

Case Report

Uncovering a Rarely Diagnosed Disease: Severe Leptospirosis with Multiorgan Failure in Slovakia

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Abstract: Leptospirosis is a zoonotic disease caused by bacteria from the genus *Leptospira*. The infection occurs mainly in developing countries in the tropical zone and countries with lower hygiene standards. The highest incidence is observed especially in environments associated with the presence of rodents, mainly rats, which are a potential source of infection. The clinical manifestations and severity of leptospirosis are highly variable. This case report describes the a 53-year-old patient (male) without comorbidities, who was admitted to an infectious disease department in the east of Slovakia for jaundice, general fatigue, weakness, and fever (health difficulties for approximately 7 days at home). The clinical laboratory picture of the patient was dominated by significant hyperbilirubinemia, acute renal failure, hepatopathy, severe thrombocytopenia, and involvement of the lung parenchyma in the sense of bilateral interstitial pneumonia on chest X-ray. A double combination of antibiotics (ceftriaxone and clarithromycin) were added to the treatment. During hospitalization, a diagnosis of leptospirosis was suspected based on medical history and the results of laboratory tests and was subsequently confirmed serologically. The antibiotic regimen was de-escalated to cephalosporin (ceftriaxone) monotherapy with adequate laboratory and clinical effects (on the 4th day). The patient was discharged after a total of 18 days in good clinical condition.

Keywords: leptospirosis; sever; pulmonary; renal injury; Slovakia



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1. Introduction

Leptospirosis is a severe, potentially life-threatening systemic bacterial disease belonging to the zoonoses caused by spirochetes of the genus *Leptospira*. The disease occurs endemically, especially in tropical zones with low hygiene standards, where it is often the cause of various epidemics, especially in a post-flood period [1].

Infection occurs after direct or indirect exposure to infected reservoir host animals. Leptospirae reside in the renal tubules, from where they are subsequently excreted in the urine. The reservoir of the infection is mostly in wild and domestic animals (rodents, cattle, pigs, buffaloes, horses, sheep, goats, squirrels, etc.). The most important and most common source of infection in humans is the brown rat (*Rattus norvegicus*). The most at-risk group are people living in urban slums with insufficient hygiene, unsuitable housing, and a high incidence of rats and other rodents. Most often, these are farmers, workers in rice fields, or travelers. Cases of leptospirosis have also been reported in children or adults after contact with infected livestock or contaminated water. In developed countries, infection occurs mainly in connection with recreational activities in freshwater (canoeing, swimming, surfing) [2,3].

2. Epidemiology, Clinical Picture, Diagnosis and Treatment of Leptospirosis

Leptospire are free-living aerobic spirochetes with characteristic hooked ends, 6–20 µm long, and approximately 0.1 µm in diameter. Only some strains are pathogenic to humans. Traditionally, the genus *Leptospira* was divided into two species: *L. interrogans sensu lato* (all pathogenic strains) and *L. biflexa sensu lato* (all saprophytic strains).

More recently, phylogenetic analysis revealed that *Leptospira* can be divided into three lineages that correlate with the level of pathogenicity of the species: saprophytic, intermediate, and pathogenic. Currently, leptospire are classified according to genotypes and serovars. A total of 64 leptospira species with more than 30 serogroups and 300 serovars are currently known. Most pathogenic strains (13 genus) belong to the *Leptospira interrogans* group [4,5].

Vincent et al. proposes to reclassify species of the *Leptospira* genus into four sub-clades, called P1, P2, S1 and S2, instead of the clusters historically named as saprophytes (S1 and S2), intermediates (P2) and pathogens (P1) [5].

The portal of entry in humans is most often skin or mucosal abrasions that come into contact with water contaminated with the urine of infected animals. The incidence is about 1 million cases worldwide, with a mortality rate of about 60,000 deaths per year. Leptospirosis is a disease of epidemic potential, especially after heavy rainfall or flooding. It is most prevalent in tropical regions but also occurs in temperate regions. Regions with the highest incidence of infections include South and Southeast Asia, Oceania, the Caribbean, parts of sub-Saharan Africa, and parts of Latin America [6].

The clinical picture of the disease is quite diverse—from a febrile condition similar to the flu, to a serious disease associated with various organ complications. Typical clinical symptoms include fever, chills, headache, myalgia, arthralgia, nausea, and vomiting [7].

