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CASE REPORT: HEMORRHAGIC VASCULITIS IN AN ELDERLY PATIENT

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Abstract

Introduction. Hemorrhagic vasculitis (HV), or Schönlein–Henoch purpura, is a systemic vasculitis of small vessels, predominantly mediated by IgA. Although the disease occurs relatively frequently in childhood, its manifestation in elderly patients is much rarer and typically presents with a more severe and torpid course. In older patients, the condition is often accompanied by pronounced visceral complications, including abdominal and renal syndromes, which significantly worsen the prognosis and require special attention in both diagnosis and treatment. This article presents a clinical case of hemorrhagic vasculitis in a geriatric patient. The disease manifested with the classical triad of symptoms: cutaneous purpura, abdominal pain syndrome, and renal involvement.

Objective. To describe a clinical case of hemorrhagic vasculitis in an 86-year-old patient with concomitant cutaneous, abdominal, and renal syndromes on the background of significant comorbidities.

Materials and Methods. This article presents the clinical case of an 86-year-old patient, V., who was admitted to the rheumatology department of the City Rheumatology Center in Almaty with complaints of hemorrhagic rashes on the upper and lower extremities, marked weakness, abdominal pain syndrome, episodes of nausea and vomiting, and oliguria. A comprehensive clinical, laboratory, and instrumental examination was conducted, including complete blood count, biochemical blood tests, urine analysis (including the Nechiporenko test), coagulation profile, immunological studies, abdominal ultrasound, ECG, and echocardiography. The diagnosis was established based on the patient's complaints, clinical presentation, laboratory and instrumental findings, and in accordance with the EULAR (2010) classification criteria for adults.

Results. The patient presented with typical symptoms of cutaneous hemorrhagic syndrome (symmetrical bluish-purple rashes that did not blanch under pressure), abdominal manifestations (nausea, vomiting, pain in the periumbilical area), and signs of renal involvement (episodes of oliguria, leukocyturia, elevated creatinine and urea levels). Under treatment with glucocorticosteroids, immunosuppressive therapy, infusion, and symptomatic therapy, positive dynamics were observed, including a reduction in the severity of skin manifestations, improvement in abdominal and renal symptoms, better laboratory values, and stabilization of the general condition. However, the disease course remained torpid, with prolonged healing of skin lesions, high sensitivity to medication changes, and the need for continuous cardiological and gastroenterological supervision.

Conclusion. Early diagnosis, timely assessment of organ involvement, appropriate use of immunosuppressive and glucocorticosteroid therapy, and careful monitoring of complications are crucial for achieving favorable outcomes.

Key words: *Hemorrhagic Vasculitis, Schönlein–Henoch purpura, Vasculitis, elderly patient, glucocorticosteroid therapy.*

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Резюме

КЛИНИЧЕСКИЙ СЛУЧАЙ: ГЕМОРРАГИЧЕСКОГО ВАСКУЛИТА У ПАЦИЕНТА СТАРЧЕСКОГО ВОЗРАСТА

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Введение. Геморрагический васкулит (ГВ), или пурпура Шёнлейна-Геноха, является системным васкулитом мелких сосудов, преимущественно IgA-опосредованным. Несмотря на относительно частое возникновение заболевания в детском возрасте, его манифестация у пациентов старческого возраста встречается значительно реже и характеризуется более тяжелым, торпидным течением. У пожилых пациентов заболевание часто сопровождается выраженными висцеральными осложнениями, включая абдоминальный и почечный синдромы, что существенно ухудшает прогноз и требует особого внимания в диагностике и лечении. В статье представлен клинический случай геморрагического васкулита у пациента старческого возраста. Заболевание манифестировало классической триадой симптомов: кожным пурпурным, абдоминальным болевым синдромами и поражением почек.

Цель. Описать клинический случай геморрагического васкулита у пациента 86 лет с сочетанным кожным, абдоминальным и почечным синдромами на фоне выраженной коморбидности.

Материалы и методы. В данной статье приведен клинический случай пациента В., 86 лет, поступивший в ревматологическое отделение ГРЦ г. Алматы с жалобами на геморрагические высыпания на коже верхних и нижних конечностей, выраженную слабость, абдоминальный болевой синдромы, эпизоды тошноты, рвоты, также олигурии. Пациенту проведена комплексная клинико-лабораторная и инструментальная диагностика, включающая общий и биохимический анализы крови, анализы мочи (в т.ч. по Нечипоренко), коагулограмму, иммунологические исследования, УЗИ органов брюшной полости, ЭКГ, ЭхоКГ. Диагноз установлен на основании жалоб, клинической картины, лабораторно-инструментальных исследований с учетом классификационных критериев EULAR (2010) для взрослых.

