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Review Article

Viscerotropic Leishmaniasis in Iran: Current Insights and Future Directions: A Narrative Review

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Abstract

This narrative review synthesizes the current evidence on the geographic distribution, diagnostic approaches, and clinical and epidemiological characteristics of Viscerotropic leishmaniasis (VTL) in both human and animal hosts in Iran. A comprehensive search of electronic databases (PubMed, Web of Science, etc.) was conducted for articles (1997 to 2025) reporting VTL in Iran with PCR-based species confirmation. Twenty-three studies met the inclusion criteria and were analyzed. The findings indicate that VTL in Iran is predominantly caused by *Leishmania tropica*, with notable cases attributed to *L. major*. A key finding is the frequent reporting of treatment failure with pentavalent antimonials in these VTL cases. Conversely, *L. infantum*, the typical agent of visceral leishmaniasis (VL), has also been implicated in CL across various regions, suggesting its potential role in dermatotropic leishmaniasis (DL) in humans. Rare cases of *L. tropica*-induced VTL in dogs highlight a potential zoonotic dimension. Reported treatment failure with first-line antimonials in several VTL cases caused by these species, highlighting a critical therapeutic challenge. VTL, primarily caused by *L. tropica*, represents an emerging public health concern in Iran with distinct epidemiological and therapeutic implications. The reviewed evidence underscores an urgent need for rapid diagnostic tools to differentiate VTL from classic VL and to guide species-specific treatment, as standard antimonial therapy often fails. A multidisciplinary approach integrating enhanced surveillance, targeted research, and revised clinical guidelines is essential to mitigate the burden of this neglected disease.



Introduction

Although *Leishmania tropica* is primarily linked to cutaneous leishmaniasis (CL) in humans, it can cause systemic infections termed "viscerotropic" leishmaniasis (VTL). Similarly, *L. major*, another CL agent, can cause VTL, species typically associated with CL, particularly in immunosuppressed individuals, such as those living with HIV (1,2). The emergence of VTL highlights the urgent need for greater awareness and understanding of this disease.

VTL, a recently classified form of neglected tropical disease, presents significant clinical challenges due to its non-specific manifestations. This relatively mild form of systemic leishmaniasis, primarily caused by *L. tropica*, represents a public health concern in various regions worldwide (3). VTL is particularly prevalent in CL-endemic areas and is more frequently observed among immunocompromised individuals, necessitating careful differentiation from other endemic diseases, especially in countries like Iran (1,2,4–7).

The epidemiology and clinical features of VL and VTL are influenced by complex interplay of environmental, socio-economic, and cultural factors, as well as pathogen tropism, genetic variability and the host immune responses (8–10). While VTL and VL cannot be differentiated clinically, VTL often presents with high fever, intermittent diarrhea, and abdominal pain, complicating diagnosis and treatment. In contrast, classic VL typically exhibits with fever, splenomegaly, lymphadenopathy, emaciation, pancytopenia, and hyperglobulinemia (11,12).

Iran's diverse climatic conditions and varied ecosystems create an ideal environment for sandfly vectors, facilitating the transmission of multiple *Leishmania* species (13–15). Although VL cases have traditionally been overshadowed by the more common cutaneous forms, the rising incidence of VTL in recent years underscores the need for comprehensive re-

search and increased awareness regarding its clinical manifestations and public health implications (16).

This narrative review aims to synthesize the existing literature on VTL in Iran, focusing on its epidemiology, clinical characteristics, diagnostic challenges, and treatment options. By evaluating the current knowledge and highlighting critical research gaps (17,18), this review seeks to inform effective strategies for the prevention and control of this neglected tropical disease in the Iranian context.

Methods

Literature search

A comprehensive literature search was conducted to identify all published studies concerning VTL and its distribution among human and animal hosts in Iran. The search utilized electronic databases including PubMed, Web of Science, Google Scholar, MEDLINE, and ScienceDirect. Titles and abstracts of retrieved articles were screened for eligibility.

