Chula Med J. May - June 2025;69(3): 179 - 183 **DOI : 10.56808/2673-060X.**5518

Case Report

A tubo-ovarian abscess in an aplastic anemia case presenting as a fever of unknown origin: A case report

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Abstract

In this case report we describe a complex case of a tubo-ovarian abscess (TOA) presenting atypically as a fever of unknown origin (FUO) in a woman in her 30s initially diagnosed with severe aplastic anemia. The case involved a protracted febrile illness, heavy menstrual bleeding, and pancytopenia. Detection of an *Escherichia coli* tubo-ovarian abscess, rather than tuberculosis, necessitated a thorough investigation of atypical presentations of TOA. Negative tuberculosis tests indicated a possibility of various microbial etiologies. The resolution of fever following drainage and tailored antibiotic therapy, as well as timely surgical intervention, demonstrated the importance of specific management strategies in such cases. Awareness of the diverse presentations of TOA, such as FUO, especially in patients presumed to have other conditions, such as aplastic anemia, is crucial for appropriate diagnosis and treatment.

Keywords: Febrile neutropenia, pancytopenia, pyrexia of unknown origin, tuberculosis, tubo-ovarian abscesses

Prolonged febrile illness or fever of unknown origin (FUO) poses a diagnostic challenge in clinical practice, as it often indicates underlying systemic disorders ranging from infectious etiologies to autoimmune conditions and neoplastic processes. [1] When FUO is accompanied by hematologic abnormalities, diagnosis becomes more complex, necessitating a comprehensive and systematic approach to evaluate the underlying pathophysiology. [2] Tubo-ovarian abscesses (TOAs) are inflammatory masses characterized by purulent collections within the fallopian tubes, ovaries, and other surrounding structures, often arising from ascending genital tract infections, such as pelvic inflammatory disease (PID). They can manifest atypically as FUO or coexist with underlying systemic conditions, posing diagnostic challenges for clinicians. [3,4] Although the incidence of TOA varies, it is a significant complication associated with PID, occurring in about 15.0% of women with PID. [5] The presentation of TOA can be nonspecific, including fever, abdominal pain, and vaginal discharge. However, in patients with underlying conditions, such as hematologic disorders, the presentation can be atypical. In immunocompromised patients, there is a broader spectrum of pathogens causing TOAs, including common aerobic and anaerobic bacteria, and opportunistic organisms. [6] This necessitates a high index of suspicion and thorough investigation to accurately identify the underlying cause. The association between aplastic anemia and high susceptibility to infections, such as TOAs, underscores the necessity for vigilance in managing febrile episodes in these patients. [7]

This report presents a case of a young woman with FUO, who was ultimately diagnosed with a TOA caused by a nontubercular infection.

A young woman in her 30s, and a resident of North India, presented at the outpatient department with a history of intermittent fever with chills in the previous year, which had increased in frequency and duration over the previous 20 days. For the past 5 months, she had been experiencing heavy bleeding with

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Received: August 10, 2024
Revised: March 19, 2025
Accepted: April 2, 2025

polymenorrhagia. She reported pinpoint reddish lesions all over her body for the previous 2 days as well as bleeding gums**.** She had no significant medical history. She reported no history of sexually transmitted infections and stated that she was in a monogamous relationship, in which she used no contraception. She denied any high-risk sexual behaviors. She was admitted to a hospital where she was diagnosed with severe anemia and thrombocytopenia and was transfused with multiple blood products. However, for further investigation, she was referred to this tertiary care center. She had no history of headache, vomiting, cough, abdominal pain, burning micturition, or vaginal discharge. Physical examination showed that the patient was vitally stable, had pallor, multiple pinpoint erythematous non-blanchable petechiae on the bilateral upper and lower limbs **(Figure 1A)**, and a pan-systolic murmur on cardiac auscultation. The rest of the systemic examination was unremarkable.

Baseline blood investigations showed hemoglobin levels of 7 g/dL (severe anemia), a total leucocyte count of 1,000/mm³ (severe leukopenia), and a platelet count of 1,000/mm³ (severe thrombocytopenia). The liver and kidney functions were within normal limits. She was examined along the lines of febrile pancytopenia. For further evaluation in lines of FUO, contrast-enhanced computed tomography revealed a left tubo-ovarian mass **(Figure 1B)**, most likely an abscess. In addition, peripheral blood smear and bone marrow examination revealed hypocellular marrow suggesting very severe aplastic anemia with an overall cellularity of 20.0%–25.0% **(Figure 1C)**. Empirical broad-spectrum antibiotics were initiated given the presence of severe neutropenia.