Результаты. Пациент предъявлял типичные жалобы на кожный геморрагический синдромы (симметричные высыпания синюшно-багрового цвета, не исчезающие при надавливании), абдоминальные симптомы (тошнота, рвота, болезненность в околопупочной области) и признаки поражения почек (эпизод олигоурии, лейкоцитурия, повышение уровней креатинина и мочевины). На фоне лечения (глюкокортикостероидная, иммуносупрессивная, инфузионная и симптоматическая терапия) отмечалась положительная динамика в виде уменьшения выраженности кожных проявлений, абдоминальных и почечных синдромов, улучшения лабораторных показателей, стабилизации общего состояния. В то же время течение заболевания оставалось торпидным, с длительным заживлением кожных элементов, высокой чувствительностью к изменению медикаментозной схемы, необходимостью постоянного кардиологического и гастроэнтерологического сопровождения.

Выводы. Ранняя диагностика, оценка органного поражения, назначение иммуносупрессивных и глюкокортикостероидных препаратов и мониторинг осложнений помогает достичь наилучшего результата.

Ключевые слова: *геморрагический васкулит, Шенлейн-Генох, пурпура, васкулит, глюкокортикостероидная терапия.*

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Түйіндеме

КЛИНИКАЛЫҚ ЖАҒДАЙ: ЕГДЕ ЖАСТАҒЫ НАУҚАСТАҒЫ ГЕМОРРАГИЯЛЫҚ ВАСКУЛИТ

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Кіріспе. Геморрагиялық васкулит (ГВ), немесе Шенлейн-Генох пурпурасы - ұсақ қантамырлардың, көбінесе IgA-мен байланысты жүйелі васкулиті болып табылады. Бұл ауру балаларда жиі кездескенімен, егде жастағы адамдарда сирек дамиды және әдетте ауыр, баяу (торпидті) ағыммен сипатталады. Қарт науқастарда аурудың ағымы жиі висцералды асқынулармен, атап айтқанда абдоминалдық және бүйректік синдромдармен қатар жүреді, бұл аурудың ағымын едәуір нашарлатып, диагностикалық және терапиялық тәсілдерге ерекше назар аударуды

талап етеді. Бұл мақалада егде жастағы науқаста дамыған геморрагиялық васкулиттің клиникалық жағдайы сипатталады. Аурудың манифестациясы классикалық үштік симптомдармен байқалды: терідегі пурпура, абдоминалдық ауырсыну және бүйрек зақымдануы.

Мақсаты. 86 жастағы, айқын коморбидтілікті иеленген науқастағы терілік, абдоминалдық және бүйректік синдромдарының үйлесуімен жүретін геморрагиялық васкулиттің клиникалық жағдайын сипаттау.

Материалдар мен әдістер. Мақалада Алматы қаласындағы «Қалалық ревматология орталығының» ревматология бөлімшесіне түскен В. есімді, 86 жастағы науқастың клиникалық жағдайы ұсынылады. Науқас аяқ-қолдарындағы геморрагиялық бөртпелерге, айқын әлсіздікке, абдоминалдық ауырсынуға, жүрек айнуы мен құсуға, сондай-ақ олигурияға шағымданған. Диагностика шеңберінде жалпы және биохимиялық қан талдаулары, зәр талдаулары (оның ішінде Нечипоренко әдісімен), коагулограмма, иммунологиялық зерттеулер, құрсақ қуысы ағзаларының ультрадыбыстық зерттеуі, ЭКГ және ЭхоКГ жүргізілді. Диагноз науқастың шағымдары, клиникалық белгілері мен зертханалық-аспаптық зерттеу нәтижелері негізінде, ересектерге арналған EULAR (2010) классификациялық критерийлеріне сүйене отырып қойылды.

