A Case Study: Leptospirosis In Malaysia

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A Case Study: Leptospirosis In Malaysia

Author(s): Lim J K, Murugaiyah V A, Ramli A, Abdul Rahman H, Mohamed N, Shamsudin N, Tan J C

Abstract

Leptospirosis is one of the common re-emerging zoonotic diseases transmitted from infected animals to humans. It is a bacterial infection caused by a species of pathogenic leptospiragenus called Spirochaetes. The very first case of human leptospirosis in Malaysia was discovered by Fletcher in the year 1925. Pathogenic leptospires can spread through the urine of carrier animals to the environment. Flu-like symptoms such as severe headache, sudden fever of 39°C and above, eyes inflammation, muscle aches, diarrhea, fatigue, nausea and vomiting, chills, rigors and maculopapular rashes can be observed. Several antibiotics that are used to treat this disease are ampicillin, ceftriazone, doxycycline and penicillin. A good prevention for the spread of leptospirosis is by practicing good sanitation and avoiding any direct contact with urine - contaminated soil and water. The recurrence of leptospirosis infection in the recent years proves to be a source for concern. Malaysian government should continue to work out measures to tackle this infection.

Introduction

Leptospirosis is a bacterial infection caused by a species of pathogenic leptospira genus called Spirochaetes. [1] This Spirochaetes are known as leptospira interrogans. [2] This disease is known worldwide as the most common re-emerging zoonotic disease [3] which is contagious and can be transmitted from an infected animal to human. [4] Classification of bacteria is based on serological analysis and there are around 200 studied serovars. However, not all of the serovars are harmful to humans. The pathogenic bacteria are generally from the leptospira interrogans genomospecies and there are eleven detected species of leptospira while thirty-seven serovars are found from wildlife and humans in Malaysia. [5, 6] Rodents are known as the main source of spirochetes that are transmitted to humans. [5] However, humans are known as “dead-end” hosts. [7] This is because the spreading of infectious bacteria between human and human or human and animal never happens. Transmission of bacteria can be direct or indirect. [1, 2] Bacteria are commonly transmitted through urine produced by carrier animal. Exposure to the bacteria in the urine of these animals enables the leptospira spirochetes to enter the body through cuts, wounds, and mucous membranes such as the eyes, mouth and nose. Besides that, ingestion of contaminated drinking water and food may cause infection as well. [3] Generally, reasons related to the rising of infection are occupational exposure, poor sanitation, climate changes, recreational activities as well as management of wild animals. [8] In Malaysia, leptospirosis is an old disease as the very first case of human leptospirosis was discovered by Fletcher in the year 1925. This discovery indicated that leptospirosis is an endemic disease in Malaysia. [5] Through the years, the number of cases continues to increase significantly from 2004 to 2010 with 263 cases and 20 deaths in the year 2004, to 1976 cases and 69 deaths in the year 2010. [4, 5, 9] In Malaysia, humid environment favors the growth of pathogenic Spirochaetes. [10] With tropical weather and flooding that occur frequently, Malaysians are always at risk of exposure to water and soil contamination with the urine of rats [4] without being aware of this. This causes records of a high incidence of leptospirosis, especially after floods. [11] Occupational targets like farm workers, campers, hikers, veterinarians, drainage cleaners and others who are frequently introduced to disease-infected environments are at a higher risk [5, 7] of acquiring this disease.

Human Leptospirosis in Malaysia

Contrary to common believe, leptospirosis is not a new disease in Malaysia. In fact the first diagnosed human case of leptospirosis in Malaysia was in 1925 by Fletcher. He was able to identify serovars leptospira icterohaemorrhagiae, leptospira hebdomadis and leptospira progres from 21 patients. [5] In 1927, Fletcher and Kanagarayer were able to demonstrate Leptospires in four patients from Kuala Lumpur General Hospital. In a study by Robinson and Kennedy in 1956, 31 cases of leptospirosis were detected among British army personnel in Malaysia. Twenty- nine of these cases were proven to be leptospirosis, either by culture or serology. [12, 13] Another study carried out by McCrumb in 1957 looked into febrile illness in 614 military personnel operating in the jungle during the
Malayan emergency. In their investigation, leptospirosis was found to be the most common cause of fever in soldiers, accounting for 35% cases admitted for fever to a military hospital. In fact, throughout the years 1953 to 1955, at least 30 pathogenic leptospiral serovars have been identified from both the military personnel and civilians. [14] These studies showed the importance of leptospirosis as a febrile disease among military personnel and civilians alike in Malaysia. [13]

