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Clinical and Immunological Overlap Between Visceral Leishmaniasis and Rheumatic Disorders: A Comparative Review on Diagnostic Challenges

Visseral Leishmaniasis ve Romatizmal Hastalıklar Arasındaki Klinik ve İmmünolojik Örtüşme: Tanısal Zorluklar Üzerine Karşılaştırmalı Bir Derleme

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ABSTRACT

Rheumatic diseases are complex systemic disorders characterized by multi-organ involvement and diverse laboratory abnormalities. The diagnostic approach involves a comprehensive evaluation of clinical and serological markers and the exclusion of conditions with overlapping presentations, such as other rheumatic diseases and systemic infections. Visceral leishmaniasis (VL) is a protozoan infection that primarily affects the reticuloendothelial system. Direct effects of parasite infiltration of the reticuloendothelial system, combined with the subsequent host immune response, produce clinical and laboratory findings that mimic those of systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA). Furthermore, the exaggerated immune response observed in a subset of VL patients overlaps clinically with primary hemophagocytic lymphohistiocytosis (HLH) and macrophage activation syndrome (MAS). VL can mimic SLE and Felty's syndrome and may trigger secondary HLH. Furthermore, in endemic regions, VL can present as a coexisting condition in patients with SLE and RA, often mimicking disease flares. In these vulnerable patient groups who are receiving immunosuppressive therapies, delayed diagnosis can significantly worsen the clinical course and prognosis of VL. This review explores the clinical and immunological manifestations of VL, providing a detailed comparative evaluation of its diagnostic overlap with HLH, MAS, SLE, and Felty's syndrome.
Keywords: Visceral leishmaniasis, rheumatic diseases, hemophagocytic lymphohistiocytosis, systemic lupus erythematosus, rheumatoid arthritis

ÖZ

Romatizmal hastalıklar, çoklu organ tutulumu ve çeşitli laboratuvar anomalileri ile karakterize, kompleks sistemik bozukluklardır. Tanısal yaklaşım, klinik bulguların ve serolojik belirteçlerin kapsamlı bir şekilde değerlendirilmesi ve diğer romatizmal hastalıklar ve sistemik enfeksiyonlar gibi benzer özelikler gösteren durumların dışlanmasını içerir. Visseral leishmaniasis (VL), öncelikle retiküloendotelyal sistemi etkileyen bir protozoon enfeksiyonudur. Retiküloendotelyal sisteme parazit infiltrasyonunun doğrudan etkileri, ardından gelen konakçı immün tepkisi ile birleştiğinde, sistemik lupus eritematozus (SLE) ve romatoid artriti (RA) taklit eden klinik ve laboratuvar bulguları üretir. Ayrıca, VL hastalarının bir alt grubunda gözlemlenen abartılı immün yanıt, klinik olarak primer hemofagositik lenfohistiyositoz (HLH) ve makrofaj aktivasyon sendromu (MAS) ile örtüşmektedir. VL, SLE ve Felty sendromunun klinik bir taklitçisi olarak hareket eder ve ikincil HLH için tetikleyici görevi görebilir. Ayrıca, endemik bölgelerde VL, SLE ve RA hastalarında sıklıkla hastalık alevlenmelerini taklit eden bir durum olarak ortaya çıkabilir. İmmünosüpresif tedaviler alan bu hassas hasta gruplarında gecikmiş tanı, VL'nin klinik seyrini ve prognozunu önemli ölçüde kötüleştirebilir. Bu derlemede, VL'nin klinik ve immünolojik belirtilerini açıklanmış, HLH, MAS, SLE ve Felty sendromu ile örtüşen tanısal özellikleri hakkında ayrıntılı, karşılaştırmalı bir değerlendirme yapılmıştır.
Anahtar Kelimeler: Visseral leishmaniasis, romatizmal hastalıklar, hemofagositik lenfohistiyositoz, sistemik lupus eritematozus, romatoid artrit

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INTRODUCTION

Leishmaniasis is a complex parasitic disease with a global distribution, affecting both human and animal populations. *Leishmania* species belong to the order Trypanosomatidae in the eukaryotic supergroup Excavata (Excavata; Discicristata; Euglenozoa; Kinetoplastea; Trypanosomatidae). Leishmaniasis is one of the world's most neglected tropical diseases according to the World Health Organization (WHO). It is caused by more than 20 protozoan parasites and is transmitted by the bite of an infected female sandfly of species *Phlebotomus* in the Old World and Lutzomia in the New World (1,2).

Its epidemiology is determined by multifaceted interactions among *Leishmania* species, sandfly vectors, host immune responses, and environmental factors. Leishmaniasis presents a broad clinical spectrum, ranging from asymptomatic and latent infections to cutaneous leishmaniasis (CL), mucocutaneous leishmaniasis, and visceral leishmaniasis (VL), traditionally known as kala-azar. VL, the most lethal form of the disease, is mainly caused by *Leishmania donovani* and *Leishmania infantum* (2). In a subset of VL patients, a clinical syndrome mimicking hemophagocytic lymphohistiocytosis (HLH) emerges, driven by the profound activation of the reticuloendothelial system (RES) (3).