Based on the patient history and examination results, FUO in a case of aplastic anemia was suspected for etiological diagnosis. This condition can be caused by various underlying conditions, including infections, bone marrow disorders, autoimmune diseases, malignancies, and certain medications or treatments. For FUO, as a potential diagnostic indication was toward TOA in the imaging, being endemic, the possibility of disseminated tuberculosis was considered first.

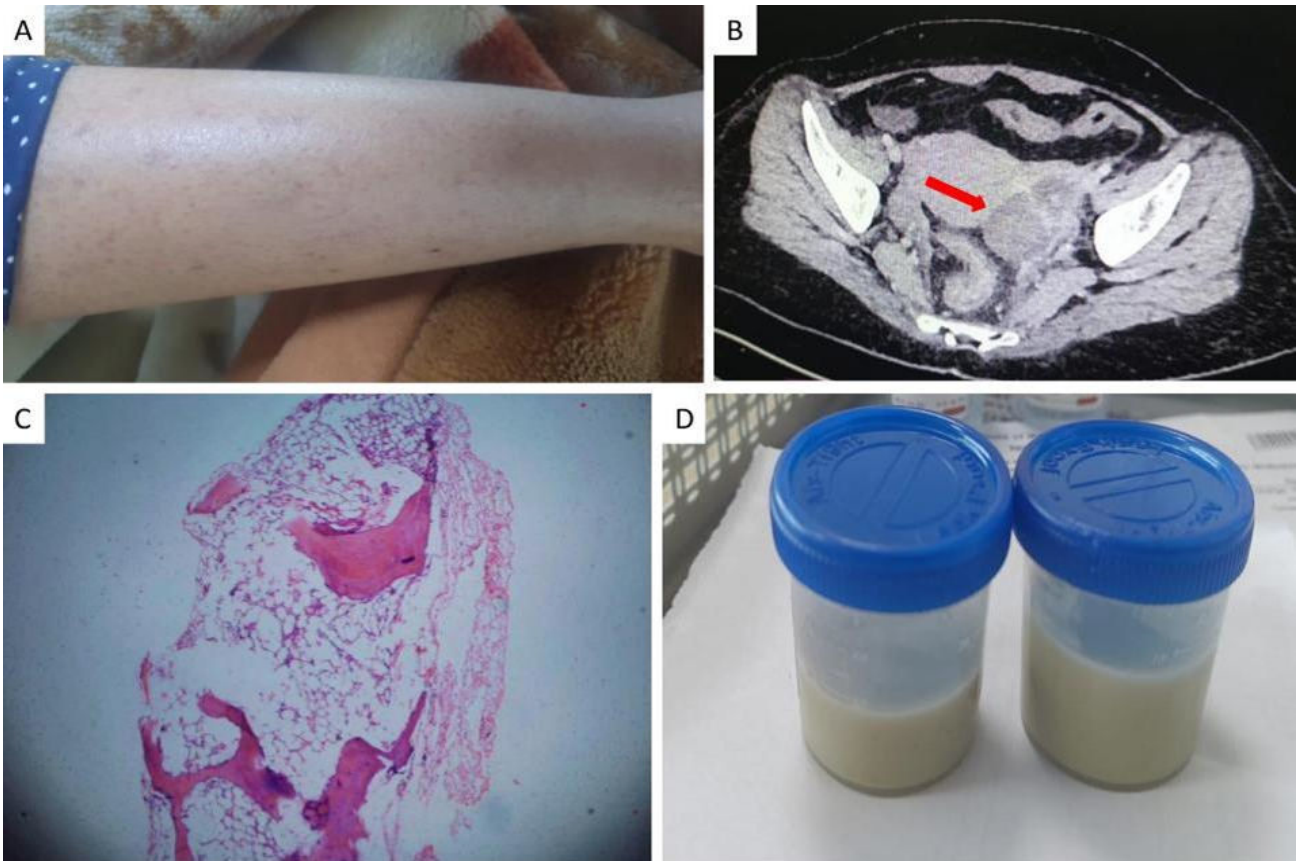

Figure 1. Skin, computed tomography (CT), bone marrow, and abscess drainage findings of the patient. **(A)** Multiple erythematous non blanchable petechiae on left lower limb; **(B)** Tubo-ovarian abscess (arrow), seen in contrast enhanced abdominal CT scan; **(C)** Bone marrow histopathology (40x magnification), suggestive of aplastic anemia with 20.0% - 25.0% cellularity; **(D)** Abscess fluid obtained from the tubo-ovarian abscess after laparoscopic drainage.

