A clinical case of Fisher-Evans syndrome

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ABSTRACT

SFE is a fairly rare disease. In cases where SFE presents with isolated ITP, and the second cytopenia develops after several months or even years, it is impossible to establish a diagnosis immediately. The described patient developed AIGA and ITP at the same time. It is also necessary to remember the importance of determining the Coombs test during the initial diagnosis of ITP and further during patient follow–up, and in patients with ITP and AIG, the basal level of serum immunoglobulins to detect PID. In all cases of SFE, it is necessary to conduct an additional examination using molecular genetic methods in order to find a possible cause of the development of secondary SFE. After the therapy, there is a positive trend in clinical signs and laboratory test results. Taking into account that the prognosis for immune cytopenia in the framework of SFE is quite unfavorable, long-term combined IST is usually recommended.

Thus, the above clinical case demonstrates the positive effect of the treatment. Taking into account the literature data on the recurrence of the disease, dynamic monitoring of the patient's condition and laboratory tests is necessary.

Keywords: Fisher–Evans syndrome, anemia, thrombocytopenic purpura, treatment.

Introduction: Fisher–Evans syndrome (SFE) is a rare autoimmune disease characterized by a combination of immune thrombocytopenia (ITP) and Coombs-positive autoimmune hemolytic anemia (AIGA), which can develop simultaneously or sequentially, and in some cases are combined with immune neutropenia (14.7-55%) associated with the formation of antineutrophil antibodies [1].

According to several retrospective studies, about half of patients develop ITP and AIG simultaneously, in other cases it is a sequential development of cytopenia (6-14%), with about 30% of cases developing ITP first, and 16-25% of cases developing AIG. On average, the period between cytopenias is 4.2 years, and there are cases when more than 16 years passed between their development [1-3].

SFE can develop at any age. Thus, in 1950-1958, out of 399 adult patients with AIG and 367 patients with ITP, only 6 of them had Fisher–Evans syndrome [1]. According to current data, SFE is diagnosed in 0.8–3.7% of all patients with newly diagnosed ITP or AIGA [2].

According to the results of a retrospective analysis of 68 adult patients in France, the average age at the time of diagnosis of SFE is 55 ± 33 years old. Women are more likely to get sick (60%) [1]. According to data from Denmark in 2016, the annual incidence was 1.8 per 1,000,000 people, and the prevalence was 21.3 per 1,000,000 people [5].

In 82% of patients, SFE was associated with other systemic conditions: positive titer of antinuclear antibodies, changes in serum immunoglobulin levels, eczema, insulin–dependent diabetes mellitus, Guillain-Barre syndrome, migraine, autoimmune hepatitis, or nephritis [6, 7].

Idiopathic SFE develops as a result of immune dysregulation of the body.

The leading role in the pathogenesis of SFE belongs to B lymphocytes and plasma cells that produce autoantibodies directed against platelets and erythrocytes [1, 4, 6]. The mechanism of development of thrombocytopenia is classically associated with the production of antiplatelet antibodies (class G immunoglobulins) directed against platelet membrane antigens – glycoproteins IIb–IIIa and glycoproteins Ib or a combination thereof. Antiplatelet antibodies opsonize platelets, which leads to their destruction by macrophages, mainly in the spleen. In most cases, immune destruction of erythrocytes develops due to the fixation of autoreactive class G immunoglobulins active at 37 °C (thermal antibodies) on the surface of the erythrocyte membrane, which leads to extravascular hemolysis. Intravascular hemolysis with the participation of class A and M immunoglobulins, which destroy red blood cells indirectly through a cascade of complement components, develops less frequently at a lower temperature (cold antibodies). In some cases, both of these mechanisms can be combined. There are some specific aspects of pathophysiology in cases of association of SFE with other autoimmune, lymphoproliferative, and myeloproliferative or immunodeficiency conditions (secondary SFE) [4].

Clinical manifestations of SFE include the usual symptoms of thrombocytopenia (hemorrhagic rash, bruising, bleeding) and hemolytic anemia (weakness, pallor, drowsiness, jaundice). The symptoms of the disease may develop acutely or gradually.

