A New Case of Weil Disease Confirmed in El Jadida, Morocco

M. Haraji, N. Cohen, H. Karib, A. Fassouane, Y. Dinar and R. Belahsen

Laboratoire de Biotechnologie, Biochimie et Nutrition, Faculté des Sciences d’El Jadida, Maroc
Laboratoire de Microbiologie et d'Hygiène des Aliments et de l'Environnement Institut Pasteur Maroc, Casablanca, Maroc
Unité HIDAOA, Département de Pathologie et de Santé Publique Vétérinaire, Institut Agronomique et Vétérinaire Hassan II, Rabat, Maroc

Corresponding Author: M. Haraji, Laboratoire de Biotechnologie, Biochimie et Nutrition, Faculté des Sciences d’El Jadida, Maroc

ABSTRACT
Leptospirosis is a bacterial disease that affects humans and animals. It is caused by bacteria of the genus Leptospira. The 23-year-old man was reported on December 15, 2010 to develop jaundice, fever, chill, headache, nausea, vomiting and muscle pain and admitted to Mohamed V Hospital in El Jadida attributed to Leptospira icterohaemorrhagiae and Leptospira hebdomadis. Residence in proximity to a landfill and an open sewer was found to be independent risk factors for acquiring disease.

Key words: Leptospirosis, jaundice, renal failure, zoonosis, diagnosis

INTRODUCTION
Leptospirosis is an emerging infectious disease and one of the most widespread zoonoses in the world (Levet, 2001) in both urban and rural areas especially during tropical climates (Vinetz, 2001), caused by pathogenic spiral bacteria that belongs to the genus Leptospira. Early diagnosis and the ability to differentiate leptospirosis from other diseases is important to reduce the risk of more serious infection or mortality (Cumberland et al., 1999). Human infections result from contact with contaminated soil, vegetation or water or with the body fluids of infected animals (Slack et al., 2006). Symptoms of leptospirosis include high fever, severe headache, chills, muscle aches and vomiting and may include jaundice, red eyes, abdominal pain, diarrhea or a rash (Mansour-Ghanaei et al., 2005). If the disease is not treated, the patient could develop kidney damage, meningitis (inflammation of the membrane around the brain and spinal cord), liver failure and respiratory distress. In rare cases death occurs. Many of these symptoms can be mistaken for other diseases. Leptospirosis is confirmed by laboratory testing of a blood or urine sample. Veterinarians, farmers and abattoir workers are at particularly increased risk for infection from contact with contaminated urine (Jaureguberry et al., 2005). Most cases occur in the warm season (Esmaeili et al., 2009) and in rural areas because leptospires can persist in water for many months. Transmission to humans occurs through penetration of the organism into the blood stream via cuts, skin abrasions or mucus membranes. Urine excretion of the organism by carrier animals, in particular rodents, is the primary environmental source of infections for humans. The Microscopic Agglutination Test (MAT) remains the reference serological method for diagnosis of leptospirosis with isolation providing the gold standard for definitive evidence of infection and is used to identify
the most probable serovar or serogroup that has caused an infection. The ELISA assays directed against IgM class antibodies are generally more sensitive than agglutination type tests but may be subject to variations in specificity (Cumberlaid et al., 1999).

The present study describes the clinical and laboratory findings of patient with leptospirosis admitted to Mohamed V hospital in El Jadida.

RESULTS AND DISCUSSION
Case definition and laboratory testing: This definition covers the multiple clinical and epidemiological features of leptospirosis and is provided on a standardised form to be filled by the clinician. This form is also used as a laboratory report sheet. For this patient, a blood sample (approximately 5 mL) was drawn 10 days after the onset of illness.

Patient with suspected clinical observation found in Mohamed V hospital, El Jadida on 15 December 2010. At admission, he was lethargic and clinically dehydrated presenting anorexia, vomiting, severe headache, failure to eat and brown urine. At physical examination, conjunctival congestion, skin and ocular jaundice (Fig. 1).

Time between onset of symptoms and admission, length of hospital stay, treatment, need of dialysis and complications were analyzed. Severity of disease was analyzed through clinical and laboratory findings. Clinical investigation included a record of all signs and symptoms presented by the patient, as well as arterial systolic and diastolic blood pressure at hospital admission. Laboratory data included an assessment of serum urea, creatinine, potassium, bilirubin, transaminases, creatinokinase, lactate dehydrogenase, total blood count and prothrombin time.

Axillary temperature was 37.8°C At ultrasonography examination, the morphology and size of kidneys and the liver were normal. Initial laboratory evaluation showed leukocyte levels of 17.6.10³ mm⁻³, haemoglobin at 12.13 g dL⁻¹, serum urea at 2.96 g dL⁻¹, creatinine at 58.6 mg dL⁻¹, direct bilirubin 189 mKat L⁻¹, indirect bilirubin 121 mKat L⁻¹. The levels of serum glucose, lactate dehydrogenase and creatine kinase were normal. Urinalysis showed microscopic haematuria and leukocyturia. As well as a leukocyte count of 17.6.10³ mm⁻³ being remarkably high for this case of leptospirosis, the size of the neutrophil fraction is noteworthy and the patient presented an acute renal failure.

