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Avascular Necrosis Complicated by Osteomyelitis in a Lupus Patient With Active Renal Disease: A Case Report

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ABSTRACT

This case highlights the importance of taking Systemic Lupus Erythematosus seriously and its possible concurrent musculoskeletal complications, as they can have significant health implications. Consulting and encouraging patients to adhere to the treatment is crucial. Although this case survived osteomyelitis without amputation, this outcome is rare and cannot be generalized.

1 | Introduction

Systemic lupus erythematosus (SLE) is classified as an autoimmune disorder that affects multiple organ systems and is marked by the production of autoantibodies. One of the prevalent clinical features of SLE is musculoskeletal system involvement. The primary manifestations of this involvement include arthralgia, arthritis, osteonecrosis, also known as avascular necrosis of bone, and myopathy [1]. The prevalence of symptomatic osteonecrosis in SLE varies between 4% and 15%, but can be up to 40% when asymptomatic patients are included [2].

Numerous risk factors have been identified concerning the onset of osteonecrosis in patients with SLE, with corticosteroid therapy being the most significant contributor. Additional factors that appear to correlate with this condition include the presence of positive anti-Cardiolipin Immunoglobulin G (aCL IgG) and anti-double-stranded deoxyribonucleic acid antibodies (anti-ds DNA), elevated levels of Low-Density Lipoprotein Cholesterol (LDL-C) in serum, and a history of osteoporotic fractures [3, 4].

Infections represent a critical source of morbidity and mortality among individuals with SLE. The heightened susceptibility to infections can be attributed to the abnormal immune response, which may stem from the underlying disease itself or as a consequence of immunosuppressive treatments. The musculoskeletal system is one of the primary sites for infections in SLE patients. The administration of immunosuppressive therapy, osteonecrosis, and stress fractures (usually at the femoral head) elevate the risk of developing osteomyelitis in these individuals [5–7]. In this study, we have reported an SLE patient with previously active lupus nephritis who has suffered from extensive osteomyelitis that was superimposed on proximal tibial osteonecrosis.

2 | Case History/Examination

First admission: A 37-year-old Iranian female with a recent diagnosis of lupus was admitted to Shariati Hospital with severe pain and swelling in her left lower extremity as her primary complaint. Two months before this admission, the patient exhibited

several clinical and laboratory indicators that led to the identification of systemic lupus erythematosus (SLE).

According to the 2019 EULAR/ACR classification criteria for SLE, the patient was diagnosed with SLE based on the positive results from the fluorescent antinuclear antibody test (the entry criterion), alongside the presence of thrombocytopenia (4 scores), oral ulcers (2 scores), type IV lupus nephritis (10 scores), elevated anti-double-stranded DNA antibody levels (6 scores), and diminished complement proteins C3 and C4 (4 scores). For the past 2 months, she has been receiving prednisolone at a dosage of 1 mg/kg/day and mycophenolate mofetil at 2 g/day as her treatment regimen. The patient had no remarkable family history. Her psychosocial history was intact. Her medical history consisted of the recently diagnosed SLE along with the medications prescribed for the disease.

On examination, she had a low-grade fever with a minimally increased heart rate; she could bear her weight but had an antalgic gait. The limb was erythematous and had tenderness to palpation.

The laboratory findings indicated thrombocytopenia, elevated Erythrocyte Sedimentation Rate (ESR), C-reactive protein (CRP) level, and 24-h urine protein excretion; elevated anti-ds DNA antibody levels; reduced complement component 3 (C3) levels; positive anti–Sjögren's-syndrome-related antigen A autoantibodies (Anti-SSA autoantibodies); negative anticardiolipin antibodies; and normal levels of lupus anticoagulant (Table 1).

According to the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) criteria [8], the patient's disease activity score on this admission was 10; hence, her disease was active.

3 | Methods (Differential Diagnosis, Investigations, and Treatment)

Our first differential diagnosis was soft tissue infections. Plain radiography of the left leg displayed evidence of gas formation, while ultrasound imaging revealed numerous gas bubbles within the posterior muscles of the left leg. Magnetic Resonance Imaging (MRI) of the pelvic region, left thigh, knee, and leg (without contrast enhancement) exhibited widespread inflammatory alterations in the left leg, along with sizable collections featuring significant air-fluid levels within the posterior subcutaneous fat of the upper thigh. No sign of bone involvement was found (Figure 1). The diagnosis of necrotizing fasciitis was confirmed, with *Escherichia coli* (*E. coli*) identified as the causative pathogen through biopsy, Gram staining, and culture of the left leg.

