

A Case of Dry Gangrene in an Autoimmune Patient: Diagnostic and Therapeutic Challenges

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ABSTRACT

Dry gangrene is a severe complication of vascular insufficiency, often associated with Peripheral Arterial Disease (PAD), diabetes, or autoimmune conditions like Rheumatoid Arthritis (RA). This case report presents a patient with RA and PAD who developed dry gangrene, highlighting the diagnostic and therapeutic challenges in such cases. A 42-year-old female patient presented with fever, joint pain, bluish discoloration, and numbness in the extremities. The patient had a history of hypothyroidism and was previously treated for lower limb thrombosis. Physical examination revealed ischemic changes in the fingers and toes. Laboratory investigations showed elevated rheumatoid factor (74.4 IU/mL), high Anti-Cyclic Citrullinated Peptide (Anti-CCP) levels (>200 RU/mL), and positive Antinuclear Antibodies (ANA). Imaging confirmed peripheral arterial disease, and echocardiography suggested mild cardiac dysfunction. Management involved anticoagulation (apixaban), vasodilators (cilostazol, nifedipine, xantinol nicotinate), and antibiotics (clarithromycin, linezolid) to prevent secondary infections. Supportive therapy included analgesics and neuropathic pain management. The patient responded well to treatment and was discharged with counseling on limb care and lifestyle modifications. This case underscores the importance of early recognition and intervention in autoimmune-related vasculopathy leading to gangrene. A multidisciplinary approach, including rheumatology, vascular medicine, and cardiology, is essential for optimal patient outcomes. This report contributes to the understanding of vascular complications in RA and emphasizes the role of aggressive management in preventing severe ischemic events.

Keywords: Dry Gangrene, Rheumatoid Arthritis, Peripheral Arterial Disease, Vasculopathy, Autoimmune Disorder, Vascular Complications.

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INTRODUCTION

Gangrene is a life-threatening condition characterized by tissue necrosis due to insufficient blood supply, often caused by Peripheral Arterial Disease (PAD), diabetes, or autoimmune disorders (Bhargava *et al.*, 2013). Dry gangrene is a non-infectious form that develops gradually due to chronic ischemia, leading to blackened, mummified tissue without significant bacterial invasion (Naji and Ranjbar, 2023). Unlike wet gangrene, which is associated with bacterial superinfection and rapid progression, dry gangrene typically results from vascular insufficiency rather than infection (Koch *et al.*, 2024).

Rheumatoid Arthritis (RA) is a systemic autoimmune disease that affects synovial joints and may also contribute to vascular complications (Sanghavi *et al.*, 2024). Chronic inflammation in RA can lead to vasculitis, endothelial dysfunction, and thrombosis, increasing the risk of PAD and subsequent ischemic events (Soufla *et al.*, 2024). In RA patients, gangrene may result from a combination of chronic inflammation, immune-mediated vasculopathy, and thrombosis, making its management complex and multidisciplinary (Jan and Dalal, 2024).

Peripheral Arterial Disease (PAD) is a significant contributor to gangrene, particularly in patients with additional risk factors such as autoimmune disorders, diabetes, hyperlipidemia, and thrombophilia (Stocco and Bailey, 2024). PAD leads to reduced blood flow, causing progressive ischemia and eventual necrosis of the affected limbs (Gaag *et al.*, 2025). This case report discusses a patient with RA and PAD who developed dry gangrene, highlighting the diagnostic approach, treatment strategies, and the role of a multispecialty team in managing vascular complications in autoimmune diseases (Wu, Y.-W., *et al.*, 2024).



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CASE PRESENTATION

A 42 years old female patient presented with complaints of fever for five days, joint pain (wrist, elbow, knee, PIP, DIP), bluish discoloration, numbness, tingling, and a burning sensation in the index and little fingers of the right upper limb and the third toe of the left lower limb (mentioned in Figure 1). Pain worsened during activities and was relieved with NSAIDs. The patient had a known history of hypothyroidism for five years and was on thyroxine 50 mcg. The patient had undergone Extracorporeal Shock Wave Lithotripsy (ESWL) on January 20, 2024, and catheter-directed thrombolysis for right lower limb thrombosis on February 18, 2025.

