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VISCERAL LEISHMANIASIS IN THE DIFFERENTIAL DIAGNOSIS OF LEUKEMIA IN CHILDREN: A CLINICAL OBSERVATION

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Abstract

Introduction. In recent years, symptoms such as anemia, fever, and general weakness have become more common, and these symptoms are often not considered the onset of serious illnesses. However, they can be clinical manifestations of serious infectious diseases. One such disease is visceral leishmaniasis, which can present with symptoms such as fever, chills, anemia, or bicytopenia with hepatosplenomegaly.

Case Study. A 15-month-old boy was hospitalized in an infectious diseases hospital. According to his mother, the patient's main complaints were weakness, nausea, a body temperature of up to 40.0°C (104.4°F), chills, tremors, abdominal distension, and no improvement despite treatment by a pediatrician and hematologist for anemia. According to the patient's mother, he has been ill for 5 months. He lives in the Angren district of the Tashkent region. His mother attributes the illness to a mosquito bite he received at the age of 10 months. He was taking Nurofen and Calcium at home. With no improvement in his condition, he sought examination at the Institute of Hematology on June 4, 2025, on the recommendation of a pediatrician. In the hematology department, the patient was diagnosed with Hemophagocytic lymphohistocytosis, anemia, hemorrhagic syndrome, hyperplastic syndrome, endogenous intoxication, hepatosplenomegaly and prescribed treatment. Despite the treatment, his condition did not improve; instead, it worsened, and an enlarged liver and spleen

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were observed. After almost 3 months of treatment in the hematology department, leishmaniasis was confirmed in the bone marrow on August 28, 2025. On August 30, 2024, he was referred to the emergency department of the Republican Specialized Scientific and Practical Medical Center for Epidemiology, Microbiology, Infectious and Parasitic Diseases for treatment. Given the signs of dehydration, test results, and the patient's condition, he was hospitalized in the intensive care unit.

Conclusion. It is necessary to increase awareness among healthcare professionals about visceral leishmaniasis in both endemic and non-endemic areas. We also believe it is appropriate to apply the same diagnostic and therapeutic approach to cases of bicytopenia and fever as to cases of pancytopenia and fever. It is important for the healthcare community to ensure the timely identification of all patients with fever and hematopoietic disorders, particularly those with hepatomegaly and/or splenomegaly, to avoid missing potential cases.

Keywords: Visceral leishmaniasis, pancytopenia, bicytopenia, anemia

Introduction

Pancytopenia is a significant clinical and hematological syndrome characterized by a simultaneous decrease in the three main cell lines of peripheral blood: red blood cells, white blood cells, and platelets. Early identification of the underlying cause of this condition significantly reduces morbidity and mortality in children. Anemia and bicytopenia are often early signs of severe illness, and if not recognized promptly, they can quickly progress to pancytopenia. However, these intermediate cases are often overlooked and underreported in the scientific literature[2,4].

The etiology of pancytopenia is broad and can range from hematological diseases to infectious, autoimmune, metabolic, and neoplastic processes. In patients with an unknown underlying cause, bone marrow aspiration and biopsy are integral to

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the diagnosis, as they play a crucial role in determining the presence of hematopoietic suppression, infiltration, or fibrosis[2]. Visceral leishmaniasis (VL) should always be considered in the differential diagnosis of children with fever, splenomegaly, anemia, or bicytopenia. The pathogenesis of VL is primarily due to the penetration of Leishmania spp. into macrophages and their accumulation in the liver, spleen, and bone marrow. This process leads to the development of anemia, bicytopenia, and ultimately pancytopenia due to hematopoietic suppression, hypersplenism, excessive cytokine production (IL-6, TNF-α), and reticuloendothelial dysfunction.

An estimated 50,000 to 90,000 new cases of VL are registered annually worldwide. Risk factors for the development of the disease include urbanization, humidity, altitude, rainy seasons, sanitary conditions, and socioeconomic factors. Children living in highly endemic areas, particularly those with malnutrition or immunodeficiency, are at risk of infection.

This clinical case demonstrates how delayed detection of pancytopenia can lead to serious consequences in a patient with fever and anemia. Hepatosplenomegaly with anemia or bicytopenia, initially identified, were missed, and differential diagnosis was delayed. Currently, such a delay is frequently observed in clinical cases associated with VL and is a major cause of diagnostic errors.

