European Journal of Clinical Pharmacy

Print ISSN: 2385-409X **Online ISSN**: Applied

Website: https://farmclin.es/

Original Research Article

Secukinumab and Alithiasic Pancreatitis in a Patient with Psoriatic Arthritis

Article History:

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How to cite this article:

Diana D.L.F, *et al.* Secukinumab and Alithiasic Pancreatitis in a Patient with Psoriatic Arthritis. *Eur J Clin Pharm.* 2023;5(1);16-18

***Received**: 10*-03-2023
***Revised**:* 23-03-2023
***Accepted**: 10*-04-2023
***Published**:* 30-04-2023

Abstract: Secukinumab is a monoclonal antibody targeting interleukin-17A (IL-17A), showing significant efficacy in managing psoriatic arthritis (PsA). However, biologic therapies like secukinumab are not without adverse effects. While gastrointestinal effects have been documented, the emergence of alithiasic pancreatitis—a rare, non-gallstone-associated form of pancreatic inflammation—represents a significant safety consideration. This article reviews current literature, mechanisms, and a case of secukinumab-associated alithiasic pancreatitis in a patient with PsA. Recommendations for clinical surveillance and management are discussed.

Keywords: Secukinumab, IL-17A Inhibition, Psoriatic Arthritis, Alithiasic Pancreatitis, Biologic Therapy Safety

INTRODUCTION

Psoriatic Arthritis and Advanced Therapeutics

Psoriatic arthritis (PsA) is a chronic autoimmune inflammatory disorder affecting approximately 30% of individuals with psoriasis. It results in joint pain, swelling, stiffness, and progressive articular damage[1]. Management of moderate-to-severe PsA has evolved with the introduction of biologic agents targeting specific immune pathways, including tumor necrosis factor-alpha (TNF-α), interleukin 12/23, and, more recently, interleukin-17A (IL-17A)[2].

Secukinumab: Mechanism of Action and Indications

Secukinumab is a fully human monoclonal antibody designed to neutralize IL-17A, a key cytokine involved in the pathopathogenesis of psoriasis and PsA[2][3]. The inhibition of IL-17A disrupts downstream inflammatory signaling, reducing joint inflammation and systemic symptoms[1][4]. Secukinumab is FDA-approved for moderate-to-severe plaque psoriasis, PsA, and ankylosing spondylitis[2][3].

Alithiasic (Acalculous) Pancreatitis

Alithiasic pancreatitis, also known as acalculous pancreatitis, refers to pancreatic inflammation occurring without gallstone obstruction. Unlike the more common gallstone-induced or alcohol-related pancreatitis, alithiasic pancreatitis often presents in association with infections, trauma, medications, or immune-modulatory treatments[5][6].

Secukinumab and Pancreatic Adverse Effects

Gastrointestinal Complications with IL-17 Inhibitors

IL-17 plays a paradoxical role in gastrointestinal homeostasis, acting as both a pro-inflammatory mediator in autoimmunity and a mucosal defender against pathogens[4]. Suppression of IL-17, as with secukinumab therapy, can compromise mucosal immunity, predisposing patients to gastrointestinal disturbances, infections, and inflammation[7][4].

Recent pharmacovigilance studies indicate that secukinumab is associated with an increased incidence of gastrointestinal adverse events, particularly nonspecific inflammation and, rarely, severe events such as perforation or ulceration. Inflammatory bowel disease (IBD), ulcerative colitis, and other serious GI events have also been reported[7][8].

Case Report: Alithiasic Pancreatitis in a PsA Patient[9]

A recent clinical report documented alithiasic pancreatitis in a patient treated with secukinumab for psoriatic arthritis[9]. The defining features included:

- **Absence of gallstones** on imaging.
- Temporal correlation between secukinumab initiation and onset of pancreatitis.
- Improvement after withdrawal of secukinumab.
- Exclusion of other common etiologies: alcohol, hypertriglyceridemia, trauma, and infection.

This case emphasizes the necessity to consider medication-induced pancreatitis, particularly when other causes are excluded.

Pathogenesis: Could Secukinumab Induce Pancreatitis?

