

## Rash, Heart Failure, Renal Insufficiency, and Eosinophilia—A Case of a Frail Elderly Patient Postprint

**Authors:** Su Linfa, Zhang Ning, Wang Qian, Zhang Lu, Yang Deyan, Xia Peng, Shao Chi, Zhu Minglei, Zhang Li, Junren Kang, Sun Xiaohong, Liu Xiaohong, Li Jun

**Date:** 2019-11-15T00:00:00+00:00

### Abstract

An 87-year-old male patient with the chief complaint of “rash for over one month, chest tightness accompanied by oliguria for 11 days” was admitted to the Department of Geriatrics at Peking Union Medical College Hospital on April 29, 2019.

### Full Text

### Preamble

**Title:** Rash, Heart Failure, Renal Insufficiency, and Eosinophilia—A Case of an Elderly Frail Patient

**Authors:** Su Linfa<sup>1</sup>, Zhang Ning<sup>2</sup>, Wang Qian<sup>2</sup>, Zhang Lu<sup>3</sup>, Yang Deyan, Xia Peng, Shao Chi, Zhu Minglei<sup>2</sup>, Zhang Li, Kang Junren, Sun Xiaohong<sup>2</sup>, Liu Xiaohong<sup>2</sup>, Li Jun

### Affiliations:

<sup>1</sup> Department of Internal Medicine, <sup>2</sup> Department of Geriatrics, <sup>3</sup> Department of Hematology, Department of Cardiology, Department of Nephrology, Department of Respiratory Medicine, Department of Immunology, Department of Clinical Nutrition, Department of Dermatology, Peking Union Medical College Hospital, Chinese Academy of Medical Sciences & Peking Union Medical College, Beijing 100730, China

**Corresponding Author:** Zhang Ning, Tel: 010-69154063, Email: 253571870@qq.com

**Keywords:** hypereosinophilia; elderly; frailty

## 1 Case Summary

### 1.1 Present Illness

The patient was an 87-year-old male who presented with a one-month history of rash and 11 days of chest tightness with oliguria, admitted to the Department of Geriatrics at Peking Union Medical College Hospital on April 29, 2019.

In March 2019, the patient developed dark red maculopapular rashes on both lower extremities without apparent cause, accompanied by dry skin, desquamation, and pruritus. The rash progressively spread from both lower extremities to both upper extremities, then to the anterior chest and back. He was diagnosed with “eczema” at a local hospital and treated with topical medications (details unknown), but the rash showed no significant improvement. From April 10, 2019, the patient self-administered traditional Chinese medicine for one week (details unknown), which also failed to alleviate symptoms.

On April 18, 2019, the patient developed chest tightness, dyspnea, and cough with small amounts of white sputum without apparent cause. His exercise tolerance decreased significantly, experiencing dyspnea after walking only 50 meters on flat ground. He also had reduced urine output (approximately 200 ml/day) and mild pitting edema of both lower extremities. On April 19, he visited the Emergency Department of Peking Union Medical College Hospital, where laboratory tests revealed: white blood cell (WBC) count  $9.04 \times 10^9$  /L, neutrophil count  $5.80 \times 10^9$  /L, eosinophil count  $1.88 \times 10^9$  /L, hemoglobin (Hb) 101 g/L, platelet (PLT) count  $201 \times 10^9$  /L; serum biochemistry showed potassium 6.8 mmol/L, blood urea nitrogen 23 mmol/L, serum creatinine 159  $\mu$ mol/L, albumin 29 g/L, cardiac troponin 0.061 g/L, and N-terminal pro-brain natriuretic peptide 10,146 pg/ml. After diuretic therapy and oral potassium-lowering resin, serum potassium decreased to 5.3 mmol/L. Further investigations revealed bilateral pleural effusion (right side deepest 10.5 cm, left side deepest 4.9 cm) on ultrasound localization, and echocardiography showed reduced left ventricular systolic function with left ventricular ejection fraction (LVEF) of 39%, myocardial disease, enlargement of both atria and right ventricle (left atrial transverse diameter 40 mm, right atrial transverse diameter 48 mm, right ventricular transverse diameter 41 mm), moderate mitral regurgitation, severe tricuspid regurgitation, estimated pulmonary artery systolic pressure of 54 mmHg, degenerative aortic valve disease, and mild aortic regurgitation. The patient continued to experience dyspnea on exertion but could lie flat at night, with urine output of 200-300 ml/day, and was admitted for further diagnosis and treatment.

