis Nomenclature (SUN) working group guidelines. Clinical examination parameters such as best-corrected visual acuity (BCVA), slit-lamp findings, intraocular pressure (IOP), and fundus examination results were recorded. Visual acuities were converted to Logarithm of the Minimum Angle of Resolution (LogMAR) units for statistical analysis. Ancillary imaging techniques included optical coherence tomography (OCT), fundus photography, fluorescein angiography (FFA) when necessary, indocyanine green angiography (ICG), B-scan ultrasonography (US), and computed tomography (CT) imaging as needed.

All statistical analyses were performed using IBM SPSS Statistics version 25 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation for normally distributed data or as median and range (minimum–maximum) for non-normally distributed data. The Kruskal-Wallis test was utilized for comparisons among multiple groups, while the Mann-Whitney U test was used for pairwise group comparisons. A p-value of less than 0.05 was considered statistically significant. Bonferroni correction was applied to control for multiple testing errors.

RESULTS

We reviewed the medical records of 785 patients diagnosed with uveitis at our clinic during the study period. Among 133 patients referred to our clinic for further evaluation, 42 patients (5.3%) were initially referred with diagnoses unrelated to uveitis but were ultimately diagnosed with uveitis following detailed clinical examination and ancillary imaging.

Of the 42 patients included in the study, 23 (54.8%) were female and 19 (45.2%) were male. The mean age at presentation was 37.3 ± 15 years. Bilateral involvement was initially suspected in 16 patients (38.1%), but clinical evaluation revealed bilateral disease in 29 patients. In total, 71 affected eyes were analyzed. Keratic precipitates were detected in 15 eyes (21.1%), varying degrees of anterior chamber cells in 28 eyes (32.4%), while vitritis was present in 52 eyes (73.2%). The most common anatomical classification was panuveitis, observed in 41 eyes (57.7%), followed by posterior uveitis in 25 eyes (35.2%) (Table 1).

The median BCVA at presentation was 0.7 LogMAR (range: 0–2.3), which improved significantly to 0.2 Log-

MAR (range: 0–2) after treatment ($p < 0.001$). There was no statistically significant difference in initial or final BCVA between infectious and non-infectious uveitis groups ($p = 0.695$ and $p = 0.133$, respectively).

The most common initial referral diagnoses were CSCR (9 patients), rhegmatogenous retinal detachment (RRD) (8 patients), retinal vein occlusion (RVO) (4 patients), and VH (4 patients) (Table 2). Final diagnoses were as follows: Behçet's disease (9 patients, 21.4%), tuberculosis (TB)-associated uveitis (8 patients, 19%) and Vogt-Koyanagi-Harada disease (VKH) (6 patients, 14.3%).

The median time from symptom onset to final diagnosis was 2 days (range: 1–15), with a mean of 3.5± 3.9 days. Non-infectious uveitis cases were diagnosed significantly earlier than infectious cases (median: 1 vs. 4 days, $p= 0.034$). The time to diagnosis was significantly different between patients with Behçet's uveitis and those with non-Behçet etiologies ($p = 0.018$). There was no statistically significant relationship between patients' age, gender and duration to diagnosis ($p> 0.05$).

Table 1. *Baseline Demographic Characteristics of Patients*

Cases (n)/ eyes (n)	42/ 71
Age (years) (mean± standard deviation)	37.3± 15
Gender (female/ male)	23/19
Laterality (unilateral/ bilateral) (n (%))	13 (30.9)/ 29 (69.1)
Presenting anatomical location (n (%), eyes)	
□ Anterior	0 (0)
□ Intermediate	1 (1.4)
□ Posterior	25 (35.2)
□ Panuveitis	41 (57.7)
□ Posterior scleritis	4 (5.6)

Table 2. *Final Diagnosis, Preliminary Diagnosis, Supporting Diagnostic Tools and Time to Uveitis Diagnosis*

