Case Report

Fuchs uveitis syndrome in a patient with primary myelofibrosis: a case report

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ABSTRACT

Background: Fuchs uveitis syndrome (FUS) is a chronic, typically unilateral, low-grade anterior uveitis characterized by diffuse stellate keratic precipitates, iris stromal atrophy, heterochromia, cataract, and vitreous inflammation. Primary myelofibrosis (PMF) is a chronic myeloproliferative neoplasm associated predominantly with thromboembolic, hemorrhagic, and infiltrative ocular manifestations, whereas inflammatory uveitic presentations are exceedingly uncommon. We report a rare coexistence of FUS in a patient with PMF and discuss potential shared inflammatory mechanisms.

Case Presentation: A 64-year-old Asian Indian man with a 2-year history of PMF, receiving oral prednisolone and hydroxyurea, presented with a 2-month history of decreased vision in the left eye (OS). Bone marrow evaluation showed evidence of hypercellular marrow with megakaryocytic proliferation and reticulin fibrosis, while next-generation sequencing identified pathogenic variants involving JAK2, TET2, and SH2B3. Best-corrected distance visual acuity was 20/20 in the right eye (OD) and 20/25 in the OS, with normal intraocular pressure in both eyes. Slit-lamp examination of the OS revealed diffuse stellate keratic precipitates, minimal anterior chamber flare, iris sphincter atrophy, and grade-2 nuclear sclerosis with posterior subcapsular cataract, whereas the OD was unremarkable. No posterior synechiae or posterior segment abnormalities were identified. Based on the characteristic unilateral clinical findings, a diagnosis of FUS was established. Systemic evaluation for infectious and granulomatous causes of uveitis, including chest radiography, Mantoux testing, Treponema haemagglutination assay (TPHA), and serum angiotensin-converting enzyme levels, were unremarkable. The patient was treated with topical nepafenac ophthalmic suspension and subsequently underwent uneventful phacoemulsification with posterior chamber intraocular lens implantation in the OS, achieving a postoperative visual acuity of 20/20. Over 3.5 years of follow-up, the eye remained clinically quiescent, with stable visual acuity and no evidence of recurrent inflammation or secondary glaucoma.

Conclusions: This case describes a previously unreported occurrence of FUS in a patient with PMF. Although a causal relationship cannot be established from a single case, this coexistence highlights the importance of careful ophthalmic evaluation and clinical documentation of atypical unilateral chronic anterior uveitis in patients with myeloproliferative neoplasms. Further studies are warranted to clarify the potential relationship between PMF and FUS.

KEYWORDS

Fuchs uveitis syndrome, case report, primary myelofibrosis, anterior uveitis, cataracts, phacoemulsification

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INTRODUCTION

Fuchs uveitis syndrome is a chronic, usually unilateral, often unnoticed inflammatory disorder that primarily affects the anterior uvea and vitreous in the eye [1]. It appears more commonly in developed countries [2], and is the second most common cause of non-infective uveitis, making it a significant contributor to uveitic cases [3]. Cataract, secondary glaucoma, and vitreous opacities are known associated complications [4, 5]. Fuchs uveitis syndrome occurs unilaterally and its characteristic features are stellate keratic precipitates, iris atrophy with or without heterochromia, mild flare and minimal cells in the anterior chamber, vitreous involvement, and absence of posterior synechiae or cystoid macular edema. Diagnosis is typically based on clinical features and may involve laboratory tests and imaging studies to rule out other infectious causes like cytomegalovirus, rubella, or herpes zoster viruses. This condition does not warrant treatment unless cataract or glaucoma develop [4–6].