Since onset, the patient’s mental status and sleep were fair, but food intake decreased to half of his usual amount. Urine output remained as described, bowel movements were normal, and recent weight showed no significant change. He denied dry mouth or eyes, oral ulcers, joint swelling or pain, and photosensitivity.

## 1.2 Past Medical History

Since 2018, the patient had intermittent mild pitting edema of both lower extremities that was lighter in the morning and heavier in the evening, worsening with activity but without other accompanying symptoms. The edema resolved after self-administration of “furosemide and spironolactone.” He had a history of sulfonamide allergy. He smoked for 30 years (average 5 cigarettes/day) but quit 20 years ago. He denied alcohol consumption. There was no recent travel, prolonged residence in other areas, or consumption of raw or undercooked fresh-water or seafood products.

## 1.3 Admission Physical Examination

On admission, temperature was 36.5°C, respiratory rate 20 breaths/min, heart rate 80 beats/min, blood pressure 115/68 mmHg (1 mmHg = 0.133 kPa), and oxygen saturation 93% on room air, improving to 96% on 2 L/min oxygen. He appeared emaciated with a weight of 48 kg and body mass index of 16.6 kg/m<sup>2</sup>, with markedly reduced muscle mass in all four limbs. Dark red maculopapular rashes were visible on the skin of all four limbs, anterior chest, and back, with local skin thickening, dryness, desquamation, scratch marks, and scabbing, but no ulceration or purulent discharge.

Cardiac rhythm was regular with normal heart sounds; no abnormal heart sounds or pericardial friction rubs were heard. Breath sounds were coarse in both lungs, diminished in both lower lung fields, with scattered moist rales audible at the lower lung borders. Hepatic and splenic borders were unclear on palpation. Shifting dullness was positive, and mild pitting edema was present in both lower extremities.

## 1.4 Comprehensive Geriatric Assessment (CGA)

Activities of Daily Living (ADL) score: 2; Instrumental Activities of Daily Living (IADL) score: 2; hand grip strength: 16 kg; Clinical Frailty Scale score: 6; Mini Nutritional Assessment score: 5 ; intermittent delirium.

## 1.5 Laboratory Tests

**Complete Blood Count:** WBC  $10.68 \times 10^9$  /L, neutrophils  $6.60 \times 10^9$  /L, eosinophils  $2.38 \times 10^9$  /L, platelets  $277 \times 10^9$  /L, hemoglobin 114 g/L, MCV 109.3 fL.

**Serum Biochemistry:** Potassium 4.5 mmol/L, albumin 34 g/L, urea 21.09 mmol/L, alanine aminotransferase 29 U/L, creatinine (enzymatic) 183 mol/L, uric acid 842 mol/L.

**Cardiac Markers:** Cardiac troponin 0.847 g/L, creatine kinase 195 U/L, CK-MB 6.3 g/L, myoglobin 255 g/L, brain natriuretic peptide >5000.00 ng/L.

**Coagulation Profile:** Prothrombin time 14.8 s, activated partial thromboplastin time 30.4 s, D-dimer 5.75 mg/L FEU.

**Urinalysis and Sediment:** Red blood cells (occult blood) 200 cells/ L, normal RBC morphology rate 60%, protein TRACE g/L. 24-hour urine total protein quantification: 0.11 g.

**Electrocardiogram:** Heart rate 93 bpm, sinus arrhythmia, flat T waves in limb leads.

**Autoantibodies:** Total immunoglobulin E >5000 KU/L; anti-extractable nuclear antigen (ENA) (4+7), anti-neutrophil cytoplasmic antibody profile: negative. Urinary 2-microglobulin, anti-glomerular basement membrane antibody-P, anti-glomerular basement membrane antibody: negative.

**Peripheral Blood Smear:** Eosinophils 18%, anisocytosis with irregular RBC morphology, macrocytes visible; other two cell lines normal. Peripheral blood flow cytometry and TCR gene rearrangement: negative.

**Bone Marrow Smear:** Myeloid series showed increased eosinophil proportion at 16.5%. Proportions and morphology of other stages were normal.