The patient underwent fasciotomy and debridement, accompanied by the administration of broad-spectrum antibiotics comprising Clindamycin, Meropenem, and Vancomycin. Although the biopsy revealed *E. coli* as the main pathogen, the invasive *E. coli* has been rarely reported in the literature as the cause of necrotizing fasciitis. This condition, combined with the patient's suppressed immunity, led us to prescribe a broad-spectrum antibiotic therapy. The aim was to cover the potentially polymicrobial involvement. Intravenous Immunoglobulin (IVIG) 2 g/Kg was administered to control both infection and active disease. Following a two-month course of antibiotic therapy during hospitalization, the patient was discharged with prescriptions for daily doses of 45 mg of oral prednisolone and 400 mg of oral hydroxychloroquine. These drugs are the cornerstone of Lupus treatment. Prednisolone reduces inflammation, and hydroxychloroquine modulates the immune response.

She was instructed to arrange a follow-up appointment at the rheumatology outpatient clinic 2 weeks later to reassess her mycophenolate mofetil dosage to treat renal involvement in Lupus.

Although she had not attended a rheumatology clinic, she self-prescribed a daily dosage of 45 mg of prednisolone for a duration of 5 months.

Second admission: Five months later, she was readmitted to Shariati Hospital, presenting with the chief complaint of pain in her left knee. She had no fever, and other vital signs were normal. Apart from tenderness and erythema observed in the popliteal cavity of the left knee, the physical examination yielded no significant findings. No observable side effect of the Prednisolone overuse was detected. Ultrasound findings indicated the presence of a Baker's cyst in the left knee, a diagnosis corroborated by MRI. Furthermore, MRI revealed bilateral avascular necrosis affecting both the distal femoral metaphysis and proximal tibial metaphysis (Figure 2).

New lab data (depicted in Table 1) along with a negative anticardiolipin result and low complement level indicated that lupus nephritis had subsided decently. The patient's disease activity score on this admission was 4. Considering the patient's past diagnosis of necrotizing fasciitis, elevated CRP levels, and the observed erythema in the popliteal cavity suggestive of a potential infectious etiology, mycophenolate mofetil was temporarily discontinued until the subsequent visit, anticipated in 2 weeks. The dosage of oral prednisolone was decreased to 30 mg per day, while oral hydroxychloroquine was

TABLE 1 | Laboratory tests.

	First admission	Second admission	Third admission	Normal range
Platelet count	97,000 per μL	169,000 per μL	—	150,000–450,000 per μL
ESR	50 mm/h	33 mm/h	112 mm/h	< 20 mm/h
CRP	183 mg/L	13 mg/L	273 mg/L	< 30 mg/L
24-h urine protein	11,550 mg	3500 mg	25,578 mg	< 100 mg per day