We recently encountered two patients in our clinic: the first was diagnosed with macrophage activation syndrome (MAS) triggered by adult-onset Still's disease (AOSD), and the second with systemic lupus erythematosus (SLE). Both patients had been initiated on systemic corticosteroids and immunosuppressive agents prior to their current presentation. Subsequent comprehensive investigations confirmed the diagnosis of VL, and a complete clinical recovery was achieved following the administration of antiparasitic therapy. In contrast, another patient managed in the intensive care unit for MAS received a delayed diagnosis of VL. Despite subsequent initiation of treatment, the patient died. Consequently, we aimed to review the literature to re-emphasize the clinical and laboratory characteristics and the differential diagnosis of VL.

Clinical Masquerade: Leishmaniasis

WHO data indicate that the annual incidence of newly diagnosed leishmaniasis cases is approximately one million. VL, the most severe clinical manifestation of the disease, accounts for an estimated 50,000 to 90,000 new cases annually. The highest disease burden is reported in Brazil, East Africa, and India, which remain the most heavily affected regions (1). Environmental factors such as temperature, precipitation, vegetation, and humidity affect the development of vectors and parasites; socioeconomic conditions such as poverty, malnutrition, conflicts, population displacement, and rapid urbanization; and factors affecting the host's immune system such as human immunodeficiency virus (HIV) infection, organ transplantation, rheumatological diseases, and drugs determine the epidemiology of the disease (4,5). It is stated that global warming may cause the disease to spread to more temperate regions and become endemic in a wider geography (6). It is endemic in the Southeastern provinces of Türkiye and the number of cases is increasing due to recent Middle Eastern migration (7). Özbel et al. (8) reported that CL cases are unexpected increasing in Türkiye, posing a risk for its spread to Europe.

The systemic inflammatory response elicited by VL pose a diagnostic challenge by mimicking the clinical manifestations and laboratory abnormalities of HLH, MAS, AOSD, SLE, and Felty's syndrome, which occurs in the context of severe seropositive rheumatoid arthritis (RA). Such clinical mimicry often leads to misdiagnosis and the initiation of inappropriate therapies (5,9). Furthermore, VL may present as a coexisting condition in patients with SLE and RA, in whom it frequently masquerades as a disease flare. While the cases published in the literature are clinically interesting, many involve patients in non-endemic regions, where diagnosis is challenging. Clinicians practicing in endemic regions are likely more proficient in diagnosing and managing VL, potentially leading to underreporting of routine cases deemed "unremarkable". Furthermore, most published cases describe successful therapeutic outcomes. It is highly probable that patients with poor ultimate outcomes, as well as those who died without being diagnosed with VL, were not reported in the literature. Because of the high mortality risk (exceeding 95%) if VL is untreated, a rapid and accurate differential diagnosis in a patient presenting with rheumatic symptoms is not merely a medical procedure but a life-saving intervention. The key factors in differential diagnosis VL and rheumatic diseases are the in-depth analysis of clinical and laboratory findings by the consultant (10-13). Similar symptoms, findings, and laboratory abnormalities are summarized in Table 1. Furthermore, Figure 1 illustrates the strength of the associations between VL and HLH, SLE, and RA-Felty's syndrome, based on their shared clinical and laboratory features.

- **Clinical Features:** Typically irregular fever, marked weight loss, hepatosplenomegaly (usually massive), and lymphadenopathy are observed in VL.
- **Laboratory Abnormalities:** Pancytopenia is a consequence of RES and spleen sequestration rather than of simple bone marrow suppression. Elevated levels of ferritin, triglycerides, and C-reactive protein (CRP) are observed, alongside increases in liver enzymes, mainly alanine transaminase and aspartate transaminase. Hypergammaglobulinemia is common in VL.
- **Immunologic Mechanisms for Mimicking Rheumatic Diseases:** By manipulating the host's immune system, VL acquires a serological profile resembling that of a full-blown autoimmune disease. *Leishmania* infection triggers autoimmunity through polyclonal B-cell activation driven by tissue damage and molecular mimicry, in which parasite antigens resemble human proteins. These processes, combined with the formation of immune complexes that cause inflammation and complement depletion, lead to a clinical profile that closely mimics systemic autoimmune diseases like SLE.

