Journal of Biomed Research

Salem et al., J Biomed Res. 2025;6(1):11-16.

Review Article

Blinding uveitis induced by secukinumab: A case-based review

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Received date: November 05, 2024
Accepted date: December 09, 2024

Abstract

Secukinumab, a human monoclonal antibody targeting interleukin-17A (IL-17A), is widely used for treating immunoinflammatory disorders. While effective, drug-induced uveitis (DIU) is a rare but serious adverse effect associated with biologic medications like TNF-α inhibitors and more recently, IL-17 inhibitors such as secukinumab. We present a case of posterior uveitis in a 42-year-old male with AS who developed ocular symptoms two years after starting secukinumab. The patient experienced a rapid decline in vision, eventually leading to blindness in one eye despite corticosteroid treatment. A literature review identified seven cases of secukinumab-associated uveitis, most of which were anterior and bilateral. In these cases, patients responded to corticosteroids, and many continued secukinumab without recurrence. However, in our case, posterior uveitis developed, and the patient's condition worsened despite standard therapy. Secukinumab-induced uveitis though rare, can be severe and may require immediate ophtalmologic intervention. Clinicians should be aware of this paradoxial effect, particularly as biologic therapies are increasingly used. Further research is essential to better understand the mechanisms and risk factors associated with this adverse event.

Keywords: Ankylosing spondylitis, Chorioretinitis, Uveitis, Secukinumab, IL17A inhibitors, Drug adverse reaction

Introduction

Secukinumab is a fully human monoclonal antibody that specifically targets interleukin-17A (IL-17A), a key cytokine involved in various immunoinflammatory disorders [1,2]. It is approved for treating psoriasis, psoriatic arthritis (PsA), and ankylosing spondylitis (AS), [1-3] demonstrating high efficacy with minimal adverse effects, such as nasopharyngitis, headache, and diarrhea [4,5].

Uveitis is a prevalent inflammatory ocular disease that encompasses various clinical forms, including anterior, intermediate, posterior, and pan uveitis [6,7]. It remains a significant cause of visual impairment globally, with up to 35% of affected patients experiencing severe visual loss leading to blindness [6]. Uveitis can result from infectious or non-infectious causes and may also be associated with systemic diseases or certain medications [6,8]. Although drug-induced uveitis (DIU) is rare with a prevalence of 0.5%, it can be a vision-threatening adverse reaction [9]. Commonly implicated medications include cidofovir, rifabutin, bisphosphonates, sulfonamides, immune checkpoint inhibitors, topical β-blockers, various vaccines and more recently tumor necrosis factor alpha (TNF-α) inhibitors [7,10,11]. While biologic therapies, including secukinumab, are used to manage ocular inflammation, paradoxical uveitis has been reported with these agents [12].

In this article, we report a case of new-onset posterior uveitis following secukinumab therapy, along with a brief review of the related literature.

Case Presentation

A 42-year-old male was diagnosed with ankylosing spondylitis (AS) 20 years ago, presenting with bilateral sacroiliitis and peripheral arthritis that initially improved with non-steroidal anti-inflammatory drugs (NSAIDs). He had no involvement of other joints, no ocular disorders, and no systemic symptoms such as shortness of breath, abdominal pain, bowel disturbances or skin lesions.

In 2013, he was started on etanercept, but he developed a skin eruption after two years of treatment. He was then switched to adalimumab 40 mg every two weeks, leading to significant improvement. However, in 2021, despite being on adalimumab, he experienced a flare-up of his disease and was started on secukinumab 150 mg weekly, along with methotrexate and meloxicam 15 mg daily.

In November 2023, he presented to an ophthalmologist with blurred vision and a painful, red left eye and was diagnosed with chorioretinitis. He denied any previous similar symptoms. A comprehensive workup, including blood tests, syphilis, toxoplasmosis, and Herpes Simplex Virus serologies, tuberculin skin test, and chest radiography, returned normal results. After excluding infectious causes, secukinumab and methotrexate were discontinued, and oral prednisone, 50 mg daily, was initiated with close monitoring. Despite corticotherapy, the patient's visual acuity rapidly deteriorated, resulting in blindness in the left eye within two weeks of symptom onset. The lesions remained stable without any improvement at the 3-month follow-up visit.