On physical examination, the patient was oriented to time, place, and person, with bilateral vesicular breath sounds present. The abdomen was soft and non-tender, and heart sounds S1 and S2 were normal. The patient's morning and evening vitals were charted during the hospital stay to assess fluctuations and trends in systemic response (mentioned in Figure 2).

INVESTIGATION

Ultrasound (USG) Abdomen

Grade 1 fatty liver, Cholelithiasis (12 mm calculus in gallbladder neck, multiple tiny calculi), Right renal calculus (4 mm at upper pole) (mentioned in Figure 3).

2D Echocardiography (ECHO)

LVEF: 55%, Normal chamber dimensions, Trivial Mitral and Tricuspid Regurgitation (MR/TR, TRPG=22 mmHg), Grade 1 Left Ventricular Diastolic Dysfunction (LVDD).

Autoimmune Markers

Anti-CCP: >200 RU/mL (Positive, suggestive of RA). ANA (IFA): Positive. Beta-2 Glycoprotein 1 (IgG, IgM) and Cardiolipin Antibodies (IgG, IgM): Negative.

Hematological and Biochemical Findings

Elevated ESR (42 mm/hr), Mild anemia (Hb: 11.3 g/dL), MCV: 77.6 fL (Microcytic anemia), RA Factor: 74.4 IU/mL (Elevated),

TSH: 0.991 mIU/L (Controlled Hypothyroidism), Lipid profile: LDL/HDL ratio: 2.24, total cholesterol: 142 mg/dL.

Medication Summary and Patient Discharge

During her hospital stay, the patient was managed with a combination of antibiotics, anticoagulants, vasodilators, neuropathic agents, and supportive medications to address infection risk, ischemic complications, and comorbidities (mentioned in Table 1). Clarithromycin and linezolid were initiated prophylactically to prevent secondary infection in the ischemic digits, given the patient's immunocompromised status due to RA and chronic inflammation. Although wound cultures were negative, the necrotic tissue posed a high infection risk, and guidelines support targeted prophylaxis in such cases. Eye drops of moxifloxacin and carboxymethylcellulose were also prescribed for ENT-related concerns. This comprehensive regimen reflected a multidisciplinary approach aimed at preventing complications and supporting systemic recovery. The patient was discharged in stable condition with comprehensive counseling on medication adherence and symptom monitoring. The discharge regimen (mentioned in Table 2) included: Clarithromycin and Linezolid (to address infection), Apixaban (anticoagulation for peripheral arterial disease), Cilostazol, Tadalafil, and Nifedipine (vasodilators to improve circulation), Pregabalin-Nortriptyline and Gabapentin (neuropathic pain management), Pantoprazole (gastroprotection), Aspirin-Atorvastatin (antiplatelet and lipid control), and Levothyroxine (hypothyroidism management). NSAIDs were discontinued to avoid interaction with Apixaban. The patient was advised to monitor for bleeding, infection, or worsening gangrene and to attend rheumatology follow-up for potential initiation of Disease-Modifying Antirheumatic Drugs (DMARDs). Lifestyle modifications and regular thyroid function tests were emphasized to ensure holistic management of comorbidities. At 2-week follow-up, gangrenous areas were stable with no new lesions. Pain scores had decreased, and the patient reported improved function in daily activities. She was awaiting rheumatology review for initiation of DMARD therapy pending vascular surgery clearance. The chronological sequence of the patient's presentation, investigations, interventions, and follow-up is summarized in Table 3.



Figure 1: Gangrene affecting the little and ring toe, and index and small finger.