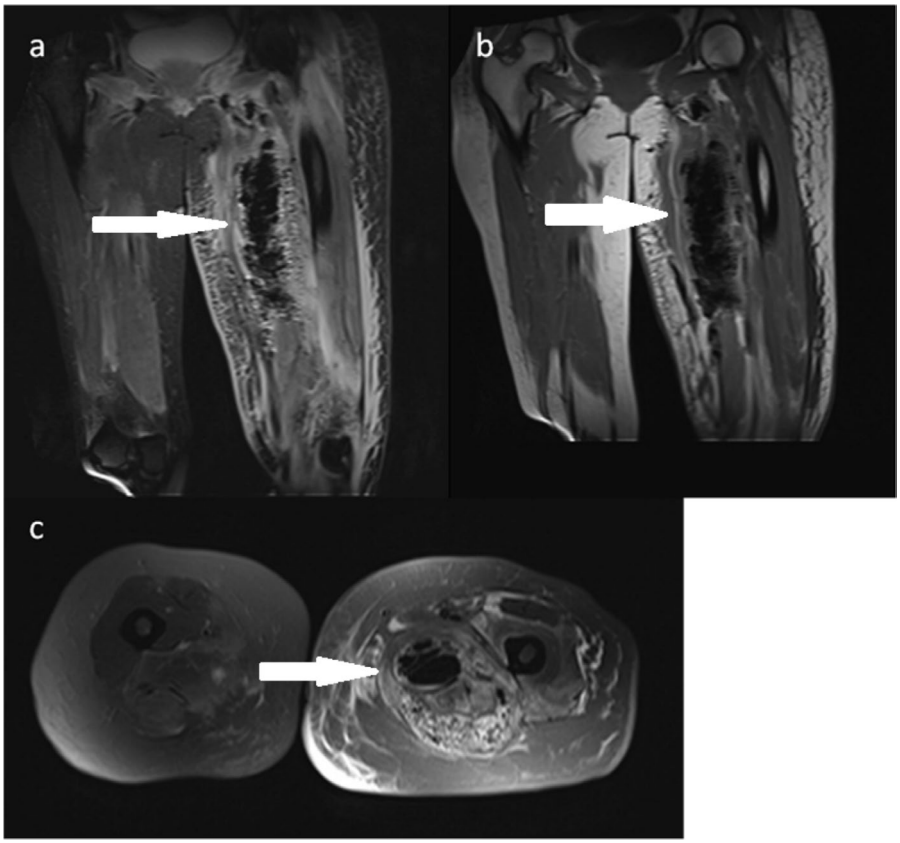

FIGURE 1 | December 22, 2019: (a) MRI coronal T2 fat saturation of both thighs: Extensive edema and inflammatory changes from the upper portion of left gluteus muscles to the lower portion of the leg involving posterior and anterior thigh muscles and adductor magnus, accompanied by a large collection of air in adductor magnus and hamstring muscles with subcutaneous edema containing some gas bubbles (Arrow indicates gas formation). Bone was not involved. (b) MRI coronal T2W of both thighs (Arrow indicates gas formation). (c) MRI axial T2 fat saturation of both thighs. (Arrow indicates gas formation).

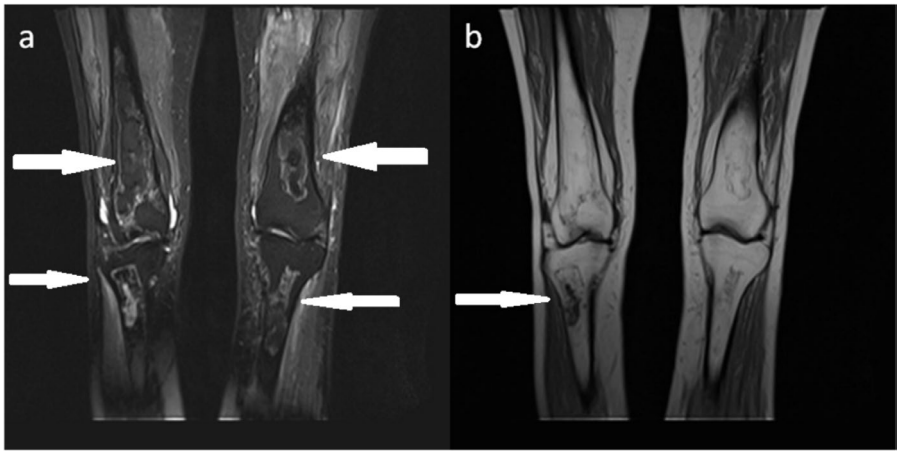

FIGURE 2 | June 2, 2020: (a) MRI coronal T2 fat saturation both legs showing the serpiginous geographic abnormal signal area in the bilateral meta-diaphysis of the tibia and femur in favor of bony infarcts. (Arrow indicates bone involvements.) (b) MRI coronal T1w of both legs. (Arrow indicates bone involvements).

maintained at 400 mg per day. Upon discharge, oral acetaminophen and naproxen were prescribed to reduce pain and alleviate inflammation.

Third admission: 3 weeks later she was admitted again with complaints of pain, swelling, and erythema of the right knee. She had a fever but no other systemic symptoms. Lab data revealed: ESR: 112 mm/h, CRP: 273 mg/L. MRI showed intense peripheral contrast enhancement in previously described intramedullary osteonecrosis in the proximal right tibial metaphysis and diaphysis with focal cortical disruption in favor of osteomyelitis (Figure 3). The patient was diagnosed with osteomyelitis