Search Strategy

We conducted a comprehensive review of the literature using primary keywords such as "Secukinumab" and "uveitis," with additional related terms including "IL-17 inhibitors," "biologics," "ocular inflammation," "behçet syndrome," and "adverse drug reaction".

The search was carried out across PubMed, Science direct, and Google Scholar databases.

We applied filters to restrict the results to English-language articles published within the last 10 years. We ruled out secukinumab induced infectious uveitis and scukinumab associated Behçet's disease cases that did not present with ophthalmologic symptoms.

Results

Our literature review identified seven relevant case reports. **Table 1** summarizes the demographic and clinical characteristics of these cases.

There were four males and three females, with a mean age of 39 years. Underlying inflammatory conditions were AS in four cases [1,3,13,14] and psoriasis associated with PsA in three cases [4,15,16]. The duration of inflammatory disease ranged from at least 8 to 25 years. All patients had previously been treated with various therapies, primarily biologics such as anti-TNF-α agents. The dose of secukinumab varied between 150 and 300 mg per week. Uveitis developed in all seven patients between 1 month and 1 year after starting secukinumab. Only one patient experienced a flare of pre-existing uveitis [1]; the remaining six patients had no prior history of ocular disease. In two cases, uveitis was associated with inflammatory disease flare [16,15]. It occurred in the context of *de novo* Behçet's disease in two other cases [4,3] and it was isolated in the remainig two cases [13,14]. All reported uveitis cases underwent laboratory testing and appropriate radiographic evaluation to exclude infectious and other immunologic causes. The uveitis was most frequently anterior in location (4 out of 7 cases) and predominantly bilateral in nearly all instances (5 out of 7 cases). Secukinumab was continued in three cases and discontinued in three cases. Patients ultimately achieved complete recovery with either topical or systemic corticosteroid therapy (n=3) [3,4,14], methotrexate (n=1) [1] or infliximab (n=1) [15]. When secukinumab was continued, no relapses or recurrences of ocular inflammation were observed during the follow-up period [1,13,14]. Similar to these previously reported cases, our patient developed new-onset unilateral posterior uveitis two years after starting secukinumab for prolonged AS, following prior treatment with TNF-α inhibitors. He had no prior history of ocular disease or similar episodes. Unfortunately, unlike the reported cases, our patient did not improve after discontinuing secukinumab and receiving oral corticosteroids. Instead, his condition worsened rapidly, resulting in blindness.

Discussion

Based on our literature review, we present the eighth reported case of secukinumab-induced uveitis.

In all reported cases in addition to ours, common potential causes of uveitis were ruled out; however, uveitis as a manifestation of the underlying systemic disease remains a consideration.

It should be noted that uveitis has an increased risk of occurrence in patients with inflammatory diseases (including psoriais and PsA) and being the most common extra-articular manifestations of AS [17,18]. The first occurrence of uveitis is typically observed between the ages of 20 and 40, aligning with the average age of the reported cases, which is 39 years. Moreover, some studies suggested that the onset of uveitis is linked to a longer duration of the underlying inflammatory disease, as seen in these cases where the condition had persisted for at least 8 years [14].

Interestingly, despite their extended history of inflammatory diseases, none of the patients had previously experienced uveitis or any ocular symptoms. They had been treated intermittently with biologics and methotrexate, managing frequent disease flares without any eye complaints. However, their first episode of uveitis emerged only within months of initiating secukinumab therapy. This raises the possibility that the recent introduction of secukinumab may be the triggering factor.