## 1.6 Imaging Studies

**Renal Ultrasound:** Diffuse bilateral renal parenchymal disease (right kidney 9.1×4.1×3.1 cm, left kidney 9.5×4.0×4.0 cm). **Carotid and Vertebral Artery Ultrasound:** Severe stenosis at the origin of the left internal carotid artery; possible stenosis of the right internal carotid artery and common carotid artery, suggesting possible occult ischemia. **Chest/Abdomen/Pelvis CT:** Diffuse pulmonary emphysema with multiple bullae [Figure 1: see original paper].

**Whole-body + Head PET/CT:** Diffusely increased metabolism in the skin, subcutaneous soft tissue, and mesenteric regions with maximum standardized uptake value (SUV) approximately 1.5; diffusely slightly increased density of subcutaneous tissue. Diffusely slightly increased metabolism in left and right atrial walls with maximum SUV approximately 4.7.

## 2 Multidisciplinary Discussion

### 2.1 Geriatrics Department

This elderly male patient presented with markedly elevated blood eosinophil counts and multi-system involvement from the early disease stage. Main manifestations included: (1) skin lesions with eczema-like rash at onset; (2) cardiac involvement manifesting as heart failure with significantly reduced LVEF; (3) renal involvement initially presenting as hyperkalemia and oliguria with markedly elevated serum creatinine and urea levels; and (4) multi-serosal effusions, primarily bilateral pleural effusion with small pelvic effusion, accompanied by hypoalbuminemia and significantly elevated inflammatory markers. According to

the 2017 Chinese Expert Consensus on Diagnosis and Treatment of Eosinophilia, this patient met the criteria for hypereosinophilic syndrome (HES) based on two peripheral blood examinations showing absolute eosinophil counts  $>1.5 \times 10^9 / L$  [1].

As an elderly patient with poor baseline organ function reserve (COPD, CKD, peripheral vascular disease), HES could further exacerbate pre-existing organ damage. Multidisciplinary consultation was requested to guide: (1) whether eosinophilia could explain the skin, cardiac, and renal involvement; and (2) next-step treatment strategies.

## 2.2 Radiology Department

Based on chest and abdominal CT imaging analysis, the patient had diffuse pulmonary emphysema with multiple bullae, which combined with his age and long-term smoking history suggested underlying COPD. Scattered cord-like opacities with calcification in both lungs were considered old tuberculous lesions. The images also revealed bilateral pleural effusion with partial atelectasis of both lower lungs, multiple lymph nodes in bilateral hilar, mediastinal, and inguinal regions with some appearing enlarged, cardiomegaly, and multiple calcifications in the aorta and its branches.

## 2.3 Dermatology Department

Eosinophils are multifunctional granulocytes distributed throughout various organs and skin, playing important roles in immune defense and tissue damage. Multiple factors can increase eosinophil production, recruitment, and survival, leading to their proliferation and infiltration into various tissues. Eosinophil infiltration commonly occurs in numerous dermatological conditions. Skin diseases related to eosinophil infiltration primarily present with polymorphic erythema, plaques, nodules, and are often accompanied by pruritus. Due to their nonspecific nature, these conditions are frequently overlooked or misdiagnosed clinically. Histopathology typically shows chronic inflammatory cell infiltration around superficial to mid-dermal blood vessels, predominantly lymphocytes and eosinophils. This patient's rash was consistent with these features, and combined with eosinophilia and organ involvement, HES could be diagnosed.

## 2.4 Cardiology Department

The patient's clinical presentation of chest tightness, decreased exercise tolerance, bilateral lower extremity edema, oliguria, and bilateral pleural effusion, combined with significantly elevated BNP and LVEF  $<40\%$ , was consistent with heart failure with reduced ejection fraction. Echocardiography showed left ventricular enlargement and myocardial disease. With no history of hypertension, myocardial infarction, congenital heart disease, or primary valvular disease, the echocardiographic findings were consistent with dilated cardiomyopathy. Additionally, with no familial cardiomyopathy or secondary myocardial damage from

infection or autoimmune disease, HES-related myocardial involvement was considered likely, which typically presents as restrictive cardiomyopathy but can manifest as dilated cardiomyopathy in end-stage or acute phases. However, the patient also had risk factors including advanced age, male sex, and smoking history, with previous echocardiography suggesting segmental wall motion abnormalities. Therefore, occult coronary artery disease could not be excluded, potentially leading to ventricular dilation due to inadequate reverse remodeling therapy, with the current episode possibly worsening chronic heart failure. HES-related myocardial involvement requires myocardial biopsy for definitive diagnosis, but this was not feasible in this patient, so current heart failure treatment was continued.