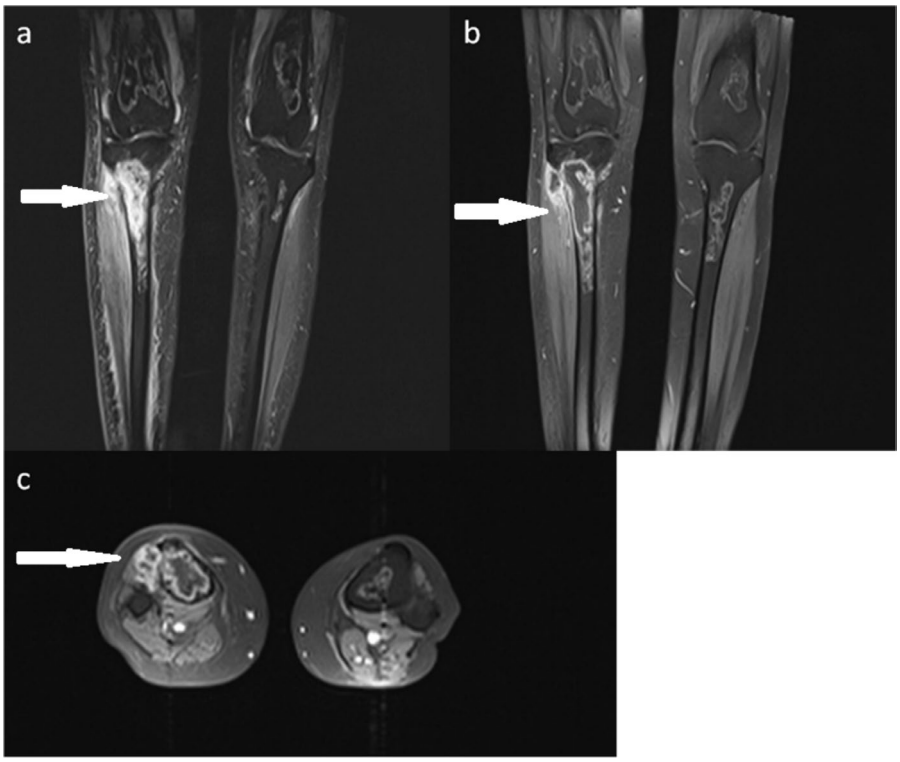

FIGURE 3 | August 19, 2020: (a) Both knees and upper legs MRI showed intense peripheral and central contrast enhancement in the previously described intramedullary bone infarct in the proximal of the right tibial meta-diaphysis with focal anterior cortical disruption. Adjacent thick rim enhancement of soft tissue in the anterior tibialis muscle was in favor of osteomyelitis and a soft tissue abscess. Mild peripheral enhancement was visualized in other infarcted areas. (Arrow indicates bone involvement and soft tissue abcess). (b) Coronal MRI of both knees and upper leg. (Arrow indicates soft tissue abcess). (c) Axial MRI of both tibiae. (Arrow indicates soft tissue abcess).

of the proximal right tibial metaphysis and underwent a four-week course of treatment with intravenous meropenem and clindamycin.

While hospitalized, the patient was diagnosed with a lupus nephritis flare-up based on laboratory data. Serious causes of fever, such as macrophage activation syndrome (MAS), were ruled out as the patient's HScore was less than 1% [9]. A significant increase in 24-h urine protein excretion to 25,578 mg was observed. Three sessions of IVIG therapy improved the kidney's condition dramatically (24-h urine protein: 4048 mg). The patient was discharged with oral cefixime 400 mg per day and oral clindamycin 300 mg every 4 h to continue the treatment and complete the course. The follow-up MRI conducted after 2 months revealed persistent osteomyelitis despite ongoing antibiotic therapy, indicating treatment failure in terms of improvement. In addition to the previously identified lesions, there were also signs of osteomyelitis observed in the posterior aspects of both femoral condyles and the medial tibial plateau. Moreover, there was a depression of about 1.5 mm at the right medial tibial plateau suggestive of subchondral bony infarct (Figure 4).

4 | Conclusions and Results (Outcomes and Follow-Up)

Considering the patient's condition, we consulted the orthopedic department, and it was determined that local surgical debridement alone would not suffice, leading to the recommendation of amputation as the most effective course of treatment. Despite this professional advice, the patient refused to undergo the amputation procedure.

Consequently, long-term oral antibiotic therapy with Ciprofloxacin 500 mg twice daily was advised for 3 months, and intravenous antibiotic therapy was recommended in case of symptom exacerbation.

At the 1-year follow-up, the patient was taking oral prednisolone 5 mg per day and 400 mg of oral Hydroxychloroquine daily. It should be highlighted that despite not taking Mycophenolate Mofetil, there were no signs of proteinuria. The activity of her lupus disease and focal signs of osteomyelitis were effectively managed with prednisolone, hydroxychloroquine, and oral Ciprofloxacin, and the SLE was under control. While the best therapeutic approach in this scenario involves surgical debridement of the necrotic bone segment or amputation followed by prolonged antibiotic treatment, our patient retained her limb by receiving prolonged oral antibiotics. Immunosuppressive therapy, along with oral antibiotic therapy, prevented the further migration of the infection. This condition is rare and is not the first treatment option; hence, it should not be generalized.