This case once again demonstrates that the possibility of VL should be seriously considered in any child with fever, enlarged spleen, anemia, or bicytopenia. When pancytopenia is detected in children living in endemic areas, laboratory confirmation of VL (PCR, microscopy) and bone marrow examination should be performed as soon as possible[1,4,5,8].

Case Study

A 15-month-old boy was hospitalized in an infectious diseases hospital. According to his mother, the patient's main complaints were weakness, nausea, a body temperature of up to 40.0°C (104.4°F), chills, tremors, abdominal

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distension, and no improvement despite treatment by a pediatrician and hematologist for anemia. According to the patient's mother, he has been ill for five months. He lives in the Angren district of the Tashkent region. His mother attributes the illness to a mosquito bite he received when he was 10 months old. He was taking Nurofen and calcium at home. With no improvement in his condition, on June 4, 2025, he sought examination at the Institute of Hematology on the recommendation of his pediatrician due to low blood counts in his complete blood count. In the hematology department, the patient was diagnosed with Hemophagocytic lymphohistocytosis, anemia, hemorrhagic syndrome, hyperplastic syndrome, endogenous intoxication, hepatosplenomegaly and prescribed treatment. Despite these treatments, his condition did not improve; instead, it worsened, and his liver and spleen became enlarged. After nearly three months of treatment in the hematology department, leishmaniasis was confirmed in his bone marrow on August 28, 2025. On August 30, 2024, the patient was referred to the emergency department of the Republican Specialized Scientific and Practical Medical Center for Epidemiology, Microbiology, Infectious, and Parasitic Diseases for treatment. Based on signs of dehydration, test results, and the patient's condition, he was admitted to the intensive care unit.

Medical History: The child was born at age 2, born at term by cesarean section. Intraocular pressure (IOP) is 3160 g. The patient is being monitored for leukemia. All preventive vaccinations have been completed. Growth and development are appropriate for his age. According to the mother, there are no allergic reactions to medications or food. The patient denies hereditary diseases.

Epidemiological History: 1. The mother denies contact with infectious diseases. 2. The patient has not left his place of residence for 6 months. 3. He has had infectious diseases: acute respiratory viral infections and colds. He has not had COVID-19. 4. The child has not received any outpatient or inpatient treatment in the past 6 months. No blood or blood components have been transfused. No surgical interventions have been performed. 5. According to the mother, all

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preventive vaccinations have been completed on time. 6. According to the child's mother, the child was bitten by a mosquito at the age of 10 months.

Objective condition: General condition is severe. The child is numb. Passive. Body build is normal, normosthenic type. Body temperature is 37.4°C. Skin tone is weak. Subcutaneous fat is moderately developed. There is no edema in the legs. The tongue is dry, coated with a white coating. The throat is slightly reddened when examined with a spatula. Nasal breathing is spontaneous. Respiratory rate is 26 per minute. SpO2-98%. Weak vesicular breathing is heard in the lungs. Pulse is 132 per minute. Heart sounds are muffled, rhythmic. The abdomen is soft, painless on palpation. The liver is enlarged by +5.5 +6.0 +6.5 cm on palpation. The spleen is enlarged by +9.0 +10.0 cm. Stool is regular. Urination is regular. Neurological status: The patient is conscious and responsive to external stimuli. Depressed mood, with normal orientation to sounds and bright colors. Meningeal signs: Neck rigidity (-). Kernig's sign (-), Brudzinski's sign (-), Brudzinski's sign (-). No abnormal reflexes. No focal signs of brain damage.

Based on all the information provided, the patient was hospitalized for further evaluation and treatment due to a potentially life-threatening condition. The initial examination at our clinic included a complete blood count, abdominal ultrasound, and consultation with a hematologist and infectious disease specialist. A complete blood count (CBC) was performed to determine several blood counts, as well as an abdominal ultrasound to detect enlarged spleen and liver. Due to positive CBC results, the patient was transferred to a specialized hospital (Republican Specialized Scientific and Practical Medical Center for Epidemiology, Microbiology, Infectious and Parasitic Diseases).

