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Spur-cell anemia in patient with acute-on-chronic liver failure: clinical case

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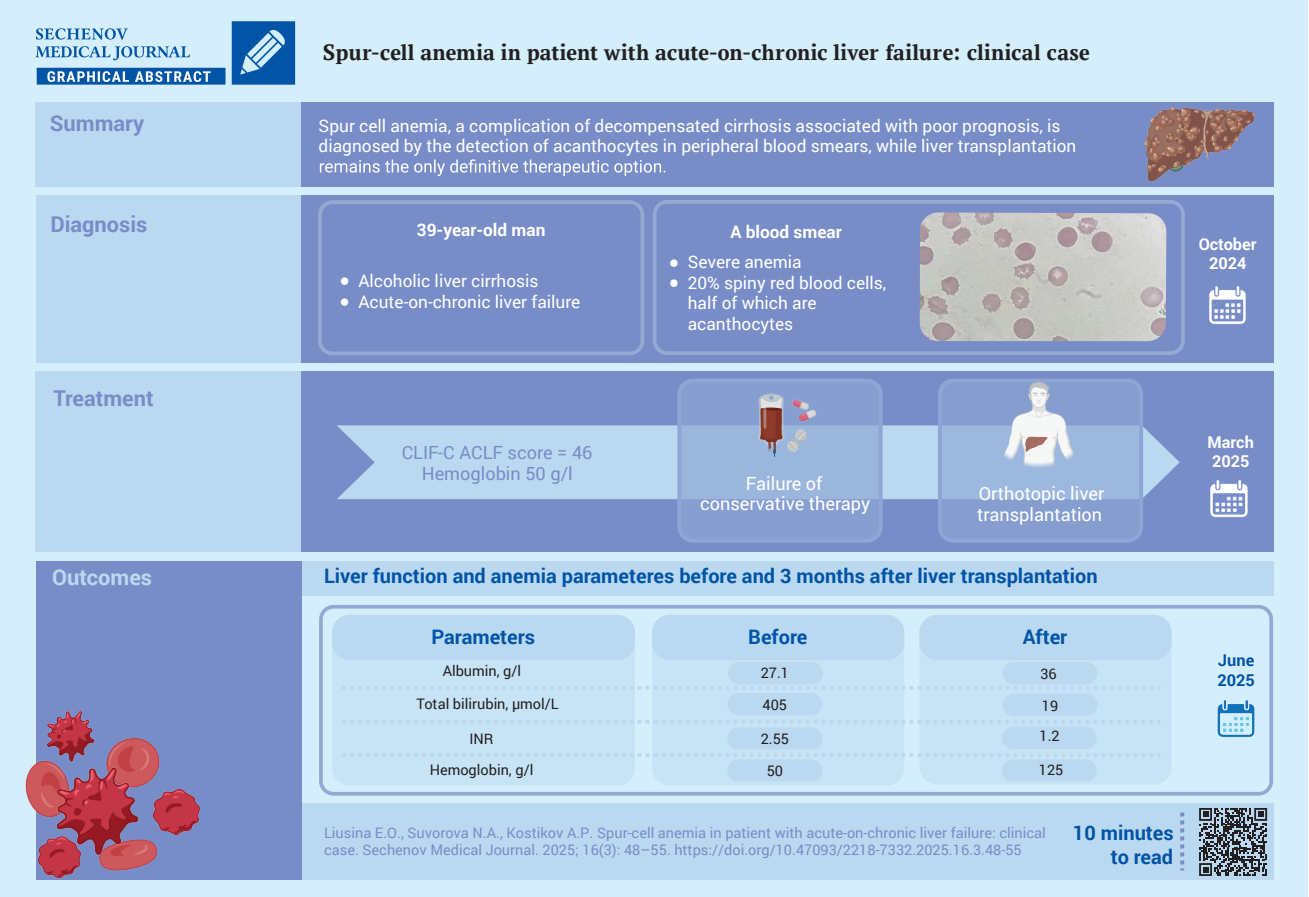

Abstract

One of the rare forms of anemia in patients with liver cirrhosis (LC) is acanthocytosis (spur cell anemia) – a non-immune hemolytic anemia caused by alterations in the lipid composition of the red blood cells membrane because of severe liver failure.