Severe forms of the disease, including meningitis, Weil's syndrome (conjunctival suffusion, icterus, and acute kidney damage), and lung involvement with hemorrhages into the lung parenchyma, occur in only about 10% of reported cases [8,9].

The disease often occurs in two phases. The clinical course of leptospirosis is classically divided into a "leptospirosis phase", or acute phase, followed by an immune phase. The incubation period of the disease is usually 7–12 days. The initial phase presents as a nonspecific acute febrile illness. The "leptospiremic" phase is followed by the immune phase, when IgM antibodies appear in the blood and the bacteria begin to be excreted in the urine. Depending on the degree of organ involvement and the organism's virulence, clinical manifestations of varying severity occur at this stage [10].

The microscopic agglutination test (MAT) is the gold standard for serodiagnosis of leptospirosis because of its unsurpassed diagnostic specificity. It uses panels of live leptospire, ideally recent isolates, representing the circulating serovars from where the patient became infected. A dilution series of the patient's serum is mixed with a suspension of live leptospire in microtiter plates. After incubating for about 2 h at 30 °C, results are read under a dark-field microscope. The titer is the last dilution in which $\geq 50\%$ of the leptospire have remained agglutinated. Seroconversion or ≥ 4 -fold titer rise in paired sera is consistent with current leptospirosis. Since performing MAT requires maintaining live leptospire, techniques such as ELISA (enzyme-linked immunoassay), indirect immunofluorescent and the slide agglutination test have been developed. Most cases of leptospirosis are diagnosed using serology (most often by the ELISA method or microscopic agglutination test). Antibodies in the Ig M class are detectable in the blood 5–7 days after the onset of symptoms. Detection of IgM antibodies specific to *Leptospira* by ELISA (IgM-ELISA) has been widely used. There is no need to test the second sample if IgM ELISA is positive, whereas paired sera testing is required for diagnostic confirmation by the MAT assay. A fourfold rise of MAT titer suggests a current *Leptospira* infection. Another possibility is the detection of leptospiral DNA using PCR (polymerase chain reaction) (methodology from various body fluids). Cultivating *leptospira* requires specialized culture media and is quite difficult in practice [11].

To detect *Leptospira* DNA in blood and urine samples of patients with clinically suspected leptospirosis, two PCR approaches were used that amplify two different target DNA sequences: the *rrs* and *lipL32* genes. According to Podgoršek et al. the RT-PCR targeting of the *lipL32* gene was shown to be preferred to conventional PCR targeting of the *rrs* gene, as it is quicker, easier to perform, less prone to contamination and more sensitive for the determination of *Leptospira* DNA in these clinical samples. This RT-PCR is also more sensitive than culture analyses and quicker than serological tests [12].

Mild and less severe forms of the disease can be treated with oral antibiotics. The drug of choice is mainly azithromycin or doxycycline [13].

Regardless of the duration of the clinical symptoms of the disease, antibiotic treatment must be started immediately after leptospirosis is suspected, as it can prevent the progression of the disease to more severe forms. In severe cases, parenteral antibiotic treatment is indicated. Intravenous penicillin G has been shown to be as effective as the 3rd generation cephalosporins—cefotaxime or ceftriaxone [14].

The disease is rare in Slovakia, and its diagnosis is often overlooked. According to data from the Public Health Office of the Slovak Republic, three cases of leptospirosis (one respiratory, one hepatic and one febrile form) caused by *Leptospira interrogans* were recorded in Slovakia in 2020 (morbidity 0.06 per 100,000 inhabitants) [15].

In 2021, three cases were also reported (one respiratory, two hepatic). As far as we know, in 2022, our case report was the only case of leptospirosis with multiorgan failure [16].

The main goal of the described case report is to draw attention to often neglected and relatively dangerous diseases such as leptospirosis. The authors point to the fact that, leptospirosis rarely occurs in the temperate climate zone; it is necessary to think about this disease, especially in patients with liver damage, fever, history of hiking, drinking water from contaminated sources, after being bitten by mammals, and after contact with soil.