Category of Final Diagnosis	Cases (n)	Eyes (n)	Preliminary Diagnosis (n, eyes)	Decisive Diagnostic Procedure	Time to uveitis diagnosis (days)
Behçet's disease	9	18	RVO (2), CSCR(1), DME (3), VH (4), HIV retinopathy (1), purtcher-like retinopathy (2), Hereditary Retinopathy (1)	Clinical examination, OCT, **FFA**	1 (1-6)
TB-related uveitis	8	14	AMD (4), CNVM (1), PEHCR (1), CSCR (2), Retinal dystrophy (2), RVO (1), Choroidal metastasis (1)	Clinical examination, FAF, **Quantiferon-TB Gold test**	4 (1-10)
VKH	6	12	CSCR (4), HTR (4), RRD (2)	Clinical examination, OCT, **FFA-ICG**	2.5 (1-10)
Bartonella neuroretinitis	4	4	CSCR (2), RAO (1), HTR (1)	Clinical examination, OCT, **Serology**	9 (1-14)
Posterior scleritis	3	5	RVO(2), RRD (2)	Clinical examination, **US**, OCT, FFA	1 (1-1)
Toxoplasma	3	4	VH (1), CSCR (1), RRD (1)	Clinical examination, OCT, **Serology**	2 (1-15)
Acute retinal necrosis	3	4	RRD (2), Intraocular lenfoma (1)	Clinical examination, **Anterior chamber PCR**	1 (1-1)
APMPPE	2	4	CSCR (4)	Clinical examination, OCT, **FAF**, FFA, ICG	3 (2-4)
Sarcoidosis	2	3	PCV (1), Choroidal hemangioma (1)	Clinical examination, OCT, FFA, ICG, Serum **ACE/lysozyme**	7 (7-7)
Fuchs' uveitis syndrome	1	1	VH (1)	Clinical examination	7
Sympathetic ophthalmia	1	2	RRD (1)	**Clinical examination**, OCT	1

RVO: Retinal Vein Occlusion, DME: Diabetic Macular Edema, VH: Vitreous hemorrhage, Human Immunodeficiency Virus, TBC: Tuberculosis, AMD: Age-Related Macular Degeneration, CNVM: Choroidal Neovascular Membrane, PEHCR: Peripheral Exudative Hemorrhagic Chorioretinopathy, CSCR: Central Serous Chorioretinopathy, VKH: Vogt-Koyanagi-Harada, HTR: Hypertensive Retinopathy, RRD: rhegmatogenous retinal detachment, RAO: Retinal Artery Occlusion, APMPPE: Acute Posterior Multifocal Placoid Pigment Epitheliopathy, PCV: Polypoidal Choroidal Vasculopathy, FA: fluorescein angiography, US: ultrasonography, OCT: Optical coherence tomography

Note: Bolded diagnostic methods represent the most contributive ancillary tests, aside from clinical examination, that played a key role in establishing the diagnosis for each disease entity.

DISCUSSION

To our knowledge, there is no previous study specifically focusing on a uveitis case series that were initially misdiagnosed as non-uveitic conditions. While several reports have addressed masquerade syndromes, those studies primarily involve patients who were initially thought to have uveitis but were ultimately diagnosed with non-uveitic diseases—a concept fundamentally opposite to the present study (10-12). Interestingly, those reports have shown misdiagnosis rates ranging from 2.5% to 3.8%, which is consistent with our findings.

Similar to previous studies, the most frequent initial misdiagnoses in our study were retinal vascular pathologies and CSCR (10-12) Although the direction of diagnostic error differs, uveitic diseases mistaken for retinal conditions in our series versus retinal diseases misinterpreted as uveitis in previous reports, the overlap in misidentified pathologies is notable. This recurring confusion underscores the importance of distinguishing these mimickers, as certain subtypes of AMD, CSCR, and true inflammatory entities may share overlapping clinical features such as subretinal fluid, pigmentary changes, or exudative retinal detachment. Therefore, both retina and uveitis specialists must remain vigilant and familiar with the subtle distinctions among these conditions, as accurate diagnosis often hinges on a combination of detailed clinical examination, multimodal imaging, and awareness of systemic associations.

Previous studies have described various inflammatory conditions that may resemble CSCR, including white dot syndromes, VKH, and posterior uveitis (13). The diagnostic challenge arises because both inflammatory and non-inflammatory disorders can present with serous retinal detachment. Careful ophthalmic examination and multimodal imaging, however, usually reveal key differences.