Primary myelofibrosis (PMF), a type of myeloproliferative neoplasm (MPN), is defined by aberrant hematopoietic stem cell proliferation in the bone marrow that results in marrow fibrosis, or scarring [7, 8]. *Janus kinase 2 (JAK2)*, *CALR (Calreticulin)*, or *myeloproliferative leukemia virus oncogene (MPL)* mutations are frequently but not always present in PMF, which is defined by aberrant growth of a clone of cells produced from hematopoietic stem cell-derived clonal myeloproliferation (*Calreticulin*, *MPL-thrombopoietin receptor genes*, *Janus kinase*). The JAK-STAT signaling system, which is essential for cell growth and survival, is dysregulated as a result of certain mutations [8, 9]. Reticulin fibrosis is a typical characteristic of the bone marrow stromal response in PMF. Disease progression is influenced by abnormal cytokine expression. Clinical features include anemia, hepatosplenomegaly, extramedullary hematopoiesis, constitutional symptoms, cachexia, and a higher risk of developing leukemia, all of which can lead to a reduced survival [8].

This case report aims to describe the rare coexistence of Fuchs uveitis syndrome in a patient with PMF, an MPN with infrequent ocular involvement. By presenting this observational single-case study, we seek to highlight a potential association and underscore the importance of considering underlying hematologic disorders in atypical uveitic presentations.

CASE PRESENTATION

A 64-year-old Asian Indian man presented with a 2-month history of decreased vision in the left eye (OS). Best-corrected distance visual acuity (BCDVA), assessed using a Snellen chart (Nidek automatic chart projector CP-670; Nidek Co., Ltd., Gamagori, Japan), was 20/20 in the right eye (OD) and 20/25 in the OS. Intraocular pressure, measured using Goldmann applanation tonometry (AT-900; Haag-Streit AG, Koniz, Switzerland) mounted on a slit-lamp biomicroscope, was 12 mmHg in the OD and 14 mmHg in the OS. Slit-lamp examination (Haag-Streit, Mason, OH, USA) of the OD was unremarkable, whereas the OS demonstrated diffuse stellate keratic precipitates distributed over the corneal endothelium (Figure 1A), minimal anterior chamber flare, iris sphincter atrophy (Figure 1B), and grade-2 nuclear sclerosis with posterior subcapsular cataract (Figure 1B). No posterior synechiae were noted. Dilated fundus examination of both eyes revealed no posterior segment abnormalities, with a cup-to-disc ratio of 0.3 in each eye. Based on the characteristic unilateral clinical findings, including diffuse stellate keratic precipitates, iris sphincter atrophy, low-grade anterior chamber inflammation, absence of posterior synechiae, and an unremarkable posterior segment examination, a diagnosis of Fuchs uveitis syndrome was established. *In vivo* confocal microscopy was not available for further characterization of the keratic precipitates.