Inclusion and exclusion criteria

Studies were included if they met all of the following criteria: (i) peer-reviewed original research articles or case reports; (ii) conducted in Iran; (iii) involved human or animal cases with systemic or visceral involvement; (iv) confirmed the *Leishmania* species using PCR-based techniques (e.g., PCR-RFLP, nested PCR, ITS1, kDNA); and (v) were published in English between 1997 and 2025. We excluded review articles, reports without PCR confirmation, and studies addressing classic visceral leishmaniasis caused by *L. infantum* unless they provided evidence of atypical dermatropic or viscerotropic behavior. Molecular confirmation formed the basis for species differentiation: we required PCR amplification of kinetoplast DNA (kDNA) or ITS/rDNA targets with species assignment by sequencing or species-specific PCR. Studies that relied solely on microscopy or serology were excluded.

Case definition and data extraction

For this review, a VTL case was defined as a clinically diagnosed visceral/systemic leishmaniasis infection where molecular methods identified a species traditionally associated with cutaneous disease (*L. tropica* or *L. major*). Data extracted from included studies encompassed host demographics, geographic location, clinical manifestations, immune status, diagnostic methods, molecular markers, treatment regimens, outcomes, and the identified *Leishmania* species.

Search terms

The search utilized specific Medical Subject Headings (MeSH) terms and keywords, including ('Visceral leishmaniasis' or 'viscerotropic leishmaniasis' or 'systemic leishmaniasis' or 'disseminated leishmaniasis') and (*L. tropica* or *L. major*) and ('Iran'). Terms like 'HIV', 'immunocompromised', and 'canine' were also used.

Screening and selection

Titles and abstracts were compiled and managed using EndNote. Duplicates were removed, and the remaining records were screened for relevance. After reviewing full texts, 23 papers met all inclusion criteria and were selected for final analysis.

Results

Global and Iranian context of VTL

Although the term "viscerotropic" is occasionally used to describe the visceralization of non-visceral species of *Leishmania*, such as *L. mexicana* or *L. major*, it primarily refers to *L. tropica* as the causative agent of VL (1,19). The first report of *L. tropica* causing VL originated from Kenya, followed by a case involving a VL patient from the Jordan Valley in Israel who was infected with both *L. donovani* and *L. tropica* (20,21). During the Persian Gulf War, particularly during Operation Desert Storm, returning military personnel from the Middle East exhibited symptoms of VL, which molecular analysis confirmed to be due to *L. tropica* infection (22). Subsequently, viscerotropic forms have been reported from India, Morocco, Iran, and Afghan immigrants to the USA (11, 23- 25).

VTL cases in Iran

In Iran, VTL cases have been reported from various regions, predominantly caused by *L. tropica*, while *L. major* also identified as a causative agent. The characteristics of these cases are summarized in Tables 1-3.

VTL cases caused by *L. tropica* in Iran

The first report of VTL in Iran was documented in 2006 in a child from Fars province, where *L. tropica* was identified among VL patients (25). Subsequent reports from southwestern Iran detailed VTL in immunocompetent and immunocompromised individuals, presenting with fever, hepatosplenomegaly, and hematological abnormalities. Diagnostic confirmation often required PCR from biopsies (spleen, duodenum) when bone marrow aspirates were negative (7, 26, 27). A notable study found *L. tropica* in 4 of 29 (14%) confirmed VL cases in the southwest, indicating its significant role (5). Cases have also been reported in immunocompetent adults (7,28), in HIV co-infected patients (30), and from southeastern Iran (29,31). A summary of human *L. tropica* VTL cases is presented in Table 1.

VTL and DCL cases caused by *L. major* in Iran

L. major occasionally has been identified as the cause of VTL and diffuse cutaneous leishmaniasis (DCL) with systemic involvement in Iran. Cases have been reported from the southwest (1), and more frequently from the northeast (2,32,33). These patients often presented with chronic cutaneous lesions and visceral symptoms, showing a marked resistance to treatment with meglumine antimoniate but responding to amphotericin B (1,32). A national report also documented several DCL cases caused by *L. major* that were unresponsive to Glucantime (36). A summary of human *L. major* VTL/DCL cases is presented in Table 2.

