EJR European Journal of Rheumatology

DOI:10.5152/eurjrheum.2025.24079

Case Report

Aseptic Abscess Associated with Ankylosing Spondylitis—Cause or Effect of Therapy? Case Report

Przemysław Borowy*[1,2], Karol Urbański*[1], Katarzyna Gołojuch*[3], Patrycja Major[4], Jakub Smyk[3], Alicja Kamińska[5], Bogdan Batko[1,2], Joanna Szpor[6]

Abstract

Neutrophil dermatitis is a group of diseases characterized by the leakage of neutrophils in the skin and subcutaneous tissue with a non-infectious, autoinflammatory etiology. These include the aseptic abscess syndrome (AA). Diagnosis is based on histopathological examination and the exclusion of infectious, allergic, and cancer causes. The paper presents the case of a 41-year-old woman with inflammatory spondyloarthropathy (HLA-B27 antigen present), treated with secukinumab, who developed a painful, inflammatory tumor in her right breast. Antibiotic treatment was ineffective, and histopathological examination detected leaching mainly from granulocytes. Infectious and oncological background changes and IgG4+ disease were excluded. After the diagnosis was confirmed, glucocorticoid therapy was started, which brought rapid improvement, but after the dose was reduced, the tumor relapsed. The re-escalation of the steroid dose and the discontinuation of secukinumab coincided with the exacerbation of ankylosing spondylitis, which forced the inclusion of upadacitinib, which was effective and well tolerated. Single studies show high efficacy of TNF inhibitors as well as IL-6 or IL-1 blockades in the treatment of AA and secondary prevention in patients with failed steroid therapy. There are no reports of AA cases in the literature during treatment with secukinumab. Treatment with upadacitinib has so far not caused AA recurrence.

Keywords: Aseptic abscess, secukinumab, SpA

ORCID iDs of the authors:
P.B. 0000-0002-6578-0699;
K.U. 0000-0002-7647-9770;
K.G. 0009-0009-8567-0591;
P.M. 0009-0002-2754-4510;
J.S. 0009-0002-7585-656X;
A.K. 0009-0007-6393-3405;
B.B. 0000-0003-3999-8093;
J.S. 0000-0002-1237-6257.

Cite this article as: Borowy P, Urbański K, Gołojuch K, et al. Aseptic abscess associated with ankylosing spondylitis—cause or effect of therapy? Case report. *Eur J Rheumatol.* 2025, 12(3), 0079, doi:10.5152/eurjrheum.2025.24079.

[1] Department of Rheumatology and Immunology, J. Dietl Hospital, Krakow, Poland
[2] Andrzej Frycz Modrzewski Krakow University Faculty of Medicine and Health Sciences, Krakow, Poland
[3] Ludwik Rydygier Memorial Hospital, Kraków, Poland
[4] 5th Military Clinical Hospital in Kraków, Kraków, Poland
[5] Stefan Żeromski Specialist Hospital, Kraków, Poland
[6] Department of Pathomorphology, Jagiellonian University Medical College, Kraków, Poland

*These authors have contributed equally to this work and share first authorship

Corresponding author:
Przemysław Borowy
E-mail: pborowy@mp.pl

Received: August 17, 2024
Revision Requested: August 23, 2024
Last Revision Received: September 26, 2024
Accepted: October 13, 2024
Publication Date: July 30, 2025

Introduction

Neutrophilic dermatoses are a group of diseases characterized by neutrophilic infiltration of various skin layers and subcutaneous tissues, which may be associated with aseptic abscess formation. Importantly, this neutrophilic inflammation is not of bacterial or other infectious origin.[1] Neutrophilic dermatoses comprise a spectrum of diseases that differ in clinical presentation. This is proposed to be influenced by the histological location of neutrophilic infiltration.[2] However, the subtypes often present considerable overlap in clinical as well as histopathological presentation, and specific diagnosis may be challenging.[3] Neutrophils play a main role in innate immunity and are responsible for the response against infections. Moreover, they regulate innate and adaptive immunity and play a dual role in the tumor microenvironment.[4] A number of cases are also connected with autoinflammatory diseases such as rheumatoid neutrophilic dermatitis. While aseptic abscess syndrome is accompanied by inflammatory bowel disease in 42% of cases, it may coexist with other autoinflammatory diseases such as ankylosing spondylitis (AS).[5] Here, we present a female patient diagnosed with neutrophilic dermatitis of the breast. The first one was a previously healthy person with no underlying disease who presented with an aseptic breast abscess. The second one was initially diagnosed with AS and treated with an anti-IL-17A monoclonal antibody, who presented with neutrophilic dermatitis affecting the dermal and hypodermal layers of the skin.