Fig. 1: Patient with jaundice
Table 1: Identification of serogroups with the MAT

<table>
<thead>
<tr>
<th>Serogroups</th>
<th>Titer</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Leptospira australis</em></td>
<td>Négatif</td>
</tr>
<tr>
<td>L. autumnalis</td>
<td>1/100</td>
</tr>
<tr>
<td>L. bataeae</td>
<td>Négatif</td>
</tr>
<tr>
<td>L. canicola</td>
<td>1/60</td>
</tr>
<tr>
<td>L. canicola</td>
<td>Négatif</td>
</tr>
<tr>
<td>L. icterohaemorrhagiae</td>
<td>1/1600</td>
</tr>
<tr>
<td>L. hardjo</td>
<td>Négatif</td>
</tr>
<tr>
<td>L. hebdomadis</td>
<td>1/400</td>
</tr>
<tr>
<td>L. icterohaemorrhagiae</td>
<td>1/1600</td>
</tr>
<tr>
<td>L. panama</td>
<td>Négatif</td>
</tr>
<tr>
<td>L. patoc (non pathogenic)</td>
<td>1/4000</td>
</tr>
<tr>
<td>L. pomona</td>
<td>Négatif</td>
</tr>
<tr>
<td>L. pyrogenes</td>
<td>Négatif</td>
</tr>
<tr>
<td>L. sejroe</td>
<td>Négatif</td>
</tr>
<tr>
<td>L. tarassovi</td>
<td>Négatif</td>
</tr>
</tbody>
</table>

We used the Cockcroft formula (Cockcroft and Gault, 1976) for calculating creatinine clearance to assess the degree of acute renal failure (Abd-El-Latif et al., 2007). ARF is a common renal complication in leptospirosis. The incidence of leptospirosis-induced ARF depends upon the criteria for diagnosis (Khositseth et al., 2008).

**Serologic case confirmation:** Sample and his respective transmission form, were referred to Cerba confirmation laboratory for second level confirmatory testing and then to Paris Pasteur Institute for MAT reference testing.

Leptospira immunoglobulin M (Ig M) was positive by Enzyme-linked Immunosorbent Assay (ELISA) 63.9 U mL⁻¹.

It is important to identify the serovars associated with human infections by leptospires. High titers of anti-leptospira antibodies were detected by the Microscopic Agglutination Test (MAT). It was also positive for *L. icterohaemorrhagiae* (titer 1/1600) and *L. hebdomadis* (titer 1/400): Human infections with this serogroup are more likely when the local population of the natural reservoir hosts, voles and field mice, is high (Table 1).

Therapy included hydration, The patient had received oral clopram ® at a dosage of 200 mg twice daily for 7 days, intravenous injection of Azantac ® twice daily in combination with intravenous injection of penicillin G sodium (10 million units day⁻¹) for 10 days. Two sessions of dialysis were indicated in this patient that remained oliguric after effective hydration. After such a therapy, the febrile illness recovered completely. Patient’s hospital discharge occurred two weeks after admission.

**Prevention** This infection should be considered in people who have occupational exposure to cattle and measures taken to minimise infection should include avoiding contamination of broken areas of skin and penicillin prophylaxis in key operators. Wet weather in summer seems to increase the incidence of this infection.

The risk factors acquiring disease are amenable to focused interventions which include provision of closed drainage systems for sewage and reduction of rodent populations in the
peri-domiciliary environment. Rodent control can also minimize the risk to humans (Massawe and Mkundi, 2011) and Environmental control of transmission may help to greatly reduce the incidence of severe leptospirosis. Human leptospirosis can be controlled by reducing its prevalence in wild and domestic animals.

Despite its limitations, this analysis may help generate hypotheses for in-depth studies that aim to identify exposure risks; it may also provide a foundation for future data collection that will lead to improvements in intervention strategies. However, further research is needed to assess the importance of other factors, including access to and quality of care. Because urban epidemics of leptospirosis are characterized by high mortality within the first 48 h of hospitalization (Ko et al., 1999), prevention and improved detection are required to improve clinical outcomes at the population level. Clearly, urban slums require adequate sewage containment and treatment urgently.

A better understanding of the clinical and para-clinical characteristics of leptospirosis (Aliyan et al., 2006) should improve the recognition and appropriate treatment of the disease. A detailed knowledge of the biology of Leptospira and the pathogenesis of leptospirosis lags behind that now elucidated for many bacterial infections.

In conclusion, prevention is largely dependent on sanitation measures that are difficult to implement, especially in developing countries.

REFERENCES

74