In our cohort, several of these entities—particularly VKH, TB-related uveitis, Bartonella neuroretinitis, ocular toxoplasmosis, and APMPPE—were initially mistaken for CSCR, highlighting how inflammatory and non-inflammatory conditions can overlap in clinical appearance. VKH typically presents with bilateral involvement, intraocular inflammation, and characteristic choroidal thickening, with findings such as RPE folds, internal limiting membrane fluctuations, and subretinal septa. In contrast, CSCR is usually unilateral and devoid of inflammatory signs (13,14). Similarly, APMPPE and other white-dot syndromes may resemble CSCR at onset but can be distinguished by multifocal placoid lesions and angiographic early-phase hypofluorescence with late staining (16). Toxoplasmic and Bartonella neuroretinitis cases, though occasionally confused with CSCR due to exudative changes, exhibit vitritis and adjacent inflammatory or optic disc findings not seen in CSCR (4,15) (Figure 1a-b). Recognizing these subtle distinctions is crucial, as inappropriate management—such as delaying corticosteroid therapy—can worsen visual outcomes. Awareness of these inflammatory mimickers, along with systematic use of OCT, FFA, and ICG when needed, is essential to prevent misclassification and ensure timely treatment. The significant improvement in BCVA ($p < 0.001$) observed in our cohort supports that functional visual recovery can be achieved, once the correct diagnosis and appropriate treatment are established.

In our series, RRD was the third most frequent initial misdiagnosis. Subretinal fluid, commonly seen in uveitic conditions, may lead to diagnostic confusion, particularly in cases misinterpreted as RRD, but the actual diagnoses include VKH disease, or sympathetic ophthalmia (SO). The presence of subretinal fluid in all these conditions contributes significantly to the overlap in clinical presentation. If corticosteroid therapy is delayed due to misdiagnosis, especially in inflammatory causes, irreversible visual complications may occur (8, 17, 18). Furthermore, dense intraocular inflammation or media opacities can obscure fundus visualization, complicating the clinical picture. This highlights the importance of a meticulous anterior and posterior segment examination. Inexperienced clinicians may misinterpret inflammatory signs such as pigment dispersion, anterior chamber cells, or vitreous haze as degenerative changes of the retinal pigment epithelium rather than true uveitis. Findings like pigment on the corneal endothelium, flare, posterior synechiae, IOP fluctuations, and vitritis may be overlooked or misattributed, further delaying the correct diagnosis (19, 20). The final diagnosis in these cases can be achieved by careful examination of the fellow eye and the use of FFA–ICG, where hypofluorescent choroiditis spots helped us distinguish VKH and SO (21). Additionally, among the patients initially referred with a diagnosis of RRD, two were ultimately diagnosed with posterior scleritis based on ultrasonographic detection of the characteristic “T-sign”. Therefore, in cases where retinal breaks are not