## 2.5 Nephrology Department

Regarding renal involvement, the patient's small bilateral kidneys suggested chronic renal insufficiency. Renal involvement in HES is rare, with reported cases primarily showing interstitial nephritis. This patient's renal injury required comprehensive consideration of multiple factors: reduced cardiac output (significantly decreased LVEF) and prerenal volume insufficiency (inadequate intake in early disease stage and currently relatively insufficient effective circulating volume). In summary, acute kidney injury was considered to involve multiple factors superimposed on chronic renal insufficiency. Current evidence for HES-related renal involvement was insufficient but could not be completely excluded. Renal biopsy would have limited significance for guiding primary disease treatment and carried extremely high risk with an unfavorable risk-benefit ratio, and was therefore not recommended. Fluid management should follow output-based intake principles with cautious diuresis permitted only if respiratory and circulatory status allowed.

## 2.6 Immunology Department

This patient was characterized by marked eosinophilia with multi-system involvement. Autoimmune markers were negative, with no history of asthma or neurological disease. Evidence was currently insufficient for eosinophilic granulomatosis with polyangiitis (EGPA). As an elderly patient with poor baseline organ function reserve, eosinophil elevation could not be excluded as a factor further exacerbating damage to vital organs including the heart and kidneys. HES is sensitive to corticosteroid therapy, and low-to-moderate dose application could be used to reduce peripheral eosinophil infiltration, with immunosuppressants (such as Tripterygium wilfordii) added if necessary to consolidate treatment.

## 2.7 Hematology Department

According to the 2017 HES diagnostic consensus, absolute eosinophil count  $>0.5 \times 10^9 / L$  is diagnosed as eosinophilia; absolute eosinophil count  $>1.5 \times 10^9 / L$  is classified as hypereosinophilia (HE), and if accompanied by organ damage,

it suggests HES [1]. HES can be categorized into several types: myeloid HES (MHES), lymphocytic variant HES (LHES), overlap HES, secondary HES, familial HES, and idiopathic HES. This patient completed relevant examinations, and all returned results showed no evidence of myeloid HE. Regarding organ assessment, considering the patient may have had underlying cardiac disease, HE could further worsen heart failure, requiring further exclusion of secondary factors. For HES treatment, prednisone is first-line therapy for most cases. Considering the patient's age, moderate-dose corticosteroids (0.5 mg/kg/day) could be used, with gradual tapering once effective. If eosinophil counts rebounded during prednisone tapering, imatinib or other second-line agents (such as interferon) could be considered based on the aforementioned examination results.

### 3 Management After First Multidisciplinary Discussion

After completing relevant examinations and comprehensive geriatric assessment, the patient was diagnosed with hypereosinophilic syndrome with skin involvement, possible cardiac involvement, and renal involvement not excluded. He also had comorbid chronic obstructive pulmonary disease, chronic renal insufficiency, and peripheral vascular disease. The geriatric assessment indicated frailty, sarcopenia, malnutrition, and fluctuating cognitive dysfunction (delirium).

From May 1, 2019, prednisone 25 mg once daily (0.5 mg/kg) was initiated, and from May 8, *Tripterygium wilfordii* 10 mg twice daily was added. Following treatment, eosinophil counts progressively decreased (EOS  $2.38 \rightarrow 0.01 \times 10^9/L$ ), and generalized rash gradually resolved after one week of treatment, leaving local post-inflammatory hyperpigmentation.

For heart failure management, fluid intake was restricted to 1000-1500 ml/day with intermittent diuretics and metoprolol 6.25 mg/day. Right-sided thoracentesis was performed intermittently for pleural effusion drainage, which significantly relieved chest tightness and dyspnea. Regarding renal function, serum creatinine, urea nitrogen, and potassium fluctuated after admission [Figure 2: see original paper], managed with intermittent diuretics and oral potassium-lowering resin. Nutritional support was provided with Nutrison 200 ml/day and Ensure 8-12 scoops/day.