Нәтижелер. Науқас геморрагиялық терілік синдромға (басқанда жоғалмайтын, симметриялы, көгілдір-қызғылт түсті бөртпелер), абдоминалдық симптомдарға (жүрек айну, құсу, кіндік маңындағы ауырсыну), сондай-ақ бүйрек қызметінің бұзылу белгілеріне (олигоурия эпизоды, лейкоцитурия, креатинин мен мочевинаның жоғары деңгейі) тән шағымдармен түсті. Глюкокортикостероидтық, иммуносупрессивті, инфузиялық және симптоматикалық терапия аясында тері көріністері мен абдоминалдық және бүйрек синдромдарының азаюы, зертханалық көрсеткіштердің жақсаруы және жалпы жағдайдың тұрақтануы байқалды. Алайда аурудың ағымы баяу (торпидті) сипатта болып, тері элементтерінің ұзақ жазылуы, дәрілік емдеу режиміне жоғары сезімталдық, тұрақты кардиологиялық және гастроэнтерологиялық бақылауды қажет етті.

Қорытынды. Геморрагиялық васкулиттің ерте диагностикасы, ағзалық зақымдану деңгейін уақтылы бағалау, глюкокортикостероидтық және иммуносупрессивті емді дұрыс тағайындау мен асқынуларды жүйелі бақылау оң нәтижелерге қол жеткізуге мүмкіндік береді.

Түйінді сөздер: *геморрагиялық васкулит, Шенлейн–Генох пурпурасы, васкулит, егде жастағы науқас, глюкокортикостероидтық терапия.*

Дәйексөз үшін:

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Introduction

The clinical association of purpura, arthralgia, and arthritis was first described in 1837 by the German physician Johann Lukas Schönlein. Schönlein–Henoch purpura, or Henoch–Schönlein purpura (HSP), also known as hemorrhagic vasculitis (HV), is a systemic hypersensitivity vasculitis caused by the deposition of immune complexes in small blood vessels, including the renal glomeruli and mesangium [8]. On the skin, it manifests as non-thrombocytopenic purpura or urticaria. Worldwide, IgA nephropathy is the most common cause of primary glomerulonephritis. The detection of IgA deposits in small blood vessels and renal glomeruli is diagnostic in the majority of cases [8]. HV (IgA vasculitis) in elderly patients is significantly less common than in children and young adults, but it has a more severe and indolent course. In patients aged over 65 years, the disease is often accompanied by marked renal dysfunction and systemic complications. A higher mortality is also observed, especially in cases of late diagnosis [8].

Studies by American researchers emphasize that skin manifestations may be less typical (sometimes without distinct purpura), which complicates early disease detection [6]. Moreover, elderly patients more often present with comorbidities that mask or intensify the clinical picture of IgA vasculitis.

Table 1 presents descriptions of clinical cases reported by foreign authors (with references and author names) [14, 11, 5, 10, 15, 13, 7, 9].

However, in some cases, as our practice has shown, the cutaneous form of hemorrhagic vasculitis (HV) in elderly patients can have a severe course and prolonged healing, even with an adequate choice of therapy. The severity of the disease is determined by systemic manifestations, particularly gastrointestinal involvement, leading to hepatitis, pancreatitis, and lesions of the intestines and kidneys. The pathogenesis of HV is currently poorly understood. In about 40% of children and adults, the disease is triggered by various risk factors or triggers. In children, these include viral and bacterial infections (Streptococcus sp., Salmonella sp., Shigella sp., Campylobacter sp., Yersinia enterocolitica, Helicobacter pylori, Mycoplasma pneumoniae, Legionella sp., Staphylococcus aureus, Mycobacteria tuberculosis, parvovirus B19, hepatitis B and C viruses, HIV, parasitic infections: Toxocara canis, amebiasis). In adults and elderly patients, in addition to infections, the disease is often triggered by medications and toxins (antibiotics, angiotensin-converting enzyme inhibitors, angiotensin II receptor antagonists, cinchona alkaloids, vitamin A, streptokinase, nonsteroidal anti-inflammatory drugs, chlorpromazine, cocaine).

Less common triggers in elderly and senile patients include comorbid conditions and the following states: paraneoplastic syndrome, lymphoma, liver diseases, diabetes mellitus, intestinal diseases, and joint disorders.

Table 1.

Descriptions of clinical cases of IgA vasculitis.