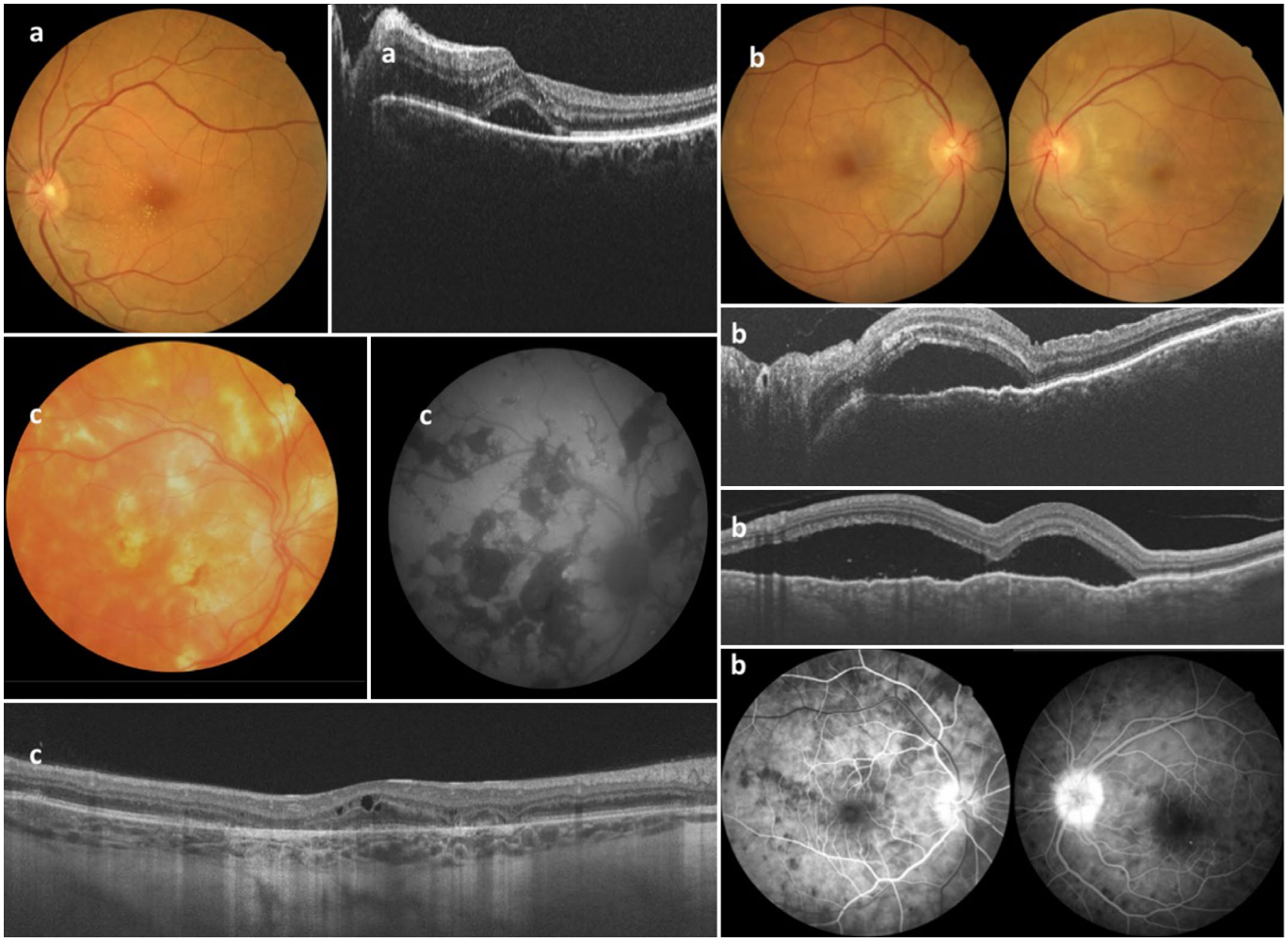

Figure 1. *Representative cases of uveitis initially misdiagnosed as non-inflammatory retinal diseases.*
(a) A patient initially diagnosed with central serous chorioretinopathy and treated with topical NSAID drops; subsequent evaluation revealed Bartonella neuroretinitis.
(b) Multimodal imaging of a patient initially diagnosed with malignant hypertension, later confirmed to have Vogt–Koyanagi–Harada (VKH) disease upon presentation to our clinic.
(c) Fundus, Fundus autofluorescence, and OCT images of a patient referred with a presumed diagnosis of hereditary retinopathy, later identified as serpiginous-like choroiditis associated with tuberculosis.

clearly visualized despite a presumed diagnosis of RRD, clinicians should reconsider the diagnosis and carefully assess for additional inflammatory clues. Findings such as choroidal folds, optic disc edema, the "T-sign" on B-scan US, pain with eye movement, and ocular hyperemia may point toward posterior scleritis or other inflammatory causes rather than RRD (13).

Another patient was initially referred with a presumed diagnosis of RRD but was ultimately diagnosed with ARN. Although RRD occurs in approximately 60% of ARN cases during the disease course, ARN was initially overlooked in this case as well (22). Even though the referring diagnosis accurately described the condition of the affected eye, careful examination of the fellow eye revealed subtle peripheral retinitis in the temporal region, leading to the correct diagnosis. Another case was misdiagnosed as intraocular lymphoma. The primary findings included anterior chamber cells of varying grades and peripheral, creamy white areas of retinitis accompanied by vasculitic changes. These findings were misinterpreted as infiltrative lesions consistent with intraocular lymphoma. Additionally, elevated IOP was noted in both affected eyes. The correct diagnosis of ARN in these cases was established through meticulous fundus examination, FFA, and detailed evaluation of the fellow eye, where additional peripheral lesions beyond the

equator were observed (23). In all cases, anterior chamber paracentesis was performed, and the diagnosis of ARN was confirmed by PCR analysis.