On May 10, the patient developed nocturnal delirium characterized by agitation, irritability, and disorientation. Serum sodium was 152 mmol/L. Delirium was considered related to hyponatremia, and after appropriate increase in oral water intake, serum sodium decreased to 147 mmol/L with improved consciousness. However, intermittent delirium persisted with nocturnal agitation and daytime apathy, poor orientation to time and place. The patient's cough and sputum production worsened from May 19, and hypoxemia developed on May 20 (SpO<sub>2</sub>: 80% on room air). Chest CT revealed new bilateral ground-glass opacities [Figure 3: see original paper].

## 4 Second Multidisciplinary Discussion

### 4.1 Geriatrics Department

After admission, the patient received glucocorticoids and immunosuppressants, with eosinophil counts normalized and rash resolved. Creatinine and urea had temporarily decreased. However, the new onset of hypoxemia and repeat chest CT showing new bilateral diffuse ground-glass opacities with increased pleural effusion raised suspicion for secondary *Pneumocystis jirovecii* infection. Additionally, the comprehensive geriatric assessment indicated frailty, sarcopenia, malnutrition, and fluctuating cognitive dysfunction (delirium), all of which are indicators of poor prognosis in elderly patients. Frailty is an important geriatric syndrome representing the decline in physiological reserve across neuromuscular, metabolic, and immune systems, reducing the ability of elderly individuals to withstand stress [2]. Multidisciplinary consultation was requested to guide: (1) treatment strategy for pulmonary infection; and (2) individualized therapy for elderly frail patients.

### 4.2 Pulmonology Department

Common pulmonary involvement in HES presents as eosinophilic pneumonia, with imaging showing migratory infiltrates, consolidative or ground-glass lesions distributed along bronchi or subpleurally, essentially caused by eosinophil infiltration into alveolar spaces. This patient lacked typical HES pulmonary infiltration manifestations, instead showing old tuberculous calcified lesions in both upper lungs and panacinar emphysema, indicating poor respiratory reserve. The recent development of hypoxemia in an elderly, immunosuppressed patient warranted vigilance for opportunistic infection. Based on the imaging appearance of new bilateral ground-glass opacities, *Pneumocystis jirovecii* pneumonia was considered highly likely, with overall poor prognosis. Given the patient's sulfonamide allergy preventing desensitization during corticosteroid therapy, empirical second-line anti-PCP treatment (clindamycin and caspofungin) was recommended while maintaining current corticosteroid dose, with adjustments based on microbiological results.

### 4.3 Cardiology Department

Regarding cardiac management, evidence supports the “golden triangle” of heart failure therapy including beta-blockers to inhibit sympathetic activation and ACEI/ARB plus aldosterone receptor antagonists to inhibit the RAAS system. However, this patient's significant renal impairment limited ACEI/ARB use, and unstable blood pressure prevented beta-blocker up-titration, making effective heart failure control difficult with poor overall prognosis.

### 4.4 Nephrology Department

The patient's recent acute kidney injury was related to prerenal hypoperfusion. Emergency dialysis indications needed to be monitored, such as markedly ele-

vated urea nitrogen with related symptoms, electrolyte/acid-base abnormalities unresponsive to medical correction, or refractory heart failure. Dialysis modality selection should consider peritoneal dialysis or CRRT, with the decision for renal replacement therapy requiring careful weighing of long-term benefits and expected survival time through thorough communication with the patient and family.

#### 4.5 Clinical Nutrition Department

As an elderly frail patient with malnutrition, nutritional status correction was essential during disease treatment. During the initial phase of nutritional support, vigilance for refeeding syndrome was necessary, first correcting electrolyte imbalances before gradually supplementing calories, starting with small doses and progressing incrementally [3].

### 5 Treatment Course and Outcome

From May 21, empirical anti-infective therapy with clindamycin 0.6 g every 12 hours and caspofungin 70 mg (loading dose) → 50 mg once daily was initiated. Sputum microbiology returned positive for PCP DNA (+). On May 23, the patient developed type II respiratory failure, hypotension, and anuria, requiring upgraded oxygen support and intravenous norepinephrine infusion to maintain blood pressure. On May 24, he developed coma with progressive decline in blood pressure and oxygen saturation. The family refused all resuscitative measures including mechanical ventilation and cardiopulmonary resuscitation. The patient died on May 24.