№	Description of clinical case	Clinical features	Reference
1	Multi-organ involvement in a 72-year-old woman	Intestinal intussusception, renal failure, cardiac complications. Treatment: steroids, hemofiltration	https://pubmed.ncbi.nlm.nih.gov/40184107/
2	Refractory IgA vasculitis in a 90-year-old patient	Purpura, anorexia, steroid resistance. Azathioprine added to treatment	https://pubmed.ncbi.nlm.nih.gov/36249629/
3	IgA vasculitis with severe complications in a 65-year-old man	GI bleeding, cutaneous vasculitis, neurological symptoms, renal involvement. Therapy: pulse steroids, cyclophosphamide	https://pubmed.ncbi.nlm.nih.gov/35637989/
4	IgA vasculitis without purpura in a 76-year-old woman	Diarrhea, pain, no skin rash; IgA deposits on biopsy. Treated with factor XIII concentrate	https://pubmed.ncbi.nlm.nih.gov/33950360/
5	IgA vasculitis with leg edema in a 74-year-old man	Edema, ankle pain, vasculitic rash	https://pubmed.ncbi.nlm.nih.gov/37529819/
6	IgA vasculitis with GI symptoms in a 78-year-old man	Bloody diarrhea, mesenteric edema, renal failure	https://pubmed.ncbi.nlm.nih.gov/39034162/
7	IgA vasculitis following rotavirus infection in an elderly patient	Small bowel inflammation, vasculitis, proteinuria; positive response to steroids	https://pubmed.ncbi.nlm.nih.gov/28174414/
8	IgA vasculitis with nephropathy in a 61-year-old man	Purpura, pain, intestinal wall thickening, ascites, IgA deposits. Improvement with steroids	https://pubmed.ncbi.nlm.nih.gov/31280225/

Triggers may also include diseases and conditions accompanied by structural abnormalities of immunoglobulin A (IgA), its subclasses, and the complement system [3]. The most typical morphological sign of renal involvement in HV is the presence of IgA deposits in the endothelium of capillaries and in the glomerular mesangium, thickening of the capillary loops, endothelial proliferation, fibrinoid swelling of the vessel walls, lymphohistiocytic infiltration of capillaries, perivascular inflammation of renal arterioles, glomeruli, tubules, and renal stroma [2].

The diagnosis of HV is based on the presence of classification criteria in patients. For adults, the criteria were developed by the American College of Rheumatology (ACR) in 1990. In children, the “gold standard” for diagnosis is the EULAR/PRINTO/PRES criteria, developed jointly by the European League Against Rheumatism (EULAR), the Paediatric Rheumatology International Trials Organization (PRINTO), and the Paediatric Rheumatology European Society (PRES) [12]. The ACR (1990) criteria and the revised 2010 EULAR/PRINTO/PRES criteria are presented in Table 2.

Table 2.

Diagnostic criteria for Henoch-Schonlein purpura.

ACR Criteria* (1990)	EULAR/PRINTO/PRES Criteria** (2010)
• Age ≤20 years	• Purpura or petechiae
• Palpable purpura	+
• Acute abdominal pain	**+** at least one of the following: - Abdominal pain - Joint inflammation or pain - Renal involvement -Leukocytoclastic vasculitis with preceding IgA deposition or proliferative glomerulonephritis
• Granulocytes in the walls of small arterioles or venules	

Note: *At least two criteria are required to establish the diagnosis. The presence of purpura or petechiae is mandatory, and at least one additional criterion is required to confirm the diagnosis.*

The same classification criteria (EULAR/PRINTO/PRES, 2010) apply to elderly patients. These include purpura or petechiae along with at least one of the following additional criteria: (1) abdominal pain; (2) joint inflammation or pain; (3) renal involvement; (4) leukocytoclastic vasculitis with prior IgA deposition or proliferative glomerulonephritis with prior IgA deposition. At least two criteria are required to establish the diagnosis: the presence of purpura or petechiae is mandatory, plus at least one additional criterion. An important addition is the assessment of target organ function, particularly renal function. The EULAR (2023) guidelines emphasize the need to screen for nephropathy and hematuria at the first suspicion of vasculitis in elderly patients.

The manifestations of HV in elderly patients are characterized by an atypical course and a high risk of complications, which require special attention in clinical practice [13]. It is important to note that elderly patients often have comorbidities and age-related features, which require an individualized approach that takes into account drug interactions, careful monitoring, and dose adjustments of selected medications. In our clinical case, the patient had several comorbid conditions, which required special attention in the treatment of HV.

Regarding geographic features of organ involvement in HV: analysis of 42 studies covering 4,064 adult patients with IgA vasculitis revealed the following regional differences ([Worldwide Distribution and Extracutaneous Manifestations of Henoch-Schönlein Purpura in Adults: Narrative Review - PMC], Table 3).

Table 3.

Diagnostic criteria for Henoch-Schonlein purpura.