Case Presentation

A 41-year-old woman with HLA-B27-positive AS was treated effectively with secukinumab from January 2021. In the beginning of November 2021, a nodule appeared in the left upper quadrant of the right breast. The affected area showed signs of inflammation. It was painful; the skin was warm and reddened. The onset was rapid, and in 2 days, the diameter reached 7 cm. The patient was initially treated with amoxicillin and clavulanic acid for 7 days, followed by ciprofloxacin for 10 days, with no improvement. The patient did not present general symptoms (fever, weakness, sweats). Secukinumab treatment was discontinued. However, after 2 weeks, inflammatory back pain relapsed, along with signs of ankle joint inflammation.

Ultrasonography revealed features of stromal oedema with dispersed, segmentally dilated milk ducts. Enlarged axillary lymph nodes were visible with a dilated cortical zone. Laboratory tests showed no

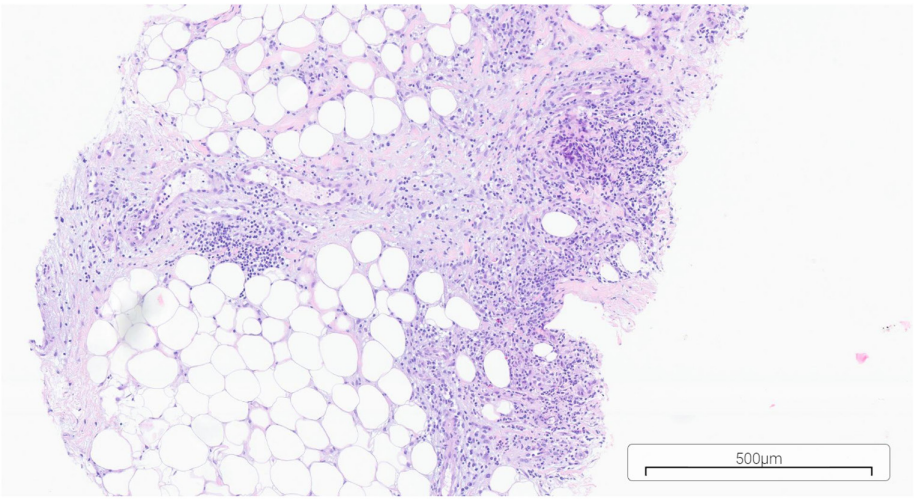

Figure 1. Surgical biopsy from a lesion of the left breast. The image contains adipocytes, inflammatory cells, fibroblasts, and connective tissue. Hematoxylin & eosin stain, magnification: 4×.

specific abnormalities. Inflammatory markers were low with negative CRP (C-reactive protein) and slightly elevated ESR (Erythrocyte Sedimentation Rate) and alpha fraction of serum proteins. Bacterial and fungal blood cultures and serological tests for bacterial etiologies were negative. Immunoglobulin fraction levels were not changed. Monoclonal proteins were not detected in serum nor urine. According to IgG subclasses, the IgG2 level was elevated whereas the IgG4 level was decreased.

Histopathological examination showed significant infiltration of lymphocytes, plasma cells, histiocytes, numerous neutrophils, and eosinophils (Figures 1-3). Visible ducts (CK7+) were surrounded by myoepithelial cells (CK5/6, p63). The analyzed specimen was classified as a benign lesion with a B2 category. Additional staining for IgG4 was performed. It showed up to 1 IgG4+ plasma cell in a high-power field (HPF) and up to 20 IgG+ plasma cells/HPF, which excluded IgG4 disease. The pathologist's opinion, based on overall histopathological findings together with the clinical context, suggested the diagnosis of neutrophilic lobular panniculitis associated with rheumatoid arthritis, rheumatoid neutrophilic dermatitis, or changes secondary to biological treatment.

Steroid treatment was started with an initial dose of 32 mg of methylprednisolone daily with rapid improvement. After dose reduction to 4 mg daily in January 2022, a relapse occurred, and steroids were again increased to 32 mg of methylprednisolone, with good results. At that time, NSAIDs (Nonsteroidal Anti-Inflammatory Drugs) were initiated because of the exacerbation of AS caused by secukinumab treatment suspension. Steroids were slowly and gradually reduced. By May 2022, the patient was receiving 4 mg of methylprednisolone daily with no signs of breast lesion recurrence. Because of the ineffectiveness of NSAIDs and high AS disease activity (BASDAI 7.5; VAS 86/100), upadacitinib was initiated as a disease-modifying antirheumatic drug. The patient continued low-dose glucocorticoids with complete resolution of dermatitis symptoms (Figure 4). Repeated laboratory test results were within normal limits. Upadacitinib was well tolerated and maintained in therapy.