The timing between secukinumab administration and onset of uveitis in our patient, along with previous case reports, ranging from 1 to 24 months, points to a possible drug-induced cause in these cases. In fact, this period aligns with the literature on drug-induced uveitis, especially with TNF-α inhibitors, where onset usually occurs between 16 and 19 months [7].

Unlike the reported cases where patients developed uveitis while on secukinumab alone, our patient was concurrently treated with both methotrexate (MTX) and secukinumab. MTX is typically the preferred disease-modifying agent for managing uveitis. A previous meta-analysis on MTX efficacy in non-infectious uveitis revealed that about 75% of patients experienced improvement in intraocular inflammation. Additionally, a separate cohort study demonstrated that methotrexate could prevent the recurrence of uveitis, whether used alone or in combination with anti-TNF-α agents [19]. Therefore, methotrexate is unlikely to be the triggering factor.

Table 1. Main characteristics of Secukinumab-induced uveitis cases reported in literature.

Author, Year	Number of uveitis cases	Gender	Age	Systematic disease/ Duration	Prior therapies	Dose of secukinumab	Total number of doses received before uveitis onset	Delay of onset after secukinumab initiation	Diagnostic	Location of uveitis	Drug status after uveitis onset	Management	Outcome
Our case, 2024	1	M	42	AS/20 years/ NS	NSAIDs, etanercept, adalimumab, methotrexate	150 mg/week	NS	2 years	New-onset Chorioretinitis	Unilateral Posterior	Discontinuation	Oral corticosteroid	Onset of total blindness
Altinisik, 2023 [13]	1	F	48	AS/NA	NA	NA	NA	NA	New-onset APMPPE	Unilateral Posterior	Continuation	NA	Recovery
Nelson*et al.*, 2023 [1]	1	F	16	JIA/11 years HLA-B27 negative	Naproxen, methotrexate, adalimumab	NS	NS	1 year	Flare of uveitis	Bilateral Anterior	Continuation	Subcutaneous methotrexate	Recovery
Nadwi *et al.*, 2020 [14]	1	M	47	AS/25 years HLA-B27 positive	NSAIDs, Methotrexate, adalimumab, etanercept	150 mg/week (five doses) then 300 mg/ month	11 doses	6 months	New-onset uveitis	Bilaeral Anterior	Continuation	Local corticosteroid eye drops	Recovery (5 weeks)
Barrado-Solís *et al.*, 2020 [4]	1	M	48	Psoriasis PA/10 years/ NS	Methotrexate, ustekinumab	NS	NS	6 months	Emergence of *de novo* BD with uveitis	Bilaeral Anterior	Discontinuation	Oral corticosteroid treatment	Recovery
Dincses *et al.*, 2019 [3]	1	M	29	AS/9 years HLA-B27 positive	Adalimumab, etanercept, certolizumab	150 mg/week	3 doses	2 months	Emergence of *de novo* BD with uveitis	Bilaeral Panuveitis	Discontinuation	3 pulses of methyl-prednisolone + infliximab 5 mg/kg	Recovery (5 months)
Hoshina *et al.*, 2018 [16]	1	F	43	Psoriasis PA/8 years	infliximab	300 mg/week	NS	1 month	Flare of psoriasis with deterioration of vision	NA	NA	NA	NA
Su *et al.*, 2017 [15]	1	M	45	Psoriasis, PA/long-history	Methotrexate, phototherapy, acitretin, cyclosporine, etanercept, adalimumab, ustekinumab, infliximab	300 mg/week	4 doses	1 month	Flare of arthritis with new-onset uveitis	Bilaeral Anterior	Discontinuation	Infliximab	Recovery

AS: Ankylosing Spondylitis; NSAIDs : Non-Steroidal Anti-Inflammatory Drugs; NS : Not Specified; NA : No Access; APMPPE : Acute Posterior Multifocal Placoid Pigment Epitheliopathy; JIA : Juvenile Idiopathic Arthritis; PA : Psoriatic Arthritis; BD : Behçet Disease; AU : Anterior Uveitis; PA : Psoriatic Arthropathy

Secukinumab is generally a well-tolerated treatment option; nevertheless, it has some adverse effects mainly increased risk of infection.