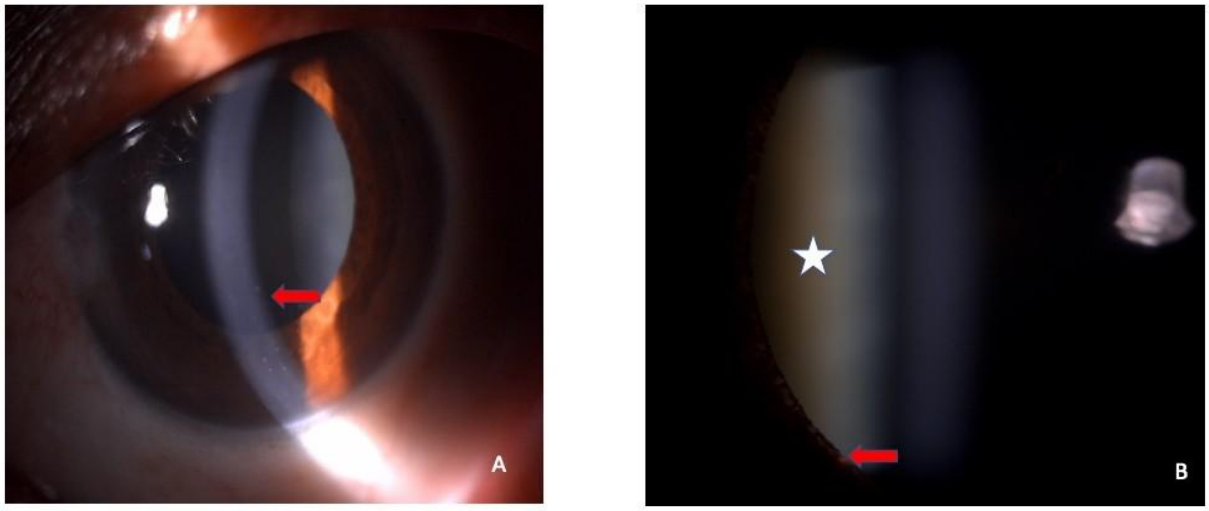


Figure 1. (A) Slit-lamp photograph of the left eye (OS) showing diffuse stellate keratic precipitates distributed over the corneal endothelium (red arrow). (B) High-magnification retroillumination slit-lamp image of the OS showing iris sphincter atrophy (red arrow) and associated cataractous lens changes (white star).

Table 1. Timeline of clinical course, investigations, treatment, and follow-up

Time	Clinical event
Approximately 2 years before ophthalmic presentation	Primary myelofibrosis was diagnosed, and systemic treatment with oral prednisolone 10 mg once daily and hydroxyurea 500 mg once daily was instituted.
2 months before ophthalmic presentation	The patient developed decreased vision in the left eye (OS).
Initial ophthalmic presentation	Best-corrected distance visual acuity was 20/20 in the right eye and 20/25 in OS. The OS showed diffuse stellate keratic precipitates, minimal anterior chamber flare, iris sphincter atrophy, grade-2 nuclear sclerosis with posterior subcapsular cataract, and no posterior synechiae or posterior segment abnormalities. Fuchs uveitis syndrome was diagnosed based on the characteristic unilateral clinical findings.
Initial diagnostic evaluation	Complete blood count, Mantoux testing, chest radiography, *Treponema pallidum* hemagglutination assay, and serum angiotensin-converting enzyme testing were performed. Rubella serology could not be performed because of financial constraints.
Following ophthalmic evaluation	Topical nepafenac ophthalmic suspension 0.1% was initiated in the OS for symptomatic relief.
Subsequently	Uneventful phacoemulsification with posterior chamber acrylic intraocular lens implantation was performed in the OS. Postoperative treatment included topical prednisolone acetate 1% and moxifloxacin 0.5%.
Postoperative period	Best-corrected distance visual acuity improved to 20/20 in the OS.
3.5-year follow-up	Visual acuity remained stable, and the OS remained clinically quiescent without recurrent inflammation or secondary glaucoma.

Table 2. Bone marrow next-generation sequencing (NGS) findings in the patient with primary myelofibrosis

Locus	Genes	Mutation	Pathogenicity	Molecular Class	Variant Allele Frequency (%)
chr9:5073770	*JAK2*	c.1849G>T p.(Val617Phe)	Pathogenic	Class I	42.7%
chr4:106193797	*TET2*	c.4259T>G p.(Leu1420*)	Pathogenic	Class IV	44.1%
chr12:111885140	*SH2B3*	c.1029_1030insT p.(Pro344fs)	Pathogenic	Class IV	95.2%

Abbreviations: chr, chromosome; *JAK2*, Janus kinase 2; *TET2*, tet methylcytosine dioxygenase 2; *SH2B3*, SH2B adaptor protein 3; NGS, next-generation sequencing. Note: The identified *JAK2* Class I variant represents a clinically significant driver mutation associated with responsiveness to targeted therapy, including ruxolitinib. NGS analysis was performed using the Oncocept HAEM™ DNA panel (Neuberg Diagnostics/NCGM, Ahmedabad, India).