Because the actual features of leptospirosis did not always conform to the generally accepted picture of Weil’s disease and clinicians sometimes fail to consider it in the differential diagnosis of febrile illnesses, leptospirosis is much more common in Malaysia than is generally realized and the disease can be mild and may even be subclinical and deceptive. [15]

Since 1986 no investigations were made on human leptospirosis in Malaysia. Institute for Medical Research (IMR) is the main institute that carried out routine work on the diagnosis of human cases based on serum samples submitted from clinics and hospitals throughout the country. A retrospective study on the incidence of human leptospirosis in Malaysia was carried out from 1983 to 1998. The incidence rate in Malaysia is estimated to be between 2– 5 per 100,000 populations. The overall incidence of leptospirosis was 13%, with the Indians showing the highest incidence (16.7%) followed by the Malays (11.5%) whilst the Chinese (5.9%) were the least affected by the disease. The percentage of males (81.1%) affected were higher than females (18.9%) in a male female ratio of 4:1. The 20-29 years age group (17.1%) showed the highest prevalence whilst the young (less than 10 years), and old (above 60 years) groups showed low prevalence. This study indicated that human leptospirosis is probably an endemic infection in Malaysia. [16]

However there has been a significant increase in recent years. According to the statistics from the Ministry of Health, in 2006, an estimated 527 cases of leptospirosis is recorded and in the following year 2007, an increase in cases is found with a total of 929 cases. The Ministry of Health had reported that the number of confirmed cases had increased from around 263 in 2004 to 1418 in 2009. [17]

Statistics released by the Ministry of Health, Malaysia in Table 2 shows a gradual increase in the cases of leptospirosis especially in the states of Perak, Selangor and Pahang. As shown in figure 1, although fatality rates have decreased, incidence rates continue to rise, drawing the attention of the government and public alike. [18]

Recently in 2010 as well, the country was taken aback by the death of 6 individuals due to leptospirosis in Hutan Lipur Lubiuk Yu, Maran. [19] In Sibu, the bacteria causing the deadly leptospirosis disease has been detected on august 2010. [20] There were two places affected by this infection which is Permai Road recreational lake and at the ponds at the Junaco Park National Service Training Camp. [20] Besides that, three national service camps in Perak have been ordered to stop water activities to prevent leptospirosis. [21]

A recent wave beginning in July 2011 when eight people, who were among a search and rescue group looking for a drowning victim in a recreational area in eastern Pahang state have died of the disease and another water-borne bacteria. The latest victim was a 17-year-old boy who died after swimming in northern Kelah state. [22] Other than that, three popular picnic spots in Kelah have been ordered to close temporarily in the wake of an outbreak of leptospirosis. [23] The three spots are Puncak Janing in Kuala Nerang, Lata Bayu in baling and Buitk Wang in Kubang Pasu. The victim who has died came for a picnic at Lata Bayu with two friends, who also contracted the disease. [23]

Thirty eight serovars have so far been described in Malaysia and these serovars are divided into 13 serogroups namely australis, autumnalis, bataviae, canicola, celledoni, grippotyphosa, hebdomanis,icterohaemorrhagiae, javanica, pomona, pyrogenes, sejroe and tarassovi. [24]

In table 2, a study done in 1975 showed the 1,362 strains isolated from soil and water in West Malaysia (Selangor and Pahang) could be divided into 13 serogroups comprising a total of 29 serovars. The most common strain of leptospirosis found in Malaysian waters is the strain Leptospirosis Icterohaemorrhagiae serotype mankarsoa, smithii and birkin. [25]

A cross-sectional serological survey of domestic animals in West Malaysia carried out in 1987 revealed that 25.5 % of the animals examined carried antibodies to the strain leptospirosa interrogans. From this strain, Sejroe serogroups was shown to be the principal one involved in cattle and buffaloes, and to a lesser extent the Tarassovi and Pomona serogroups. Almost 16% of the domestic animals examined also had evidence of infection to serovar Hardjo. [26]

In a more recent study in 2009, a single leptospira strain (designated Bejo-Iso9T) was isolated from a soil
sample taken in Johor, Malaysia by researchers at the Faculty of Veterinary Medicine, Universiti Putra Malaysia. This is a new discovery of a novel serovar because serologically, the strain Bejo-Isos9T produced titers towards several members of the tarassovi serogroup, but was found to be serologically unique by cross-agglutinin absorption test. [10]