3. Case Report

A 53-year-old male, previously healthy and not treated for any medical issue, was transported by the emergency medical service at the end of August 2021 to the Infectious disease department for jaundice. During the medical examination, the patient stated that he had been ill for about 7 days, had aches through his whole body, chills, and rigors. During the previous few days, he noticed he was urinating less; the maximum body temperature measured at home was 37.1 °C. For the last three days, he observed a gradual yellowing of the skin and sclera, darker urine, and a lighter shade of stool. He has repeatedly vomited three times, but he did not have diarrhea. As part of the medical history, he reported alcohol consumption (beer several times a day), working as a university teacher in the field of natural sciences, negative epidemiological anamnesis (contact with animals, intravenous drug use, ingestion of mushrooms, drinking and bathing in fresh water sources, tattoos and piercings, administration of blood products, ingestion of hepatotoxic drugs, hard alcohol, etc.).

During the subsequent examination, the patient was afebrile, cardiopulmonary compensated, mildly hypotensive (100/70 mmHg), heart rate of 85/minute and oxygen saturation of 93%. The clinical picture was dominated by icterus of the skin and sclera, signs of dehydration (dry, coated tongue), palpable enlarged liver (+3 cm), and audible crepitus over the lungs on auscultation, bilaterally.

As part of the routine outpatient examination, an ultrasound examination of the abdomen was performed. The gallbladder was without signs of acute cholecystitis, there was no dilatation of the bile ducts. There was a small amount of free fluid detected in the cavum Douglasi, gastric ectasia, and the hyperechoic edematous parenchyma of both kidneys as a sign of acute renal failure.

The patient was subsequently admitted to the Department of Infectology and Travel Medicine with jaundice for differential diagnosis. In the initial laboratory screening (Tables 1 and 2), there was hyperbilirubinemia, hepatic lesion (slightly isolated elevation of AST and GMT), significant elevation of urea, creatinine, and inflammatory parameters (leukocytes, C-reactive protein, procalcitonin, interleukin-6), mineral disorder

(hyponatremia—126 mmol/L, hypokalemia—2.7 mmol/L), and severe thrombocytopenia (without coagulopathy).

Table 1. Results of biochemical examinations—during the first 8 days and on the day of discharge (18. day) (abbreviations: AST—aspartate aminotransferase, ALT—alanine transaminase, GGT—gamma-glutamyltransferase, ALP—alkaline phosphatase, CRP—C-reactive protein, PCT—procalcitonin, MYO—myoglobin).

Laboratory Parameters Day of Hospitalization	Urea (mmol/L) (2.8–7.2)	Creatinine (μmol/L) (64.0–104.0)	Total Instead Bilirubin (μmol/L) (5.0–21.0)	Conjugated Bilirubin (μmol/L) (0.5–3.4)	AST (U/L) (3–51)	ALT (U/L) (3–51)	GGT (U/L) (3–55)	CRP (mg/L) (0–5)	PCT (μg/L) (0–0.5)	MYO (μg/L) (28–72 μg/L)
1.	32.8	344	404.2	340.8	98	51	58	139.8	2.0	1155
2.	31.8	246	498.1	310.8	107	48	47	187.4	4.5	560
3.	27.9	138	693.1	379.4	110	49	55	140.1	3.4	313
4.	18.2	93	1046.0	700.2	117	66	94	103.6	1.8	-
5.	15.0	80	955.4	638.6	108	84	78	64.6	0.9	53
6.	11.8	86	900.5	606.2	78	89	80	55.3	0.7	-
7.	9.5	84	883.2	577.9	61	89	67	41.6	0.5	-
8.	6.5	78	833.5	523.0	84	92	65	33.3	0.5	-
18.	3.7	67	237.4	107.5	72	180	61	3.6	0.3	-

Table 2. Results of hematological examinations during the first 8 days and on the day of discharge (18. day) (abbreviations: HGB—hemoglobin, WBC—white blood count, PLT—platelets, INR—prothrombin time, Fib.—fibrinogen).

Laboratory Parameters Day of Hospitalization	HGB (g/dL)	WBC ($\times 10^9$ /L)	PLT ($\times 10^9$ /L)	INR (s)	APTT (s)	Fib. (g/L)
1.	10.2	11.6	27	1.03	28.2	2.2
2.	9.9	19.6	30	1.19	26.3	2.6
3.	9.0	18.8	18	0.96	26.9	2.9
4.	8.5	22.6	65	0.99	30.5	3.5
5.	10.8	20.3	155	1.03	33.6	3.0
6.	9.9	19.9	209	1.07	31.9	2.9
7.	10.7	17.8	303	-	-	-
8.	10.5	16.6	323	-	-	-
18.	11.2	5.4	300	-	-	-

At the beginning of the patient's hospitalization, an X-ray examination of the chest was performed, concluding that there were numerous speckled and reticular opacifications, predominantly peripherally located and sometimes confluent, in both lung wings (Figure 1A).