There is no widely available data documenting secukinumab as the underlying cause of uveitis. Our literature research have found, however, seven reported cases of uveitis under secukinumab teatment in addition to our eighth case.

Secukinumab was initially investigated for its efficacy in various inflammatory diseases, including non-infectious uveitis and uveitis associated with Behçet's disease (BD) [17]. Early clinical trials revealed that secukinumab was not only ineffective in controlling non-infectious uveitis and preventing ocular attacks associated with BD but also identified uveitis as one of the most frequently reported adverse events caused by secukinumab [3,20]. However, in contrast to these earlier findings, more recent studies have shown that secukinumab can be effective in treating non-infectious uveitis. Despite these positive outcomes, uveitis has still emerged as a potential adverse event, raising concerns about the drug's complex role in uveitis development. This implies that although secukinumab may initially alleviate uveitis, it might later inadvertently cause the condition as a paradoxical reaction [17].

In light of this, a multicenter study on long-term secukinumab therapy has demonstrated its safety, with no reported ocular exacerbations in patients with a history of ophthalmological involvement. Additionally, recent research has shown that intravenous administration of secukinumab is effective and well-tolerated. These findings reveal a nuanced and evolving understanding of secukinumab's role in uveitis management, highlighting the need for careful consideration of administration methods and monitoring for paradoxical reactions [21].

Following these findings, there has been increased interest among physicians in studying the incidence of uveitis associated with secukinumab treatment. Subsequent research has indicated that secukinumab is linked not only to the recurrence of pre-existing uveitis but also to the emergence of new-onset uveitis.

In previous clinical trials of secukinumab, the most common ocular adverse effects reported were eye pain and blurred vision, each affecting 8.1% of participants [22]. Deodhar *et al.* reported an exposure-adjusted incidence rate (EAIR) for uveitis of 1.4 per 100 patient-years in patients with ankylosing spondylitis (AS) throughout the treatment period. Of the total uveitis cases (3.3%), 14 (1.8%) were flares and 12 (1.5%) were new-onset cases [17]. This aligns with findings by Lindström *et al.*, who observed a rate of anterior uveitis diagnoses at 6.8 per 100 patient-years, with 1.3% of new-onset anterior uveitis cases associated with secukinumab. Notably, the mean age of patients who developed uveitis following secukinumab treatment in Lindström's study was 38.2 years, closely matching the mean age reported in published case reports. Similar to our literature review, these studies revealed a predominance of males (sex ratio M/F = 2.7). The mean duration of AS among these patients was 6 years, which contrasts with the 13 years reported in our review. Most new-onset uveitis cases involved patients previously treated with anti-TNF-α drugs, with a few having been treated with methotrexate [23]. Importantly, while secukinumab clinical trials reported some cases of uveitis, only one out of 26 reported cases in Dheodar's study was severe, with the remainder classified as mild or moderate [17].

Conversly, postmarketing data indicates a cumulative total of 29 uveitis cases, with an exposure-adjusted reporting rate (EARR) of 0.03 cases per 100 patient-years. However, many of these postmarketing cases were poorly documented, with at least half being relapses of preexisting uveitis. An observational cohort study in 2024 found an incidence rate of uveitis during secukinumab therapy at 9.4 per 100 patient-years, with no statistically significant difference between the incidence rates before and during secukinumab treatment (p=0.74) [24].