L. tropica, in its VTL form, generally demonstrates mild pathogenesis (3) while *L. major* shows moderate to acute manifestation (37).

Table 1: Summary of human Viscerotropic Leishmaniasis (VTL) cases caused by *L. tropica* in Iran

Province	City	Year	Age/Sex	Clinical Features	Diagnostic method	Clinical finding	Treatment outcome	Ref.
Fars, Bushehr, Kohgiluyeh and Boyer Ahmad, Kerman	Shiraz, Bushehr, Yasuj, Kerman	2018	----	VL case series, 29 BM aspiration smears from patients with microscopically confirmed <i>L. infantum</i> (n = 25) and <i>L. tropica</i> (n = 4) were identified	PCR	-----	-----	(5)
Kohgiluyeh and Boyer-Ahmad	Yasuj	2016	50 M	Spleen biopsies positive	PCR	Fever, splenomegaly	Sodium stibogluconate (responsive)	(7)
Fars	Shiraz	2006	2 M	Part of a VL case series. BM/spleen aspirate positive, PCR	PCR	-----	Not specified	(25)
Fars	Shiraz	2006	2.5 F	BM negative, Liver/Duodenal biopsies positive	Semi-nested PCR	Fever, edema, hepatosplenomegaly, generalized lymphadenopathy, anemia, thrombocytopenia	Amphotericin B	(26)
Fars	Shiraz	2008	15 F	Skin lesion and BM PCR positive	Isoenzyme analysis	DCL, hepatosplenomegaly	Failed meglumine antimoniate; Responsive to Miltefosine	(27)
Fars	Marvdasht	2005	M	Microscopic Positive	Histopathological	Fleshy mass on tongue	cured spontaneously	(28)
Kerman	Kerman	2014	----	BM positive with amastigote (1/10 cases)	Nested-PCR	Fever, splenomegaly	Sodium stibogluconate	(29)
Tehran	Tehran	2003 2006	32 M 49 M	HIV+ with (CD4 count was 180/mm ³) • Negative serological tests (DAT, IFA, rK39 dipstick) and blood culture • BM and skin lesions positive	RAPD-PCR	Disseminated lesions, Mild splenomegaly	Glucantime + ART (zidovudine, lamivudine, nelfinavir)	(30)
Nationwide	Tehran	2013	----	BM microscopy (2/49 cases)	PCR	Fever, weakness, hepatosplenomegaly	Not specified	(31)

Table 2: Summary of human viscerotropic and disseminated cutaneous leishmaniasis cases caused by *L. major* and cutaneous cases caused by *L. infantum* in Iran