Case report. A 37-year-old patient with decompensated alcoholic LC (Child-Pugh class C, the MELD-Na (Model for End-Stage Liver Disease – Na) score was 34 points) presented with severe weakness and dyspnea. Acute-on-chronic liver failure was diagnosed: CLIF-C ACLF (Chronic Liver Failure Consortium Acute-on-Chronic Liver Failure Score) score was 46. Severe macrocytic anemia with reticulocytosis was detected: hemoglobin – 50 g/L, red blood cells – 1.26×10^{12}/L, reticulocytes – 77.9 ‰. Other causes of anemia, such as blood loss, iron deficiency, vitamin B_{12} and folate deficiencies were excluded. The Coombs test was negative, and bone marrow examination ruled out myelodysplasia. Blood smear analysis revealed that 20% of red blood cells had the shape of spur cells, with approximately half of them being acanthocytes. Orthotopic liver transplantation was performed. Follow-up examination after three months showed normalization of liver function tests and absence of anemia and acanthocytosis.
Discussion. This case report highlights the need for blood smear examination to detect acanthocytes – a rare but prognostically unfavorable cause of anemia in patients with LC. Liver transplantation remains the only effective treatment option.

Keywords: spur-cell anemia; hemolysis; cirrhosis; alcohol; acanthocytes; MELD-Na; CLIF-C ACLF; liver transplantation
Рубрики MeSH:
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Акантоцитоз – редкая причина анемии у пациента с острым повреждением печени на фоне хронического: клинический случай

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Аннотация

Одной из редких форм анемии у пациентов с циррозом печени (ЦП) является акантоцитоз (шпороклеточная анемия) – неиммунная гемолитическая анемия, обусловленная изменением липидного состава мембраны эритроцитов в результате тяжелой печеночной недостаточности.

Описание случая. У пациента 37 лет с декомпенсированным алкогольным ЦП (класс С по шкале Child – Pugh, индекс MELD-Na (Model for End-Stage Liver Disease – Na, модель для оценки терминальной стадии заболевания печени с учетом натрия) составил 34 балла) появилась выраженная слабость и одышка. Диагностировано острое повреждение печени на фоне хронического: показатель по шкале CLIF-C ACLF (Chronic Liver Failure Consortium Acute-on-Chronic Liver Failure Score, шкала оценки острого повреждения печени на фоне хронического Консорциума по изучению хронической печеночной недостаточности) составила 46 баллов. Выявлена тяжелая макроцитарная анемия с ретикулоцитозом: гемоглобин – 50 г/л, эритроциты – $1{,}26\times10^{12}$/л, ретикулоциты – 77,9 ‰. Исключена кровопотеря, дефицит железа, витамина B_{12} и фолатов, проба Кумбса отрицательная, исследование костного мозга исключило миелодисплазию. В мазке крови 20% эритроцитов имели форму шпоровидных клеток, из них около половины – акантоциты. Выполнена ортотопическая трансплантация печени. Контрольное обследование через три месяца показало нормализацию печеночных функциональных тестов и отсутствие анемии и акантоцитоза.

Обсуждение. Представленный случай демонстрирует необходимость изучения мазка крови для выявления акантоцитов как редкой, но прогностически неблагоприятной причины анемии у пациентов с ЦП. Единственным эффективным методом лечения является трансплантация печени.

Ключевые слова: шпороклеточная анемия; гемолиз; цирроз; алкоголь; акантоциты; MELD-Na; CLIF-C ACLF; трансплантация печени

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АНЕМИЯ ГЕМОЛИТИЧЕСКАЯ – ПАТОЛОГИЯ
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Abbreviations:
ACLF – acute-on-chronic liver failure
LC – liver cirrhosis
SCA – spur cell anemia

HIGHLIGHTS

Acanthocytosis (spur cell anemia) in a patient with liver cirrhosis is a rarely diagnosed but highly unfavorable prognostic form of anemia, particularly in the setting of acute-on-chronic liver failure.