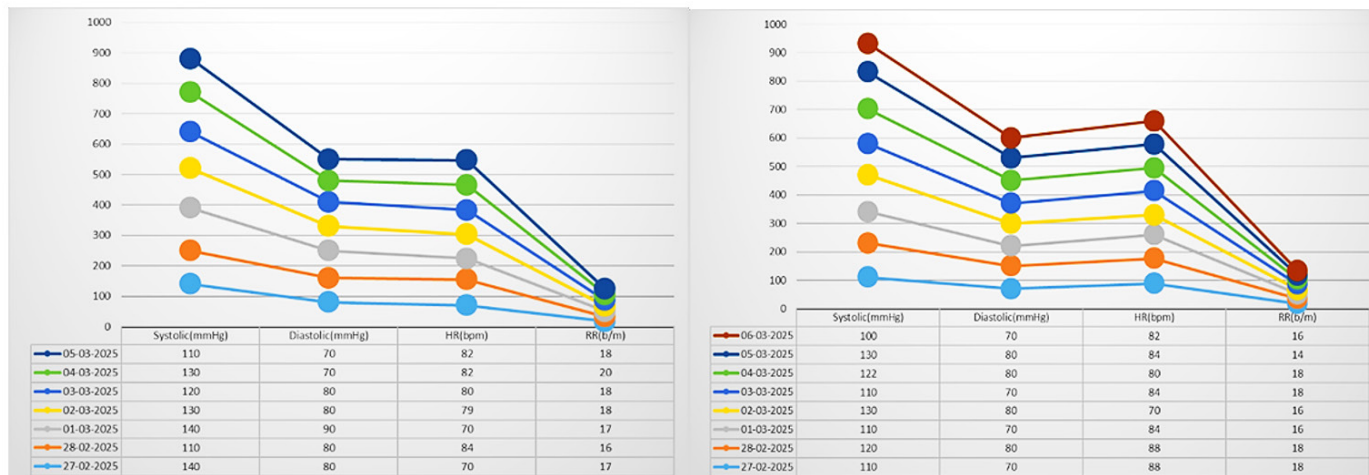


Figure 2: Morning and evening vital signs during hospital stay.

Table 1: Medications administered during hospital stay.

Sl. No.	Brand Name	Generic Name	Dose	Frequency
1	INJ. Claribid	Clarithromycin	500 mg	BD
2	INJ. Linzolid	Linezolid	600 mg	BD
3	INJ. Pantop	Pantaprazole	40 mg	OD
4	TAB. Eliquis	Apixaban	5 mg	BD
5	TAB. Ecosporin AV	Aspirin+Atorvastatin	75 mg+10 mg	OD
6	TAB. Pletoz	Cilostazol	100 mg	BD
7	TAB. Pregaba NT	Pregabalin+Nortriptyline	75 mg + 10 mg	HS
8	TAB Gabapin	Gabapentin	150 mg	HS
9	TAB Thyroxine	Levothyroxine	50 mcg	AC before breakfast
10	E/D Moxicip	Moxifloxacin	0.5% w/v	2 drops TDS
11	E/D CMC	Carboxymethylcellulose	0.5% w/v	2 drops TDS

Table 2: Medications Discharge Summary.

Sl. No.	Brand Name	Generic Name	Dose	Frequency
1	TAB. Claribid	Clarithromycin	500 mg	BD
2	TAB. Linzolid	Linezolid	600 mg	BD
3	TAB. Pantop	Pantaprazole	40mg	OD
4	TAB. Eliquis	Apixaban	5 mg	BD
5	TAB. Ecosporin Av	Aspirin+Atorvastatin	75 mg+10 mg	OD
6	TAB. Pletoz	Cilostazol	100 mg	BD
7	TAB. Pregaba NT	Pregabalin+Nortriptyline	75 mg+10 mg	HS
8	TAB. Gabapin	Gabapentin	150 mg	HS
9	TAB. Thyroxine	Levothyroxine	50 mcg	AC before breakfast
10	TAB. Tadact	Tadalafil	10 mg	HS
11	TAB. Nicarda	Nefedipine	20 mg	BD
12	TAB. Complamina	Xantinol Nicotinate	500 mg	BD

Table 3: Timeline of Events, Investigations, and Interventions.

