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An interesting case of concomitant anemia secondary to prolonged PARP inhibitor toxicity, and a hemolytic anemia with non-specific warm autoantibodies

Julien Dereme[1], Konstantinos Asonitis[2], Francesco Grandoni[1], Aikaterini Liapi[2], Apostolos Sarivalasis[2], Gerasimos Tsilimidos[1]

1. Service and Central Laboratory of Haematology, Department of Oncology and Department of Laboratories and Pathology, Lausanne University Hospital (CHUV) and University of Lausanne (UNIL), Lausanne, Switzerland.

2. Medical oncology Unit, Department of Oncology, Lausanne University Hospital (CHUV) and University of Lausanne (UNIL), Lausanne, Switzerland.

CHUV Centre hospitalier universitaire vaudois

UNIL | Université de Lausanne

Background

This case report discusses a 71-year-old female patient with a history of high-grade serous ovarian adenocarcinoma. After receiving neoadjuvant chemotherapy, surgery, and subsequent adjuvant therapy, she was placed on olaparib maintenance. The patient developed severe progressive anemia starting in March 2022, culminating in a hemoglobin level as low as 55 g/L, which presented significant clinical challenges.

Results

Initial investigations ruled out common causes of anemia, including iron and vitamin deficiencies, autoimmune disorders, and infections. Bone marrow biopsies were conducted, revealing a clonal cytotoxic T-LGL population and a DNMT3A mutation, but no signs of myelodysplasia or metastatic infiltration. A mixed anemia profile was observed, characterized by declining reticulocyte counts and the presence of warm autoantibodies (IgG). Despite discontinuing olaparib, the patient remained transfusion-dependent, with no initial improvement in her condition. The identification of warm autoantibodies (initial DAT positive for C3d, detection of IgG autoantibodies only about two months after stopping olaparib), and a progressive drop in reticulocyte counts suggested a complex anemia with both central hyporegenerative components from prolonged PARP inhibitor toxicity and an extravascular hemolytic mechanism. High-dose corticosteroids were administered secondarily to address the hemolytic part of the anemia. Following corticosteroid treatment, the patient experienced rapid hematological improvement, achieving transfusion independence and reduced hemolysis markers.

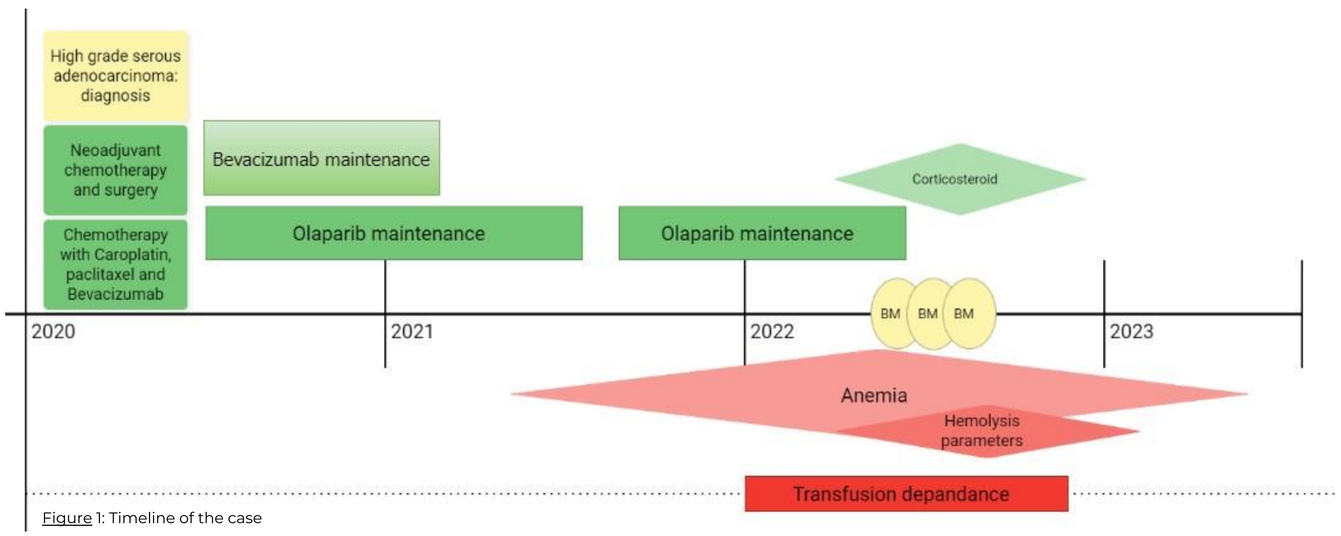

Figure 1: Timeline of the case

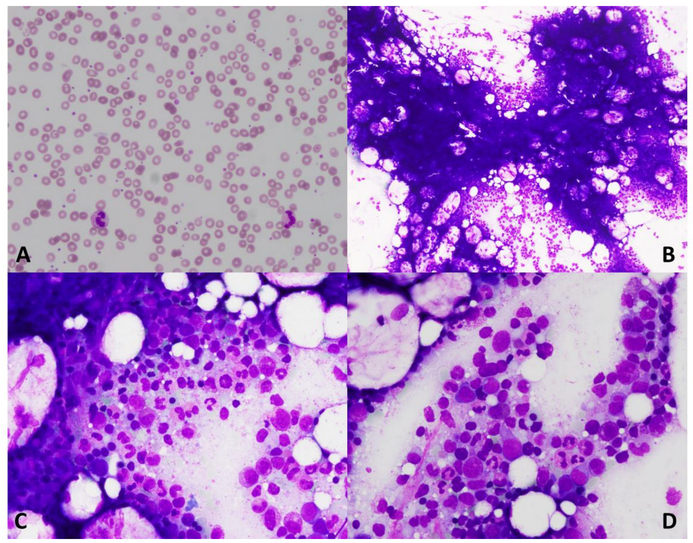

Figure 2: Peripheral blood smear with anispoikylocytosis, 40x magnificiance (A), Bone marrow aspiration image with May-Grunwald-Giemsa showing hypercellularity without dysplastic evidence with 10X magnificiance (B) and 40x magnificiance (C-D)

This case illustrates a severe mixed anemia stemming from prolonged olaparib toxicity combined with warm autoantibody-mediated hemolysis. The prolonged toxic effect of olaparib, in conjunction with confounding factors (the DNMT3A mutation, warm antibodies, LGL clone), underscores the need for careful hematologic monitoring. The patient's response emphasizes the complexity of managing drug-induced hematologic toxicity and the potential for overlapping hematologic conditions.