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Case Report

Severe Microcytic Anaemia and Acute Respiratory Distress Syndrome in a Rheumatoid Arthritis Patient on Long-Term Corticosteroids: A Case Report

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ABSTRACT

Rheumatoid Arthritis (RA) is a chronic autoimmune disorder associated with multiple extra-articular manifestations, including hematological abnormalities and pulmonary complications. Anaemia in RA is often multifactorial, and prolonged corticosteroid therapy further increases the risk of infections and systemic complications. A 38-year-old female with a one-year history of rheumatoid arthritis on long-term deflazacort therapy presented with generalized weakness, fatigue, and progressive dyspnoea. Laboratory investigations revealed severe microcytic hypochromic anaemia with a haemoglobin level of 3.6 g/dL and low reticulocyte count. During hospitalization, the patient developed worsening respiratory symptoms and was diagnosed with bilateral lower lobe pneumonia progressing to Acute Respiratory Distress Syndrome (ARDS). Management included packed red blood cell transfusion, broad-spectrum antibiotics, oxygen therapy, and supportive care, resulting in gradual clinical improvement with haemoglobin rising to 8.9 g/dL. This case highlights the complex interplay among chronic inflammation, severe anaemia, and corticosteroid-induced immunosuppression in patients with rheumatoid arthritis. Early identification of anaemia and prompt management of infections are essential to prevent life-threatening complications such as ARDS. Multidisciplinary care plays a crucial role in improving clinical outcomes.

Keywords: Acute Respiratory Distress Syndrome, Corticosteroid Treatment, Extra-articular Manifestations, Microcytic Anaemia, Pulmonary Infection, Rheumatoid Arthritis.

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INTRODUCTION

Rheumatoid Arthritis (RA) is a chronic systemic autoimmune disorder marked by ongoing synovial inflammation, symmetric polyarthritis, and progressive joint damage, frequently accompanied by various extra-articular manifestations. Among these manifestations, hematologic issues such as anemia are commonly observed and have a significant effect on patient morbidity and overall quality of life. Anemia in RA is generally multifactorial, with Anemia of Chronic Disease (ACD) being the most prevalent type, driven by systemic inflammation that disrupts erythropoiesis and iron metabolism. The occurrence of anemia in RA populations is notably high, with numerous studies indicating rates between approximately 40% and over 60% of affected individuals, correlating with disease activity and increased inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein (Wilson *et al*., 2004; Shah *et al*., 2024).

Pathophysiologically, the chronic inflammation associated with RA results in heightened production of pro-inflammatory cytokines, including Interleukin-6 (IL-6), which stimulates the liver to produce the peptide hepcidin. Hepcidin restricts iron release from macrophages and intestinal absorption, leading to reduced iron availability for erythropoiesis, even when iron stores are normal or elevated, a characteristic feature of ACD. This condition is often exacerbated in RA by concurrent iron deficiency and by cytokine inhibition of erythropoietin production, further reducing red blood cell production (Nemeth and Ganz, 2022). Clinically, anemia leads to fatigue, decreased exercise capacity, and a lower quality of life, and is associated with increased disease severity and worse functional outcomes in RA patients (Weiss and Goodnough, 2005).

In addition to the inflammatory mechanisms associated with diseases, therapeutic approaches in Rheumatoid Arthritis (RA), especially prolonged corticosteroid treatment, can affect both systemic and organ-specific functions. Glucocorticoids are a cornerstone in the management of RA across various clinical scenarios due to their strong anti-inflammatory properties and

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their capacity to deliver swift symptom relief, particularly during exacerbations of the disease (Saag *et al*., 1994). Nevertheless, even the use of low-dose corticosteroids over an extended period is linked to a heightened risk of various adverse effects, such as metabolic disturbances, immunosuppression, and an increased vulnerability to infections. Retrospective cohort studies involving RA patients undergoing chronic low-dose corticosteroid therapy have revealed a greater occurrence of adverse events, including severe infections, which may lead to acute respiratory complications (Curtis *et al*., 2006).

The respiratory manifestations of RA can range from chronic interstitial lung disease to acute pulmonary issues. Although rheumatoid lung disease affects only a small percentage of patients, acute respiratory failure may occur in the setting of significant systemic inflammation, infection, or treatment-related immunosuppression. Acute Respiratory Distress Syndrome (ARDS) has been documented in autoimmune disorders, including RA, where immune dysregulation and external factors such as infections can trigger a rapid decline in respiratory function (Poli *et al*., 2024). These respiratory issues, particularly when compounded by severe anaemia, present considerable diagnostic and therapeutic challenges, as anaemia itself can lead to diminished oxygen delivery and heightened cardiopulmonary strain.