Region	Mean age (years)	Men (%)	Gastrointestinal involvement (%)	Musculoskeletal symptoms (%)	Genitourinary involvement (%)
Europe	49.3	62.2	58.2	57.9	67.2
America	48.6	47.7	31.4	47.7	47.0
Asia	29.8	51.4	44.7	47.4	72.3

According to Table 3, gastrointestinal (GI) and musculoskeletal involvement are more frequently observed in Europe (58.2% and 57.9%, respectively), whereas in Asia genitourinary involvement is more common (72.3%). Regarding prevalence and incidence in children:

USA: 6.1–20.4 cases per 100,000 children per year;

UK and France: 20–70 cases per 100,000 children per year;

Asia: up to 56 cases per 100,000 children per year.

In adults globally: 1.4–5.1 cases per 100,000 adults per year. The peak incidence in adults occurs between 50 and 60 years of age [6].

Objective: To present a clinical case description of hemorrhagic vasculitis in an 86-year-old patient with combined cutaneous, abdominal, and renal syndromes on the background of pronounced comorbidity.

Clinical case

Patient V., 86 years old, presented to the City Rheumatology Center (CRC) of Almaty with complaints of a hemorrhagic rash on the upper and lower limbs, heaviness in the lower limbs, nausea, vomiting, diarrhea up to 3 times per day, cramping pain in the periumbilical area, loss of appetite, fever up to 38°C, and marked general weakness.

The patient provided informed consent to participate in the study and did not object to the publication of the treatment results with personal data anonymized.

From the patient's history:

According to the patient, two weeks before the onset of the disease he had an acute respiratory viral infection (ARVI). In early September 2024, a rash appeared on the upper limbs and in the umbilical area. He consulted his district general practitioner accompanied by his son and was referred to an allergist and dermatologist. On 12.09.2024, the allergist suggested the diagnosis "dermatitis?", and the dermatologist's conclusion was "hemorrhagic vasculitis?"

On 13.10.2024, symptoms of nausea, vomiting, episodes of oliguria, fever up to 38°C, and general weakness appeared, and he was examined by a rheumatologist at the City Rheumatology Center (CRC) of Almaty. Laboratory tests: Complete blood count (CBC) on 11.09.2024: ESR – 28 mm/h, hemoglobin – 133 g/L, leukocytes – 10.1×10^3/μL, platelets – 424×10^3/μL, eosinophils – 0.06×10^3/μL, lymphocytes – 3.1×10^3/μL. Biochemical blood test: urea – 13.9 mmol/L, creatinine – 194 μmol/L, CRP – 27 mg/L. Urinalysis on 13.09.2024: protein negative, leukocyturia. Based on complaints and clinical-laboratory data, the patient was hospitalized at the CRC of Almaty with the diagnosis: Hemorrhagic vasculitis.

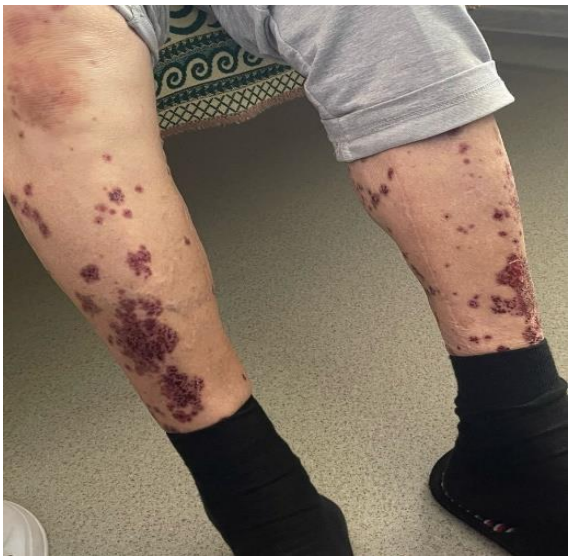
Medical history:

The patient denies viral hepatitis, tuberculosis, and sexually transmitted diseases. Family history is unremarkable. Bad habits: denies smoking, does not consume alcohol. No history of allergies. The patient is followed up by a cardiologist with the diagnosis: ischemic heart disease (IHD), unstable angina IIIB according to Braunwald. Myocardial revascularization with stenting of the right coronary artery (RCA) and posterolateral branch (PLB) on 25.07.2024. Status post coronary artery bypass grafting (CABG) of the left anterior descending artery (LAD), posterior interventricular artery (PDA), and diagonal branch (DB) (October 2019, 3 grafts). Status post pacemaker (PM) implantation due to sick sinus syndrome (SSS) (December 2022). Persistent atrial flutter. CHA2DS2-VASc score 5. HAS-BLED score 2. EHRA class IIb. Acute heart failure, type 1. Grade III arterial hypertension, very high cardiovascular risk. Cerebrovascular disease (CVD). Atherosclerosis. Stenotic lesions of the carotid arteries. On 02.08.2024, the patient was urgently hospitalized at the City Cardiology Center (CCC) of Almaty with the diagnosis: acute coronary syndrome (ACS) without ST-segment elevation. Emergency coronary angiography with stenting of the RCA and its posterolateral branch (PLB-RCA) was performed. The patient is on standard IHD therapy. Surgeries: CABG in October 2019. Pacemaker implantation for SSS in December 2022. Coronary angiography with stenting of RCA and PLB-RCA in August 2024. The patient is also followed up by a gastroenterologist with the diagnosis: chronic gastroduodenitis (currently in remission). Cystectomy in 1999. Epidemiological history: no contact with infectious patients.

Objective findings at the time of examination:

General condition: moderate severity due to cutaneous, abdominal, and renal syndromes. Consciousness: clear. Body type: normosthenic. Skin: widespread, symmetrical hemorrhagic rash on the skin of the upper and lower limbs, acute inflammatory in nature, bluish-purple color, confluent, does not blanch on pressure, with telangiectasias (Figure 1).

Peripheral lymph nodes are not palpable. Peripheral edema is moderate in the legs. Respiratory system: the



Picture 1. Hemorrhagic rash on the legs.

chest is of normal shape, participates symmetrically in the act of breathing. Percussion: clear lung sounds over all fields. Auscultation: vesicular breathing, no wheezing. Respiratory rate: 16 per minute.

Cardiovascular system: no visible abnormalities in the heart area. The borders of relative cardiac dullness are normal. Auscultation: irregular rhythm (atrial flutter). Heart rate: 76–135 bpm. Pulse rate: 76-135 bpm. Blood pressure: 110/70 mmHg. Heart sounds are muffled. No pathological murmurs. Digestive system: the tongue is dry, coated with a white film. The abdomen is soft, moderately tender. The liver is at the costal margin. The spleen is not palpable. Tendency to constipation noted. Musculoskeletal system: the joints show no significant visible deformities or exudative changes. Urinary system: negative costovertebral angle tenderness on both sides. Urination is painless, the patient notes decreased urine output.

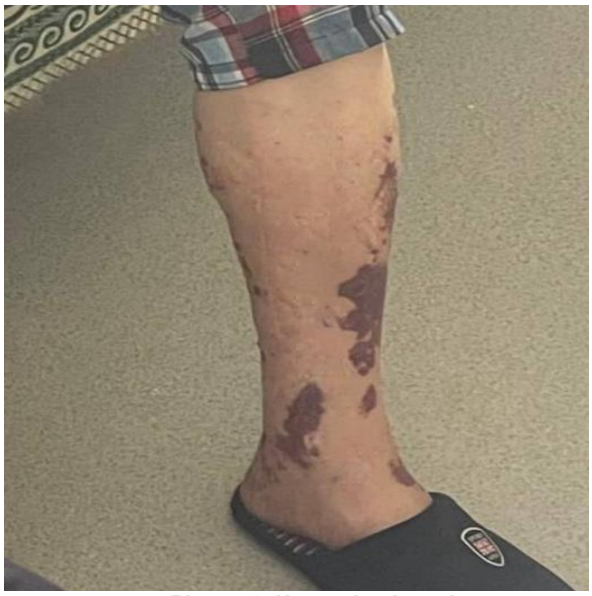
Laboratory tests:

