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CASE REPORT

An Elderly Female Systemic Lupus Erythematosus Patient Presented with Severe Agranulocytosis as Initial Symptom

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ABSTRACT

Background: **Agranulocytosis refers to a severe reduction in neutrophils (with an absolute value less than 0.5 x 10^9/L) in peripheral blood, and a small portion of acquired agranulocytosis is caused by systemic lupus erythematosus (SLE). SLE is a chronic autoimmune disease involving multiple systems, mainly occurring in young women. Its clinical manifestations include polyarthralgia and arthritis, Raynaud's syndrome, rashes on the cheeks and other parts, pleurisy or pericarditis, involvement of the kidneys or central nervous system, and autoimmune cytopenia.**

Methods: **Blood routine and bone marrow routine tests were used to detect the cell proliferation condition in peripheral blood and bone marrow. Complement, immunoglobulin, etc. were detected by biochemical instrument, and antinuclear antibody (ANA), anti-dsDNA antibody, etc. were detected by immunofluorescence method.**

Results: **Blood routine indicated a WBC of 0.49 x 10^9/L with neutrophil count 0.09 x 10^9/L. Bone marrow routine showed bone marrow nucleated cells decreased, granulocyte granules increased and thickened. Biochemistry indicators: immunoglobulin G (IgG), immunoglobulin A (IgA), C-reactive protein (CRP), amylase (AMY), aspartate transaminase (AST), lactate dehydrogenase (LDH) were elevated, and complement C3, complement C4, albumin, eGFR were decreased. ANA.IgG positive (+), anti-dsDNA antibody positive (++).**

Conclusions: **This postmenopausal SLE patient presented with agranulocytosis as the initial symptom are rarely seen. Therefore, clinicians should enhance their understanding of SLE, conduct a comprehensive analysis of various clinical manifestations, and conduct relevant tests timely to reduce misdiagnosis.**

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KEYWORDS

systemic lupus erythematosus, agranulocytosis, initial symptom

INTRODUCTION

Agranulocytosis refers to a severe reduction in neutrophils (with an absolute value less than 0.5 x 10^9/L) in peripheral blood, which is divided into hereditary and acquired types. The causes of acquired agranulocytosis include drugs, chemical toxins, radiation, infections, etc., and a small portion is caused by autoimmune diseases (such as systemic lupus erythematosus, SLE). SLE is a chronic multisystem autoimmune disease characterized by the production of a large amount of auto-

antibodies and immune complexes, which in turn cause damage to tissues and organs [1,2]. With the continuous deepening of understanding of the disease, it has been found that SLE not only presents on the skin (butterfly-shaped erythema on both cheeks) but also causes damage to multiple systems such as the kidneys, heart, nervous system, blood system, joints, and oral cavity [3]. In addition, SLE can present as a chronic process and recur frequently. Exposure to sunlight, infection, surgery or pregnancy can all trigger its outbreak. In this article, we report a rare case of an elderly female patient with SLE who presented with agranulocytosis.

CASE PRESENTATION

The patient, a 76-year-old female, experienced fatigue and anorexia after uterine prolapse surgery one month ago (she reported that her blood count was normal at that time). She had no other discomforts and did not pay much attention. Two days ago, she felt significantly more fatigue and anorexia. A blood routine test at the local health center indicated pancytopenia. After treatment with cefmetazole for anti-infection and Leucogen tablets for increasing blood cells, there was no improvement. Now the patient developed fever with no chills or shivering, nausea or vomiting, abdominal pain or distension, and thus came to our hospital for treatment. Physical examination: clear consciousness, soft spirit, anemic appearance, no obvious enlargement of lymph nodes throughout the body, coarse breath sounds in both lungs, regular heart rhythm, no obvious edema in both lower extremities. Blood routine test: white blood cell count 0.49 x 10^9/L, neutrophil count 0.09 x 10^9/L, hemoglobin 83.0 g/L, platelet count 101.0 x 10^9/L; urine routine: urine protein +1, occult blood 2+. Plasma D-D: 3.38 mg/L; biochemistry: immunoglobulin G 19.52 g/L, immunoglobulin A 6.30 g/L, complement C3 0.27 g/L, complement C4 0.08 g/L, CRP 14.00 mg/L, amylase 311.9 U/L, AST 75.9 U/L, lactate dehydrogenase 307.0 U/L, albumin 24.1 g/L, eGFR 44.7 mL/minute. Anti-nuclear antibody: ANA. IgG positive (+), mainly nuclear homogeneous type, fluorescence intensity 2+, anti-double-stranded DNA antibody positive (++), anti-nucleosome antibody positive (++), anti-histone antibody weakly positive (±), U1-nRNP antibody positive (+). Anemia indicators: ferritin 2,707.16 ng/mL. Chest CT: suggests local inflammatory lesion in the lower lobe of the right lung. The cause of pancytopenia in this patient is unknown; a bone marrow biopsy was performed. Bone marrow routine: bone marrow nucleated cells decreased, granulocyte granules increased and thickened, megakaryocyte function deviated. It is recommended to conduct clinical examinations for viral factors, drugs, and immune-related factors.