The interaction between chronic inflammation, medication-induced immunomodulation, and hematological irregularities highlights the intricate nature of Rheumatoid Arthritis (RA) that extends beyond mere joint pathology (Swaak, 2006). In cases of severe microcytic anaemia associated with RA, both chronic disease mechanisms and external factors such as iron deficiency or the effects of medications can play a significant role, potentially worsening systemic symptoms. While corticosteroids are effective in reducing inflammation, their prolonged use may impair the body's defenses and increase the risk of infections, particularly affecting the pulmonary system, which could result in acute respiratory distress (Dixon *et al*., 2011; Curtis *et al*., 2006).

Despite extensive research on anaemia within RA populations, there is a scarcity of comprehensive reports detailing its coexistence with acute respiratory complications in patients undergoing long-term corticosteroid treatment, particularly when the anaemia is of a mixed or multifactorial nature. This situation is clinically significant, as the combined impact of severe anaemia and respiratory failure can greatly affect patient outcomes, treatment plans, and hospitalization patterns in RA. By documenting and analyzing such cases, healthcare providers can gain a deeper understanding of the systemic complexities of RA and enhance multidisciplinary strategies tailored to high-risk cases.

CASE REPORT

A 38-year-old female patient was admitted to the hospital, presenting with complaints of generalized weakness persisting for five days and progressive shortness of breath classified as Grade II for one month. Additionally, she reported experiencing oligomenorrhoea, with menstrual cycles occurring every three to six months. Associated symptoms included fatigue, cough, nausea, occasional palpitations, and instances of syncope. There was no reported history of fever, orthopnoea, paroxysmal nocturnal dyspnoea, pedal oedema, vomiting, diarrhoea, or previous similar episodes. The chronological sequence of events is summarized in Table 1.

The patient had a known diagnosis of rheumatoid arthritis for the past year and had been undergoing long-term oral corticosteroid therapy with deflazacort at a dosage of 6 mg once daily. Furthermore, she had been receiving oral iron supplementation (Orofer XT) for the last six months. There was no history of prior hospitalizations for similar complaints, no use of inhalers, and no known drug allergies. It was also noted that she had hypertension, although a comprehensive history of antihypertensive therapy was not documented.

Upon physical examination at the time of admission, the patient was found to be conscious, alert, and oriented. Her vital signs were stable, with a blood pressure reading of 110/70 mmHg, a pulse rate of 98 beats per minute, a respiratory rate of 18 breaths per minute, a temperature of 99°F, and an oxygen saturation of 97% on room air. The general physical examination indicated marked pallor, with no signs of cyanosis, clubbing, icterus, lymphadenopathy, oedema, or dehydration. The cardiovascular examination revealed normal heart sounds without any murmurs. The respiratory examination showed bilateral normal vesicular breath sounds, accompanied by the presence of rhonchi and crepitus. The abdominal examination was unremarkable, and the neurological assessment indicated normal higher mental functions, reflected by a Glasgow Coma Scale score of 15/15.

Initial laboratory assessments indicated the presence of severe anaemia, with a critically low haemoglobin level of 3.6 g/dL. The red blood cell indices were consistent with microcytic hypochromic anaemia, characterized by diminished mean corpuscular volume and mean corpuscular hemoglobin values. The reticulocyte count was low (0.4%), indicating reduced marrow response. The white blood cell count was elevated, suggesting an underlying inflammatory or infectious process, while platelet counts were initially found to be increased. Inflammatory markers were significantly elevated, with the erythrocyte sedimentation rate reaching 113 mm/hr and C-reactive protein levels also elevated. Iron studies indicated that ferritin values fell within the normal reference range; however, an iron deficit of approximately 1540 mg was calculated, implying functional iron deficiency likely due to chronic inflammation. A stool examination for occult blood

returned negative, effectively ruling out overt gastrointestinal bleeding. The laboratory findings are summarized in Table 2.