Name	Indicators
CBC from 13.09.2024	Hemoglobin – 125 g/L, erythrocytes – 4.0×10^{12}/L, color index – 0.93, platelets – 327×10^{9}/L, leukocytes – 9.5×10^{9}/L; segmented neutrophils – 81%, monocytes – 2%, lymphocytes-17%, ESR-28 mm/h. **Note**: slight leukocytosis, elevated ESR.
CBC from 18.09.2024	Hemoglobin – 137 g/L, erythrocytes – 4.4×10^{12}/L, color index – 0.93, platelets – 344×10^{9}/L, leukocytes – 14.9×10^{9}/L, segmented neutrophils – 71%, monocytes – 7%, lymphocytes-22%, ESR – 6 mm/h. **Note**: leukocytosis.
Urinalysis from 13.09.2024	Volume: 60.0 mL, color – straw yellow, clarity – clear, specific gravity – 1015 g/L, reaction – acidic, protein – absent, squamous epithelium – 3-4 units, leukocytes – 6-7 per field of view. **Note**: leukocyturia.
Nechiporenko urine test from 13.09.2024	Leukocytes – large number; erythrocytes: 1000.
Biochemical blood test from 13.09.2024	Glucose – 9.1 mmol/L, cholesterol – 3.3 mmol/L, total protein – 68 g/L, CRP – 27 mg/L, urea – 13.9 mmol/L, creatinine – 194 μmol/L, potassium – 4.0, sodium – 140, ALT – 15.3 U/L, AST – 14.8 U/L, total bilirubin – 9.1 μmol/L, amylase – 28.3 U/L. **Note**: elevated glucose, urea, creatinine, and CRP levels.
Biochemical blood test from 18.09.2024	C-reactive protein – 2.7 mg/L.
Coagulogram from 13.09.2024	APTT – 29 sec, prothrombin index – 126%, fibrinogen A – 2.88 g/L, thrombin time – 26 sec, INR – 1.08, prothrombin time – 12.7 sec, D-dimer – 1.65, INR – 1.05, fibrinogen – 3.5 g/L.
Immunological tests from 13.09.2024	ANCA-screen – 0.4, antibodies to mitochondria AMA-M2 – 1.2 IU/mL.
CBC from 18.10.2024	Hemoglobin – 152 g/L, erythrocytes – 4.93×10^{12}/L, color index – 0.92, platelets – 329×10^{9}/L, leukocytes – 8.9×10^{9}/L, ESR - 2mm/h. **Note**: within normal limits.
Biochemical blood test from 18.10.2024	Glucose – 5.64 mmol/L, cholesterol – 4.16 mmol/L, ALT – 25 U/L, AST – 20 U/L, creatinine – 117 μmol/L, urea – 8.60 mmol/L, CRP – 2.7 mg/L. **Note**: decreasing levels of creatinine and urea in dynamics.
Urinalysis from 18.10.2024	Volume: 60.0 mL, color – straw yellow, clarity – clear, specific gravity – 1024 g/L, reaction – acidic, protein – absent. **Note**: within normal limits.
From 15.09.2024: microreaction, ELISA for hepatitis B and C viruses, Wright-Hedelson reaction, HIV – negative results.	

Instrumental studies:

Name	Indicators
Chest X-ray in 2 projections	Conclusion: no pulmonary pathology detected.
ECG:	Atrial fibrillation with an average ventricular rate of 83 bpm, normosystole, fine-wave form. Rightward deviation of the electrical axis. Decreased metabolism in the lower-lateral region of the VS.
Echocardiography:	CABG. Right heart pacemaker. Aortic root is not dilated, wall thickened. Heart chambers are not dilated. Valves are thickened. Left ventricular contractility is reduced. Left ventricular wall hypertrophy. Type 1 diastolic dysfunction of the left ventricle. Mitral valve regurgitation grade 1, aortic valve regurgitation grade 1. Tachycardia. Hypokinesia zone in the LV walls.
Abdominal and kidney ultrasound	Diffuse changes in liver parenchyma. Signs of portal hypertension. Reactive pancreatitis. Thickened renal pelvis walls bilaterally.
Doppler ultrasound of veins and arteries of lower limbs	Conclusion: atherosclerosis of the arteries of the lower limbs. Altered blood flow in the right posterior tibial artery. No evidence of venous or arterial thrombosis of the lower limbs. Edema of soft tissues of both lower limbs.
EGD	Conclusion: erosions of the duodenal bulb. Mixed gastritis, cardia insufficiency.

In the inpatient setting, the diagnosis was established: hemorrhagic vasculitis, activity grade II, mixed form (cutaneous-abdominal, renal), acute course. Recommended: anti-inflammatory immunosuppressive therapy with methylprednisolone 8 mg/day, followed by dose reduction by ¼ tablet every 7 days under control of blood sugar and blood pressure. Basic anti-inflammatory therapy (BAIT): sulfasalazine: 1st week – 500 mg, 1 tablet once a day after meals for 7 days. 2nd week – 500 mg, 1 tablet twice a day (morning and evening) after meals for 7 days. 3rd week – 500 mg, 1 tablet three times a day after meals, long-term, under rheumatologist supervision. Gastroprotective therapy: rabeprazole (Rabemac) 20 mg, 1 tablet once a day 30 minutes before meals during the period of steroid intake. If epigastric pain intensifies, use of antacid therapy was recommended.