### 6 Final Diagnosis

HES (idiopathic type, skin involvement, probable myocardial involvement, renal involvement not excluded), *Pneumocystis jirovecii* pneumonia, septic shock.

### 7 Discussion

This elderly male patient presented with rash as the initial manifestation, subsequently developing cardiac and renal dysfunction with persistently elevated peripheral blood eosinophils, responding to corticosteroid therapy, suggesting possible HES with multi-system involvement. Pardanani et al. [4] conducted a retrospective study of HES patients (n=98, median follow-up 70 months) and identified independent predictors of poor prognosis: age >60 years (HR=8.1, P=0.0006), Hb<10 g/L (HR=5.5, P=0.01), cardiac involvement (HR=3.9, P=0.03), and hepatosplenomegaly (HR=12.1, P=0.004). This patient had at least two adverse prognostic factors: advanced age and cardiac involvement. The Mayo Clinic [5] followed 247 patients with idiopathic HES between 1990 and 2008, among whom 23 died during follow-up (15 with known causes) at a mean age of 60 years (range 27-85). Primary causes of death were cardiac

involvement, secondary infection, malignancy, and thromboembolism. This patient's HES organ involvement mainly manifested as heart failure and acute kidney injury.

Reviewing previous clinical cases of elderly HES patients, Navarro et al. [6] reported a case of idiopathic HES with acute kidney injury (73-year-old female) in 2009, where renal biopsy showed tubular atrophy with massive eosinophil infiltration in the renal interstitium, confirming eosinophilic renal involvement. After corticosteroid treatment (starting at 1 mg/kg/day and tapering to 15 mg/day), renal function improved significantly (creatinine decreased from 659 to 262 mol/L) after 3 months. Another case of HES with cardiac involvement [7] (83-year-old male) with idiopathic HES (peripheral blood eosinophils 26.7%) presented primarily with dyspnea and heart failure symptoms. Echocardiography showed LVEF of 35%, and myocardial biopsy revealed massive eosinophil infiltration. After corticosteroid therapy (1 mg/kg/day), peripheral blood eosinophils decreased by >50% within 1 week, and LVEF improved to 45% after 1 month.

Comprehensive geriatric assessment is essential in elderly patients. Frailty is a common diagnosis in geriatrics, representing a nonspecific state of decreased physiological reserve leading to increased vulnerability and reduced stress resistance, distinguishing physiological from biological age in elderly individuals. Frail elderly patients can develop a series of adverse clinical events from minor external stressors. Assessment typically uses scales including Activities of Daily Living (ADL), Instrumental Activities of Daily Living (IADL), and the Clinical Frailty Scale (CFS) to comprehensively evaluate frailty severity [8]. This patient had a CFS score of 5 (mild frailty with relatively preserved ADL but impaired IADL) before onset, which progressed to 6 (moderate frailty with impaired ADL and IADL) after admission, indicating poor prognosis. Nutritional status is fundamental to organ function reserve in elderly patients and an important factor affecting long-term quality of life, typically assessed using the Mini Nutritional Assessment-Short Form (MNA-SF), with <7 points diagnosing malnutrition [9]. This patient scored 5 on MNA-SF, indicating poor nutritional status. Delirium is an acute cognitive disturbance characterized by impaired arousal and cognition, with common symptoms including decreased consciousness, agitation, visual hallucinations, disorganized thinking, and orientation and memory deficits. Its acute onset and fluctuating course are important features, commonly seen in geriatrics and intensive care units [10]. This patient exhibited delirium episodes sensitive to electrolyte disturbances or volume abnormalities, accelerating cognitive deterioration and disability in a patient with dementia, serving as an alarming symptom of poor prognosis.

Geriatric syndromes influence treatment decisions. Clinical decision-making for elderly patients requires both adherence to disease-specific guidelines and assessment of overall functional status and life expectancy for comprehensive decision-making. For "young-old" patients with >10 years expected survival and few comorbidities, treatment primarily follows disease-specific guidelines. For frail patients with multiple comorbidities and shorter expected survival, treatment

should prioritize patient preferences, comorbidity management principles, and goal-directed care. Finally, for end-stage disease patients with <6 months expected survival, treatment should focus primarily on symptom management to alleviate physical symptoms and improve quality of life.

