Derna Academy Journal for Applied Sciences

E-ISSN: 3006-3159

DAJAS

EVANS SYNDROME TRIGGERED BY COVID-19 INFECTION

Rudhuway Abdulqadir Mtawil .[1*]

Physiology Department, Tobruk Medical University, Tobruk

Crossponding Author: G-mail: Radwaemtawil2@gmail.com

Volume: 5 **Issue: 1** **Page Number: 50 - 52**

Keywords:
Evans syndrome, Autoimmune Cytopenia, COVID-19, Autoimmune Hemolytic Anemia, Immune Thrombocytopenia, Hematology

Received: 15\10\2025
Accepted: 11\11\2025
Published: 12\11\2025

https://doi.org/10.71147/49d6hw11

ABSTRACT

Evans syndrome is a rare autoimmune disorder characterized by the coexistence of autoimmune hemolytic anemia (AIHA) and immune thrombocytopenia (ITP). The pathogenesis involves immune dysregulation leading to autoantibody-mediated destruction of multiple blood cell lineages. We report the case of a 40-year-old female presenting with jaundice, pancytopenia, hemodynamic instability, and serologically confirmed Evans syndrome associated with acute COVID-19 infection. The patient showed elevated AST and ALT, complete recovery after corticosteroid therapy, and has remained in remission for two years under regular follow-up.

1. INTRODUCTION

Evans syndrome (ES) is an uncommon autoimmune hematologic disorder defined by the simultaneous or sequential presence of autoimmune hemolytic anemia (AIHA) and immune thrombocytopenia (ITP), with or without neutropenia. *(Jaime-Pérez JC et al ,2018)***.** The disease may occur as a primary (idiopathic) condition or secondary to infections, autoimmune diseases (such as systemic lupus erythematosus), or lymphoproliferative disorders. Since the emergence of SARS-CoV-2, several reports have described autoimmune cytopenias triggered by COVID-19 infection *(Aladily TN et al ,2021).* However, Evans syndrome associated with COVID-19 remains rare and poorly understood. *(Taherifard E, et al.;2022)*

2.Methods

Data were collected retrospectively from the patient's medical records at the Hematology and Oncology Department, Tobruk Medical Center, Libya.

Clinical information, laboratory results, imaging findings, and treatment outcomes were extracted directly from the hematology department and by the attending hematologist.

Laboratory investigations need to diagnosis even syndrome included a complete blood count, reticulocyte count, liver function tests, peripheral blood smear, and direct antiglobulin (Coombs) test. Serological assays were performed to exclude autoimmune and infectious causes (anti-dsDNA, rheumatoid factor, and COVID-19 PCR test).

CASE PRESENTATION

A 40-year-old female, previously healthy and with no history of diabetes or hypertension, presented to the internal medicine department with complaints of headache, nausea, generalized joint pain, and jaundice for several days. On examination, she was pale, febrile (38°C), and diaphoretic, with a blood pressure of 70/40 mmHg and a pulse rate of 110 beats/min. No rash or lymphadenopathy was noted. Cardiovascular and respiratory examinations were normal, and the abdomen was soft with a palpable spleen two fingerbreadths below the left costal margin. Neurological examination was normal.

Laboratory findings revealed: decrease Hemoglobin level ، Hemoglobin was 7 g/dL (Normal 11 -12g /dL), White blood cells 3 × 10^9/L was decrease than normal range (5 _ 10×10) ,and Platelets 15 × 10^9/L lower than normal range (150_450 ×10 , Reticulocyte count increased, ESR elevated then normal level about 112 , Direct Coombs test positive, with increase in direct bilirubin level was 4.5mg/ dl (0.1-1.5 mg /dl) , and elevated liver enzymes (AST 145 U/L and ALT 150 U/L) above the normal level(normal AST and ALT8-33 U/L) .

Peripheral smear showed microcytic hypochromic RBC, nucleated RBCs with schistocytes and acanthocytes, WBC was leukopenia mainly neutropenia, low platelets normal ،blood film was resulting pancytopenia, Bone marrow was hypocellular bone marrow, decrease production megakaryocytes' White blood cells with red blood cells.