The diagnosis of leishmaniasis involves various methods, including microscopy, culture, and polymerase chain reaction (PCR); however, it is recommended that laboratories employ a multimodal approach rather than relying on a single diagnostic tool (2). In the diagnosis of VL, tissue aspiration smears are used for histopathological examination, parasite culture, and molecular testing. While bone marrow is the most commonly preferred site, liver, spleen, enlarged lymph nodes, and whole blood (buffy coat) samples are also viable options (14). While splenic puncture in VL offers a high diagnostic yield, it carries a significant risk of splenic rupture. The sensitivity of microscopic evaluation varies between 15% and 70% as it is observer-dependent (2). Serological methods are widely utilized in the diagnosis of VL. Currently,

the rK39-based immunochromatographic rapid test is the most commonly employed diagnostic tool for VL (3). Cunningham et al. (15) evaluated multiple commercial rK39- and rKE16-based immunochromatographic rapid diagnostic tests for VL using serum samples from the Indian subcontinent, Brazil, and East Africa. While most tests showed high sensitivity and specificity in the Indian subcontinent, their sensitivity was considerably lower in Brazil and East Africa, indicating substantial regional variability and reduced reliability of some rapid diagnostic tests when used alone in these settings. In a meta-analysis of diagnostic tests for VL, the direct agglutination test (DAT) and ELISA exhibited higher sensitivity (93.0-93.8%) than rapid diagnostic tests (89.1%) and immunofluorescence assays (82.0%). Notably, all methods maintained high specificity, ranging from 95.5% to 96.9%. Furthermore, the overall diagnostic performance of DAT was found to be comparable to that of molecular assays (16). Antibody-based tests must always be used in combination with a standardized clinical case definition for the diagnosis of VL. While the sensitivity and specificity of PCR vary depending on the methodology employed (median: 78.9-97.0% and 98.4-99.4%, respectively), these molecular techniques generally surpass microscopy, culture, and histology in diagnostic yield. However, their clinical utility remains constrained by a lack of standardization and the ongoing need for validation, particularly within large hospital and clinical settings (2,16).

Rheumatological Disorders Mimicked by or Overlapping with VL

a) VL and HLH: Clinical Overlap and Differential Diagnosis

HLH involves excessive inflammation characterized by immune activation and uncontrolled cytokine release, which can lead to multiorgan failure. HLH develops as a result of inherited genetic mutations that disrupt the function of immune system cells—especially cytotoxic T-cells and natural killer cells; this form is called primary or familial HLH. Secondary HLH is triggered by infections, malignancies, or rheumatic diseases. HLH, which develops on the basis of rheumatic diseases such as AOSD and systemic juvenile idiopathic arthritis, is also described in the literature as MAS. VL shows many similarities to HLH due to its clinical presentation, which is characterized by the triad of persistent fever, splenomegaly, and pancytopenia. Numerous case reports emphasize that VL patients often fully meet the HLH-2004 diagnostic criteria, making the two diseases clinically indistinguishable (17-34). Table 2 presents a summary of case reports and case series published in the PubMed database since 2015. In a study involving pediatric VL patients in Brazil, 35 out of 127 children (27.5%) were found to meet the diagnostic criteria for HLH (35). Another Brazilian study reported that 39 out of 258 children (15.1%) diagnosed with VL met the HLH criteria, with the majority of these cases residing in urban areas (36). Diagnostic confusion represents a critical clinical threshold that directly impacts patient survival. In cases of VL misdiagnosed as primary or secondary HLH, the initiation of aggressive immunosuppressive therapy without first ruling out underlying VL can lead to uncontrolled parasite proliferation and fatal outcomes (19-21). However, with appropriate and timely antiparasitic treatment, survival rates in VL-associated HLH cases are significantly higher than those observed in primary HLH.

Pathophysiology: Immune Evasion and Cytokine Storm

The pathogenesis of VL-associated HLH is driven by a complex interplay between the survival strategies of *Leishmania* and a dysregulated host immune response. Immune response to VL has been discussed in detail by Lodi et al. (3). The immunopathophysiology of VL is characterized by the parasite's sophisticated evasion of host defenses, resulting in systemic infection involving the spleen, liver, and bone marrow. *Leishmania* creates a protected niche for replication within macrophages by manipulating Toll-like receptors, inhibiting phagosome-lysosome fusion, and utilizing a "Trojan horse" strategy via apoptotic neutrophils. While approximately 70% of individuals mount an effective Th1/M1 pro-inflammatory response that confines the parasite within granulomas, disease progression is driven by a shift toward an interleukin (IL)-10-dominant anti-inflammatory milieu (M2/Th2 polarization). This environment, exacerbated by B-cell-mediated hypergammaglobulinemia and programmed death-ligand 1expression, results in T-cell exhaustion and impaired microbicidal capacity. Ultimately, the failure of these regulatory mechanisms can precipitate an "HLH-like" state, characterized by uncontrolled macrophage activation, a cytokine storm, and high mortality, particularly in immunosuppressed patients (3).

Hemophagocytosis is the most frequently observed pathological feature in bone marrow smears from VL patients. This finding raises a critical diagnostic question: should it be interpreted as a component of VL-associated dyserythropoiesis or as a distinct HLH syndrome triggered by the infection? Lodi et al. (3) reported that it would be more accurate to define the condition called VL-HLH as "VL-related HLH-mimic". Indeed, clinical and laboratory abnormalities often resolve completely with anti-parasite therapy alone, without the need for specific HLH-directed treatment (Table 2). This suggests that the presence of histopathological hemophagocytosis may represent a reactive bone marrow phenomenon within the natural course of VL, rather than a separate, self-sustaining syndrome.