This patient was elderly with poor baseline multi-organ function reserve. Hypereosinophilia caused acute exacerbation of chronic cardiac and renal dysfunction, with HES multi-system involvement not excluded. After corticosteroid and immunosuppressant therapy, peripheral blood eosinophils normalized and rash resolved. However, pulmonary PCP infection developed during treatment. With sulfonamide allergy, second-line anti-PCP therapy was ineffective, infection could not be controlled, and the patient ultimately died of septic shock. This case demonstrates that even low-to-moderate dose corticosteroids can cause severe complications in elderly frail patients, suggesting that individualized treatment plans should be formulated with greater caution for frail, elderly patients in future clinical practice.

## 8 Expert Commentary

**Associate Professor Zhu Minglei, Department of Geriatrics, Peking Union Medical College Hospital**

Diseases in elderly patients often involve multiple contributing factors. Treatment selection for elderly patients is challenging, primarily based on three aspects: First, prognosis assessment—this patient's prolonged bed rest, frailty, malnutrition, vital organ involvement, and numerous treatment complications all indicated poor prognosis. Second, frailty—elderly patients have reduced adaptive capacity and compensatory ability, with poor treatment tolerance, requiring dose adjustment and individualized protocols. Additionally, vigilance for secondary adverse events during hospitalization such as falls, nosocomial infections, and delirium is essential. Third, communication—despite meticulous clinical planning, elderly patients' conditions are variable and treatment may not be completely reversible. Thorough communication with patients and families regarding treatment preferences and expectations, with comprehensive assessment of long-term benefits, is necessary to maximize clinical benefit.

In summary, for elderly patients with poor disease prognosis, clinicians should recognize that frailty and delirium are also risk factors for adverse outcomes. Elderly patients have weak compensatory capacity, poor tolerance to treatment adverse effects, and are prone to poor outcomes. Clinical decision-making must weigh treatment complications, long-term benefits, expected survival time, and other factors comprehensively.

## References

[1] Leukemia and Lymphoma Group, Chinese Society of Hematology, Chinese Medical Association. Chinese expert consensus on diagnosis and treatment of

eosinophilia (2017 edition) [J]. Chinese Journal of Hematology, 2017, 38(07): 561-565.

[2] Kurpas D, Gwyther H, Szwamel K, et al. Patient-centred access to health care: a framework analysis of the care interface for frail older adults [J]. BMC Geriatr, 2018, 18(1): 273.

[3] Zhu Changzhen, Li Kang, Yu Jianchun, et al. Refeeding syndrome: a case report [J]. Journal of Peking Union Medical College Hospital, 2015, 03: 234-236.

[4] Pardanani A, Lasho T, Wassie E, et al. Predictors of survival in WHO-defined hypereosinophilic syndrome and idiopathic hypereosinophilia [J]. Leukemia, 2016, 30(9): 1924-1926.

[5] Podjasek JC, Butterfield JH. Mortality in hypereosinophilic syndrome: 19 years of experience at Mayo Clinic with a review of the literature [J]. Leuk Res, 2013, 37(4): 392-395.

[6] Navarro I, Torras J, Gomà M, Cruzado JM, Grinyó JM. Renal involvement as the first manifestation of hypereosinophilic syndrome: a case report [J]. NDT Plus, 2009, 2(5): 379-381.

[7] Khalid F, Holguin F. Idiopathic Hypereosinophilic Syndrome in an Elderly Female: A Case Report [J]. Am J Case Rep, 2019.

[8] Rockwood K. A global clinical measure of fitness and frailty in elderly people [J]. Canadian Medical Association Journal, 2005, 173(5): 489-495.

[9] Rubenstein LZ, Harker JO, Salvà A, Guigoz Y, Vellas B. Screening for undernutrition in geriatric practice: developing the short-form mini-nutritional assessment (MNA-SF) [J]. J Gerontol A Biol Sci Med Sci, 2001, 56(6): M366-M372.

[10] Marcantonio ER. Delirium in Hospitalized Older Adults [J]. N Engl J Med, 2017, 377(15): 1456-1466.

*Note: Figure translations are in progress. See original paper for figures.*

*Source: ChinaXiv – Machine translation. Verify with original.*