Further inquiry about the patient's medical history revealed periodontitis recently, no obvious hair loss, no recurrent oral ulcers, no joint swelling and pain, no Raynaud's phenomenon in both hands, and a possibility of systemic lupus erythematosus was considered. Further tests indicated erythrocyte sedimentation rate (ESR): 111 mm/hour, ANA titer: anti-nuclear antibody 1:2,560 (+), anti-dsDNA quantification: 104.00 IU/mL, direct antiglobulin test (Coombs) positive. Based on the above examination results, SLE was considered, and hematological system was mainly involved. The patient had hematuria (4 points), pleurisy (2 points), low complement (2 points), elevated anti-dsDNA (2 points), decreased white blood cells (1 point), and a SLEDAI score 11 points, moderately active. Methylprednisolone sodium succinate was given for anti-inflammation, cefoperazone sodium sulbactam for anti-infection, mecobalamin and folic acid tablets for hematopoietic raw materials supplementation, recombinant human granulocyte stimulating factor injection for increasing white blood cells, hydroxychloroquine for regulating immunity, etc. After the condition stabilizes, cyclosporine soft capsules are added to regulate immunity. After treatment, re-examination of blood routine: white blood cell count 3.46 x 10^9/L, neutrophil count 2.75 x 10^9/L, hemoglobin 78.0 g/L, platelet count 225.0 x 10^9/L, and the patient's fatigue improved significantly, without fever, cough, sputum, chest tightness, or shortness of breath.

DISCUSSION

The etiology of SLE is not fully understood yet. It is believed to occur in individuals whose genes are activated by environmental stimuli and trigger inflammatory responses, and studies have shown that endocrine, infection, and immune abnormalities are also closely related to SLE [4,5]. Under the interaction of genetic and environmental factors, patients have a decrease in T lymphocytes, excessive proliferation of B lymphocytes, and production of large amounts of antibodies. These antibodies bind to antigens to form immune complexes and deposit them in various parts of the body, leading to tissue necrosis and acute or chronic inflammation with the participation of complement. In addition, gender hormones are involved in the occurrence of innate and adaptive immune responses, and estrogen has been confirmed to be the cause of susceptibility to SLE, which is also the reason why SLE is more common in young and middle-aged women [6]. Studies have shown that compared with premenopausal female patients, postmenopausal SLE patients present with significantly reduced manifestations such as rashes, renal damage, leukopenia, positive antinuclear antibodies, positive anti-double-stranded DNA, and hypocomplementemia [7]. However, the rare SLE patient reported in this case is newly diagnosed after menopause, and has obvious manifestations such as granulocytopenia and positive anti-double-stranded DNA.

Additionally, neutrophils are also involved in the pathogenesis of SLE. The activation of neutrophils can induce the release of proteases, tissue damage factors and

