A Case of Pulmonary Tuberculosis with Underlying Renal Tubular Acidosis, Complicated with Acquired Haemophilia A: A Case Report

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Introduction

Acquired haemophilia A (AHA) is a rare autoimmune bleeding disorder caused by antibodies against factor VIII, leading to sudden bleeding in individuals without prior bleeding history. It commonly presents with spontaneous or post-procedural soft tissue haemorrhage and a prolonged activated partial thromboplastin time. Although often idiopathic, AHA may be associated with infections, autoimmune diseases, malignancy, and medications. Tuberculosis-related immune dysregulation may complicate diagnosis and management.

Case Report

We report a rare case of AHA occurring during treatment for pulmonary tuberculosis, highlighting the diagnostic challenges and need for multidisciplinary care. A 50-year-old Malay woman with poorly controlled diabetes mellitus, dyslipidaemia, urolithiasis, and type 1 renal tubular acidosis was diagnosed with bacteriologically confirmed pulmonary tuberculosis (PTB) and commenced on fixed-dose combination anti-tuberculosis therapy (Akurit-4) in October 2024. After three weeks of treatment, she developed asymptomatic transaminitis (ALT 94 U/L, AST 42.5 IU/L), with negative viral hepatitis screening and normal hepatobiliary ultrasonography. Subsequently, she presented with swelling of the right lower limb and left anterior chest wall. Although initially suspected to be an abscess, incision and drainage revealed an intramuscular haematoma. Further spontaneous swelling following intravenous cannulation raised suspicion of an underlying bleeding disorder. Coagulation studies demonstrated severely reduced factor VIII activity (1%) with a markedly elevated factor VIII inhibitor titre (38.5 Bethesda units), while factor IX activity was preserved (125%). She was also noted to have anaemia (haemoglobin 8.3 g/dL), thrombocytosis (554 × 10^9/L), and hypokalaemia. A diagnosis of AHA, likely secondary to PTB, was established, along with chronic metabolic acidosis related to underlying renal tubular acidosis. She was treated with high-dose oral prednisolone (1 mg/kg/day) and oral cyclophosphamide (100 mg daily) for inhibitor suppression, while anti-tuberculosis therapy was continued. The patient showed good clinical recovery. At the latest review, she remained clinically stable without further bleeding episodes. Prednisolone was gradually tapered, cyclophosphamide was continued, and her PTB demonstrated clinical improvement, allowing transition to the maintenance phase of anti-tuberculosis therapy.

AHA is a rare autoimmune disorder where the body makes antibodies against clotting Factor VIII, leading to a deficiency and causing abnormal bleeding, even in people with no personal or family history of bleeding disorders. It should be suspected when someone develops unexplained acute bleeding and shows a prolonged activated partial thromboplastin time (aPTT) on lab test (1). This case highlighted the diagnostic challenge of identifying AHA in a patient being treated for active infection, who was initially misdiagnosed as TB-related abscesses, but finding an intramuscular hematoma during surgical drainage prompted suspicion for bleeding diathesis. AHA typically presents with spontaneous or post-procedure bleeding into soft tissues rather than joints (2). Although over 50% of AHA cases are idiopathic, they are usually associated with other conditions, including infections, malignancies, postpartum state, and autoimmune diseases like SLE and rheumatoid arthritis. Certain medications are also known triggers (1). In TB-endemic areas, about one-third of patients with active tuberculosis (32%) have elevated autoantibodies such as anticardiolipin IgG and anti-Scl-70. These are likely reactive to chronic infection, not signs of a primary autoimmune disease, as they are unrelated to typical autoimmune symptoms and usually return to normal after successful

TB treatment without immunosuppression (3). Diagnosing AHA requires investigating a prolonged aPTT with mixing studies and measuring factors VIII, IX, XI, XII, von Willebrand factor (VWF) activity, and VWF antigen. The Bethesda assay confirms the diagnosis by detecting and quantifying factor VIII inhibitors in Bethesda Units per millilitre (BU/mL); the higher the value, the stronger the inhibitor (4). Treatment requires potent immunosuppression, commonly high-dose prednisolone combined with cyclophosphamide to eradicate the inhibitor. Using these drugs in a patient with active TB is high risk, so continuing Akurit-4 while starting immunosuppression requires careful risk-benefit balancing. This case underscores the importance of a multidisciplinary approach, in which coordinated decision-making, prompt evaluation, and close monitoring enabled the treatment of both AHA and TB and resulted in a good clinical outcome despite overlapping risks.

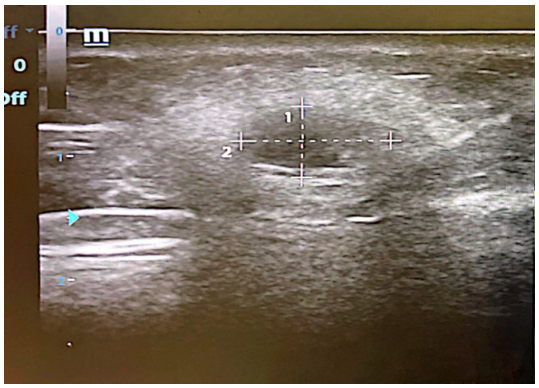

Figure 1. Ultrasonography of the left anterior chest wall, showing an ill-defined hypoechoic subcutaneous collection, measuring 0.6 x 1.0 x 0.9 cm

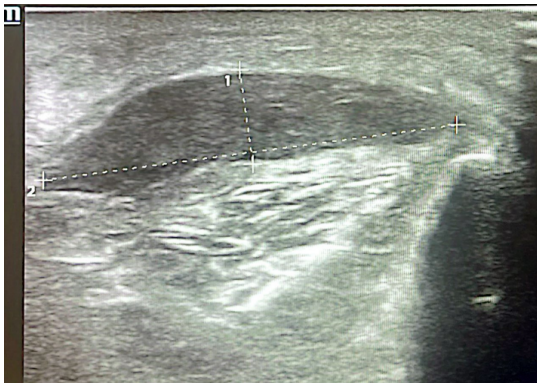

Figure 2. Ultrasonography of the right leg, showing an intramuscular collection 0.7 x 3.2 x 5.0 cm, with minimal subcutaneous tissue oedema

Keywords: Acquired Haemophilia A**,** tuberculosis, autoantibody

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