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	30.08	31.08	01.09	05.09	08.09	12.09	19.09	27.09	29.09
Leukocyte (WBC) - 10^9/L	6.0	6.9	6.0	7.3	6.2	3.8	6.5	6.4	7.0
Red blood cell anisocytosis with standard deviation (RDW-SD) - %				47.7					57.6
Hemoglobin (HGB) - g/L,	103	98	88	82	81	78	78	84	78.0
Lymphocyte count (LYMPH#) - 10^9/L,				4.5					4.6
Erythrocyte sedimentation rate (ESR) - mm/s				15	12	14	11	4	16
Mean hemoglobin concentration in erythrocytes (MCHC) g/L	317			324					305
Mean size cells-%				10.9					7.8
Granulocyte count (Gran#) 10^9/L				2.1					1.9
Lymphocyte (LYM%) %				61.0					64.5
Thrombocrit (RST) %	0.109			0.134					0.202
Mean hemoglobin content in erythrocytes (MCH) - pg	26.2			26.7					27.4
Red blood cell (RBC) - 10^12/L	3.92	3.11	2.92	3.06	2.8	2.7	2.7	2.6	2.84
Hematocrit (HCT) -%	32.4			25.3					25.5
Mean corpuscular volume (MCV) fL	82.9			83.0					90.1
Platelet (PLT) - 10^9/L	115			156					207
Red blood cell anisocytosis (RDW-CV) - %	15.1			16.6					19.1
Medium-sized cells - 10^9/L,				0.7					0.5
Granulocytes (Gran%) - %				28.1					27.7
Mean platelet volume (MPV) - µm³	9.5			8.6					9.8
Platelet anisocytosis (RDW)	15.3			14.8					15.5
Rodular neutrophil-%					2	1	1	1	
Segmented neutrophil-%					65	61	61	59	
Eosinophils-%					1	1	1	3	
Monocytes-%					7	8	5	8	
Lymphocytes-%					25	29	32	29	

Table 1. This table shows the results of a complete blood count.

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Complete Blood Count (CBC). The first CBC showed a normal white blood cell (WBC) count (6.0 × 109/L) and red blood cell (RBC) count (3.92 × 1012/L). The hemoglobin level also decreased (103 g/L), but the mean corpuscular volume (MCV) was normal. The hematocrit (HCT) also decreased (32.4%). (Table 1).
The second and subsequent CBCs showed virtually the same WBC count (6-7.3 × 109/L), with only one showing 3.8 × 109/L. The RBC count further decreased (from 3.11 × 1012/L to 2.6 × 1012/L). The hemoglobin also decreased (98-78 g/L), and the MCV remained normal (Table). No elevated erythrocyte sedimentation rate was detected in the first and second blood tests, and no significant changes were observed during a later examination. Although the platelet count was low (115 x 109/L), subsequent laboratory data showed that platelet counts increased due to the plasma transfusion received (twice) (156-207 x 109/L). Therefore, by this time, we had bicytopenia, not pancytopenia, although this could have been related to the treatment received in hematology. Even before admission to our clinic, the patient had anemia (according to his mother).

Table 2. This table presents the results of the blood biochemistry analysis.

	30.08.25	**08.09.25**	**12.09.25**	**29.09.25**
Total bilirubin -µmol/L	12,5	10,5	8.3	20.0
Conjugated bilirubin- µmol/L	2,5	2,5	2.5	3.0
Free bilirubin- µmol/L	10,0	8,0	5.8	17.0
Ast (aspartate aminotransferase)- U/L	0,35	0,34	0.32	0.85
Alt (Alanine aminotransferase)- U/L	0,70	0,68	0.64	1.67
Total protein- g/l	50,3	52,5	57.5	56.5
Glucose- Mmol/L	2.8	4.81	4.3	4.8
Urea- Mmol/L	6.8	8.4	8.0	7.9
Creatinine- Mmol/L	78.3	91.2	84,7	81.2
Diastase- One/L				148
Potassium- Mmol/L				3.4
Sodium- Mmol/L				138
Calcium- Mmol/L				1,59