In patients with HLH or MAS, serum sCD25 (soluble IL-2 receptor) levels are significantly increased. Its association with elevated ferritin levels yields approximately 100% diagnostic accuracy for MAS. Additionally, sCD25 levels were increased in VL cases. In endemic regions, while sCD25 exhibits high sensitivity, its specificity remains limited. Consequently, differentiating between "isolated VL" and "VL-associated HLH" based solely on sCD25 elevation can be clinically challenging. Nevertheless, markedly elevated levels of both ferritin and sCD25 should strongly suggest the presence of a secondary HLH syndrome (37).

The identification of the parasite in bone marrow remains the most common and definitive diagnostic standard for VL. A primary challenge in reporting VL cases that mimic HLH is the frequent failure to detect amastigotes on initial bone marrow aspiration. In 36% to 64.7% of cases, the parasite is not identified in the first examination; detection is often only achieved through repeat aspirations or more exhaustive diagnostic evaluations (19). Liver biopsies are used for diagnosis only in rare instances. Serological assays are essential for diagnosing leishmaniasis in endemic areas. In the majority of cases presented in the literature, these tests were used in conjunction with bone marrow aspiration (Table 2).

The treatment of HLH is tailored to its underlying etiology. In primary HLH, the standard induction regimen follows the HLH-94/2004 protocols, primarily utilizing corticosteroids and etoposide; hematopoietic stem cell transplantation remains the definitive cure. Rituximab is highly effective in managing HLH triggered by Epstein-Barr virus because it targets infected B-cells. In MAS secondary to systemic inflammatory diseases, corticosteroids and intravenous immunoglobulin (IVIG) constitute the mainstays of therapy and are often supplemented by calcineurin inhibitors. Finally, in the cytokine storm induced by coronavirus disease 2019, targeted biologicals such as anti-IL-1 (e.g., anakinra) and anti-IL-6 (e.g., tocilizumab) therapies have proven effective (38). However, in the presence of VL and associated HLH, the primary therapeutic approach must be anti-parasitic therapy. With appropriate treatment strategies, mortality rates remain remarkably low. Recovery was achieved in 27 of 30 cases (90%), as summarized in Table 2. Studies conducted in Brazil have reported cures in nearly all VL-HLH patients, emphasizing that early diagnosis is life-saving and that liposomal amphotericin B is the treatment of choice. While anti-parasitic therapy is paramount, corticosteroids may still be required at anti-inflammatory doses in some patients, or at immunosuppressive doses in a limited number of cases (35,36). According to the literature, in certain cases where clinical improvement is not achieved despite anti-parasitic therapy, clinicians have resorted to agents included in the standard HLH protocols—such as glucocorticoids, IVIG, and cyclosporine A—and have observed therapeutic efficacy (Table 2).

b) VL and SLE: Comparative Evaluation

SLE is a multisystemic autoimmune disease characterized by a broad spectrum of clinical manifestations, often mimicking various systemic conditions due to its diverse organ involvement. VL is also recognized as a "great mimicker" due to its diverse clinical presentation. Both SLE and VL can manifest with overlapping features such as fever, splenomegaly, and cytopenias, often leading to significant diagnostic confusion-particularly in cases where VL triggers secondary immunological phenomena that mirror autoimmune disorders (5,39-45). Table 3 summarizes some cases of concurrent SLE and leishmaniasis reported in the literature since 2015. Notably, in five of eleven patients (45.5%), SLE was the provisional diagnosis. However, following diagnosis and successful treatment of VL, all clinical and serological abnormalities resolved, and the diagnosis of SLE could not be confirmed during follow-up. In another six patients (54.5%) with a pre-existing diagnosis of SLE, VL emerged as a comorbid infection, presenting with a clinical profile that closely mimicked an acute SLE flare (Table 3).

Shared Symptoms and Clinical Findings, Distinct Phenotypes: The involvement of the RES in both conditions accounts for their overlapping clinical presentations, characterized by hepatosplenomegaly, lymphadenopathy, and cytopenias. While splenomegaly in VL is typically massive (often exceeding 20 cm) and characterized by firm consistency, it is generally mild in SLE and only rarely reaches such significant proportions (Table 1 and Figure 1).

Irregular or high fever, significant weight loss and weakness common features of both VL and SLE (5,39-42). High-grade fever is more frequently associated with VL; however, significant thermal elevations may also occur in a subset of SLE patients during acute flares.

Arthralgia is common in VL, but arthritis/synovitis is rare, whereas non-erosive arthritis is a significant clinical finding in SLE (5). SLE is characterized by photosensitive skin rashes, such as malar rash, discoid rash, and subacute cutaneous lupus dermatitis. CL can present with facial lesions that closely mimic discoid lupus erythematosus. Specifically, lupoid leishmaniasis—a distinct clinical variant of CL—is characterized by its striking resemblance to lupus vulgaris or discoid lupus, as its nomenclature implies. In immunocompromised individuals, including SLE patients, CL can present as unusual or widespread lesions, such as ulcerated nodules and crusted plaques, which can be confused with cutaneous lupus. In some VL cases, skin lesions mimicking malar rash and vasculitis, combined with fever, pancytopenia, and antinuclear antibody (ANA) positivity, may lead clinicians to initially consider SLE as a provisional initial diagnosis (39-45).