The diagnosis of acanthocytosis is based on microscopic examination of a peripheral blood smear.

Liver transplantation is the only effective treatment option for spur cell anemia in patients with liver cirrhosis.

Anemia is a common complication of liver cirrhosis (LC), occurring in 66–75% of patients, and is associated with a higher rate of cirrhosis decompensation, hospital admissions, development of acute-on-chronic liver failure (ACLF), reduced survival, and increased mortality [1, 2]. The most common etiological factors of anemia in LC patients are iron, folic acid, and vitamin B_{12} deficiencies, as well as chronic inflammation.

One of the least studied and rarely diagnosed forms of anemia in patients with LC is spur cell anemia (SCA) — a type of non-immune hemolytic anemia that develops because of abnormalities in the lipid composition of red blood cells membranes. In particular, there is an increase in the cholesterol-to-phospholipid and protein ratio, which in turn increases membrane rigidity, reduces its fluidity, and leads to mechanical damage of the cells. In addition, fatty acid metabolism is disrupted: their incorporation into phosphatidylethanolamine decreases, while incorporation into acylcarnitine increases. Such disturbances are characteristic of all forms of SCA, as they impair the restoration of membrane lipids and lead to the formation of characteristic protrusions (spurs) on the red blood cells surface, which can be observed under blood smear microscopy [3].

Spur-shaped red blood cells (acanthocytes) undergo hemolysis and are eliminated by the mononuclear phagocyte system. The standard diagnostic criterion for SCA is the detection of more than 5% acanthocytes in a blood smear with a hemoglobin level below 100 g/L and the exclusion of other causes of anemia [4].

The development of SCA in patients with LC is associated with an unfavorable prognosis, frequent episodes of decompensation, and higher mortality rates [5–7].

Currently there is no effective treatment for SCA; various pharmacological agents with different mechanisms of action and plasmapheresis have been described, although it is underlined that only liver transplantation leads to resolution of the anemia [8].

The aim of this case report is to demonstrate the development of secondary acanthocytosis in a patient with severe hepatic dysfunction and to increase clinicians' awareness of this rare etiology of anemia.

CASE REPORT

A 37-year-old male patient, technical specialist with a history of alcohol abuse over the past 3–5 years first noted scleral icterus in November 2023. Laboratory tests revealed elevated levels of bilirubin, alanine aminotransferase, aspartate aminotransferase, and gamma-glutamyl transpeptidase. Viral and autoimmune etiologies of liver disease, Wilson's disease, and hereditary haemochromatosis were excluded. The patient had no metabolic risk factors, and ultrasonography of the liver showed no signs of steatosis, which allowed the exclusion of metabolic dysfunction-associated steatotic liver disease. Upon alcohol abstinence, jaundice regressed.

In April 2024, the patient developed recurrent jaundice. Laboratory tests revealed hyperbilirubinemia, elevated alanine aminotransferase, aspartate aminotransferase, and gamma-glutamyl transpeptidase, hypoalbuminemia, moderate thrombocytopenia, and a slight decrease in hemoglobin to 121 g/L (Table). Imaging investigations showed signs of portal hypertension (grade 2 esophageal varices, minimal ascites, splenomegaly). A diagnosis of severe alcoholic hepatitis (Maddrey's discriminant function was 41) and LC was established. The patient responded to treatment with prednisolone 40 mg/day: on the day 7th, the Lille score was 0.06, which after prednisolone was gradually tapered and discontinued. During therapy, the severity of jaundice decreased, although complete regression was not achieved.

In August 2024, the patient's condition deteriorated, with the onset of weakness, dyspnea, ascites, and oedema. Laboratory tests revealed hyperbilirubinemia, marked hypoalbuminemia, severe anemia (hemoglobin – 79 g/L), elevated C-reactive protein, and deficiencies of vitamin B_{12} (159 pg/mL) and folates (1.9 ng/mL) (Table). Iron metabolism indices were within the reference ranges. The severity of LC was classified as Child–Pugh C, MELD-Na (Model for End-Stage Liver Disease – Na) was 25 points. The anemia was initially attributed to vitamin B_{12} and folate deficiency. Treatment included albumin infusions, a single erythrocyte transfusion, spironolactone, furosemide, rifaximin, vitamin B_{12}, folic acid, and thiamine. Improvement was observed, with reduction in oedema and ascites, some alleviation of weakness and dyspnea, and a transient increase in hemoglobin to 85 g/L.