In our series, there were 8 TB-related and two sarcoidosis-related final diagnoses. Circumscribed choroidal hemangioma typically appears as an orange-red mass in the posterior pole and is often accompanied by serous detachment involving the macula. It is possible for TB and sarcoidosis to mimic such masses (24). Tuberculomas were fewer in number, more yellow in color, and may be associated with subretinal fluid. On the other hand, sarcoid granulomas were more likely to be multiple, smaller, dark yellow, well-demarcated or diffusely distributed, and often associated with retinal vasculitis, retinal vascular leakage, cystoid macular edema, and disc hyperfluorescence/leakage on FFA (25). Another 15-year-old patient was misdiagnosed with retinal dystrophy, whereas the actual diagnosis was serpiginous-like choroiditis due to yellow-white retinal lesions (Figure 1c). The asymmetry of the lesions, the presence of an inflammatory response, and the absence of typical dystrophic features ruled out a hereditary retinal disorder. A positive family history of pulmonary tuberculosis further supported the diagnosis. One sarcoidosis case was initially suspected to have polypoidal choroidal vasculopathy (PCV) due to macular edema and subretinal exudation. PCV typically occurs in older individuals and is characterized by reddish-orange, polyp-like or aneurysmal lesions located in the macula or peripheral retina (13). The condition often presents with recurrent episodes of exudative retinal detachment, along with serous or hemorrhagic pigment epithelial detachments. However, the absence of polypoidal vascular structures on FFA-ICG, along with the presence of vitritis, vasculitis and peripheral choroidal lesions, suggested an inflammatory rather than a degenerative process (26).

The most frequently misdiagnosed uveitis cases in our series were those related to Behçet's disease. Given that we practice in a region where Behçet's disease is endemic, this finding is not unexpected. Among the nine patients with Behçet's uveitis, one case had been initially misdiagnosed as CSCR at the time of referral. This misinterpretation was exceptional and not considered a common diagnostic confusion. None of the Behçet's uveitis patients were under systemic corticosteroid therapy at presentation, suggesting that the incorrect diagnosis likely reflected limited familiarity with Behçet-related ocular inflammation among ophthalmologists who do not routinely manage uveitis. In a previous study focusing on Behçet's uveitis, it was reported that uveitis specialists demonstrated high diagnostic accuracy for Behçet's disease (27). While this may seem to contradict our findings, the discrepancy can be attributed to the fact that the previous study was conducted exclusively among physicians with a dedicated focus on uveitis. In contrast, our study specifically evaluated the reverse scenario—cases in which Behçet's disease was initially misdiagnosed as non-uveitic retinal pathologies—and, to our knowledge, this is the first study to specifically address this perspective in the literature. Supporting this, our study also showed that the time to diagnosis for Behçet's uveitis was significantly shorter than for other uveitic entities, reinforcing the notion that clinicians familiar with uveitis can recognize Behçet's disease more rapidly.

In clinical practice, Behçet uveitis can often mimic a variety of retinal and neuro-ophthalmological pathologies, leading to frequent misdiagnoses. For example, Purtcher-like retinopathy and HIV-associated microangiopathy both present with retinal hemorrhages, cotton wool spots, and areas of retinal ischemia, but these entities typically progress without significant intraocular inflammation. Behçet uveitis, on the other hand, is almost invariably accompanied by active vitritis, anterior chamber cells, and a diffuse inflammatory response. The presence of dense vitreous cells and perivascular sheathing, as well as the tendency for rapid inflammatory progression, are hallmarks that favor Behçet disease over these primarily ischemic or microvascular conditions (28). A variety of inflammatory signs may emerge throughout the disease course in Behçet's uveitis, including hypopyon, iridocyclitis, diffuse vitreous haze, white retinal infiltrates, retinal hemorrhages, optic disc inflammation, and cystoid macular edema all of which are considered characteristic features of the disease (28).