When comparing secukinumab to other TNF-α inhibitors, a systematic literature review on the efficacy and safety of biological disease-modifying antirheumatic drugs (bDMARDs) indicated that both secukinumab and etanercept were associated with a higher risk of uveitis compared to other anti-TNF-α agents [25]. Some researchers have suggested that TNF-α inhibitors may provide better protection against recurrent anterior uveitis flares than secukinumab [23]. In observational studies, adalimumab and infliximab were associated with a lower risk of uveitis flares (0.5%) compared to secukinumab (1.3%) and etanercept (1.2%). However, new-onset anterior uveitis was rare across all treatments [23,26]. Conversely, a recent study examining the impact of IL-17 inhibitors, including secukinumab and netakimab, on uveitis found that uveitis occurred in 7% of patients during secukinumab therapy and in 4% of patients during netakimab therapy. This study revealed no significant differences in the incidence rates of uveitis between IL-17 inhibitor therapy and non-biological treatments [24].

These findings suggest that while secukinumab and other IL-17 inhibitors may have a comparable or slightly higher incidence of uveitis compared to some TNF-α inhibitors, the overall risk of new-onset uveitis remains relatively low.

When comparing the incidence of secukinumab induced uveitis across different studies, it is crucial to consider the proportion of patients with a history of uveitis, as prior episodes are a significant risk factor for new flares [23]. In our literature review, we noted that a patient with juvenile idiopathic arthritis (JIA) experienced a flare of uveitis after transitioning to secukinumab therapy, despite showing improvement in their joint disease. This case highlights that while secukinumab may benefit arthritis; it may not always prevent or may even exacerbate uveitis in some patients.

The incidence of uveitis—both new-onset and flares—remains low, making it difficult to draw definitive conclusions, especially given the discrepancies between clinical trial results and postmarketing observations. These findings highlight the need for continued vigilance and further research to fully understand the relationship between secukinumab and uveitis. Nevertheless, the increasing number of documented cases suggesting secukinumab as a potential cause of new-onset or exacerbated uveitis indicates that it may induce uveitis as a paradoxical effect.

The exact mechanism behind secukinumab-induced uveitis remains largely unclear. It is believed that drug-induced uveitis could result from direct drug toxicity or indirect mechanisms such as immune-mediated vasculitis [27]. The complex immunopathogenesis of adverse reactions to biologic agents is influenced by the local cytokine environment, which can condition T helper 17 cells to adopt either proinflammatory or regulatory roles. This variability may help explain the paradoxical reactions observed [15].

The strength of this review lies in its aggregation of current data on secukinumab-induced uveitis from clinical trials and case reports. It aims to identify key characteristics of patients who developed uveitis during secukinumab treatment and provide a general overview of this adverse effect. However, several limitations should be noted. The primary limitation is the small number of case reports and the lack of comprehensive information in some of these reports. Additionally, the incidence of uveitis reported in many cases was based on observations by prescribing physicians rather than confirmed by specialized ophthalmological consultations. The large number of contradictory results across studies further complicates the analysis. Moreover, distinguishing uveitis caused by secukinumab from that due to ongoing active inflammatory diseases is challenging and may confound the evaluation of a causal relationship.

Conclusion

Secukinumab-associated uveitis, though rare, may become more commonly encountered, and healthcare practitioners should be vigilant and refer any suspected cases for ophthalmological evaluation. Although a definitive cause-and-effect relationship has not yet been established, it is crucial for physicians to be aware of this potential paradoxical effect. Continued research and additional published experiences are needed to fully understand and confirm this possible adverse reaction.

Author Contribution Statement

YSM: data collection, literature review, conceptualization, design of the study, data analysis, interpretation of the results, and writing the manuscript; **IA:** data collection, and assisted in the analysis and interpretation of the findings; **ID:** design of the study and assisted in the interpretation of the findings; **FZ:** literature review and critical revision of the manuscript; **OC:** provided methodological support and critically revised the manuscript; **SEA:** provided overall supervision of the study, contributed to data interpretation, and critically reviewed the manuscript.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

Consent to Participate

Given the retrospective nature of the study, written consent was not required. However, oral consent was obtained from the patient, and the anonymity was strictly preserved in accordance with ethical guidelines.

Source(s) of Support and Funding

This research received no funding.

Conflicts of Interest

The authors have no conflicts of interest.

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