Investigations, including complete blood count, Mantoux skin test, chest radiography, Treponema pallidum hemagglutination assay (TPHA), and serum angiotensin-converting enzyme (ACE) levels, were within normal limits. The erythrocyte sedimentation rate (ESR) was 31 mm/hour. The principal differential diagnostic considerations included infectious and granulomatous causes of chronic anterior uveitis; the available systemic investigations were unrevealing, although rubella serology could not be performed because of financial constraints. The patient was initiated on topical nepafenac ophthalmic suspension 0.1% (Nevanac®, Alcon Laboratories [India] Pvt. Ltd., Bengaluru, Karnataka, India) in the OS for symptomatic relief. Systemic medications for PMF included oral prednisolone (Wysolone®, Pfizer Ltd., Mumbai, Maharashtra, India) 10 mg once daily in the morning and oral hydroxyurea (Hydrea®, Bristol-Myers Squibb India Pvt. Ltd., Mumbai, Maharashtra, India) 500 mg once daily. The patient subsequently underwent uneventful phacoemulsification with implantation of a posterior chamber acrylic intraocular lens in the OS. Postoperative management included topical prednisolone acetate ophthalmic suspension 1% (Pred Forte®, Allergan India Pvt. Ltd., Bengaluru, Karnataka, India) and topical moxifloxacin ophthalmic solution 0.5% (Vigamox®, Alcon Laboratories [India] Pvt. Ltd., Bengaluru, Karnataka, India), administered with tapering frequency over the postoperative period. The postoperative BCDVA improved to 20/20. The patient was followed up for 3.5 years, during which the eye remained clinically quiescent with stable visual acuity and no evidence of recurrent inflammation or secondary glaucoma at the final visit. The key clinical events, investigations, interventions, and outcomes are summarized chronologically in Table 1.

The patient had a 2-year history of PMF and was receiving systemic therapy with oral prednisolone and hydroxyurea. Bone marrow aspiration showed normal bony trabeculae surrounding a hypercellular marrow. Megakaryocytes were increased in number and predominantly exhibited large hyperlobulated forms, with occasional cells showing hyperchromatic nuclei. Focal megakaryocytic clustering, comprising approximately 3–5 megakaryocytes per cluster, was also observed. The erythroid lineage appeared suppressed. The myeloid series demonstrated normal maturation patterns with preserved cellular distribution. Reticulin staining revealed a fine reticulin fiber network diffusely involving most areas of the section, without evidence of coarse fiber formation, consistent with grade 2/4 reticulin fibrosis (WHO MF-1). The original histopathology slide was unfortunately unavailable for review at the time of presentation.

To further characterize the underlying hematologic disorder, molecular genetic testing and cytogenetic analysis were performed at the discretion of the treating oncologist. Conventional cytogenetic evaluation showed a normal karyotype. However, next-generation sequencing (NGS) of the bone marrow identified pathogenic variants involving the *JAK2* gene on chromosome 9, *TET2* on chromosome 4, and *SH2B3* on chromosome 12 (Table 2).

Written informed consent was obtained from the patient for the publication of this case report. In accordance with institutional guidelines and local regulations, formal institutional ethical approval was not required for the publication of a single, de-identified retrospective case report. This work adhered to the ethical principles of the Declaration of Helsinki, and every effort has been made to ensure complete anonymization of patient details.

DISCUSSION

Here we described the rare coexistence of Fuchs uveitis syndrome in a patient with PMF, a chronic MPN with infrequently reported ocular involvement. The patient presented with characteristic unilateral features of Fuchs uveitis, including diffuse stellate keratic precipitates, iris sphincter atrophy, low-grade anterior chamber inflammation, and cataract formation. Hematologic evaluation demonstrated established PMF with pathogenic *JAK2*, *TET2*, and *SH2B3* mutations.

Ocular manifestations of MPN [7, 10–38] are elaborated in Table 3. PMF is the least common disease among chronic myelofibrotic neoplasms [39]. Ocular manifestations in PMF patients occur due to hematological abnormalities causing microvascular disturbances and hyperviscosity, thus leading to thrombotic and ischemic events. Central nervous system involvement leads to decreased vision and other symptoms due to such events [10, 18]. None of these were present in our patient.