The authors certify that they have obtained all appropriate patient consent (for patient details and images).

Main Points

- Due to the unknown possible autoinflammatory pathomechanism of neutrophil activation, there is a possible causal relationship with autoimmune diseases, in our case with SpA.
- The appearance of AA during treatment with an IL-17A inhibitor (secukinumab) has been reported in a patient. There is no information regarding the possible effects of anti-cytokine therapy triggering AA. Our observation is the first.
- Switching to ustekinumab did not lead to AA recurrence, suggesting a different mechanism for AA formation than that associated with IL-17.
- The publication of such observations will broaden our knowledge in this field and gather more data on possible causal relationships.

Discussion

Aseptic abscess syndrome is a rare manifestation of a variety of systemic inflammatory conditions. The pathophysiology of AA is not clearly understood. The characteristic pathological finding is sterile neutrophilic infiltration of the deep tissues, similar to neutrophilic dermatoses, which are characterized by sterile neutrophilic infiltration of the skin. Though the pathogenesis of AA has not been elucidated, it presents with autoinflammatory features: absence of disease-specific autoantibodies, activation of the innate immune system (neutrophils), and a long gap without any complaints between symptoms.[6]

There are no formal diagnostic criteria for AA. Ultimately, AA may be suspected in a person with radiological evidence of deep abscesses in the absence of infection, allergy, or neoplasm. The diagnosis is based on histopathological examination and exclusion of infectious, allergic, and neoplasmatic causes. The lesion localization and depth are the main issues used in neutrophilic dermatoses classification.

André[7] proposed that neutrophilic dermatoses should be divided into 3 subgroups. Our

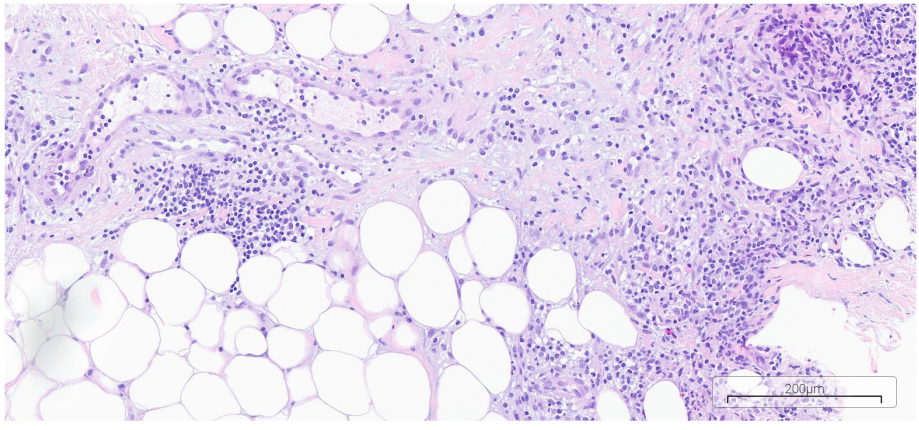

Figure 2. An extensive inflammatory infiltrate composed of neutrophils, eosinophils, macrophages, and lymphocytes. Additionally, necrotic debris and areas of fibrosis are visible. Hematoxylin & eosin stain, magnification: 10×.

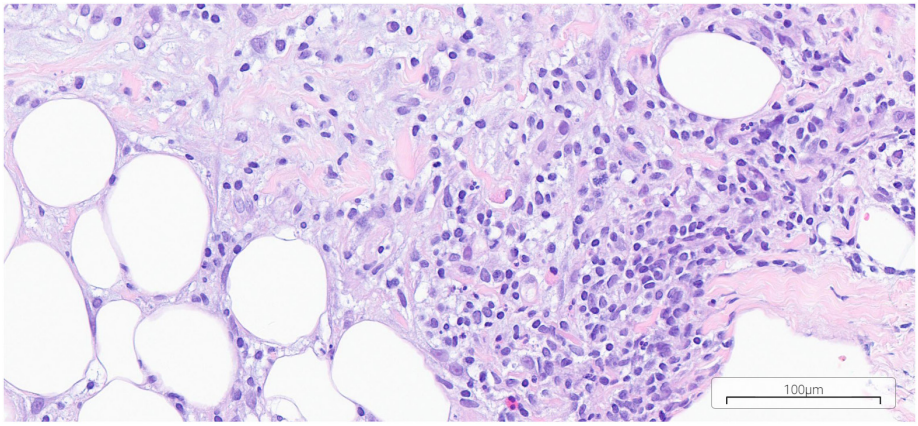

Figure 3. An extensive inflammatory infiltrate composed of neutrophils, eosinophils, macrophages, and lymphocytes. Hematoxylin & eosin stain, magnification: 20×.