Date / Day	Event	Intervention / Findings
January 20, 2024	Underwent Extracorporeal Shock Wave Lithotripsy (ESWL)	Procedure completed successfully, uneventful recovery.
February 18, 2025	Developed right lower limb thrombosis	Catheter-directed thrombolysis performed.
February 25, 2025	Onset of bluish discoloration, numbness, tingling, and burning sensations in the digits	Hospital admission for evaluation.
February 26, 2025	Detailed clinical examination and investigations	Laboratory tests (elevated RA factor, positive ANA, high Anti-CCP), USG abdomen, 2D ECHO, autoimmune profile; diagnosis: RA + PAD with dry gangrene.
February 27-March 5, 2025	Hospital stay	Multidisciplinary management including anticoagulation (apixaban), vasodilators (cilostazol, nifedipine, tadalafil, xanthinol nicotinate), prophylactic antibiotics (clarithromycin, linezolid), neuropathic agents, and supportive care.
March 19, 2025	Follow-up visit	Gangrenous areas stable, no spread of necrosis, pain improved, patient ambulatory, DMARD initiation planned pending vascular surgery clearance.

DISCUSSION

This case illustrates the intricate interplay between autoimmune inflammation and vascular compromise that culminates in dry gangrene. Laboratory data and imaging findings were pivotal in establishing the diagnosis and guiding management (Chen *et al.*, 2025). An elevated Erythrocyte Sedimentation Rate (ESR) of 42 mm/hr underscores the high systemic inflammatory burden characteristic of active Rheumatoid Arthritis (RA) (Javaid, Z. and M. Luqman). Such inflammation is known to contribute to endothelial dysfunction and accelerate atherosclerosis, thereby predisposing patients to Peripheral Arterial Disease (PAD) (Nyúl-Tóth, *et al.*, 2024).

The significantly elevated Anti-Cyclic Citrullinated Peptide (Anti-CCP) levels (>200 RU/mL) and rheumatoid factor (74.4 IU/mL) are highly specific for RA and have been associated with more aggressive disease and extra-articular manifestations, including vasculitis and digital ischemia. These autoantibodies not only confirm the diagnosis but also serve as prognostic markers for vascular complications. Additionally, the positive Antinuclear Antibody (ANA) test supports an underlying autoimmune dysregulation, further contributing to a prothrombotic state and vascular injury (Jung and Paul, 2021).

Moreover, the presence of Microcytic Anemia (MCV 77.6 fL, Hb 11.3 g/dL) is consistent with anemia of chronic disease or iron deficiency, conditions that can exacerbate tissue hypoxia and contribute to the ischemic cascade (OH, H., O. CHAPTER, and C. BRUCE, 2022). Differential diagnoses included systemic sclerosis, thromboangiitis obliterans, antiphospholipid antibody syndrome, cryoglobulinemia, embolic events, and frostbite. These were excluded through targeted investigations-including negative autoimmune serologies (except for RA-related markers), absence of smoking history or cold exposure, and a negative thrombophilia screen.

Management was multifaceted and aimed at addressing both the autoimmune and vascular components. Anticoagulation with apixaban was initiated to mitigate the heightened thrombotic risk inherent in patients with RA and PAD, in line with current cardiovascular guidelines. Vasodilatory therapy with cilostazol and nifedipine was employed to enhance peripheral blood flow, thereby reducing ischemic injury (Sahinturk, 2023). Prophylactic antibiotics (clarithromycin and linezolid) were administered to prevent secondary infections-a critical intervention in cases of necrotic tissue where infection can precipitate rapid deterioration (Chen *et al.*, 2024 and Tuan *et al.*, 2024).

Furthermore, optimal management of RA through the early use of Disease-Modifying Antirheumatic Drugs (DMARDs) is essential for controlling systemic inflammation and preventing further vascular complications (Giachi *et al.*, 2022 and Smolen *et al.*, 2023). Recent treatment recommendations stress the importance of a multidisciplinary approach that includes rheumatology, vascular medicine, and cardiology to tailor long-term management and improve outcomes (Bekarissova *et al.*, 2024 and Narkhede *et al.*, 2024). At discharge, the patient was ambulatory without assistance, able to perform self-care, and reported moderate residual pain, well controlled on prescribed neuropathic agents.

Initiation of DMARD therapy was deferred until resolution of acute ischemic changes and reduction in infection risk. The rheumatology team planned to start methotrexate or biologic agents during outpatient follow-up.