Due to the high inflammatory parameters and findings on the chest X-ray, dual antibiotic treatment (as for pneumonia) was initiated with ceftriaxone 2 g every 24 h and clarithromycin 500 mg every 12 h intravenously. The patient was parenterally rehydrated, given symptomatic treatment (antipyretics, analgesics, vitamins, bronchodilators, replenishment of minerals), low-flow oxygen therapy, and monitored for fluid balance.

During hospitalization (3rd day), there was a further decrease in the number of platelets. these results were discussed with a hematologist, who recommended the administration of platelet concentrate (a total of two transfusion units were administered) and hemostyptics (etamsylate 250 mg every 8 h).

Examination of a swab from the nasopharynx for SARS-CoV-2 PCR was negative. As a part of the differential diagnosis of jaundice, additional microbiological examinations were completed (Table 3) with positive antibodies in the Ig M class by ELISA against *Leptospira*

interrogans (commercial serological “Panbio™ *Leptospira* IgM ELISA” rapid test, manufactured by Abbott). The disease was subsequently confirmed through another microbiological test—an immunochromatographic test with the finding of positivity of antibodies of the Ig M class against *Leptospira interrogans* (commercial, qualitative rapid immunochromatographic test “Prevent ID® *Leptospira*”, manufactured by Preventis GmbH).

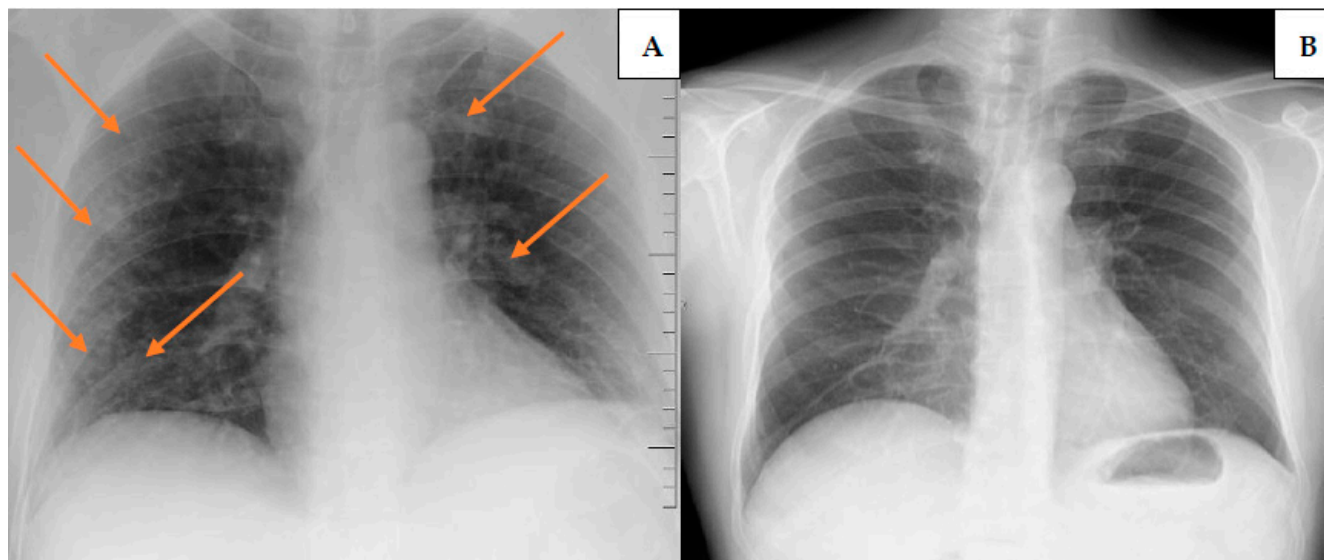


Figure 1. (A)—X-ray of the chest showing bilateral opacifications (arrow), (B)—Control X-ray of the chest (after 2 weeks)—regression of the finding.

Table 3. Results of microbiological examinations (*—repeated examination).