In some patients, enlarged lymph nodes, liver, and/or spleen are described, which may be permanent or intermittent and manifest themselves during periods of exacerbation of the disease.

Patients with neutropenia may develop mucositis, recurrent upper respiratory tract infections, diarrhea, and more severe bacterial infections, including sepsis [2, 3, 6-9].

From the moment of the first description to the present, SFE remains a diagnosis of exclusion. Before establishing primary SFE, it is necessary to exclude other conditions against which secondary SFE could develop, and diseases in which SFE may be their first manifestation [10-12]. For the diagnosis of SFE, it may be useful to assess family predisposition to autoimmune diseases, malignancies, and associations with medications and vaccinations that could trigger the disease.

During laboratory examination of patients, anemia, thrombocytopenia ± neutropenia is detected in a general blood test. Morphological examination of peripheral blood can describe abnormal red blood cell forms characteristic of AIG, and exclude other possible diseases (malignant, microangiopathic hemolytic anemia, congenital hemolytic and thrombocytopenic conditions). To diagnose anemia, the count of the number of reticulocytes and schizocytes should be included in the analysis. Red blood cell agglutination by the type of "coin columns" and false macrocytosis as a result of such agglutination can be detected. In the biochemical analysis of blood serum, an increase in LDH, unconjugated bilirubin and a decrease in haptoglobin are determined. When conducting direct and indirect Coombs tests, the reaction can be positive, weakly positive, or even negative (by 10%).

When determining antiplatelet and antineutrophil antibodies, a negative result also does not exclude the diagnosis of SFE. The study of serum immunoglobulins and their subclasses is recommended for all patients not only to exclude OVIN and IgA deficiency, in which cytopenia may develop, but also to determine their basal level before starting immunosuppressive therapy. To confirm/exclude other autoimmune conditions, in particular SLE, antinuclear antibodies, double-stranded DNA, and rheumatoid factor are being studied [13-16].

Bone marrow puncture is performed to exclude infiltrative and myelodysplastic processes, the first manifestation of which may be cytopenia. The picture of the bone marrow in SFE is non-specific, and its normal or increased cellularity may be detected [4].

SFE therapy still remains a difficult task. The disease is characterized by a chronic course with frequent exacerbations of AIG and/or ITP.

According to the literature, ITP relapses occur more frequently (on average, 3-5 episodes of ITP per 2-3 episodes of AIG) and worse than AIGA, they are controllable [1].

Accordingly, therapy aimed at treating severe ITP is required more often. Only isolated cases of spontaneous remissions have been described among patients with SFE. Most patients require long-term combined treatment with multiple adverse events to maintain a hematological response. At the same time, the responses to therapy can be highly variable even in one patient. With the consistent development of ITP and AIG, therapy aimed at initial cytopenia, including splenectomy, does not prevent the development of a second cytopenia, which can occur several months or even years later [1].

Due to the small number of patients, randomized trials of various therapeutic approaches for SFE have not been conducted, and there is no recommended therapy algorithm for adults. Usually, the therapy of patients with SFE is based on the individual experience of the medical institution in managing patients with ITP and AIG [1-3]. By analogy with ITP and AIGA therapy, the drugs used to treat SFE are divided into first- and second-line drugs. The goal of first–line therapy is to achieve rapid control of cytopenia and prevent the development of life-threatening bleeding and anemia. Unlike ITP and AIGA, when an initial response is achieved, dose reduction of first-line drugs is carried out much more slowly. Second-line therapy is required in patients with resistance to first-line therapy, with recurrent SFE, in order to maintain long-term remission and minimize the doses of glucocorticosteroids (GCS) recommended in the first line.

The duration of maintenance therapy is usually at least 2 years. In cases of continued remission, discontinuation of therapy is carried out gradually, no faster than 6 months. Cyclophosphamide, alemtuzumab, bortezomib, vincristine, danazol, hematopoietic stem cell transplantation (HSCT), etc. are used in patients with severe disease who have not responded to first- and second-line therapy for several years [1, 4, 17-19].

In general, there is no specific and non-toxic therapy, and all the proposed therapy options are palliative rather than curative.