Radiological assessment via chest X-ray revealed patchy opacities in the left mid zone, indicative of an infectious cause. An abdominal ultrasonography demonstrated mild hepatomegaly with altered echotexture, while the kidneys appeared normal apart from small cortical cysts in the right kidney. Liver function tests indicated a mild elevation of transaminases, whereas renal function parameters remained within normal limits.

Based on clinical assessments and investigations, the patient was tentatively diagnosed with severe anemia and a lower respiratory tract infection. She was started on packed red blood cell transfusions, intravenous fluids, oral iron supplements, and broad-spectrum antibiotics. Despite the transfusion, the patient experienced an exacerbation of shortness of breath and a dry cough, which led to a consultation with pulmonology. Further assessments, including arterial blood gas analysis, revealed hypoxemia accompanied by developing metabolic acidosis. The pulmonology team diagnosed the patient with Acute Respiratory Distress Syndrome (ARDS) alongside bilateral lower lobe pneumonia, in addition to severe microcytic hypochromic anemia and pre-existing rheumatoid arthritis. The treatment regimen is detailed in Table 3.

The patient was treated with oxygen therapy and intravenous antibiotics, including ceftriaxone, sulbactam, levofloxacin, and doxycycline, as well as supportive measures such as proton pump inhibitors and bronchodilators. Continuous monitoring of vital signs, oxygen saturation, and fluid intake-output was conducted. Throughout the hospitalization, the patient exhibited gradual clinical improvement. Hemoglobin levels rose to 8.6-8.9 g/dL following the transfusion, respiratory parameters stabilized, and the need for oxygen decreased.

Upon discharge, the patient exhibited hemodynamic stability, along with enhanced oxygen saturation and diminished respiratory distress. She was provided with oral antibiotics, iron supplements, and supportive medications, and was instructed to have regular follow-ups in the outpatient department for ongoing assessment and management of anaemia and rheumatoid arthritis.

Table 1: Timeline of Clinical Events.

Time Point	Clinical Events
1 year before admission	Diagnosed with rheumatoid arthritis; started on oral deflazacort 6 mg once daily.
6 months prior	Initiated oral iron supplementation (Orofer XT) for anaemia.
1 month before admission	Developed progressive shortness of breath (Grade II dyspnoea).
5 days before admission	Onset of generalized weakness and fatigue.
Day 1 (20 January 2025)	Admitted with severe anaemia (Hb: 3.6 g/dL); chest X-ray showed patchy opacities; started on PRBC transfusion and antibiotics.
Day 2-3	Worsening respiratory symptoms; diagnosed with bilateral pneumonia progressing to ARDS.
Day 3-5	Managed with oxygen therapy, intravenous antibiotics, and supportive care; haemoglobin improved to ~8.9 g/dL.
Discharge day	Clinical improvement with stable vitals, improved oxygen saturation; discharged with medications and follow-up advice.

Table 2: Laboratory Parameters during Hospitalization.

Parameter	Day 1	Day 3	Normal Range
Hemoglobin	3.6 g/dL	8.9 g/dL	12-15 g/dL
RBC Count	2.92 million	3.25 million	3.8-4.8 million
MCV	70.9 fL	74.4 fL	83-101 fL
MCH	23.9 pg	20.8 pg	27-32 pg
MCHC	31.5 g/dL	29.5 g/dL	31.5-34.5 g/dL
ESR	113 mm/hr	64 mm/hr	0-13 mm/hr
Reticulocyte Count	0.4%	-	0.5-2%
Ferritin	61.3 ng/mL	-	11-306 ng/mL

Table 3: Drug Therapy During Hospitalization.

Drug Name	Dose	Route	Frequency	Indication
Deflazacort	6 mg	Oral	Once daily	Management of rheumatoid arthritis (pre-existing therapy).
Orofer XT (Iron supplement)	-	Oral	Once daily	Treatment of iron deficiency anaemia (pre-existing therapy).
Packed Red Blood Cells (PRBC)	As required	Intravenous	As per protocol	Correction of severe anaemia.
Ceftriaxone + Sulbactam	1.5 g	Intravenous	Twice daily	Bacterial infection (pneumonia).
Levofloxacin	500 mg	Intravenous	Once daily	Broad-spectrum antibiotic for respiratory infection.
Doxycycline	100 mg	Oral/Intravenous	Twice daily	Coverage for atypical pathogens.
Pantoprazole	40 mg	Intravenous/Oral	Once daily	Gastric protection.
Bronchodilators	-	Inhalational	As prescribed	Relief of respiratory distress.
Oxygen therapy	-	Inhalational	Continuous/as needed	Management of hypoxaemia and ARDS.