In the early stages of disease, thrombotic events are more common due to erythrocytosis and thrombocytosis [10]. In later stages, blood cell depletion causes anemia, which further causes ischemic retinopathy, neovascularization, retinal hemorrhages, and cotton wool spots [10]. PMF cases with extramedullary hematopoiesis in orbit, lacrimal gland, and conjunctiva [40–42]; pachydermoperiostosis in which there is soft tissue hyperplasia resulting in tarsitis and ptosis; severe iritis and profound vision loss secondary to occlusive vasculopathy reported in essential thrombocytosis (which is a variant of myeloproliferative disease) [10, 16, 42–44]; angle closure glaucoma secondary to paraneoplastic infiltration of uveal tract [19]; neovascular glaucoma secondary to vascular occlusions [17, 19]; bilateral retinal hemorrhages [45]; inflammatory central retinal vein occlusion [39]; bilateral neovascularization with vitreous hemorrhage [18]; bilateral papilledema [46]; and cytomegalovirus retinitis in patients receiving ruxolitinib [33] are reported so far in PMF patients.

Reported ocular associations of Fuchs uveitis syndrome are infections like herpes simplex virus, rubella, ocular toxoplasmosis, cytomegalovirus, varicella zoster virus, chikungunya virus, and Horner's syndrome [47–49]. Ocular involvement in PMF is rare, with very few cases reported [10, 19, 50, 51].

In Fuchs uveitis syndrome, inflammation in the eye leads to changes in iris color (heterochromia) and potential cataract and glaucoma development. In PMF, inflammation and clonal myeloid cell proliferation drive excessive bone marrow fibrosis [48, 52–54]. Figure 2 summarizes the immunopathogenic mechanisms in both conditions [55–58].

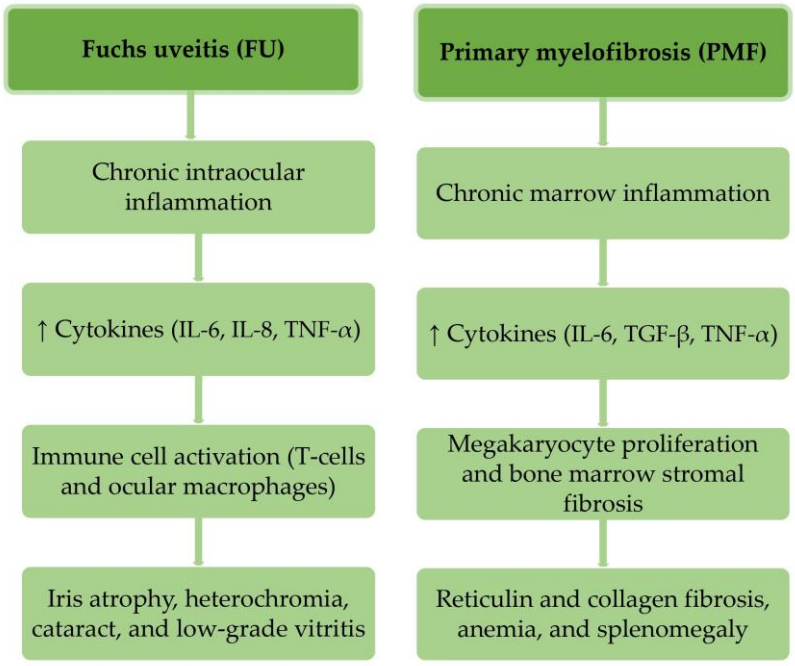


Figure 2. The simplified pathway of pathogenesis of FU and PMF [53–58]. Abbreviations: IL, interleukin; TNF-α, tumor necrosis factor-alpha; TGF-β, transforming growth factor-beta.

Table 3. Ocular manifestations associated with myeloproliferative neoplasms and related therapies