patient belongs to the first, which includes superficial, epidermal, and pustular diseases, such as Sneddon–Wilkinson disease. Unfortunately, there are no specific features nor markers of AA that may help us to set a proper diagnosis. This results in long lag times and sometimes years of antibiotic treatment.[8]

Aseptic abscess cases described in the literature are found in patients with autoimmune diseases such as rheumatoid arthritis, inflammatory bowel disease, Cogan syndrome, recurrent polychondritis, Behçet's disease, systemic lupus erythematosus, or nodular arteritis. However, about 60% of patients with AA do not present any concomitant diseases.[9] It seems that the number of AA limited to the skin and subcutaneous tissue may be underestimated. Benign lesions may disappear spontaneously, which may be incorrectly attributed to antibiotic treatment. On the other hand, patients suffering from autoimmune diseases treated with immunosuppressive agents are under increased surveillance and have a greater chance of proper diagnosis. Even though, treatment-induced immunodeficiency usually draws attention to bacterial infection.

Single studies show high effectiveness of TNF-α inhibitors, as well as IL-6 or IL-1 blockade, in AA treatment and secondary prevention in patients with steroid treatment failure.[10] In our case, AA occurred during IL-17A inhibitor treatment. To our best knowledge, there are no published cases of AA during secukinumab treatment. There are no available data about AA treatment with IL-17/17A inhibitors.

There are no data assessing the influence of the underlying disease treatment on AA morbidity. It is not elucidated whether patients treated with biological agents such as TNF-α, IL-6, or IL1 blockers are more susceptible to AA than those treated with classical DMARDs (Disease-Modifying Antirheumatic Drugs).

We cannot exclude the potential relationship of AA with secukinumab treatment in our patient. After the AA diagnosis, secukinumab was switched to ustekinumab. The latter, however, blocks IL12 and IL23, which induces Th17 cells. In that regard, it reflects to some extent secukinumab's action. New biological treatment has not resulted in AA recurrence so far, which may suggest beyond IL17 in AA occurrence. The patient remains in remission for both AA as well as AS.

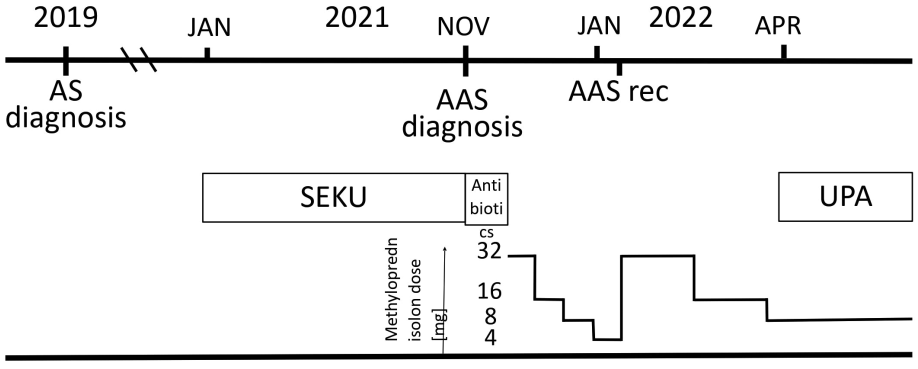

Figure 4. Patient's treatment history (AS, ankylosing spondylitis, AA, aseptic abscess syndrom;, rec, recurrence; SEKU, secukinumab; UPA,upadacitinib).

Informed Consent: Written informed consent was obtained from the patient who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – P.B., K.U., B.B.; Design – K.G., P.M., J.S., A.K.; Supervision – P.B., K.U., B.B.; Resources – P.B., K.U., K.G., P.M., J.S., A.K., B.B., J.SZ. ; Materials – J.SZ., K.G.; Data Collection and/or Processing – P.B., K.U., K.G., P.M., J.S., A.K.; Analysis and/or Interpretation – P.B., K.U., K.G., P.M., J.S., A.K., J.SZ.; Literature Search – K.G., P.M., J.S., A.K.; Writing – P.B., K.U., K.G., P.M., J.S., A.K.; Critical Review – P.B., K.U., B.B.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study has received no financial support.

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