This case is clinically significant as it represents the rare intersection of autoimmune-mediated vasculopathy and peripheral arterial disease culminating in dry gangrene. It demonstrates



Figure 3: Ultrasound images of right and left kidney showing Grade 1 fatty liver, gallstones, and renal calculus.

the importance of early vascular assessment in patients with rheumatoid arthritis, careful selection of prophylactic antibiotics to prevent secondary infections without promoting antimicrobial resistance, and the indispensable role of a multidisciplinary team. The educational value lies in reinforcing diagnostic vigilance, antibiotic stewardship, and timely referral to rheumatology and vascular surgery specialists.

Patient Perspective

The patient expressed relief at receiving a clear diagnosis after weeks of uncertainty and distress over the discoloration and pain in her fingers and toes. She was initially anxious about the possibility of amputation but felt reassured by the multidisciplinary team's explanations and treatment plan. At follow-up, she reported improvement in pain, confidence in managing her condition, and gratitude for the coordinated care received.

CONCLUSION

This patient's presentation underscores the need for integrated care in autoimmune diseases with vascular sequelae. Early initiation of RA-specific therapies, alongside vascular and metabolic management, is critical to prevent progression of complications like gangrene. Further follow-up for DMARD initiation and surgical evaluation for gangrene is recommended.

This case highlights the importance of an integrated diagnostic and therapeutic strategy in managing complex cases where autoimmune disorders intersect with vascular pathology. Continued research and clinical vigilance are necessary to further elucidate the mechanisms linking RA and PAD and to refine therapeutic interventions for preventing critical ischemic events. The patient expressed relief at the improvement in pain and was motivated to follow limb care advice, stating, "I am thankful that my fingers and toes have not worsened, and I hope to avoid surgery with proper care."

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ABBREVIATIONS

RA: Rheumatoid Arthritis; **PAD:** Peripheral Arterial Disease; **ANA:** Antinuclear Antibodies; **Anti-CCP:** Anti-Cyclic Citrullinated Peptide; **ESR:** Erythrocyte Sedimentation Rate; **Hb:** Haemoglobin; **MCV:** Mean Corpuscular Volume; **TSH:** Thyroid Stimulating Hormone; **LDL:** Low-Density Lipoprotein; **HDL:** High-Density Lipoprotein; **DMARDs:** Disease-Modifying Antirheumatic Drugs; **NSAIDs:** Nonsteroidal Anti-Inflammatory Drugs; **LVDD:** Left Ventricular Diastolic Dysfunction; **MR:** Mitral Regurgitation; **TR:** Tricuspid Regurgitation; **TRPG:** Tricuspid Regurgitant Pressure Gradient; **USG:** Ultrasonography; **ECHO:** Echocardiography.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

FUNDING

This case report was gathered from the Shri Mahant Indiresch Hospital (SMIH), a multispecialty hospital in Dehradun, with no additional external funding.

ETHICAL COMPLIANCE

Institutional permission for publication was obtained from the Head of Department, Shri Guru Ram Rai University, and written informed consent was obtained from the patient.

SUMMARY

Dry gangrene is a serious vascular complication often associated with autoimmune conditions like Rheumatoid Arthritis (RA) and Peripheral Arterial Disease (PAD). This case report presents a 42-year-old female with a history of hypothyroidism and recent lower limb thrombosis who developed dry gangrene in her fingers and toes. The patient presented with fever, joint pain, and bluish discoloration of the extremities. Laboratory investigations revealed high rheumatoid factor (74.4 IU/mL), elevated anti-cyclic citrullinated peptide (Anti-CCP >200 RU/mL), positive ANA, and raised ESR. Imaging confirmed PAD and mild left ventricular diastolic dysfunction.

The patient was managed with a multidisciplinary approach, including anticoagulation (Apixaban), vasodilators (Cilostazol, Tadalafil, Nifedipine, Xanthinol Nicotinate), prophylactic antibiotics (Clarithromycin, Linezolid), neuropathic agents (Pregabalin, Nortriptyline, Gabapentin), and supportive therapy. NSAIDs were discontinued to avoid interaction with anticoagulants. She was discharged in a stable condition with counseling and advised to follow up for DMARD initiation and limb care.

This case highlights the importance of early recognition of vascular complications in autoimmune diseases and demonstrates the role of integrated pharmacological and clinical interventions to prevent irreversible tissue damage and improve outcomes.

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