When analyzing the outcomes of SFE in 68 adult patients who were observed for an average of 4.8 years, 32% of patients remained in complete or partial remission without therapy at the time of analysis; 56% continued to receive therapy (in the vast majority of cases, combined therapy) and had complete or partial remission; 12% continued to receive therapy without remission of the disease; 24% of patients died; the most common causes of death were septic shock and bleeding [2].

In general, hemorrhage mortality in SFE is much higher than in isolated ITP (1-3% in adults) [3].

RESEARCH MATERIALS AND METHODS

The results of a clinical examination of a 50-year-old patient with Fisher-Evans syndrome, who was treated and examined in the hematology department of the Tashkent City Clinical Hospital No. 1, were analyzed. Clinical research methods included the collection of complaints, the identification of anamnesis of the disease, and clinical signs. Laboratory research methods included a general blood test on a hematology analyzer, a biochemical blood test, a general urine test, a myelogram study, and a direct and indirect Coombs test.

RESULTS AND DISCUSSION

A 50-year-old patient went to the hematologist of the consultative polyclinic with complaints of general weakness, fatigue, dizziness, shortness of breath and palpitations when walking, jaundice of the sclera.

From anamnesis: He does not associate his condition with anything, the disease has developed gradually over the past 2-3 months with the above complaints.

Pregnancies -3, deliveries-3. Menopause. Coronary artery stenting was performed in 2022. He has been taking cardiomagnil 75 mg and clopidogrel 75 mg for several years. He suffers from cholelithiasis. Chronic stone cholecystitis.

Laboratory research data.

A general blood test. 24.07.2025. HGB- 72 g/L, RBC- 2.8 x 10^{12}/L, HCT- 24.1%, MCV- 86 f/L, MCH- 23p/g, MCHC- 274 g/L, RDV-CD- 55.4%, RDV-CV- 17%, WBC - 8.96 x 109/l, PLT - 81.0x 10^9/L, Gran%- 30%, eosinophil-1%, lymphocytes- 51%, monocytes- 18%, ESR- 33 mm/hr.

Upon repeated examination in the general blood test dated 07.26.2025 Hemoglobin levels decreased to 59 g/l, erythrocytes- 2.21 x 10^{12}/L, hematocrit- 20.7%, MCV- 93.7 f/l, MCH- 26.7 p/g, MCHC- 285.0 g/l, RDV-CD- 55.4%, RDV-CV- 18.7%, WBC - 4,31x10^9/l, PLT -106,4x10^9/L, Myelogram.

Gran%- 32%, eosinophils-2%, basophils- 0%, LYM%- 47%, monocytes-8%, ESR- 33 mm/hour. Pronounced anisocytosis.

In the biochemical analysis of blood: Serum iron- 5.1 mmol/l, ferritin-1499 ng/ml, vitamin B_{12}-1952 pg/ml, total bilirubin- 30.0 mmol/l, direct- 12.8 mmol/l, indirect- 17.2 mmol/L.

The direct Coombs test is positive 1:32, the indirect Coombs test is positive 1:2.

Taking into account the changes in blood tests, a diagnostic sternal puncture was performed. The results are shown below.

Performance	**Indicators**
Blasts	0,2
Promyelocytes	0,8
Myelocytes	19,8
Metamyelocytes	11,4
Rod-shaped	20,6
Segment-core	22,8
Eosinophils	1,2
Lymphocytes	5,4
Monocytes	0,2
Plasma cells	0,4
Pronormocytes	0,6
Basophilic normocytes	1,4
Polychromatophilic normocytes	12,0
Oxyphilic normocytes	3,2
Leukoerythroblastic ratio	4,78:1
Erythrocaryocyte maturation index	0,88
Neutrophil maturation index	0,74

The presented bone marrow preparation is hypercellular with the presence of adipose tissue. The erythroid sprout is normoblastic - 17.2%. Morphologically, there are dysplastic signs (no more than 10%) in the form of intercellular joints and internuclear bridges. The myeloid granulocyte range is expanded- 75.4%, with delayed maturation at the myelocyte stage-19.8%. Cytoplasmic degranulation and toxogenic granularity of neutrophils occur. Megakaryocytes in sufficient quantity (about 11-12 pieces).