Category	Ocular manifestation	Reported association / mechanism
Anterior segment	Conjunctival hemorrhage, thrombosis, conjunctivitis [7, 10–12].	Conjunctival hemorrhage may occur secondary to thrombocytosis-associated acquired von Willebrand syndrome, whereas conjunctival infarction/thrombosis is rare because of rich vascular supply; conjunctival extramedullary hematopoiesis has also been reported in PMF [7, 10–12].
	Bloody tears (hemolacria) [13].	Hemorrhagic ocular manifestation associated with chronic myelogenous leukemia [13].
	Conjunctival injection [10, 14, 15].	Likely related to hyperviscosity, plethoric vascular congestion, and increased erythrocyte mass in myeloproliferative disease [10, 14, 15].
	Iritis / anterior uveitis [10, 16].	Likely secondary to ocular ischemia from recurrent microvascular occlusive events associated with essential thrombocytosis [10, 16].
	Neovascular glaucoma [10, 17].	Secondary ischemic ocular complication [10, 17].
Posterior segment	Retinal neovascularization, hemorrhages, vascular occlusions, venous dilatation and tortuosity, cotton wool spots, Roth spots [10, 18, 19].	Hyperviscosity and thromboembolic retinal vascular changes [10, 18, 19].
	Age-related macular degeneration and increased drusen prevalence [10, 20, 21].	Chronic systemic inflammation and complement dysregulation [10, 20, 21].
	Reduced macular sensitivity [22].	Associated with elevated systemic C5a inflammatory marker despite absent overt macular degeneration [22].
	Choroidal blood-flow alterations [10, 23].	Delayed blood flow [10, 23].
Optic nerve / neuro-ophthalmic manifestations	Anterior ischemic optic neuropathy, papilledema [10, 24, 25].	Ischemia caused by vascular and hematologic complications [10, 24, 25].
	Optic disc blurring [26].	Secondary hypertensive optic disc changes [26].
	Transient visual obscurations, amaurosis fugax, episodic monocular vision loss, scintillating visual phenomena, hemianopic disturbances, blurred vision, and visual hallucinations [10].	Central nervous system-related visual manifestations [10].
Orbital manifestations	Unilateral or bilateral orbital involvement [10, 27–29].	Extramedullary hematopoiesis and orbital infiltration [10, 27–29].
Treatment-related manifestations	Unilateral anterior uveitis following ruxolitinib (JAK 1/2 inhibitor) administration/discontinuation [30].	Potentially attributable to primary myelofibrosis itself, ruxolitinib administration, ruxolitinib discontinuation, or less likely frequent blood transfusions [30].
	Oculomotor dysfunction [31, 32].	Reported with fedratinib (selective JAK2 inhibitor) therapy [31, 32].
	Opportunistic ocular infections associated with ruxolitinib [10, 33, 34].	Immunomodulation-related infectious complications [10, 33, 34].
	Interferon-α–associated retinopathy, irritative conjunctivitis, anterior ischemic optic neuropathy [10, 35–38].	Drug-related ocular adverse effects [10, 35–38].

Abbreviations: PMF, primary myelofibrosis; C5a, complement component 5a; JAK, janus kinase.

The strengths of this report are detailed multimodal clinical characterization, molecular confirmation of PMF, long-term follow-up, and an extensive literature review summarizing the spectrum of ocular manifestations associated with MPN. Limitations are the single-case design, absence of rubella serology and *in vivo* confocal microscopy, and inability to establish a causal relationship between PMF and Fuchs uveitis syndrome. Future multicenter prospective studies integrating ophthalmic, molecular, and immunologic profiling are warranted to determine the true prevalence of uveitic manifestations in PMF and to further elucidate shared inflammatory and cytokine-mediated pathogenic mechanisms.

CONCLUSIONS

This case highlights the occurrence of clinically characteristic Fuchs uveitis syndrome in a patient with established primary myelofibrosis, representing—to the best of current knowledge—a previously unreported association. Although a causal relationship cannot be established from a single case, further studies are warranted to clarify the potential relationship between primary myelofibrosis and Fuchs uveitis syndrome and to better elucidate the underlying immunopathogenic mechanisms.

ETHICAL DECLARATIONS

Ethical approval: Written informed consent was obtained from the patient for the publication of this case report. In accordance with institutional guidelines and local regulations, formal institutional ethical approval was not required for the publication of a single, de-identified retrospective case report. This work adhered to the ethical principles of the Declaration of Helsinki, and every effort has been made to ensure complete anonymization of patient details.

Conflict of interests: None.

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