DISCUSSION

Rheumatoid Arthritis (RA) is a chronic systemic inflammatory disorder frequently associated with extra-articular manifestations, including hematological abnormalities and pulmonary complications. Among these, anaemia is one of the most common comorbidities and is typically multifactorial in origin, involving chronic inflammation, impaired iron metabolism, and reduced erythropoietic response.

In the present case, the patient exhibited severe microcytic hypochromic anaemia with a markedly low haemoglobin level (3.6 g/dL) and reduced reticulocyte count, indicating inadequate bone marrow response. Although serum ferritin levels were within the normal range, the presence of functional iron deficiency due to inflammation is strongly suggested. Chronic inflammatory states in RA are known to elevate cytokines such as interleukin-6, which increase hepcidin production and subsequently impair iron availability for erythropoiesis. Similar findings have been reported by Wilson *et al*., who demonstrated a high prevalence of anaemia in RA patients, particularly associated with inflammatory activity and altered iron homeostasis.

The severity of anaemia in this patient likely played a significant role in clinical deterioration. Reduced oxygen-carrying capacity may have contributed to tissue hypoxia and increased cardiopulmonary stress, thereby predisposing the patient to rapid decompensation during the acute infectious episode. Previous studies, such as those by Weiss and Goodnough, have emphasized that anaemia of chronic disease significantly impairs oxygen delivery and is associated with poor clinical outcomes in inflammatory disorders.

Another critical aspect of this case is the development of Acute Respiratory Distress Syndrome (ARDS) secondary to bilateral pneumonia. Patients with RA receiving long-term corticosteroid therapy are at an increased risk of infections due to immunosuppression. In this case, prolonged use of deflazacort may have contributed to increased susceptibility to lower respiratory tract infection, which progressed to severe pneumonia and subsequently ARDS. This observation is consistent with findings from Dixon *et al*., who reported an increased risk of serious infections in RA patients receiving long-term glucocorticoid therapy.

The coexistence of severe anaemia and acute respiratory compromise presents a complex clinical scenario. Anaemia may exacerbate hypoxaemia by limiting oxygen delivery, while pulmonary infection further impairs gas exchange, creating a synergistic effect that can precipitate respiratory failure. This interaction underscores the need for prompt and aggressive management, including blood transfusion, antimicrobial therapy, and supportive respiratory care.

The patient's favorable outcome can be attributed to early intervention, including timely blood transfusion and appropriate antimicrobial therapy. This case emphasizes the importance of a multidisciplinary approach involving physicians, pulmonologists, and clinical pharmacists to optimize patient management, especially in individuals with chronic inflammatory diseases receiving long-term immunosuppressive therapy.

Overall, this case highlights the need for routine monitoring of hematological parameters, cautious use of corticosteroids, and early identification of infectious complications in RA patients. Recognizing the interplay between anaemia, immunosuppression, and respiratory pathology is essential for

preventing life-threatening complications and improving clinical outcomes.

CONCLUSION

This case highlights the complex interplay between chronic inflammation, severe microcytic anaemia, and corticosteroid-induced immunosuppression in patients with rheumatoid arthritis. The presence of profound anaemia significantly contributed to clinical deterioration and increased susceptibility to acute respiratory complications such as ARDS. Early recognition of anaemia, careful monitoring of patients on long-term corticosteroid therapy, and prompt management of infections are essential to prevent life-threatening outcomes. A multidisciplinary approach plays a crucial role in optimizing patient care and improving clinical prognosis in such high-risk cases.

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ABBREVIATIONS

RA: Rheumatoid arthritis; **ARDS:** Acute respiratory distress syndrome; **ACD:** Anaemia of chronic disease; **IL-6:** Interleukin-6; **Hb:** Haemoglobin; PRBC**:** Packed red blood cells; **RBC:** Red blood cell; **MCV:** Mean corpuscular volume; **MCH:** Mean corpuscular haemoglobin; **MCHC:** Mean corpuscular haemoglobin concentration; **ESR:** Erythrocyte sedimentation rate.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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DECLARATION OF PATIENT CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical information. The patient has been informed that personal identifiers will not be disclosed and that all efforts have been made to ensure confidentiality. The authors have ensured that the patient's identity has been adequately protected in accordance with ethical standards and journal guidelines.

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