

ЛИТЕРАТУРА

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UNDIFFERENTIATED NECROTIZING ULCERATIVE VASCULITIS IN A PATIENT WITH CHRONIC KIDNEY DISEASE

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Relevance. Cutaneous necrotizing vasculitis (CNV) can present as palpable purpura, urticarial or erythematous macules, papules, nodules, blisters or ulcers. Cutaneous manifestations are common and skin is the organ which is frequently damaged, but other systemic involvement may also occur. The cause of CNV varies from idiopathic to variety of underlying conditions such as infections, food allergens, chronic inflammatory systemic disorders, drugs, chemicals, or malignant neoplasms. *S. aureus*, *E. coli* and *Streptococcus* are the common infectious agents which can trigger vasculitis. CNV is a segmental inflammation of venules, characterized by two main histologic patterns represented by the leukocytoclastic form [leukocytoclastic vasculitis (LcV)], and a lymphocytic form. Biopsy the skin lesions is necessary to confirm the diagnosis. CNV resolves spontaneously within 3-4 weeks in most of the patients without any systemic treatment. But, in case of severe, chronic and recurrent vasculitis systemic therapy is indicated and glucocorticoids serve as an effective treatment of choice.

Object. To propose a novel hypothesis that elevated levels of IgE and *Staphylococcus aureus* infection can be a precipitating factor for LcV, especially in patients with CKD.

Research methods. A 36-year-old male patient with Chronic Nephritic Syndrome and CKD 5 presents to the Grodno University Clinic with weakness and hemorrhagic rashes on the lower extremities. Since 2020, he was on haemodialysis. He also has a history of right-sided hydrothorax of an unspecified etiology and secondary right-sided

pneumonia with anemia of chronic disease for which he was treated with multiple antibiotics and was discharged after resolution of the symptoms. Few days after discharge, he returned with complaints of generalised weakness and difficulty breathing. The patient was re-admitted and a nares swab was withdrawn from which *Staphylococcus aureus* was isolated, seed rate – 10^6 . Coagulase-negative Methicillin Resistant – *Staphylococcus aureus* (MRSA) and *Candida* were isolated from the sputum. The patients condition deteriorated, complicated by bacteremia and bacterial sepsis. He was treated aggressively for sepsis. A few days after following the treatment for bacterial sepsis, the patient developed a high IgE antibody count, and ELISA showed elevated levels of IgE (1650.4 UI/ml, normal 150 to 1,000 UI/ml). The patient developed hemorrhagic rashes with central foci of necrosis on the legs. A provisional diagnosis of Hemorrhagic Necrotizing vasculitis (skin form) was made. ANCA and ANA tests were performed which turned out to be negative. Methylprednisolone 24mg/day under the cover of his near conclusion antibiotic therapy for sepsis was prescribed. The skin and subcutaneous tissue biopsy concluded the presence of leukocytoclastic vasculitis with characteristic histological features on skin flap biopsy. By this time the bacterial sepsis had already subsided and as there was an expansion of the necrotic zone, it was decided to increase the dose of glucocorticoids. He was put on pulse therapy- IV Methylprednisolone 1000 mg for 3 days.. Elevated IgE antibodies and IgE immune complex deposition was treated with FFP albumin plasmapheresis (one session) and anti-immunoglobulin sorption (2 sessions). The patient showed a rapid resolution of symptoms over a few days and was discharged.

Results and discussion. The patient was started on steroid pulse therapy followed by plasmapheresis for elevated IgE count, leading to rapid resolution of symptoms.

Conclusions. Elevated IgE and IgE immune complexes can be a triggering factor for CNV-LcV form. . High-dose corticosteroid therapy with plasmapheresis and anti-immunoglobulin sorption can be the mainstay treatment for aggressive manifestations of similar forms of necrotizing vasculitis.

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