As is well known, inflammation in patients with hereditary retinal disorders is not uncommon. Several studies have demonstrated elevated levels of proinflammatory cytokines and chemokines in aqueous and vitreous samples in such patients (29). In our study, one patient was referred with a preliminary diagnosis of hereditary retinopathy. The patient had no history of decreased visual acuity during

childhood but reported progressive vision loss beginning in their 30s. Fundus examination revealed bilateral atrophic retinal changes. Rheumatology consultation did not confirm Behçet's disease due to the absence of systemic findings. However, the literature indicates that diffuse vitritis is a consistent feature of posterior segment inflammation in active uveitis and is typically absent in eyes with end-stage Behçet uveitis (28). Based on this, the presence of recurrent oral ulcers along with bilateral atrophic retinal changes—without accompanying vitritis—strongly supported the diagnosis. The patient was ultimately considered to have Behçet's disease.

STUDY LIMITATIONS

This study has several limitations that should be acknowledged. First, its retrospective design may introduce inherent biases related to documentation and data completeness. As our data relied on existing medical records, the consistency and accuracy of symptom onset reporting and referral diagnoses may vary. Second, although we aimed to evaluate the timeline from symptom onset to final diagnosis, this estimation was based on patient-reported history, which may be subject to recall bias. Additionally, some of the uveitis diagnoses were made clinically without confirmatory testing, particularly in cases with characteristic findings, potentially limiting the generalizability of diagnostic criteria.

CONCLUSION

In conclusion, our findings underscore the diagnostic challenges posed by uveitis, especially when it mimics more common non-inflammatory ocular pathologies. A significant proportion of patients referred to our retina unit with non-uveitic diagnoses were ultimately found to have uveitis, highlighting the need for heightened clinical suspicion in atypical presentations. Retinal vascular disorders, CSCR, and RRD were among the most common initial misdiagnoses, often due to overlapping features such as subretinal fluid and macular edema. Multimodal imaging, detailed anterior and posterior segment evaluation, and systemic assessment were crucial in establishing the correct diagnosis. Collaboration among subspecialists and access to supportive diagnostic tools facilitated timely intervention and, in many cases, prevented irreversible visual damage. These findings underline the importance of uveitis expertise within retina referral units to minimize diagnostic delays.

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Ethical Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Conflicts of Interest

The authors declare that they have no conflict of interest.

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Authors' role in this study was as follows:

Design of the study : SCH, YCY
Writing: SCH, YCY
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REFERENCES

1. Acharya NR, Tham VM, Esterberg E, et al. Incidence and prevalence of uveitis: results from the Pacific Ocular Inflammation Study. JAMA Ophthalmol. 2013;131(11):1405-12.
2. Suhler EB, Lloyd MJ, Choi D, et al. Incidence and prevalence of uveitis in Veterans Affairs Medical Centers of the Pacific Northwest. Am J Ophthalmol. 2008;146(6):890-6.e8.
3. Garcia-Aparicio A, Garcia de Yebenes MJ, Oton T, et al. Prevalence and incidence of uveitis: a systematic review and meta-analysis. Ophthalmic Epidemiol. 2021;28(6):461-8.
4. Paez-Escamilla M, Caplash S, Kalra G, et al. Challenges in posterior uveitis—tips and tricks for the retina specialist. J Ophthalmic Inflamm Infect. 2023;13(1):35.
5. Lin ML, Hall AJ. Uveitis masquerade syndromes: an approach to diagnosis. Clin Exp Ophthalmol. 2024;52(1):91-105.
6. Kubicka-Trzaska A, Romanowska-Dixon B. Non-malignant uveitis masquerade syndromes. Klin Oczna. 2008;110(4-6):203-6.
7. Shoughy SS, Tabbara KF. Initial misdiagnosis of Vogt-Koyanagi-Harada disease. Saudi J Ophthalmol. 2019;33(1):52-5.