Microbiological Examination	Biological Material	Result
PCR SARS-CoV-2, influenza A/B	nasopharyngeal swab	negat.
anti-Ebstein–Barr antibodies (ELISA)	serum	IgM negat./IgG positive
anti-Cytomegalovirus antibodies (ELISA)	serum	IgM negat./IgG positive. *
anti-HIV antibodies (ELISA)	serum	negat.
anti-Hepatitis A antibodies (ELISA)	serum	IgM negat./IgG positive.
anti-Hepatitis E antibodies (ELISA)	serum	IgM negat./IgG positive.
Hepatitis B surface antigen	serum	negat.
anti-Hepatitis C antibodies (ELISA)	serum	negat.
anti- <i>Leptospira interrogans</i> (ELISA+ immunochromatographic test) antibodies	serum	IgM positive
<i>Candida</i> antigen	serum	negat.
<i>Aspergillus</i> antigen	serum	negat.
antigen of <i>Legionella pneumophila</i>	urine	negat.
antigen of <i>Streptococcus pneumoniae</i>	urine	negat.
antigen of adenovirus, rotavirus, norovirus	stool	negat.
anti- <i>Mycoplasma pneumoniae</i> antibodies (ELISA)	serum	Ig M/Ig A/Ig G negat.
anti- <i>Chlamydia pneumoniae</i> antibodies (ELISA)	serum	Ig M/Ig A/Ig G negat.
stool/rectal swab culture	stool	negat.
urine culture	urine	sterile *
<i>Clostridioides difficile</i> —toxin A/B, antigen	stool	negat.
blood culture	blood	sterile *
anti- <i>Brucella</i> sp. antibodies (ELISA)	serum	negat.
anti- <i>Toxoplasma gondii</i> antibodies (ELISA)	serum	negat.
anti- <i>Hantavirus</i> antibodies (ELISA)	serum	Ig M/IgG negat.
anti- <i>Francisella tularensis</i> antibodies (ELISA)	serum	Ig M negat.

Due to these microbiological findings, the epidemiological anamnesis was supplemented with an emphasis on being in nature and drinking fresh water. The patient subsequently states that approximately two weeks prior to the onset of symptoms, as part of field research at work, he took soil samples (from the deeper layers) and drank water from a stream.

Based on results of serological tests (ELISA, rapid immunochromatographic test), epidemiological history, and the patient's condition, diagnosis of acute leptospirosis with acute renal failure and lung involvement was made. Because of the above, we continued the antibiotic ceftriaxone. However, due to the negative result of the other serological tests (especially *Mycoplasma pneumoniae* and *Chlamydia pneumoniae*), we did not continue the treatment with clarithromycin (we administered the antibiotic for a total of 3 days).

A transthoracic echocardiographic examination was performed by a cardiologist, which showed no systolic and diastolic dysfunction of the ventricles, no valve defects, sinus rhythm on the electrocardiogram, and no rhythm disorder.

During hospitalization, the patient was examined by a nephrologist, with the conclusion that this is acute kidney failure of combined etiology (pre-renal caused by dehydration and from direct renal damage by leptospirosis). Because of paresthesias and pain in the cervical spine, a neurological examination was completed, with the conclusion that it was an exacerbated vertebrogenic algic syndrome with a recommendation for symptomatic treatment (analgesics, myorelaxant agents).

During the hospitalization, a hepatological examination was completed. The laboratory tests for autoimmune diseases (anti-nuclear, anti-mitochondrial, antibodies against granulocyte cytoplasm, glomerular basement membrane, and microsomes by *immunofluorescence testing*), were negative. This also supported the diagnosis of hepatopathy caused by infectious disease.

With treatment, the patient's condition improved, there was a decrease in inflammatory activity, the serum bilirubin level gradually decreased, renal parameters were adjusted, and the number of platelets in the blood count increased (Figure 2). Antibiotic treatment was administered for 14 days. After ceftriaxone treatment, we performed a follow-up X-ray examination of the chest (Figure 1B), which showed complete regression of inflammation bilaterally. After 18 days of hospitalization, the patient was discharged in stable condition to outpatient care.