From the patient's perspective, she was satisfied with the treatments she received for 1 year after the diagnosis of necrotizing fasciitis. She had slight pain in both knees.

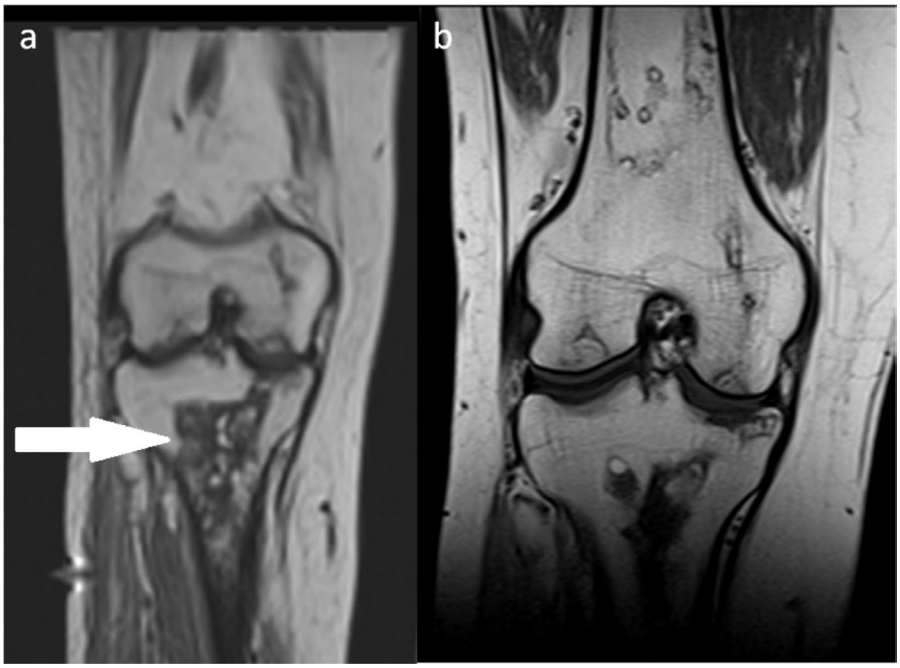

FIGURE 4 | (a) November 30, 2020: Follow-up MRI after 5 months from the right knee (Coronal T1w) showed multi-focal avascular and subchondral necrosis in the epiphysis of femorotibial joint accompanied with 1.5 mm depression at medial tibial plateau in the site of subchondral bony infarct. (Arrows indicate bone necrosis). (b) August 19, 2020: The previous right tibial osteomyelitis was partially resolved.

5 | Discussion

We have described a patient with poor compliance to treatment after a soft tissue infection which possibly resulted in bone involvement. She declined amputation as the preferred treatment option. However, she preserved her limb successfully, with no deterioration in kidney function upon discontinuation of Mycophenolate Mofetil.

Our patient experienced a severe case of necrotizing fasciitis caused by an *E. coli* infection while undergoing immunosuppressive therapy. Necrotizing fasciitis is an infection of the deep soft tissues characterized by the gradual destruction of subcutaneous fascia and surrounding tissues, with subsequent involvement of the skin typically occurring at a later stage [10].

Based on a literature review, active disease, nephropathy, lymphopenia, anemia, decreased serum albumin levels, immunosuppressive treatment, and a history of significant infections could serve as predisposing factors for the onset of necrotizing fasciitis in individuals with SLE [2, 11, 12]. In immunosuppressed patients, necrotizing fasciitis can rapidly progress to death due to sepsis, concomitant infections, or pulmonary embolism. Once necrotizing fasciitis is suspected, surgical exploration of the soft tissue, with the respective surgical cleaning and antibiotic therapy, should be performed without delay [13].

The most important aspect of our patient was severe osteomyelitis superimposed on previous osteonecrotic lesions. Lupus nephritis is a life-threatening and serious disease [14]. Therefore, the patient had been undergoing treatment for lupus nephritis with high-dose corticosteroids for 2 months, subsequently developing osteonecrosis following corticosteroid administration, with a cumulative dose totaling 4 g.