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Table 2 shows a number of significant changes in the patient's key biochemical parameters between August 30, 2025 and September 29, 2025. Bilirubin metabolism showed changes within the normal range. AST and ALT values were close to normal between August 30 and September 12. However, on September 29, 2025, AST increased to 0.85 U/L and ALT to 1.67 U/L. This indicates a gradual increase in liver cell damage. The total protein content gradually increased from 50.3 g/L to 57.5 g/L, and by September 29, 2025, it stabilized at 56.5 g/L. This indicates a slow recovery of protein synthesis in the patient. Initially, it was 2.8 mmol/L, and hypoglycemia was observed. In subsequent tests, glucose levels increased to 4.3-4.8 mmol/L and stabilized within the normal range. Urea levels increased from 6.8 to 8.4 mmol/L, then maintained at around 7.9 mmol/L. This may indicate an increase in metabolic load or a change in renal function. Creatinine was initially 78.3 mmol/L, increasing to 91.2 mmol/L, and then decreasing to 81.2 mmol/L. This indicates minor changes in renal function, but no significant renal failure. Additional indicators: Diastase: 148 U/L - pancreatic function may be slightly activated. Potassium: 3.4 mmol/L - below normal, moderate hypokalemia. Sodium: 138 mmol/L - normal. Calcium: 1.59 mmol/L - decreased, possible signs of hypocalcemia.

Enzyme-linked immunosorbent assay (ELISA) 09/29/2025 - procalcitonin - 0.038 ng/ml, ultrasound 09/29/2025 - Hepatomegaly, gallbladder flexure, splenomegaly, echocardiogram of liver hemangioma? Hepatomegaly, splenomegaly with splenic vein ecstasy. 09/08/2025 Prothrombin time - 18 seconds, prothrombin index - 100%. 09/09/2025 X-ray - signs of bronchitis. Ultrasound 09/03/2025 Hepatomegaly, splenomegaly with splenic vein ecstasy.

During the ultrasound examination of the abdominal cavity, the child had a significantly increased oblique vertical size of the left lobe of the liver (99 mm) and craniocaudal size of the right lobe (112 mm), craniocaudal size of the left lobe (49 mm) (normal at 1 year: right lobe - ~60 mm, left lobe - ~33-40 mm), the

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size of the spleen was 126x42 mm (approximately 6.8 x 5 cm for children 1-3 years old).

Treatment: semi-bed position. Diet No. 15. For oral rehydration: ORSA 100.0 ml per os max. 3. For rehydration: Ringer's lactate -100.0 ml intravenously. For detoxification: 5% glucose solution 100.0 ml + 4% KCl solution 2.0 ml intravenously. For antioxidant therapy: 5% ascorbic acid solution -1.5 intravenously. For hemostatic therapy: Didsicon 250 mg 0.5 ml intravenously. For etiotropic therapy: Glucontin 1.5 g 5.0 ml intravenously up to 2.0 ml intravenously. To improve microcirculation: Suximed 100 ml intravenously. Thiotriazoline 2.4% intravenously 2.0 ml intravenously. Antibacterial therapy: Ceftriaxone 0.5 g + Novocain 0.5% intravenously, 5.0 ml intravenously. Plasma transfusions twice. Symptomatic treatment. Before diagnosis was confirmed, the patient was immediately hospitalized and given supportive therapy and empirical intravenous antibiotics. After confirmation of the VL diagnosis, the patient was prescribed Glucontin 1.5 g, 5.0 ml, and 2.0 ml intravenously, which is first-line therapy according to the protocol proposed by the Ministry of Health. The following tests are required to monitor side effects: complete blood count, AST/ALT, UTT, and chest X-ray. No complications were identified, supportive therapy is ongoing; two plasma transfusions were administered to correct anemia and blood clotting disorders.

The patient underwent treatment for one month at the Republican Specialized Scientific and Practical Medical Center for Epidemiology, Microbiology, Vector-Borne, and Parasitic Diseases and was discharged home in relatively satisfactory condition.

Discussion

Visceral leishmaniasis is a severe parasitic disease caused primarily by Leishmania donovani and Leishmania infantum. Most cases are reported in South Asia, East Africa, the Mediterranean region, and parts of Latin America. Fewer

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than 100,000 cases are reported annually worldwide, with more than 95% occurring in high-incidence countries such as Brazil, India, Ethiopia, and Sudan [2,4,9].