During the following month, progressive weakness, dyspnea, and oedema recurred. The patient was readmitted in October 2024. On examination: body temperature 36.5 °C, oxygen saturation 98%, clear consciousness

***Table*. Dynamics of laboratory parameters in a patient with liver cirrhosis**

Parameter	Ref. range	Nov. 2023	Apr. 2024	Aug. 2024	Oct. 2024	Jul. 2025
Complete Blood Count						
hemoglobin, g/l	130–170	132	121	79	50	125
red blood cells, $\times10^{12}$/l	4.3–5.7	4.2	3.5	2.3	1.3	4.0
reticulocytes, ‰					77.9	5.1
white blood cells, $\times10^{9}$/l	4.5–11.0	6.5	6.9	8.4	15.6	4.1
Neutrophils, $\times10^{9}$/l	1.78–5.38			5.2	11.1	2.0
Plateles, $\times10^{9}$/l	150–400	137	80	47	86	124
Biochemistry						
albumin, g/l	35–52		31.2	27.6	27.1	36.0
ALT, U/l	<35	80	58	27	13	12
AST, U/l	<35	230	72	82	52	17
LDH, U/l					671	182
GGTP, U/l	<49	938	526	621	749	32
total bilirubin, μmol/l	3.4–20.5	42	214	273	405	
direct bilirubin, μmol/l	<8.6	34	176	207	172	
creatinine, μmol/l	49–90	70	95	110	152	127
cholesterol, mmol/l	<5.0	6.7		2.5	1.9	3.2
C-reactive protein, mg/L	<5.0			36	20	3
Coagulation panel						
INR	0.8–1.2	1.13	1.75	1.65	2.55	1.20
prothrombin time, sec	9.4–12.5		18.7		23.4	
fibrinogen, g/l	2.0–4.0	3.1	2.6	1.5	1.8	2.4

Note: ALT – alanine aminotransferase; AST – aspartate aminotransferase; GGTP – gamma-glutamyltranspeptidase; INR – international normalized ratio; LDH – lactate dehydrogenase.

(Glasgow Coma Scale score was 15), no signs of overt hepatic encephalopathy. The skin, sclerae, and visible mucosae were icteric, lower extremities oedema were present. Heart rate was 83 bpm, blood pressure – 115/75 mmHg. The abdomen was enlarged due to non-tensed ascites, non-tender on palpation, hepatosplenomegaly was noted.

Abdominal ultrasound showed grade 2 ascites, hepatosplenomegaly, and dilation of the portal and splenic veins. Upper endoscopy showed grade 2 oesophageal varices.

Laboratory tests demonstrated persistent marked hyperbilirubinemia involving both fractions, elevated gamma-glutamyltranspeptidase, impaired hepatic protein-synthetic function (hypoproteinemia, hypoalbuminemia, hypocholesterolemia, hypocoagulation), elevated C-reactive protein, and increased creatinine up to 152 µmol/L.

A complete blood count revealed severe macrocytic anemia with reticulocytosis: hemoglobin – 50 g/L, red blood cells – 1.26×10^{12}/L, reticulocytes – 77.9 ‰, MCV (mean corpuscular volume) – 126 fL; moderate thrombocytopenia (platelets – 86×10^{9}/L), and leukocytosis (white blood cells – 15.6×10^{9}/L) with a left shift (Table).

Urinalysis showed increased urobilinogen and direct bilirubin, but no free hemoglobin, proteinuria or urinary sediment abnormalities.

A diagnostic workup for the causes of anemia was conducted. Occult blood was not detected in stool samples (tested twice). Iron metabolism parameters — serum iron (26 µmol/L), transferrin saturation (47%), and latent iron-binding capacity (24.9 µmol/L) — were within reference limits, excluding iron deficiency. Serum vitamin B_{12} (201.4 pg/mL) and folic acid (9.2 ng/mL) concentrations were within normal ranges. Lactate dehydrogenase levels were elevated, and the direct Coombs test was negative.