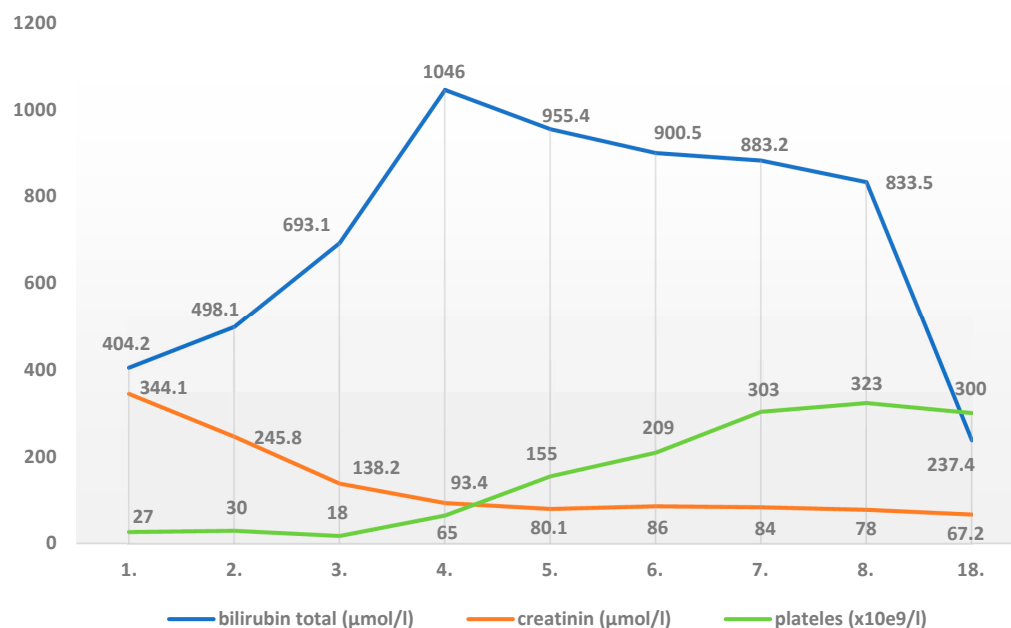


Figure 2. Dynamics of monitored laboratory parameters during hospitalization.

4. Discussion

The incidence of leptospirosis in the temperate climate zone is approximately ten times lower than in the tropics. Human infection most often occurs through contact with infected animals' urine (rodents) or after contact with infected soil. The point of entry is mainly through various skin injuries. The disease is also transmitted after drinking water contaminated with the urine of infected animals [17].

According to the patient's history, it is possible to assume two possible means of infection. The infection was acquired either by ingesting contaminated water, as the patient stated, or it is also possible that the infection may have occurred through contact with contaminated soil while collecting samples as part of the patient's work in the field.

We consider our case report to be exceptional and rare, as a similar serious clinical course of leptospirosis has not yet occurred in our department. Most of the patients who were hospitalized in our department with a diagnosis of leptospirosis had only liver or renal involvement.

According to data from the Public Health Office, there is an average of 3–7 cases of leptospirosis per year (2017–2021) in Slovakia. In most cases, it was a disease that manifested in weakness ($n = 9$) and hepatopathy ($n = 9$). During this period, only one case of pneumonia was recorded (in 2020). As far as we know, no case of leptospirosis with multi-organ involvement has been recorded and described in Slovakia [18–20].

In Europe, leptospirosis is seasonal. Most cases occur mainly in August and September [21]. The patient in our case report was infected at the end of August, correlating with the highest seasonal incidence in Europe.

It is reported in the literature that the cause of liver damage in leptospirosis is a disruption of cellular cohesion, blockage of bile ducts, and acute inflammatory damage. This pathophysiology is consistent with the laboratory findings in the patient in this case report (hyperbilirubinemia, elevation of transaminases, and inflammatory parameters) [22].

According to O'Neil et al. pulmonary complications occur in 20–70% of cases [23].

Matos et al. analyzed the chest radiographs of 139 patients with leptospirosis. A total of 74.3% of patients had infiltrates on the X-ray image. Bilateral findings were present in 54.3% of cases [24].

Gulati et al. reported that leptospirosis is characterized by rapidly evolving, predominantly peripheral, diffuse, nodular, or confluent lung lesions, pleural effusions are uncommon, and pulmonary findings usually resolve within 15 days without permanent lung tissue damage. Lung damage was more common in smokers [25].

We observed a similar finding in the case of our patient, who was not a smoker. On the X-ray image of his chest, he had bilateral, numerous, spotted and reticular opacifications, mainly peripherally located and sometimes confluent.

In connection with the findings on the chest X-ray, we repeatedly carried out a PCR examination for the presence of the SARS-CoV-2 virus, as this radiological finding gave an impression of interstitial viral pneumonia and the patient's illness happened during the coronavirus pandemic (August 2021). At the end of the antibiotic treatment (14 days), the follow-up X-ray showed no pathological findings.