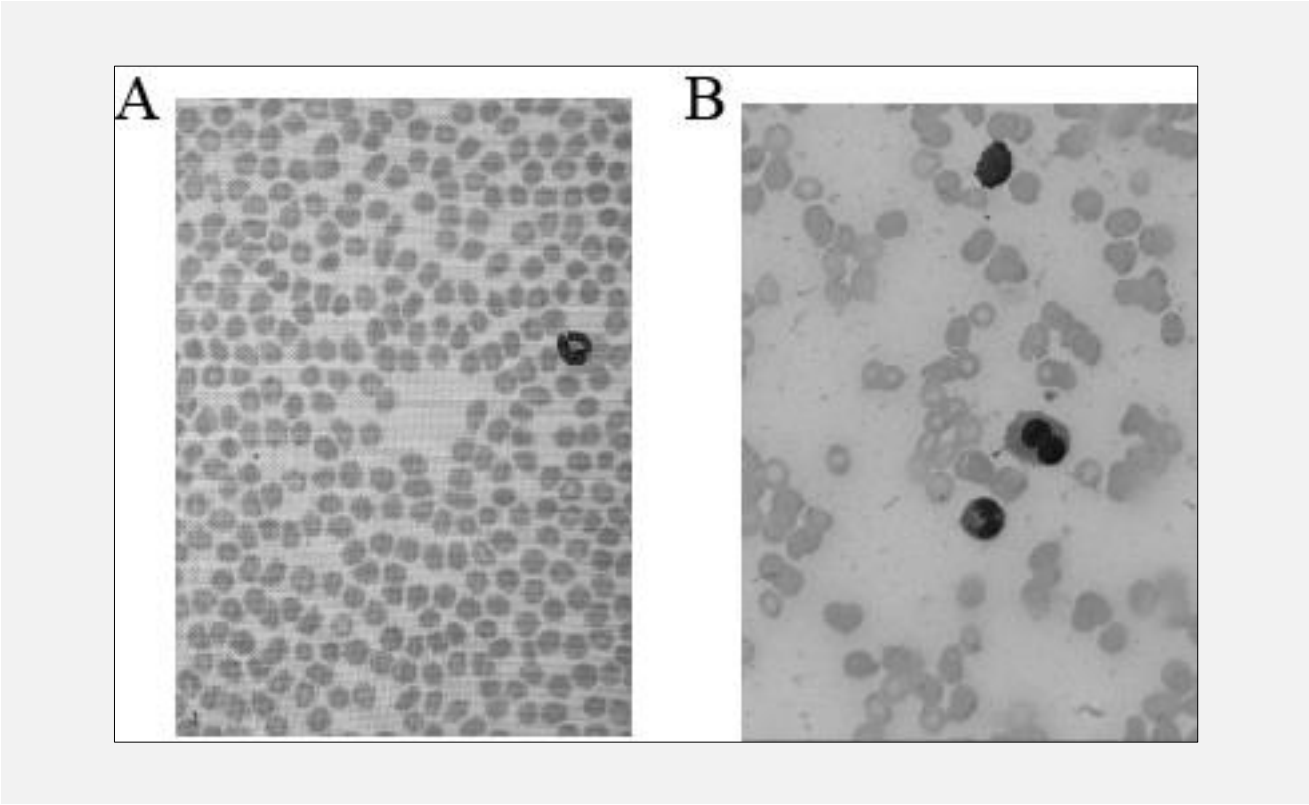

Figure 1. The examination results of the case in this paper.

A: Microscopic examination of blood routine examination. B: Microscopic examination of bone marrow routine.

reactive oxygen, leading to tissue damage in SLE. Moreover, active neutrophils also release neutrophil extracellular traps (NETs), which are an important component of the pathogenesis of SLE. Studies have shown that a large number of NETs have been found in the kidneys, skin and blood of SLE patients, and their presence is related to disease activity [8,9].

Research by Li et al. indicates that for SLE patients in China the most frequently affected are hematological system and kidneys [10]. The hematological manifestations of SLE are very extensive, including autoimmune hemolytic anemia, leukopenia, and thrombocytopenia. Leukopenia, especially neutropenia, is one of the common symptoms in SLE patients and of disease activity, and about 5% of patients may experience moderate to severe neutropenia [11]. The mechanisms include increased destruction of neutrophils by anti-neutrophil antibodies in peripheral blood and reduced neutrophil production in the bone marrow [12,13]; however, agranulocytosis is rare. Moreover, many studies have shown that periodontitis is associated with SLE [14,15], and chronic infection is an inducing and aggravating factor for autoimmune diseases. Hence the presence of periodontitis is very likely to be the main factor of inducing and increasing activity of SLE.

Most SLE patients will experience hematological abnormalities during the course of the disease, and some patients even present with hematological system abnormalities as the initial manifestation, which is very likely to be misdiagnosed as hematological diseases or other diseases. This case is a postmenopausal SLE patient with initial manifestations of agranulocytosis and periodontitis, which is rarely reported. Therefore, doctors should integrate multidisciplinary knowledge to diagnose and treat patients early to reduce misdiagnosis.

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