A peripheral blood smear demonstrated anisocytosis (macrocytosis), poikilocytosis, spherocytes, target cells, and 20% of red blood cells with spur-like morphology, approximately half of which were acanthocytes. Howell–Jolly bodies were observed in some red blood cells (Figure). The patient was examined by a hematologist, and sternal puncture revealed erythroid hyperplasia. To exclude hypothyroidism as a potential cause of secondary acanthocytosis, thyroid-stimulating hormone was measured (2.5 mIU/L), which was within the reference range.

Thus, in a patient with toxic etiology liver cirrhosis (Child–Pugh class C, MELD-Na was 34), ACLF was diagnosed: CLIF-C ACLF (Chronic Liver Failure Consortium Acute-on-Chronic Liver Failure score)[1] score was 46. The severity of ACLF was grade 2 with the development of hepatic failure and acute kidney injury of prerenal origin stage 1 according to KDIGO (Kidney Disease: Improving Global Outcomes). Based on hematological evaluation, secondary acanthocytosis was diagnosed in the patient with severe hepatic failure.

Treatment of LC and its complications was conducted in accordance with the clinical guidelines of the Ministry of Health of the Russian Federation[2]. To correct anemia, three packed red blood cell transfusions were performed, achieving a maximum hemoglobin level of 71 g/L.

The patient met the criteria for severe decompensated LC with signs of poor prognosis, which justified consultation with a transplant specialist and placement on the liver transplantation waiting list. In spring 2025, orthotopic liver transplantation was performed. The surgical procedure was completed without complications, appropriate immunosuppressive therapy was administered, and three months post-transplantation, anemia resolved: hemoglobin was 125 g/L, red blood cells – 4.0×10^{12}/L, and no acanthocytes were detected (Table).

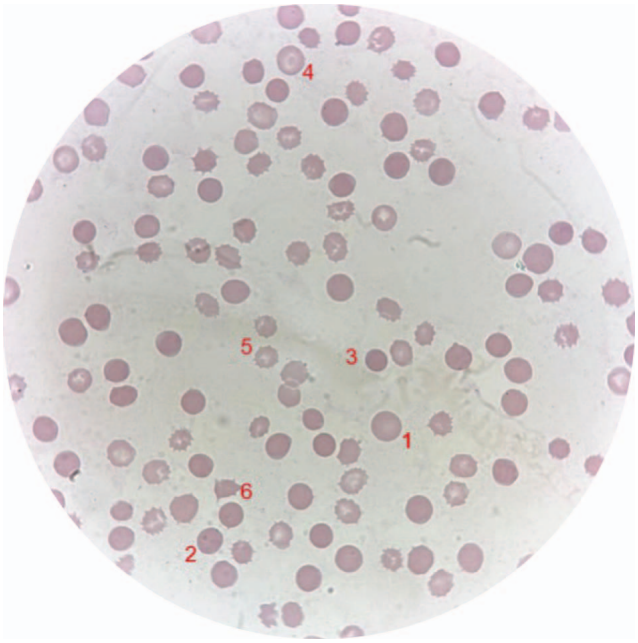

FIG. Peripheral blood smear, Romanowsky–Giemsa stain, magnification ×1000.

Note: 1 – normal red blood cell; 2 – pathological red blood cell inclusions (Howell–Jolly bodies); 3 – spherocyte; 4 – target cell; 5 – spur cell: echinocyte (uniform, evenly distributed spicules); 6 – spur cell: acanthocyte (irregular, unevenly distributed spicules).

[1] Chronic Liver Failure Consortium Acute-on-Chronic Liver Failure score. https://efclif.com/research-infrastructure/score-calculators/clif-c-of-aclf-ad/ (access date: 05.06.2025).

[2] Ministry of Health of the Russian Federation. Clinical Guidelines. Liver Cirrhosis and Fibrosis (approved by the Ministry of Health of the Russian Federation, 2022) https://cr.minzdrav.gov.ru/view-cr/715_2 (access date: 05.06.2025).