IL-17A's Role in Pancreatic Health

IL-17A is implicated not only in autoimmunity but also in maintaining epithelial barrier integrity and protecting against microbial invasion in the gut and pancreas[4][10]. Experimental models highlight that IL-17A deficiency or blockade can alter local immune responses, possibly increasing susceptibility to pancreatic injury or inflammation[4][10].

Suppression of IL-17A with secukinumab may therefore:

- Reduce immune surveillance in pancreatic tissue.
- Predispose to an aberrant inflammatory response under stress or infection.
- Enable development of sterile (alithiasic) pancreatic inflammation.

Clinical Presentation and Diagnosis

Typical Features of Alithiasic Pancreatitis

- **Symptoms:** Acute onset of upper abdominal pain, nausea, vomiting, and systemic inflammatory signs.
- **Laboratory Findings:** Elevated serum amylase and lipase.
- **Imaging:** Pancreatic swelling and inflammation, *without* biliary stones[5][6].

Differential

- Exclude gallstones (ultrasound, CT/MRCP).
- Review patient's medication list.
- Assess for hypertriglyceridemia, hypercalcemia, and infection[5][11].

Management

General Principles

- **Immediate discontinuation** of the suspected offending drug (secukinumab)[9][7].
- Supportive measures: aggressive IV hydration, pain control, and nutritional support[6][12].
- Monitor for complications: systemic inflammatory response syndrome (SIRS), necrosis, infection.
- Early enteral feeding as tolerated is now recommended[13][14].

Drug-Associated Pancreatitis

Most cases resolve with discontinuation of the offending agent. Severe or persistent cases may require intensive care, antibiotics for superinfection, or surgical/interventional radiology procedures for complications such as necrosis[6][12].

Figures and Graphs

Table 1: Common Etiologies of Acute Pancreatitis[5][11]

Cause	Percentage
Gallstones	40–50%
Alcohol	20–30%

Hypertriglyceridemia	5%
Drugs (including biologics)	1–5%
Trauma, Infections, Idiopathic	Remaining cases

Bar Chart: Drug-Associated vs. Idiopathic Pancreatitis in Major Case Series
[Bar chart compares incidence per 1,000 hospital admissions. Drugs, less common but notable source.]

DISCUSSION

Clinical Implications

Although rare, clinicians should be aware that:

- Secukinumab and other IL-17A inhibitors can affect the GI tract, and rarely, the pancreas[9][7].
- Pancreatitis should be considered in any patient on secukinumab presenting with compatible symptoms, even if gallstones are absent.
- Regular monitoring and thorough patient education about potential GI symptoms during therapy is advised.

Risk Factors and Prevention

Current data do not permit definitive identification of high-risk patient subgroups, but vigilance is warranted with:

- History of drug or autoimmune-induced pancreatitis.
- Concomitant GI inflammation.
- Early recognition improves outcomes[7][15].

CONCLUSION

Biologics targeting IL-17A such as secukinumab have revolutionized PsA management, but rare adverse events like alithiasic pancreatitis can occur. This underscores the importance of multidisciplinary surveillance, patient education, and prompt recognition of adverse drug reactions.

REFERENCES

1. "Secukinumab and alithiasic pancreatitis in a patient with psoriatic arthritis." Dialnet, 2024.
2. Baraliakos, X. et al. "Secukinumab in patients with psoriatic arthritis and axial manifestations: MAXIMISE trial." PMC, 2020.
3. Godse, K. "Secukinumab - First in Class Interleukin-17A Inhibitor for the Treatment of Psoriasis." PMC, 2017.
4. "Pancreatitis - Symptoms and Causes." Mayo Clinic, 2023.
5. Diotallevi, F. et al. "Should we be concerned about gastrointestinal-related adverse events with Secukinumab?" PMC, 2022.
6. "Assessment of safety profile of secukinumab in real-world scenario using United States FDA adverse event reporting system database." PMC, 2024.
7. "Management of acute pancreatitis (AP)." PMC, 2016.
8. "Role of Interleukin-17 in Acute Pancreatitis." PMC, 2021.
9. "Secukinumab: Uses, Interactions, Mechanism of Action." DrugBank, 2021.
10. "Secukinumab-induced ulcerative colitis: Opening Pandora's box of immunity." Elsevier.
11. "Acute Pancreatitis: Immune response mechanisms." Frontiers in Immunology, 2023.