Laboratory Overlap and Diagnostic Challenges: Cytopenias are common in both SLE and VL, but the mechanisms by which these reductions in cell lines develop are different. In VL, cytopenias are primarily due to parasitic bone marrow infiltration and RES activation, whereas in SLE they result from peripheral immune-mediated destruction. Bone marrow examination provides a definitive distinction. Bone marrow findings in SLE vary but may present as hyper- or normocellular, often reflecting peripheral immune-mediated destruction. Conversely, VL is characterized by macrophages containing intracellular amastigotes and the presence of hemophagocytic histiocytes—indicative of associated HLH—which demonstrate the phagocytosis of erythrocytes and neutrophils (5,10,12,40).

VL causes polyclonal B-cell activation, leading to positive results for many autoantibodies used in the diagnosis of SLE. ANA, which is essential for the diagnosis of SLE, can also be detected in 82% to 94% of VL patients. In addition, rheumatoid factor (RF), anti-

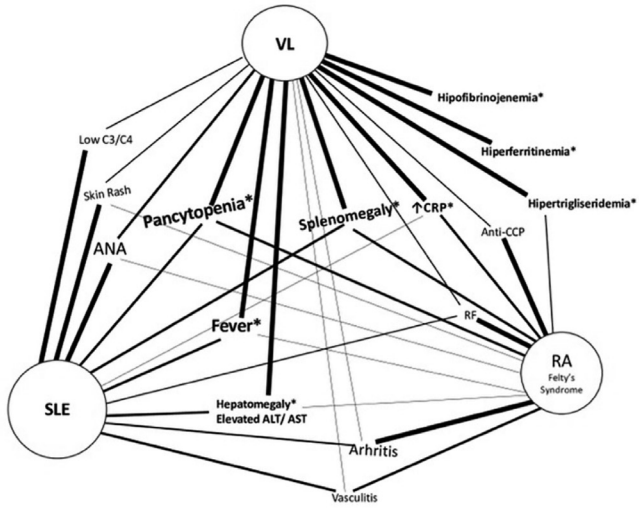

Figure 1. The figure illustrates the overlapping clinical and laboratory features of visceral leishmaniasis (VL), systemic lupus erythematosus (SLE), and Felty's syndrome. The thickness of the lines and writings represents the strength of the association between these conditions and their shared findings. Features marked with an asterisk (*) also serve as diagnostic criteria for hemophagocytic lymphohistiocytosis

RA: Rheumatoid arthritis, CRP: C-reactive protein, Anti-CCP: Anti-cyclic citrullinated peptide, ALT/AST: Alanine aminotransferase/ aspartate aminotransferase, ANA: Antinuclear antibody

dsDNA (to a lesser extent), anti-Sm, and a positive direct Coombs test can be detected in both diseases (5,11-13,40).

A significant disparity exists in inflammatory markers: VL is characterized by markedly elevated CRP levels (often >100 mg/L), whereas SLE typically presents with only mild-to-moderate CRP levels. In the context of SLE, a disproportionately high CRP should alert the clinician to concomitant infections or serositis (5,13). Serum ferritin levels are also extremely high in VL (>4000 ng/mL), but usually normal in SLE (<500 ng/mL). Serum ferritin levels may not reach characteristically high levels in the presence of concurrent iron deficiency, and conversely, low ferritin measurements may still be observed despite a systemic inflammatory state.

Serum complement levels (C3 and C4) are normal in VL. They are usually low in patients with active SLE, especially lupus nephritis (5,41,42). Hypergammaglobulinemia and low albumin levels are observed in both SLE and VL patients (10,12,41).

Due to symptom overlap with other autoimmune diseases, the diagnosis of SLE relies on specific classification criteria that combine clinical and immunological markers and are anchored by mandatory ANA positivity. Even if the patient fully meets the SLE classification criteria, in a newly diagnosed SLE patient—or one undergoing follow-up—who receives high-dose corticosteroids or immunosuppressive therapy the persistence of fever and lack of hematological improvement should strongly raise suspicion of underlying other diseases, including VL (10,40). Misinterpreting an underlying VL infection as an SLE flare and subsequently intensifying immunosuppressive therapy can lead to the rapid dissemination of the parasite, potentially resulting in fatal outcomes. Consequently, in SLE cases presenting with refractory fever and pancytopenia, particularly in endemic regions, VL should be considered before increasing the dose of immunosuppressive therapy (5,11,12,39-42). In conclusion, because leishmaniasis can act as a mimic, a trigger, or a co-infection with SLE, it must remain a high-priority consideration in the differential diagnosis of patients presenting with multisystem inflammatory features.