The first reports of visceral leishmaniasis (VL) in Uzbekistan date back to the early 20th century and were recorded in Tashkent, Andijan, Bukhara, Samarkand, and Termez. Among the republics of the Soviet Union in the early and middle periods of the 20th century, the highest incidence rates of VL were observed in Uzbekistan. According to epidemiological analysis, approximately two-thirds of patients were children under three years of age. The incubation period lasted from one to six months, and in most cases, it lasted from two to three months. As Mirzoyan noted, the bite of an infected sand fly caused visible spots or papules to form on the skin of infants and young children. Microscopic examination revealed leishmanial amastigotes one to two months after the appearance of these papules. He called this lesion the "primary effect," as it was the entry point of infection and the initial center of inflammation. Approximately half of the patients experienced this "primary effect" and subsequently (within 4-8 months) amastigotes were detected in bone marrow smears. Patients with clinical signs, including fever, were hospitalized [10].

The predominant clinical manifestations of the disease include fever, weight loss, and enlargement of the liver and spleen. Symptomatic disease develops in a small number of infected individuals when the parasites overcome the host's defenses and replicate in target tissues (liver, spleen, lymph nodes, bone marrow, etc.). The mortality rate for this disease in immunocompromised patients reaches 10% [14]. In the case of the described patient, the clinical picture was characterized by nonspecific symptoms, mainly hepatosplenomegaly, fever, weight loss, and anemia. Hepatosplenomegaly, which is the main feature of the disease, is caused primarily by lymphoid hyperplasia. Histopathological studies have shown that both the spleen and liver are infiltrated by parasites, which disrupts their cytoarchitecture.

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This disease, mediated by proinflammatory and regulatory cytokines, leads to an increase in organ size, with splenomegaly being more common than hepatomegaly [1.2].

The spleen can enlarge to more than ten times its normal size, which plays a central role in the pathogenesis of the disease. Splenectomy significantly improves the clinical condition of patients, although it does not cure HIV coinfection. Some liver hypertrophy is also observed, associated with an increase in the number of Kupffer cells infected with L. donovani or L. infantum [4,5]. In the described patient, physical examination and imaging studies (UTT) revealed splenomegaly, which decreased from a maximum of +9.0 +10.0 cm to +3.0 cm with antiparasitic treatment. The largest recorded liver volume was +5.5 +6.0 +6.5 cm, which is an increase of 1.6 times, and after treatment it decreased to +2.5 +2.5 +2.5 cm. Considering that parasites multiply not only in the liver but also in the bone marrow, pancytopenia or bicytopenia, anemia with thrombocytopenia are common concomitant symptoms of the disease. According to Aljurayyan et al., its causes are multifactorial - suppression of hematopoiesis, sequestration by the spleen and hemolysis. In our case, pancytopenia played a key role in the diagnosis of the disease, since a bone marrow biopsy was performed based on this discovery [6]. VL is a disease that can affect any human organ, including the gastrointestinal tract, peritoneum, lungs and pleural cavity. Atypical lesions of internal organs are more common in progressive HIV infection (CD4 T-lymphocyte count <50 cells/mm³) and in immunocompromised individuals [7]. This is a good reminder that febrile anemia or bicytopenia, like pancytopenia, also require attention.

Severe leishmaniasis (SL), one of the most dangerous infections in the world, is caused by the parasites Leishmania donovani and Leishmania infantum, which are transmitted through the bites of Phlebotomine sandflies; the incubation period of the disease is reported to range from a few days to 10 years [8, 9]. Once infected, the protozoan parasites begin to attack the lymph nodes, the immune