Lung damage can also manifest as bleeding and is related to the activation of the toll-like receptor by the *Leptospira*'s lipoproteins, often along with a decrease in platelets. Toll-like receptor 2 plays a major role in the development of pulmonary (and renal) manifestations of leptospirosis. *Leptospira* lipoprotein LipL32 triggers an inflammatory response by activation of Toll-like receptor 2. Toll-like receptor 2 may contribute to myocarditis in sepsis and may be involved in the pathogenesis of acute pancreatitis [26,27].

Pulmonary hemorrhages in our patient could not be ruled out with certainty since we did not perform a chest CT scan for technical reasons. However, this finding had no influence on treatment selection. Finally, heart involvement can be manifested by non-specific electrocardiogram abnormalities (prolongation of the PR interval, T-wave inversion, first-degree atrioventricular block) or by pericarditis or myocarditis. We did not find any signs of cardiac damage on electrocardiographic and echocardiographic examinations [28].

Acute renal failure is also associated with toll-like receptor activation and is reported to occur in 16%–40% of cases. Oliguria is a significant predictor of death [29].

In our case, acute renal failure was noted, characterized mainly by elevation of renal parameters (urea, creatinine), but with an excellent response to treatment. Although oliguria was not noted, renal parameters have gradually decreased during the course of hospitalization.

Thrombocytopenia has been described in the literature as a significant predictor of the development of acute renal failure. At the same time, a factor involved in the pathogenesis of hemorrhagic diathesis in leptospirosis, which is the leading cause of death in patients. Therefore, it is crucial to be aware of this pathology early in the course of the disease and to administer platelet transfusions early to prevent fatal complications [30,31].

In our patient, we administered two transfusion units of platelets at the beginning of hospitalization (on the second day). We observed the nadir of the platelet count on the third day of hospitalization ($18 \times 10^9/L$).

The authors Thalji et al., Cardoso et al. and Rao et al. also describe a similar course of human leptospirosis with multiorgan failure [32–34].

In our case, the diagnosis of leptospirosis was confirmed using two different microbiological tests. Using the ELISA method, the presence of antibodies of the Ig M class against *Leptospira interrogans* was established. The result was subsequently confirmed with an immunochromatographic test and the presence of Ig M antibodies against *Leptospira interrogans*.

In clinical practice, it would be appropriate to confirm the diagnosis with another microbiological method, especially using the “gold standard” in diagnosing leptospirosis. Unfortunately, this microscopic agglutination test is not available in our laboratory. However, it is important to note that the unavailability of this laboratory method did not negatively affect the patient’s management.

Another diagnostic option is serum testing using PCR, which we were also unable to implement due to its unavailability in our region (this applies to PCR of a urine sample as well).

In addition, it is necessary to mention that leptospiremia occurs only during the first phase of the disease and before the onset of symptoms and usually lasts no more than 7 days. The patient in this case, was admitted to the hospital at the stage of organ complications already, i.e., during the second so-called immune phase of the disease. Therefore, it was possible that the PCR test would already be negative, however, it also depended on the type of PCR test used [35].

5. Conclusions

Leptospirosis is a rare but potentially life-threatening disease in the temperate climate zones of Slovakia and Europe. The clinical course of the disease is very variable, ranging from a mild disease with flu-like symptoms to a severe disease with various organ complications. Due to the variability in symptoms, it can mimic a wide variety of other infectious diseases. In clinical practice, epidemiological history (focusing on being in nature and drinking water from a freshwater source) and disease symptoms (fever, mild hepatopathy, hyperbilirubinemia, etc.) play a critical role in early management. The aforementioned case report describes a severe course of leptospirosis in a patient that was not immunocompromised, with renal failure, pulmonary and liver involvement and the development of severe thrombocytopenia. The disease was confirmed with the ELISA method demonstrating positivity of *Leptospira interrogans* Ig M antibodies and the microscopic agglutination test positive as well. The treatment regimen consisted of 3rd generation cephalosporin (ceftriaxone) and symptomatic treatment. In conclusion, it is essential to note that even though this disease is rare in Slovakia, it is important to consider this zoonosis, especially in the differential diagnosis of a febrile illness with liver involvement.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki, and approved by the Institutional Review Board (or Ethics Committee) of Louis Pasteur University Hospital (protocol code 88/EK/22, approval 9.9.2022) for studies involving humans.

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: Not applicable.

Conflicts of Interest: The authors declare no conflict of interest.

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