Empirical treatment for tuberculosis was initiated as imaging studies revealed TOA, suggestive of tuberculosis as it was associated with prolonged fever and endemicity. Despite treatment, her condition worsened, necessitating a multidisciplinary approach involving hematologists, gynecologists, and infectious disease specialists. The patient underwent laparoscopic drainage of the abscess **(Figure 1D)** with left salpingo-oophorectomy. Microbial cultures from this abscess revealed the presence of *Escherichia coli* (*E. coli)*, and the cartridge-based nucleic acid amplification test for tuberculosis was negative.

After abscess drainage and initiation of culture-guided antibiotic therapy, antitubercular therapy was ceased. Her fever resolved, and specific management for aplastic anemia was initiated in the department of hematology.

Discussion

This case illustrates the diagnostic complexities associated with FUO, particularly in patients with underlying hematologic disorders, such as aplastic anemia.[7] The patient exhibited no typical symptoms or risk factors, including lower abdominal pain, high-risk behavior, or history of intrauterine manipulation. The initial evaluation focused on the assessment of fever and pancytopenia, resulting in a confirmed diagnosis of aplastic anemia. This highlights the necessity for increased clinical suspicion and thorough investigation for similar cases. [8]

Neutropenic fever is defined as a single oral temperature greater than or equal to 101°F (38.3°C) or a temperature greater than or equal to 100.4°F (38°C) for at least an hour, with an absolute neutrophil count of less than 1,500 cells/microliter. [9] In most cases, the infectious etiology cannot be determined and the condition is diagnosed as FUO. [10] The patient's risk of developing an infection-related complication must be determined so that appropriate early management can be initiated. Because patients may have minimal or no symptoms of bacterial infections, detection of neutropenic fever requires close examination of the most commonly infected sites. Suspected neutropenic fever should initially be investigated for infection on sites of previous procedures or catheters, as well as on or in the skin, alimentary tract, oropharynx, gastrointestinal tract, genitourinary, and respiratory system. [11] Although the present patient examined up for the same, no potential diagnostic indications were found.

Extrapulmonary tuberculosis is a common differential diagnosis in patients with prolonged fever, especially in endemic regions, such as India. [12] There are several risk factors for tuberculosis of the female genital tract. Most of these are host factors causing impaired immunity, whereas high exposure to the infection is also considered a risk factor. [13] Patients suffering from immunosuppressive diseases, such as HIV, aplastic anemia, and alcoholism, are at a high risk of genital tuberculosis. [14] Secondary infection through hematogenous spread is common in these patients, resulting in conditions such as TOA. [15] Thus, regarding tuberculosis as a differential diagnosis, our patient was started on empirical antitubercular therapy. However, even after 2 weeks of treatment, she showed clinical deterioration, with persistent fever spikes and worsening hematologic parameters, prompting further investigations into alternative etiologies for her condition. [16]

Aplastic anemia substantially increases the risk of TOA development due to compromised immune function and prolonged illness, rendering patients more susceptible to infections. [17, 18] Delayed diagnosis of aplastic anemia exacerbates this risk, prolonging the duration of illness and increasing susceptibility to secondary infections such as TOAs. [19] In this case, the delayed diagnosis most likely contributed to the progression of the tubo-ovarian infection to abscess formation, highlighting the importance of prompt recognition of underlying hematologic disorders and vigilance for infectious complications in patients with hematologic abnormalities. [20] Early intervention is crucial to prevent severe sequelae and optimize patient outcomes.

The management of TOAs in immunocompromised patients often requires a combination of surgical intervention and broad-spectrum antibiotics tailored to the specific microbial etiology. [21] The presence of *E. coli* in this case highlights the requirement for precise microbial identification to guide appropriate antibiotic therapy or to indirectly de-escalate the regimen. [22] Misdiagnosis or delayed diagnosis can result in significant morbidity and highlights the necessity of a multidisciplinary approach in complex cases involving multiple systems.