Abdominal and pelvic ultrasound revealed splenomegaly (15 cm) more than normal size of spleen (12cm) with normal size of liver, without lymph node enlarge and no free fluid in abdominal. All of these laboratory finding were indicative of hemolysis.

Rheumatoid factor was negative, Anti–Nuclear antibody (ANA) was negative, Chest x- rays was bilateral infiltration of lung, COVID-19 PCR positive by nasopharyngeal swab, based on these findings, the diagnosis of Evans syndrome associated with acute COVID-19 infection was established.

The patient was managed with intravenous fluids, intra venous antibiotic and corticosteroid therapy for 14 days, with supportive management as packed RBCs transfusion, antipyretic therapy. She showed rapid clinical and hematologic improvement after 14 days of mangment , with normalization of blood counts and resolution of splenomegaly after 3 months. No complications were observed. She continued regular follow-up every three months for two years after diagnosis, last follow up before one month back, and remained in complete remission throughout the observation

3. ETHIC APPROVAL

In order for this case report to be considered ethical, I obtained ethical approval for a scientific case report from the patient, on whom the case report is being conducted. The patient's permission was taken and it was said that treatment and research had begun at Hematology department, Tobruk Medical Center November 2021

4. DISCUSSION

This case illustrates a rare instance of Evans syndrome occurring concurrently with COVID-19 infection. SARS-CoV-2 has been increasingly recognized as a trigger for immune dysregulation, including autoimmune cytopenias such as ITP and AIHA. The proposed mechanisms include molecular mimicry, hyperactivation of B-cells, and cytokine-mediated immune dysregulation.

The coexistence of AIHA, thrombocytopenia, and neutropenia, together with a positive direct Coombs test and exclusion of other autoimmune disorders (negative RF and anti–dsDNA), strongly supported the diagnosis of Evans syndrome.

The patient's excellent response to corticosteroids highlights the role of early immunosuppressive therapy in COVID-associated autoimmune cytopenias. *(Godeau B, et al 2002).*

Previous studies have reported that up to one-third of Evans syndrome cases can relapse; however, our patient remained in long-term remission for two years, suggesting a transient immune activation secondary to viral infection rather than a chronic autoimmune process. *(,Michel M, et al 2009). .(Jaime-Pérez JC et al ,2018).*

6. CONCLUSION

Evans syndrome should be considered in patients presenting with multi-lineage cytopenia, jaundice, and a positive Coombs test, particularly during or following viral infections such as COVID-19. Early recognition and corticosteroid therapy can result in rapid recovery and prevent life-threatening complications. This case adds to the growing evidence linking SARS-CoV-2 infection with immune-mediated hematologic disorders.

7. REFERENCES

Aladily TN, Salama A. Autoimmune cytopenias in the context of COVID-19: mechanisms and management. Front Immunol. 2021;12:758514.

Audia S, Mahévas M, Ouandji S, et al. Evans' syndrome: from pathophysiology to treatment. Autoimmun Rev. 2022;21(1):103039.

.Evans RS, Takahashi K, Duane RT, Payne R, Liu C. Primary thrombocytopenic purpura and acquired hemolytic anemia: evidence for a common etiology. Arch Intern Med. 1951;87(1):48–65.

Godeau B, Caulier MT, Decuypere L, et al. Evans syndrome in adults: clinical presentation and outcome. QJM. 2002;95(9):597–607.

Khelif A, Beyaert R, Michel M. Recent advances in the understanding and management of Evans syndrome. Br J Haematol. 2023;203(4):457–470.

Michel M, Chanet V, Dechartres A, et al. The spectrum of Evans syndrome in adults: new insight into the disease based on the analysis of 68 cases. Blood. 2009;114(15):3167–3172.

Taherifard E, et al. COVID-19 associated Evans syndrome: A case report and review of the literature. Clin Case Rep. 2022;10(7):e06111.

Jaime-Pérez JC, Aguilar-Calderón PE, Salazar-Cavazos L, Gómez-Almaguer D. Evans syndrome: clinical perspectives, biological insights and treatment modalities. J Blood Med. 2018;9:171–184.