Based on the above, a diagnosis was made: Acquired hemolytic anemia.

Recommended: inpatient treatment with a hematologist after presentation of an order (electronic).

The patient was admitted to the hematology department.

Diagnosis upon admission: Other autoimmune hemolytic anemia.

Competing diagnosis: Immune thrombocytopenic purpura. Evans-Fisher syndrome (autoimmune hemolytic anemia and thrombocytopenia).

Concomitant: Condition after coronary artery stenting (2022). Cholelithiasis. Chronic stone cholecystitis.

Complaints upon admission: dizziness when moving, darkening in front of the eyes, palpitations, puffiness around the eyes.

Objectively: The condition is serious. Conscious. The position is passive. The questions are answered to the point. The skin and visible

mucous membranes are pale, slightly icteric. There are no hemorrhages. Peripheral lymph nodes are not palpable. Subcutaneous fat is moderately expressed. There is vesicular respiration in the lungs. The heart tones are muffled, rhythmic, and a functional systolic murmur is heard at the top of the heart. The pulse rate is 94 beats per minute. Blood pressure is 100/60 mmHg. The tongue is moist and clean. On palpation, the abdomen is soft and painless. The liver and spleen are not palpable. Stools and urination are regular. There is no swelling on the lower extremities.

Laboratory blood test data. 30.07 2025

Blood type A (11) Rh+.

General urinalysis. Protein in urine abc. The relative density is -1019. The color of urine is light. The transparency of urine is transparent. The amount of urine is 60 ml. The epithelium is flat -3-4 in the field of view. White blood cells 2-4 in the field of vision. Abc red blood cells.

Coagulogram. The ethanol test is negative. The recalcification time is 62 seconds. Fibrinogen A-5.4 g/L. INR-0.9. Prothrombin time is 16 seconds. Hematocrit is 18 g/l.

Biochemical analysis. Glucose-5.9 mmol/L. AST-11 U/L. Total bilirubin- 24.9 mmol/l, indirect-16.5 mmol/l, direct-8.4 mmol/L. Total protein-63.4 g/l.

A general blood test. HGB-54 g/L, RBC-2.02 x 10^{12}/L, HCT-18.9%, MCV-93.4 f/L, MCH-26.7 p/g, MCHC-286 g/L, RDV-CD-54.4%, RDV-CV-16.8%, WBC- 5.6 x 10^9/l, PLT-46x10^9/L, Gran%-30%, LYM%-60%, Monocyte-10%, ESR- 32 mm/hr.

Against the background of pulse therapy with prednisone 500 mg per day for 3 days intravenously, there is a gradual positive trend in the overall blood count.

08/01/2025 HGB- 77 g/L, RWC- 3.15 x 10^{12}/L, HCT- 28.4%, MCV- 90.3 f/L, MCH- 24.3p/g, MCHC- 269 g/L, RDV-CD- 64.9%, RDV-CV-17.5%, WBC - 31.5 x 10^9/l, PLT-340x10^9/L, Gran%-87.5%, LYM%- 12.5%, ESR- 33 mm/hour.

General urinalysis. There are traces of protein in the urine. The relative density is 1020. The color of urine is light. The transparency of urine is transparent. The amount of urine is 200 ml. The epithelium is flat-3-4 in the field of view. White blood cells 4-6 in the field of vision. Red blood cells unchanged abc. Uric acid salts ++. Bacteria+.

08/02/2025 HGB-81 g/L, RBC- 3.04 x 10^{12}/L, HCT- 27.9%, MCV- 91.7 f/L, MCH- 26.6p/g, MCHC- 290 g/l, RDV-CD- 51.3%, RDV-CV-16.2%, WBC- 11,1x10^9/l, PLT -78x10^9/L, Gran%- 53%, LYM%- 38.7%, ESR- 33 mm/hour, granulocytes 4.3 10^{*9}/1. The ESR is 6 mm/h.