c) VL and RA/Felty's Syndrome: Comparative Analysis

RA typically presents with a clinical picture dominated by erosive arthritis. Its autoimmune pathogenesis is characterized by the development of RF and anti-cyclic citrullinated peptide (CCP) positivity. Diagnosis is established based on the evaluation of the distribution of affected joints, symptom duration, acute-phase reactant levels, and serological markers (RF and/or anti-CCP). A fundamental requirement of these criteria is that the observed synovitis cannot be better explained by an alternative diagnosis. Felty's syndrome is characterized by the triad of splenomegaly, neutropenia, and typically severe seropositive RA, which often precedes systemic symptoms by over a decade. Furthermore, compared with isolated RA, this syndrome is associated with a significantly higher prevalence of extra-articular manifestations, including rheumatoid nodules, vasculitis, and persistent skin ulcers. Rather than being a primary differential diagnosis for RA, VL is more frequently confused with Felty's syndrome. In a patient with established RA, the emergence of hepatosplenomegaly and pancytopenia strongly suggests Felty's syndrome; however, these findings also necessitate the exclusion of VL, especially in endemic regions (46-50) (Table 3).

Table 1. Comparison of clinical and laboratory features in visceral leishmaniasis and its mimics (hemophagocytic lymphohistiocytosis, systemic lupus erythematosus, and rheumatoid arthritis-Felty's syndrome) (Ref. 9,11,12,13,17,19,35,36,47-50)

	VL	HLH	SLE	RA
Fever	Irregular, persistent	Persistent and prolonged	Sometimes (>38.3 °C)	Rarely (subfebrile)
Splenomegaly	Massive	Massive	Mild/moderate	If Felty's syndrome present
Pancytopenia	Bone marrow infiltration by parasite and RES activation-related pancytopenia	RES activation, overstimulation of lymphocytes and macrophages, pancytopenia associated with cytokine storm	Immune cytopenias (isolated or combined) -Autoimmune hemolytic anemia -Immune thrombocytopenia, -Immune neutropenia or lymphopenia	Chronic disease anemia Leukocytosis Bicytopenia and pancytopenia in Felty's syndrome
Arthralgia arthritis	Artralgia (generally)	Arthritis associated with triggering diseases; AOSD, sJIA	Non-erosive arthritis	Erosive arthritis accompanied by synovitis involving symmetrical, small joints
Elevated liver enzymes	Often accompanied by hepatomegaly (~60%)	Oftenly (~80%)	Rarely	Rarely
Skin lesion	If cutaneous Leishmaniasis present	Non-pathognomonic	Typical photosensitive; malar rash, discoid lesion	None OR vasculitic lesions
Ferritin level	Very high (>4000 ng/mL)	Very high (>4000 ng/mL)	Normal*	Normal*
CRP	Very high (>100 mg/L)	Very high	Normal/mild	Moderately
Serum C3/C4	Normal	Normal	Normal/low (in active disease and nephritis)	Normal
RF/anti-CCP	Both are measurable as positive	None	RF (+) (20-60%) Anti-CCP(+), (2-15%)	RF (+) (70-80%) Anti-CCP (+) (60-70%)
Triglycerides	High	Very high	Normal/mild	Normal/mild
Fibrinogen	Normal/high	Very low	Normal	Normal

*: It is measured as low in the presence of iron deficiency, VL: Visceral leishmaniasis, HLH: Hemophagocytic lymphohistiocytosis, SLE: Systemic lupus erythematosus, RA: Rheumatoid arthritis, CRP: C-reactive protein, Anti-CCP: Anti-cyclic citrullinated peptide antibody, RES: Reticuloendothelial system, AOSD: Adult-onset Still's disease, sJIA: Systemic juvenil idiopathic arthritis, RF: Rheumatoid factor, OR: Odds ratio

Table 2. Summary of cases representing visceral leishmaniasis-associated hemophagocytic lymphohistiocytosis (HLH)[δ]