Province	City	Year	Age/Sex	Key Clinical Findings	Diagnostic method	Etiological agent	Clinical finding	Treatment outcome	Ref.
Bushehr	Bushehr	2007	31 M	BM and skin lesions positive with amastigote	Nested-PCR	<i>L. major</i>	Chronic ulcer, fever, pancytopenia, anorexia, fatigue, weight loss, lymphadenopathy, hepatosplenomegaly, anemia	Failed meglumine antimoniate; Amphotericin B (responsive)	(1)
Khorasan Razavi	Mashhad	2014	40 M	Positive amastigote in bone-marrow (BM)	Nested-PCR	<i>L. major</i>	HIV+, VL	HAART therapy (zidovudine, lamivudine, nevirapine) Anti-microbial treatment given (CMX + Azit)	(2)
North Khorasan	Zard village near the Mane and Samalghan	2018	26 M	Skin positive with amastigote	ITS1-PCR.RFLP	<i>L. infantum</i> / <i>L. major</i>	HIV+, DCL, mild splenomegaly	Failed meglumine antimoniate; Amphotericin B (responsive)	(4)
Tehran/Isfahan	Tehran	1997	36 F	Negative smear but positive culture	Isoenzyme	<i>L. infantum</i>	Chronic CL lesions	Not specified	(6)
	Isfahan		12 F						
North Khorasan	Esfarayen	2018	78 M	Amastigote positive in skin but negative on BM	Identified ITS1-PCR.RFLP,	<i>L. major</i>	DCL (severe)	Failed meglumine antimoniate; Amphotericin B (responsive)	(32)
North Khorasan	Bojnurd, Mashhad	2025	3 F	BM aspiration (1/50 cases)	ITS 1- PCR-RFLP	<i>L. major</i>	Malabsorption	Not specified	(33)
Ilam/Semnan/Northern Khorasan	Dehloran from Ilam	2009	41 M	Skin smears positive	PCR-RFLP	<i>L. major</i>	DCL with papulonodular cutaneous lesions	Failed Glucantime	(36)
	Damghan, Semnan	2010	59 M						
	Bojnoord city, northern Khorassan	2009	46 M						
	Isfahan	2003	40 F						
Fars	Jahrom	2009	30 M	Lymphadenopathy, BM/Skin smear positive with amastigote	PCR	<i>L. infantum</i>	HIV+, papulonodular lesions	Glucantime (antimonial drug) for 1 month)	(41)
East Azerbaijan	Ardabil	2013	≤5 M	Microscopy positive	kDNA with nested-PCR	<i>L. infantum</i>	CL/PKDL lesions	Meglumine antimoniate	(42)
Fars and Kerman		2013	10-45 M/F	Amastigote positive in the cytology smears	kDNA-nested PCR	<i>L. major</i> / <i>L. infantum</i>	Mucosal lesions	Amphotericin B	(43)
Tehran	Tehran	2011	27 M	BM and lamina propria positive with amastigote	Latex agglutination test and IFA+	<i>L. infantum</i>	HIV+, Fever, abdominal pain, vomiting and diarrhea, severe weight loss	Not specified	(44)

Cutaneous and mucosal leishmaniasis caused by *L. infantum* in Iran

In Iran, the primary causative agents of CL are *L. major* and *L. tropica*, while *L. infantum* is the main causative agent of VL (38–40). Conversely, *L. infantum*, the primary agent of VL, has been reported to cause atypical cutaneous (6,41), post-kala-azar dermal leishmaniasis (PKDL) (42), mucosal (43), and intestinal diseases (44), particularly in immunocompromised hosts. A case of mixed *L. infantum*/*L. major* cutaneous co-infection in an HIV-positive patient has also been documented (4).

L. tropica-induced VTL in animals in Iran

Dogs, recognized as primary reservoirs for *L. infantum*, have also been found infected with *L. tropica*, causing visceral disease. Cases have been reported from central and northeastern Iran (31, 45–47), suggesting a potential secondary reservoir role for dogs in the *L. tropica* cycle. A summary of canine *L. tropica* VTL cases is presented in Table 3.

Table 3: Summary of Canine Viscerotropic Leishmaniasis (VTL) cases caused by *L. tropica* in Iran

Province	Year	Animal Details	Patient Age	Clinical/Pathological Findings	Diagnostic method	Species Confirmation	Ref.
Nation-wide		4 dogs from 80 VL cases		Canine VL	Spleen/Liver positive	PCR-RFLP <i>L. tropica</i> (4/80 cases)	(31)
Alborz	2011	5-month puppy		Mucocutaneous lesions, systemic	Smear, DAT, rK39 positive	PCR-RFLP: <i>L. tropica</i>	(45)
Kerman	2015	2 dogs (>5 years)		Alopecia, hyperkeratosis, systemic	Histopathology, kDNA-PCR	kDNA-PCR: <i>L. tropica</i>	(46)
Tehran, Alborz, Golestan, Ardebil	2014	4 dogs		Canine VL	Culture, DAT and rK39 Positive	PCR, RFLP, Sequencing (6/10): <i>L. Infantum</i> and (4/10): <i>L. Tropica</i>	(47)

Discussion

This narrative review consolidates evidence on the emerging phenomenon of viscerotropic leishmaniasis in Iran, revealing a complex epidemiological picture where traditional host-parasite associations are frequently disrupted.