08/05/2025. Hemoglobin 90 g/L, RBC – 3.38 10^{12}/L, HCT – 39.8%, MCV – 91.1 fl, MSN – 26.6 pg, MCHC - 287 g/L, WBC – 11.51 x 10^9/L, PLT - 148, Gran% - 76.0%, LYM% – 17.1 %, Medium–sized cells - 6.9 %, ESR - 8 mm/h.

08/05/2025. General urinalysis.

The epithelium is flat - 1-2 in n /a. Urine protein-traces, urine quantity - 124 ml. The color of urine is light yellow. The relative density is - 1019 mmol/L. Urine leukocytes - 2-3 per day. Red blood cells are abs. Hyaline - 0 in the field of view.

The treatment performed in the department. Prednisone 500 mg No. 3, Potassium chloride 4% 10 ml, Levoflaxacin 500 mg 100 ml No. 3, Pantoprazole 40 mg per day.

Upon discharge, the patient's condition improved significantly on the background of the therapy.

Discharge recommendations. Methylprednizalon (Slideron) 16 mg should be reduced according to the scheme. Omez 20 mg 2 times a day for 10 days. Folic 5 mg once a day for 1 month. Macamin 1500 once a day under the tongue for 1 month. Asparkam 1 tablet 3 times a day for 10 days.

A month after the patient's discharge from the hospital, all the indicators of the general blood test recovered to normal values.

09/08/2025. Hemoglobin 122 g/L, RBC – 4.37 10^{12}/L, HCT - 31.3%, MCV - 92.5 fl, MCH – 27.9 pg, MCHC – 307.0 g/L, WBC – 11,51x109/L, PLT -256, NEUT% - 76.0%, LYM% – 17.1 %, medium–sized cells - 6.9%, ESR - 8 mm/h.

SFE is a fairly rare disease. In cases where SFE presents with isolated ITP, and the second cytopenia develops after several months or even years, it is impossible to establish a diagnosis immediately. The described patient developed AIGA and ITP at the same time. It is also necessary to remember the importance of

determining the Coombs test during the initial diagnosis of ITP and further during patient follow–up, and in patients with ITP and AIG, the basal level of serum immunoglobulins to detect PID. In all cases of SFE, it is necessary to conduct an additional examination using molecular genetic methods in order to find a possible cause of the development of secondary SFE.

After the therapy, there is a positive trend in clinical signs and laboratory test results. Taking into account that the prognosis for immune cytopenia in the framework of SFE is quite unfavorable, long-term combined IST is usually recommended.

Thus, the above clinical case demonstrates the positive effect of the treatment. Taking into account the literature data on the recurrence of the disease, dynamic monitoring of the patient's condition and laboratory tests is necessary.

CONCLUSION

Fisher–Evans syndrome is a chronic, recurrent disease, a condition caused by profound dysregulation of the immune system. SFE is often associated with other autoimmune conditions or is a manifestation of other autoimmune, lymphoproliferative diseases or primary immunodeficiency. In all cases, it is necessary to conduct a thorough diagnostic search in order to identify secondary forms of SFE and select the appropriate therapy. ITP may be the first manifestation of the disease and develops in relapses of SFE, it is necessary to keep in mind the possibility of developing a second cytopenia over a long period (possibly for life), especially in patients with severe, therapy-resistant or recurrent ITP and in association with other autoimmune conditions and diseases.

In most patients, the course of SFE is severe and recurrent. Thrombocytopenia in the framework of SFE is significantly more aggressive and more often associated with the development of life-threatening hemorrhagic complications than in primary ITP. Cytopenias in SFE may be resistant to IST and splenectomy. Long-term immunosuppressive therapy of SFE is associated with significant toxicity and is one of the risk factors for the development of life-threatening infections. In general, SFE is characterized by significant mortality from infectious and hemorrhagic complications. It is necessary to further study the etiology and pathogenesis of SFE, monitor patients, identify possible predictors of the severity of the disease and develop an optimal therapy strategy.

Ethics approval and consent to participate- The patient has given written informed consent to participate in the study.

Consent to publication is valid, and recognition by the organization is not required. The author agrees to open the publication.

Availability of data and materials- Existence of competitive interests -Lack of funding- No financial support was provided for this work

Conflict of interest-The author declares that there is no conflict of interest.

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