N	Age	Sex	Country*	Diagnosis	Ferritin	Treatment	Outcome	Ref.
1	1	F	Italy	A/BM+PCR (BM)	912	Lipo. Amp. B	Cure	17
2	16	F	Italy	A/BM+PCR (BM)	593	Lipo. Amp. B	Cure	17
3	2	M	Ethiopia	rK39	1500	Lipo. Amp. B+Dex	Cure	18
4	58	M	China	A/BM+ rK39	>500	Sodium stibogluconate+MP	Cure	19
5	58	M	China	A/BM+rK39	>500	Sodium stibogluconate	Died	19
6	38	M	China	A/BM+rK39	>500	Sodium stibogluconate+MP	Cure	19
7	43	M	China	A/BM+rK39	>500	Sodium stibogluconate+MP	Cure	19
8	47	M	China	A/BM+rK39	>500	Sodium stibogluconate	Cure	19
9	27	M	China	A/BM+rK39	>500	Sodium stibogluconate+MP	Cure	19
10	60	M	China	A/BM+rK39+mNGS	>1650	Pentavalent antimonial	Died	20
11	46	M	China	PCR+rK39	>1650	Pentavalent antimonial	N/A	20
12	26	M	Brazil	A/BM+rK39	NA	Lipo. Amp. B	Died	21
13	20	F	Brazil	rK39	10770	Lipo. Amp. B+MP+IVIG	Cure	21
14	41	F	Brazil	A/BM	577	Lipo. Amp. B+MP	Cure	21
15	48	F	Saudi Arabia	A/BM	5324	Lipo. Amp. B+MP	Cure	22
16	50	F	Türkiye	A/BM+IFAT+PCR	764	Lipo. Amp. B	Cure	23
17	83	F	Greece	A/BM+PCR+anti-*Leishmania* Ab	7146	Lipo. Amp. B+MP	Cure	24
18	64	M	Belgium	PCR	13418	Lipo. Amp. B	Cure	25
19	22	M	Armenia	A/BM+Anti-*Leishmania* Ab	2563	Lipo. Amp. B	Cure	26
20	1	M	Armenia	A/BM	>2000	Lipo. Amp. B+Dex+IVIG	Cure	26
21	6	M	Russian	rK39+Anti-*Leishmania* Ab	>3000	Lipo. Amp. B+Dex	Cure	26
22	7	M	Greek/Bulgarian	A/BM+IFAT	>2000	Lipo. Amp. B	Cure	27
23	40	F	Bulgaria	A/BM	NA	Meglumine antimoniate+ MP+IVIG+Cs	Cure	28
24	2	M	Türkiye	A/BM	14000	Lipo. Amp. B	Cure	29
25	3	N/A	Türkiye	IFAT	573	Lipo. Amp. B+IVIG	Cure	30
26	35	M	Deutschland/Spain	A/BM+PCR	896	Lipo. Amp. B	Cure	31
27	2	F	China	A/BM+rK39	40000	Antimony	Cure	32
28	1	F	China	A/BM+rK39	69445	Antimony	Cure	32
29	21	F	Portugal	A/BM	32573	Lipo. Amp. B	Cure	33
30	14	M	Ethiopia	A/BM+rK39	30000	Lipo. Amp. B+Dex	Cure	34

δ : Includes case reports and case series published in PubMed since 2015, *: The country where the case was published and/or where the potential transmission occurred, N: Number, F: Female, M: Male, BM: Bone marrow, A/BM: Amastigotes on bone marrow, PCR: Polymerase chain reaction, mNGS: metagenomic next-generation sequencing test, IFAT: Indirect fluorescent antibody test, Lipo. Amp. B: Liposomal amphotericin B, Dex: Dexamethasone, MP: Methylprednisolone, IVIG: Intravenous immunoglobulin, Cs: Cyclosporin-A, N/A: Not available

Constitutional symptoms, including malaise, fatigue, anorexia, and weight loss, can manifest in both VL and RA. However, fever is one of the primary symptoms in VL and is usually irregular, higher, and prolonged, it is rare and mild levels in RA (9,46). In patients with active RA who have not progressed to Felty's syndrome, anemia of chronic disease is a common finding, whereas hepatosplenomegaly remains rare (see Table 1 and Figure 1).

The clinical picture of RA is typically dominated by symmetrical small-joint polyarthritis, characterized by active synovitis and erosive changes, with the latter being an infrequent feature of VL. Nevertheless, in endemic regions, the presence of polyarthralgia or polyarthritis combined with RF and/or anti-CCP positivity can create significant diagnostic ambiguity between these two conditions. The release of self-antigens resulting from parasite-induced tissue destruction may trigger an autoimmune response. Specifically, this process can initiate the citrullination of proteins—a key factor in RA pathogenesis—thereby potentially acting as a catalyst for the development of RA (14). In patients with VL, RF positivity occurs at remarkably high rates (63-90%), primarily due to polyclonal B-cell activation. Furthermore, anti-CCP antibodies—traditionally regarded as having up to 95% specificity for RA—may also yield positive results in VL cases. This phenomenon is attributed to the citrullination of host proteins induced during the course of the infection. Consequently, both diseases share a common laboratory profile characterized by ANA positivity, hypergammaglobulinemia (predominantly elevated immunoglobulin G), and significantly increased acute-phase reactants, such as erythrocytes sedimentation rate and CRP (11-13).