The critical importance of differentiating *L. tropica* VTL from classical VL

Differentiating VTL caused by *L. tropica* from classical VL caused by *L. infantum* is critical for several reasons:

Epidemiology and control:

L. tropica VTL arises in urban/peri-urban foci of anthroponotic cutaneous leishmaniasis

(ACL), implicating a human-to-human transmission cycle. This contrasts sharply with the rural, zoonotic cycle of *L. infantum* involving canine reservoirs. Control strategies for ACL (e.g., targeting human cases) differ fundamentally from those for zoonotic VL (e.g., reservoir control). The finding of *L. tropica* in dogs (46,47) further blurs this distinction and complicates control policies.

Clinical presentation and course:

While both VTL and VL forms present with systemic involvement, reviewed cases suggest *L. tropica* VTL may have a milder or more protracted course. However, it can also present severely, especially in immunocompromised hosts. The common occurrence of concurrent

cutaneous lesions in *L. tropica* VTL cases (5,27) is a distinguishing clinical clue not typical of *L. infantum* VL.

Diagnostic challenges:

Standard serological tests (e.g., rK39 dipstick) developed for *L. infantum* may have lower sensitivity for *L. tropica* infections, as indicated by some case reports (30). This can lead to diagnostic delays or misdiagnosis. Therefore, molecular confirmation (PCR) from relevant tissues is essential for accurate species identification, which has profound implications for management.

Therapeutic implications:

The most striking finding from this review is the high frequency of treatment failure with pentavalent antimonials (e.g., meglumine antimoniate, Glucantime) in both *L. tropica* and *L. major* VTL/DCL cases (1,7,27,32,36). In contrast, amphotericin B (and in one case, miltefosine (27)) proved effective. This pattern of antimonial unresponsiveness necessitates a re-evaluation of first-line treatment protocols in regions where these species are prevalent. Treating *L. tropica* VTL with protocols optimized for *L. infantum* may lead to poor outcomes and increased morbidity.

The plasticity of *Leishmania* species in Iran

The reviewed literature underscores significant parasite plasticity. Not only can *L. tropica* and *L. major* cause visceral disease, but *L. infantum* can also manifest cutaneously. This bidirectional "tropism switching" suggests that factors such as host immune status (e.g., HIV co-infection), parasite genetic adaptation, and possibly vector characteristics may be more influential than previously thought in determining disease outcome. The genetic homology noted between *L. tropica* and *L. infantum* haplotypes (47) may provide a biological basis for this plasticity.

Limitations and future directions

This review is limited by the inherent nature of narrative syntheses and the reliance on published case reports/series, which may be subject to publication bias. The true prevalence of VTL in Iran remains unknown due to a lack of systematic, nation-wide studies employing molecular speciation in all suspected VL cases.

Future research must prioritize: 1) Large-scale, prospective studies to determine the true burden and geographic distribution of VTL; 2) Development and validation of rapid, cost-effective, and species-specific diagnostic tests for field use; 3) Clinical trials to establish evidence-based treatment guidelines for *L. tropica* and *L. major* VTL; and 4) Ecological studies to better understand the reservoir hosts and transmission dynamics of *L. tropica*, particularly its zoonotic potential.

Conclusion

VTL, primarily caused by *L. tropica* and, to a lesser extent, *L. major*, is an under-recognized but clinically significant form of leishmaniasis in Iran with serious public health implications. This review highlights its distinct epidemiological niche, frequent diagnostic complexity, and concerning pattern of resistance to conventional antimonial therapy. Moving forward, a paradigm shift is needed: visceral leishmaniasis in Iran should not be automatically attributed to *L. infantum*. Integrating molecular diagnostics into routine practice is crucial to unmask the true spectrum of disease, guide appropriate treatment, and develop effective, species-tailored control strategies to reduce the burden of this emerging health challenge.

Competing interests

The authors declare no competing interests.

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