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system, and other reticuloendothelial organs such as the liver, spleen, and bone marrow, destroying white blood cells and red blood cells [10,13,14]. Because this disease can also affect the immune system, it is also called the parasitic form of AIDS [2]. In children, SSL may present with episodic fever and other mild symptoms or be asymptomatic [4,5]. In 25% of untreated cases, the disease may reactivate within a few months with an enlarged spleen and intermittent fever [4,8]. Prolonged fever, hepatosplenomegaly, pancytopenia, weight loss, and pallor are classic clinical features of visceral leishmaniasis and should be excluded as soon as possible [12]. One study reported that 15% of children with pancytopenia had visceral leishmaniasis [2]. Amphotericin B and pentavalent antimony are the most commonly used drugs to treat this condition [4,5,6]. The diagnosis of VL can be made by detecting parasites in spleen or bone marrow aspirates or by detecting antibodies using serological tests [4,5,8]. Leishmania parasites can also be detected using polymerase chain reaction (PCR), which has high sensitivity and specificity [13]. Early diagnosis of visceral leishmaniasis is essential for a better prognosis [2]. Malaria and acute leukemia are important differential diagnoses, as both can present with fever, splenomegaly, and pancytopenia [2]. Other hematological etiologies of pancytopenia in children include megaloblastic anemia, myelodysplastic syndrome, or aplastic anemia. Cytostatic drugs and certain viral agents, such as cytomegalovirus (CMV), Epstein-Barr virus (EBV), human immunodeficiency virus (HIV), influenza, rubella, parainfluenza, hepatitis A, adenovirus, parvovirus B19, and others, can also present with pancytopenia in children. In endemic areas, tuberculosis and brucellosis may also be suspected in patients with fever, splenomegaly, and pancytopenia. Leishmaniasis can easily be misdiagnosed as an autoimmune disease, as various autoantibodies may be present during the course of the disease [15]. VL with positive antinuclear antibodies and multiorgan involvement may also be misdiagnosed as systemic lupus erythematosus [16]. Antineutrophil and platelet antibodies combined with a positive direct Coombs test may be

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misdiagnosed as an autoimmune disease. Autoimmune pancytopenia, such as hemophagocytic lymphohistiocytosis syndrome (HLH), should also be considered as a variant [2,4].

It should be noted that VL is one of the most dangerous and life-threatening diseases, so rapid diagnosis is essential. However, this is a challenging task. Even in endemic areas, many cases of late diagnosis are observed. According to the National Center for Disease Control and Public Health of Georgia, 35 and 20 cases of VL were registered in 2020 and 2021, respectively[14]. Unfortunately, it should be noted that one of the reasons for late diagnosis is ignoring the possibility of VL, such as anemia and bicytopenia, which often progress to pancytopenia. A detailed physical examination is also necessary to detect enlargement of the spleen and/or liver, and abdominal ultrasound followed by a complete blood count allows monitoring the progression of the disease, especially in cases of prolonged fever [2]. Literature reviews show that the time to diagnosis after the onset of symptoms is very long: an average of 30 days, with a peak of 5 months [2].

One article reported that the patient was diagnosed 8 days after admission to the hospital, which corresponds to the 28th day after the onset of symptoms [2,12]. In many articles, the time to diagnosis of VL can vary from several days (the minimum period described is 7 days) to several months [2].

Since VL is a serious disease, without timely diagnosis and treatment, it can lead to a fatal outcome for most patients [6]. Diagnosis can be delayed for a number of objective and subjective reasons. Microscopic examination (spleen, bone marrow, lymph nodes) is considered the gold standard [3,8,11], but in some cases it can be delayed due to the invasive nature of the procedure. Therefore, if a patient presents with a clinical picture consistent with VL, it may be prudent to consider serologic testing as soon as possible and then rely on positive results, although it is widely known that serologic tests can be positive in asymptomatic patients or in patients with a history of the disease [5–7].

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One of the challenges in diagnosing VL is the need for a broad and thorough differential diagnosis. Typical symptoms of VL include fever, hepatomegaly and/or spleen enlargement, and pancytopenia. However, these symptoms are nonspecific for VL and can be indicative of a number of other diseases, such as rheumatic, infectious, malignant, and others. In the absence of other specific signs, symptoms, and indications (use of certain medications, etc.), differential diagnosis can be difficult.

Conclusion

This disease is easily confused with other conditions, especially hematological malignancies, where the diagnosis is confirmed only by bone marrow histopathology.

It is necessary to raise awareness of VL among healthcare professionals in both endemic and non-endemic regions. We also recommend that our colleagues apply the same approach as for pancytopenia and fever when anemia or bicytopenia is accompanied by fever. The medical community must ensure that no case of fever and pancytopenia is overlooked, especially in the presence of hepatomegaly and/or spleen enlargement.

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