8. Panigrahi PK, Mahapatra MM, Minj A, et al. Central serous chorioretinopathy mimicking choroiditis. Clin Exp Optom. 2018;101(3):420-1.
9. Gonzalez-Gonzalez LA, Rodriguez-Garcia A, Foster CS. Pigment dispersion syndrome masquerading as acute anterior uveitis. Ocul Immunol Inflamm. 2011;19(3):158-66.
10. Hsu YR, Wang LU, Chen FT, et al. Clinical manifestations and implications of nonneoplastic uveitis masquerade syndrome. Am J Ophthalmol. 2022;238:75-85.
11. Rothova A, Ooijman F, Kerkhoff F, et al. Uveitis masquerade syndromes. Ophthalmology. 2001;108(2):386-99.
12. Svozilkova P, Rihova E, Heissigerova J, et al. Benign masquerade syndromes in differential diagnosis of uveitis. Cesk Slov Oftalmol. 2008;64(5):175-84.
13. Sahoo NK, Singh SR, Rajendran A, et al. Masqueraders of central serous chorioretinopathy. Surv Ophthalmol. 2019;64(1):30-44.
14. Tayal A, Daigavane S, Gupta N. Vogt-Koyanagi-Harada disease: a narrative review. Cureus. 2024;16(4):e58867.
15. Garweg JG. Ocular toxoplasmosis: an update. Klin Monbl Augenheilkd. 2016;233(4):534-9.
16. Li AL, Palejwala NV, Shantha JG, et al. Long-term multimodal imaging in acute posterior multifocal placoid pigment epitheliopathy and association with coxsackievirus exposure. PLoS One. 2020;15(8):e0238080.
17. Nguyen NV, Khan F, Emig M, et al. Management of atypical central serous chorioretinopathy mimicking Vogt-Koyanagi-Harada disease. J Vitreoretin Dis. 2023;7(3):249-53.
18. Papadia M, Jeannin B, Herbort CP. Central serous chorioretinopathy misdiagnosed as posterior uveitis and the vicious circle of corticosteroid therapy. J Ophthalmic Vis Res. 2015;10(3):303-8.
19. Jarrett WH 2nd. Rhegmatogenous retinal detachment complicated by severe intraocular inflammation, hypotony, and choroidal detachment. Trans Am Ophthalmol Soc. 1981;79:664-83.
20. Ivanisevic M. The natural history of untreated rhegmatogenous retinal detachment. Ophthalmologica. 1997;211(2):90-2.
21. Stanga PE, Lim JI, Hamilton P. Indocyanine green angiography in chorioretinal diseases: indications and interpretation: an evidence-based update. Ophthalmology. 2003;110(1):15-21.
22. Putera I, Ridwan AS, Dewi M, et al. Antiviral treatment for acute retinal necrosis: a systematic review and meta-analysis. Surv Ophthalmol. 2024;69(1):67-84.
23. Mapelli C, Milella P, Dona C, et al. Acute retinal necrosis: clinical features, diagnostic pitfalls, treatment, and outcome of an insidious disease in children. Front Pediatr. 2022;10:854325.
24. Ashour DM, Abdel Aziz NAS, Eltonbary K, et al. Bilateral intraocular masses in a child as a first presentation of disseminated tuberculosis: case report. Ther Adv Ophthalmol. 2025;17:25158414251356373.
25. Agarwal A, Aggarwal K, Pichi F, et al. Clinical and multimodal imaging clues in differentiating between tuberculomas and sarcoid choroidal granulomas. Am J Ophthalmol. 2021;226:42-55.
26. Giorgiutti S, Jacquot R, El Jammal T, et al. Sarcoidosis-related uveitis: a review. J Clin Med. 2023;12(9).
27. Tugal-Tutkun I, Onal S, Ozyazgan Y, et al. Validity and agreement of uveitis experts in interpretation of ocular photographs for diagnosis of Behçet uveitis. Ocul Immunol Inflamm. 2014;22(6):461-8.
28. Tugal-Tutkun I, Onal S, Stanford M, et al. An algorithm for the diagnosis of Behçet disease uveitis in adults. Ocul Immunol Inflamm. 2021;29(6):1154-63.
29. Dutta Majumder P, Menia N, Roy R, et al. Uveitis in patients with retinitis pigmentosa: 30 years' consecutive data. Ocul Immunol Inflamm. 2018;26(8):1283-8.