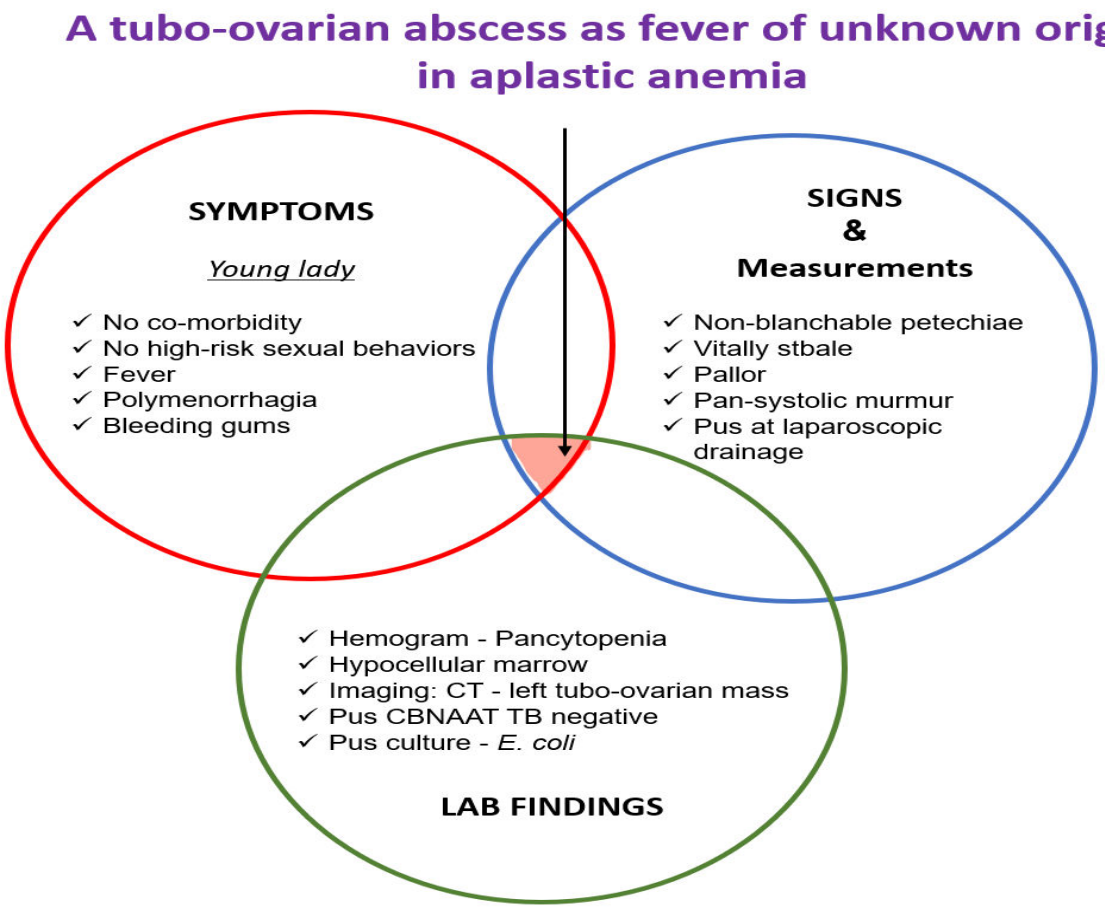

Figure 2. Symptoms, signs, and lab findings of Tubo-ovarian abscess in aplastic anemia.

Furthermore, this case illustrates the importance of considering nontubercular causes of TOAs in endemic regions, especially in patients with hematologic disorders **(Figure 2)**. The initial empirical treatment for tuberculosis, although reasonable given the clinical context, was inappropriate. This highlights the potential pitfalls of empirical therapy without definitive microbiological evidence.

Conclusion

In this case, the diagnosis of a tubo-ovarian abscess in a patient with aplastic anemia and fever of unknown origin required careful diagnosis beyond the typical etiologies, such as tuberculosis, which are common in endemic regions. This case emphasizes the necessity for a high index of suspicion of intraabdominal abscesses, such as TOA, even in the absence of typical risk factors or clinical features. The detection of *E. coli* emphasizes the diversity of pathogens apart from tuberculosis that must be considered in immunocompromised patients. This case advocates for the use of comprehensive diagnostic imaging early in the evaluation process, tailored specifically to the unique challenges presented by atypical infectious agents in patients with underlying hematologic disorders, and de-escalating antimicrobials as early as possible.

Acknowledgements

The authors would like to thank the patients who provided informed consent to have their information published in this report.

Conflict of interest statement

Each of the author has completed and ICMJE disclosure form. The authors declare that they do not have any potential or actual relationship, activity, or conflict of interest related to the content of this article.

Data sharing statement

Data generated or analyzed for the present report are included in this published article. Further details are available from the corresponding author on reasonable request after the deidentification of the patient whose data are included in the report.

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