Modes of transmission

Leptospirosis is spread by Leptospires which are pathogenic or saprophytic. [27] Pathogenic leptospires are normally found in the renal tubules of host animals while saprophytic leptospires are commonly isolated from wet or humid environments. Saprophytic leptospires species normally contaminate surface waters and do not cause infections to humans as they only feed on organic matter in water. [28] Therefore, they do not require a host. On the other hand, pathogenic Leptospires need a host in order for them to survive and reproduce. [28] Pathogenic Leptospires can spread through the urine of carrier animals to the environment. The animal hosts that carry this specific pathogenic leptospires are not harmed themselves. They are also known as natural maintenance hosts. [27, 29]

Leptospirosis can spread directly or indirectly from hosts in the contaminated environment. Excretion of urine from the animal host causes the soil and water to be contaminated. [30] Contaminated water is then ingested by humans who then become infected. Besides that, food that is contaminated by urine can also transmit the pathogenic Leptospires to human. Other than the indirect ingestion of food and water, the Leptospires can invade the body when in direct contact with Leptospires through cuts as well as wounds. [30] It also enters the human body through nasal, oral and conjunctiva mucosa if humans are in contact with contaminated water for a long period of time. [30, 31] After penetrating the human body, Leptospires then pass into the blood stream and from the blood stream the Leptospires attacks body tissues and organs. [27]

Leptospirosis can spread through air as well. [19] Evaporation of urine containing Leptospires into air droplets can be inhaled by humans and thus cause infection. Even though transmission of the disease between human and human rarely happen, it should be noted that sexual intercourse as well as breast feeding can cause the transmission of the pathogenic Leptospires from human to human. [30, 19] In addition, an infected mother is able to pass the pathogenic Leptospires to her foetus. Excretion of Leptospires in the urine of humans also occurs continuously up to 11 months. [31]

The sources of pathogenic Leptospires are generally rodents and wild animals such as cattle, pigs, rats, buffaloes and dogs. They serve as main reservoirs for the pathogenic Leptospires. These infected maintenance hosts may excrete leptospires via their urine for up to a few months or years. [30]

The incidence of leptospirosis is seasonal at which most cases are reported during the rainfall season and after floods. [11] Occupational exposures as well as recreational activities are mostly related to the incidence of cases. Veterinarians, zoo keepers, pet shop owners, slaughterhouse workers and any other occupations that frequently come into contact with wild animals are at a higher risk as compared to others. Populations that work in an environment with water, soil and mud for example, military, farm workers, fishing industry and plumbers are equally at risk. [19, 30]

Signs and symptoms of Leptospirosis infection

The incubation period for leptospirosis is normally 5-14 days and within a range of 2-30 days. [27] However, unless the volume of bacteria invading the body is higher than the usual volume, a patient basically does not show signs and symptoms in less than 24 hours. [28] The infection is normally systemic and infects the whole body. Generally, there are two types of infection that portray different signs and symptoms, which are anicteric leptospirosis and icteric leptospirosis. [30, 31] 90 percent of cases are anicteric leptospirosis, which means that jaundice does not occur in this infection. Patients who suffer from anicteric leptospirosis infection undergo phase 1 and phase 2 of the illness. [28, 31] In anicteric form of leptospirosis, the illness is always related with aseptic meningitis. [30] This less severe type of leptospirosis is rarely fatal but is associated with pulmonary haemorrhage even without jaundice and may lead to death.

In phase 1, also known as acute or septicemic phase, flu-like symptoms such as severe headache, sudden fever of 39°C and above, eyes inflammation, muscle aches, diarrhea, fatigue, nausea and vomiting, chills, rigors and maculopapular rashes are seen. [32] If the signs and symptoms mentioned above only lasts around 3-5 days, the patient is said to have recovered. However recovery may not be permanent. During phase 2 or the immune phase, anti-leptospira antibodies start to multiply such that organisms of
Leptospirosis can be found in the patient's urine excretion. During this phase, infected patients fall sick again lasting for up to 30 days or more. However, some patients do not develop into phase 2 of the illness. The other 10 percent of cases develop into icteric leptospirosis which is also known as Weil's syndrome. This second phase is much more severe than anicteric leptospirosis. A majority of the signs and symptoms are similar to anicteric leptospirosis but can develop into a more fatal condition. Organs including the liver, kidneys, brain, heart and central nervous system are harmed within 10 days. Jaundice and other signs of icteric leptospirosis become obvious at the early third week and late ninth week after the infection. This fatal infection tends to prolong the period of severe fever, jaundice, azotemia, hypotension as well as haemorrhagic vasculitis.