Table 3. Characteristics of patients with SLE and RA coexisting with or mimicking leishmaniasis[δ]

N	Age	Sex	Country*	Drugs	Disease	Diagnosis	Treatment	Outcome	Ref.
SLE									
1	25	F	Brazil	MP	Not confirmed	A/BM	Lipo. Amp. B	Cure	5
2	27	F	Brazil	MP	6 years prior	A/BM	N/A	Died	5
3	18	F	Brazil	MP, MMF	1 year prior	A/BM+rK39	Lipo. Amp. B	Cure	5
4	53	M	Brazil	-	Not confirmed	A/BM+IFAT	Lipo. Amp. B	Cure	14
5	73	M	India	MP, HQ, MMF, Belimumab	Not confirmed	A/BM+anti *Leishmania Ab*	Sodium stibogluconate Lipo. Amp. B	Cure	39
6	36	F	India	MP, Cs, HQ, Tofasitinib, Telitacicept	Not confirmed	A/BM+PCR	Lipo. Amp. B	Cure	40
7	60	F	Portugal	MP, Mtx	40 years prior	PCR (blood, BM)	Lipo. Amp. B	Cure	41
8	48	F	Türkiye	MP, MMF	20 years prior	A/BM	Lipo. Amp. B	Cure	42
9	21	M	Türkiye	MP	Not confirmed	A/BM	Lipo. Amp. B	Cure	43
10	22	M	Greece	MP	Prior	N/A	N/A	Cure	44
11	40	M	Colombia	-	7 years prior	A/BM	Miltefosine Pentamidine	Cure	45
RA									
1	50	M	Brazil	Mtx, ABA	3 years prior	A/BM (+liver)+PCR+anti-*Leishmania* Ab	Lipo. Amp. B	Cure	9
2	72	N/A	Deutschland	Mtx, MP, ETA	9 years prior	Amastigotes (liver)+PCR+ELISA	Unknown	N/A	46
3	64	F	Spain	Mtx, MP	14 years prior	A/BM	Lipo. Amp. B	Cure	47
4	71	F	Greece	Mtx, MP	5 years prior	A/BM +IFAT+PCR	Lipo. Amp. B	Cure	48
5	66	M	Spain	Mtx, ADA	7 years prior	A/S+ PCR (BM)+serology	Lipo. Amp. B	Cure	49
6	79	M	Spain/Türkiye	Mtx, MP	Several years	A/BM+serology	Lipo. Amp. B	Relaps/died	49
7	83	M	Spain	Mtx, MP	6 month prior	Amastigotes (spleen)+PCR (blood and spleen)+serology	Lipo. Amp. B	Cure	49
8	84	M	Spain	Mtx	6 years prior	A/BM+serology+PCR	Lipo. Amp. B	Cure	50

δ : Includes case reports and case series published in PubMed since 2015, *: The country where the case was published and/or where the potential transmission occurred, N: Number, F: Female, M: Male, SLE: Systemic lupus erythematosus, RA: Rheumatoid arthritis, MP: Methylprednisolone, MMF: Mycophenolate mofetil, HQ: Hydroxychloroquine sulfate, Cs: Cyclosporin-A, Mtx: Methotrexate, ABA: Abatacept, ETA: Etanercept, ADA: Adalimumab, BM: Bone marrow, A/BM: Amastigotes on bone marrow, A/S: Amastigotes on skin, IFAT: Indirect fluorescent antibody, Ab: Antibodies, PCR: Polymerase chain reaction, Lipo. Amp. B: Liposomal amphotericin B, N/A: Not available

It is crucial to emphasize that drugs used to treat RA—including corticosteroids, disease-modifying antirheumatic drugs such as methotrexate, and biological agents, particularly anti-tumor necrosis factor (TNF) drugs (e.g., adalimumab, infliximab)—can significantly impair host immunity. Such treatments may trigger the reactivation of latent *Leishmania* infections or exacerbate atypical or severe clinical presentations of VL. Compared to immunocompetent individuals, those who are immunosuppressed may lack typical VL findings—such as fever, splenomegaly, and pancytopenia—or exhibit these symptoms in much milder form. Furthermore, because immunosuppressive drugs inhibit antibody production, serological tests for VL may yield false-negative results, leading to significant diagnostic delays. If immunosuppressive therapy is mistakenly intensified under the assumption of an underlying disease flare, the parasitic load increases rapidly, resulting in severe clinical deterioration. TNF-α antagonists, in particular, disrupt granuloma formation—a critical mechanism for sequestering the parasite—thereby triggering latent infections. This effect is notably more pronounced with monoclonal antibody-based agents, such as infliximab and adalimumab. In contrast, the risk appears to be lower with etanercept, which acts via soluble receptor blockade rather than direct monoclonal binding (9,46,49).

Secondary prophylaxis with liposomal Amphotericin B is indicated for patients co-infected with HIV and leishmaniasis, provided that $CD4^+$ T-cell counts are less than 200 cells/mm^3. A definitive consensus has yet to be established regarding the necessity of prophylactic treatment for patients with a history of leishmaniasis who require ongoing immunosuppressive therapy for rheumatic diseases. Specifically, it remains unclear whether prophylaxis is mandatory upon the resumption of immunosuppression. Nevertheless, regular monitoring via PCR assays is strongly recommended in this high-risk group to ensure the early detection of reactivation (1).

* Ethics

*Footnotes

*Authorship Contributions

Concept: H.Y., V.Y, Design: H.Y., V.Y., Data Collection or Processing: H.Y., V.Y., Analysis or Interpretation: H.Y., V.Y., Literature Search: H.Y., V.Y., Writing: H.Y., V.Y., Literature Search: H.Y., V.Y., Writing: H.Y., V.Y.

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