A meta-analysis including 57 studies (23,561 patients) revealed that with a cumulative corticosteroid dose of more than 2 g, the incidence of osteonecrosis was 6.7%. This study also indicated that for every 10 mg/d increment, there is a notable 3.6% escalation in the occurrence of osteonecrosis, with dosages surpassing 20 mg/d leading to a significantly elevated risk of developing this condition [15]. A literature review by Koshi et al. concluded that the elevated administration of both daily and cumulative doses of Corticosteroids correlates with an increased risk of complications, such as avascular necrosis. Therefore, it is essential to conduct counseling with the patient and secure informed consent before prescribing moderate to high doses of corticosteroids [16].

While osteomyelitis and osteonecrosis have overlapping predisposing factors, their concurrent presentation in patients with SLE has been infrequently documented. SLE patients with elevated anti-phospholipid antibodies and high-dose corticosteroid consumption seem to be at higher risk of developing osteonecrosis with superimposed osteomyelitis [17]. The history of septic arthritis seems to play a major role in the development of osteonecrosis [18]. On the other hand, medullary infarctions may act as sequestra, increasing the susceptibility of patients to osteomyelitis and soft tissue infections [19].

To address osteomyelitis overlaid on osteonecrotic bone, the recommended therapeutic approach involves surgical debridement of the necrotic bone segment followed by prolonged antibiotic treatment. Both SLE and its pharmacological management (specifically corticosteroids) are significant predisposing factors for the development of osteonecrosis and concurrent

osteomyelitis. Therefore, long-term follow-up with imaging studies (preferably MRI) is recommended. In rare instances, such as the one described in our case, debridement and antibiotic therapy alone may prove insufficient for managing concurrent osteomyelitis. In such circumstances, surgical amputation of the affected limb may be considered the main treatment option. Despite refusing amputation and only receiving steroids and hydroxychloroquine, the patient retained her limb. Without mycophenolate mofetil, her renal disease did not worsen and even showed improvement.

5.1 | Limitations

As this study is a case report and only focuses on a single patient, we cannot generalize the outcome to a larger population. In addition, we cannot establish the causal inference for the condition of the patient.

Strengths: There are a few articles reporting soft tissue infections in SLE patients. Given the suppressed immunity of these patients, the management of these conditions could be challenging. Hence, by exploring a relatively under-researched area, our study provides a foundation for future research.

5.2 | Strengths

There are a few articles reporting soft tissue infections in SLE patients. Given the suppressed immunity of these patients, the management of these conditions could be challenging. Hence, by exploring a relatively under-researched area, our study provides a foundation for future research.

Author Contributions

Nejadhosseinian Mohammad: conceptualization, writing – original draft, writing – review and editing. **Hadighi Pouya:** conceptualization, data curation, formal analysis, visualization, writing – original draft. **Aghaghazvini Leila:** investigation, methodology, writing – original draft, writing – review and editing. **Hakemi Monirsadat:** investigation, project administration, writing – original draft, writing – review and editing. **Atef Yekta Reza:** investigation, methodology, validation, writing – original draft, writing – review and editing. **Akhlaghi Maassoumeh:** formal analysis, investigation, methodology, supervision, writing – original draft, writing – review and editing. **Kavosi Hoda:** conceptualization, formal analysis, methodology, project administration, supervision, writing – original draft, writing – review and editing. **Alikhani Majid:** conceptualization, formal analysis, methodology, supervision, writing – original draft, writing – review and editing. **Loghman Maryam:** investigation, methodology, supervision, writing – original draft, writing – review and editing. **Salehi Samira:** data curation, methodology, supervision, writing – original draft, writing – review and editing. **Mozaffari Mohammad Ali:** conceptualization, formal analysis, methodology, project administration, writing – original draft, writing – review and editing. **Babagoli Mazyar:** conceptualization, data curation, formal analysis, writing – original draft, writing – review and editing. **Mobina Taghva Nakhjiri:** conceptualization, validation, writing – original draft, writing – review and editing. **Faezi Seyedeh Tahereh:** conceptualization, methodology, project administration, writing – original draft, writing – review and editing.

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The authors have nothing to report.

Ethics Statement

The research methodology adhered to the principles outlined in the Declaration of Helsinki, and ethical standards were met throughout the study. Furthermore, the study protocol received approval from the ethics committee of Tehran University of Medical Sciences, Tehran, Iran. The registered code of ethics of the study is IR.TUMS.SHARIATI.REC.1402.019.

Consent

Written informed consent was obtained from the patient to publish this report following the journal's patient consent policy, and A copy of the written consent is available for review by the Editor of this journal.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

All data generated or analyzed during this study are included in this published article.

Research Reporting Guidelines

The study has followed the CARE guidelines for Case Reports.

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