DISCUSSION

Approximately in 50% of anemia cases observed in patients with liver cirrhosis, as reported by B. Scheiner et al. [1], the underlying etiology remains unknown even after comprehensive laboratory and instrumental diagnostic work up.

The presented clinical case shows the diagnostic workup for anemia, during which all causes of anemia (iron-deficiency, megaloblastic, and hemolytic anemias (including autoimmune anemia and anemia associated with Wilson's disease), as well as anemia due to bone marrow aplasia) were excluded. Based on the combination of clinical, laboratory, and morphological data, a diagnosis of secondary acanthocytosis due to LC was established. Another potential mechanism contributing to anemia could be chronic inflammation in the patient with LC; however, iron metabolism parameters did not indicate functional iron deficiency.

Microscopic examination of the peripheral blood smear revealed diverse red blood cells morphologies: hyperchromic macrocytes, spherocytes, codocytes, echinocytes, acanthocytes, and red blood cells containing Howell–Jolly bodies. This combination may reflect both the severity of the underlying disease and the effects of therapeutic interventions. The presence of spherocytes is likely related to prior blood transfusions, while Howell–Jolly bodies and pronounced poikilocytosis indicate functional hyposplenism developing in LC and during hemolysis [9].

In this case, the absence of hematological pathology prior to liver disease manifestation excludes hereditary forms of acanthocytosis. Normal thyroid-stimulating hormone levels and bone marrow findings ruled out hypothyroidism and myelodysplasia as causes of secondary acanthocytosis.

In routine clinical practice, distinguishing acanthocytes from echinocytes in blood smears may present certain diagnostic challenges. Acanthocytes are pathological, irreversibly altered red blood cells with unevenly distributed spicules, formed due to lipid metabolism disorders, membrane damage from hemolysis, and impaired splenic clearance of defective red blood cells [10]. Echinocytes, by contrast, have evenly distributed projections and are reversible; their appearance may result from osmolarity disturbances, smear preparation technique, or sample transport [11].

SCA in patients with alcoholic LC must be differentiated from Zieve's syndrome, which presents with the triad of hemolysis, jaundice, and dyslipidemia. In Zieve's syndrome, improvement may occur with alcohol cessation and conservative therapy, whereas SCA is irreversible and serves as a marker of poor prognosis [12], as observed in the presented case.

Pharmacotherapy for SCA remains insufficiently studied: few case reports describe positive effects of flunarizine, pentoxifylline, cholestyramine, high-dose steroids, and plasmapheresis [2, 13, 14]. Blood transfusions provide temporary correction but may contribute to iron overload and progression of liver damage.

In the presented patient, the development of ACLF and secondary acanthocytosis were extremely poor prognostic factors. It should be emphasized that among all available interventions, only liver transplantation has demonstrated the ability to reverse both hepatic decompensation and the characteristic red blood cells changes observed in acanthocytosis [15]. This was clearly illustrated by the outcome in our patient.

Given the high prognostic significance of acanthocytosis even in the absence of anemia in patients with ACLF [7], it can be anticipated that this parameter may be evaluated for potential inclusion in future ACLF severity scoring systems.

CONCLUSION

The presented clinical case of SCA highlights the complexity of diagnosing rare causes of anemia in patients with decompensated LC and ACLF. Morphological assessment of peripheral blood smears is a simple and accessible method for detecting acanthocytosis. Liver transplantation remains the only intervention capable of achieving regression of both hepatic insufficiency and acanthocytosis.

AUTHORS CONTRIBUTIONS

Ekaterina O. Liusina, Natalia A. Suvorova and Andrey P. Kostikov participated in the examination, treatment of the patient, literature analysis, preparation of the manuscript text, and critical revision of the manuscript. All the authors approved the final version of the article.

ВКЛАД АВТОРОВ

Е.О. Люсина, Н.А. Суворова и А.П. Костиков принимали участие в проведении обследования, лечения пациента, анализе литературы, подготовке текста рукописи, критическом пересмотре рукописи. Все авторы утвердили окончательную версию статьи.

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