Picture 2. Hemorrhagic rash on the legs during treatment.

Discussion

At present, there are no specific diagnostic serological laboratory tests or biomarkers for IgA vasculitis. However, serum IgA levels are elevated in 50–70% of patients with IgA vasculitis [10]. In this clinical case, the patient exhibited a rapidly progressive form of the disease, accompanied by hemorrhagic rashes on the upper and lower limbs, heaviness in the legs, nausea, vomiting, diarrhea up to 3 times a day lasting 4 days, transient cramping pain in the periumbilical region, and episodes of oliguria. Following anti-inflammatory and immunosuppressive therapy, a marked reduction was observed in cutaneous, abdominal, renal, intoxication, and asthenic syndromes. Considering the history of transient pain in the umbilical area, nausea, vomiting, diarrhea, and the primary cardiovascular disease, basic anti-inflammatory therapy included sulfasalazine 1,500 mg and methylprednisolone 8 mg in combination with antiplatelet, angioprotective therapy and antacid medications. In dynamics, normalization of ESR to 2 mm/h, leukocyte levels to 8.9/L, CRP to 2.7 mg/L was noted, and leukocyturia disappeared in urinalysis. Early corticosteroid therapy is an important aspect of treatment aimed at reducing inflammation and preventing complications. However, it is important to consider age-related features and complications of steroid therapy, especially in elderly patients, such as increased susceptibility to infections and femoral neck fractures [1].

Methylprednisolone was prescribed in the initial phase of the disease to control symptoms and reduce the activity of the inflammatory process, to achieve rapid regression of skin lesions, strictly under the supervision of a rheumatologist, with monitoring of blood pressure, heart rate, and clinical-laboratory parameters. The steroid therapy was short-term and at the lowest effective dose to reduce the likelihood of side effects. Sulfasalazine, a folic acid antagonist, acts by blocking endothelin receptors (ET-A and ET-B) on the smooth muscle lining of blood vessels. This action reduces vasoconstriction, improves oxygen delivery to tissues, and alleviates symptoms such as shortness of breath and fatigue. The drug demonstrates a rapid onset of therapeutic effect, is effective in patients regardless of age or disease duration and seroreactivity, does not interact with other medications, and can be used in combination with other antirheumatic drugs [4].

The clinical features of this case include male sex, age 86 years, cutaneous-abdominal and renal syndromes, and comorbid conditions in the medical history. The use of sulfasalazine, which is not part of the standard treatment regimen for Henoch-Schönlein purpura, made it possible to achieve a reduction in skin lesions and abdominal syndrome in a short time. According to our observations, sulfasalazine is a relatively safe and effective antirheumatic drug and may serve as an alternative therapeutic option for the treatment of IgA vasculitis.

Conclusion

IgA vasculitis (Henoch-Schönlein purpura) is traditionally considered a childhood disease; however, its manifestation in old age represents a clinically significant phenomenon with a number of features. In elderly patients, the course of the disease is often severe, with more pronounced visceral involvement and a higher risk of complications. Unlike children, in elderly individuals hemorrhagic vasculitis is less often accompanied by the typical triad of symptoms (purpura, arthralgia, abdominal syndrome) and often presents predominantly as nephropathy [5]. Moreover, advanced age is associated with a weakened immune response, multiple comorbidities, and polypharmacy, which can mask the clinical picture and influence therapeutic strategy. Immunosuppressive therapy (glucocorticosteroids and cytostatics), which is standard for severe IgA vasculitis, requires caution in elderly and senile patients due to the increased risk of infections, osteoporosis, hyperglycemia, and other side effects [14].

It should also be noted that in the elderly and senile population, IgA vasculitis may have a paraneoplastic nature associated with oncological diseases, especially in cases of late onset and refractory course. Therefore, in the diagnosis of IgA vasculitis in elderly patients, oncological vigilance and comprehensive evaluation are essential.

Thus, the presence of comorbidities, the high risk of drug interactions, and the increased mortality in cases of late diagnosis emphasize the clinical significance of timely recognition of IgA vasculitis in geriatric practice, requiring an interdisciplinary approach. Early diagnosis, assessment of organ involvement, individualized treatment selection, and monitoring of complications help achieve the best outcomes in the management of this category of patients.

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Preparation of the original project, writing, reviewing and editing, project administration *- Nogayeva Maral, Bakytova Aidana, Utepova Roza, Dayrabai Symbat.*

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