Consequences of Leptospirosis

Leptospirosis is a systemic infection with the primary lesion being endothelial cell membrane defects of small vessels leading to haemorrhage, ischaemia and secondary organ pathology. The most famous consequences of this infection is Weil's disease or syndrome which has more severe manifestation with symptomatic hepatic and renal failure and bleeding diathesis associated with a 10%-50% mortality rate. The complications are normally illnesses like meningitis, kidney failure and jaundice. However there are also instances where patients develop bleeding of the lung. Weil syndrome is the severe form of leptospirosis and primarily manifests as profound jaundice, renal dysfunction, hepatic necrosis, pulmonary dysfunction, and haemorrhagic diathesis. It occurs at the end of the first stage and peaks in the second stage. However, the patient's condition can deteriorate suddenly at any time. Often, the transition between the stages is obscured. Criteria to determine the development of Weil disease are not well defined. Weil syndrome carries a mortality rate of 5-10%. The most severe cases of Weil syndrome, with hepatorenal involvement and jaundice, have a case-fatality rate of 20-40%. The mortality rate is usually higher for older patients. Jaundice as its name implies is a prognostic factor and is related with liver function failure at a subcellular level, thought to be enzyme or toxin mediated. Vascular and renal dysfunction accompanied by jaundice develops for 4-9 days after onset of disease, and jaundice may persist for weeks. Patients with severe jaundice are more likely to develop renal failure, haemorrhage, and cardiovascular collapse. Haemorrhagic diathesis is infection is associated with serovars L. icterohemorrhagiae and Copenhagen. Hepatomegaly and tenderness in the right upper quadrant may be present. Oliguric or anuric acute tubular necrosis may occur during the second week due to hypovolemia and decreased renal perfusion. Multiorgan failure, rhabdomyolysis, adult respiratory distress syndrome, haemolysis, splenomegaly, congestive heart failure, myocarditis, and pericarditis may also occur. Leptospirosis is a common cause of acute renal failure, which occurs in 16% to 40% of cases. A distinction may be made between patients with prerenal azotemia, and those with acute renal failure. Patients with prerenal azotaemia may respond to rehydration, and decisions regarding dialysis can be delayed for up to 72 h. In patients with acute renal failure, oliguria was a significant predictor of death. Serum amylase levels are often raised significantly in association with acute renal failure but clinical symptoms of pancreatitis are not a common finding. Necrotizing pancreatitis has been detected at autopsy. Thrombocytopenia, platelet count of severe vasculitis amplified by impaired liver production of prothrombin, albumin and globulin causes haemorrhagic and circulatory manifestations. Anaemia, thrombocytopenia, leucocytosis with neutrophilia is common haematological laboratory findings. Renal pathology secondary to ischaemia, results in azotemia, oliguria and anuria. Rhabdomyolysis especially of the gastrocnemius muscle, hence tender calves, releases creatinine kinase and myoglobin which have been suggested to increase the severity of renal failure.

Diagnosis of Leptospirosis

A patient is suspected to acquire leptospirosis when experiencing symptoms for about 1-2 weeks after exposure to the urine of the carrier animals. This disease can only be confirmed through blood tests to detect the presence of leptospira bacteria. Leptospira bacteria can be isolated from blood, urine or cerebrospinal fluid (CSF) during the first phase of the illness. They are several criteria to confirm if a patient has the leptospirosis disease. Firstly, it may be confirmed through the isolation of leptospira from a clinical specimen. Besides, leptospirosis infection is confirmed if there is fourfold or greater increase in leptospira agglutination titer between acute and convalescent phase serum specimens obtained in 2
weeks or more apart and studied at the same laboratory. [36] Other than that, urine analysis is also reliable. If a urine test gives a positive result during the second week of illness and continues on up to 30 days, the patient is confirmed to have leptospirosis. [37]

If there is a single case or household cluster case, some information should be collected. Firstly, the occupation of the suspected person must be identified. This is because farmers, veterinarians, sewer and abattoir workers have a higher risk of contracting the disease. Next, recent exposure to water or soil potentially contaminated with the urine of the carrier animal can be determined. Besides that, recent contact of the individual with animal tissue or fluids is looked into. Lastly, the patient is checked to have recently travelled to endemic or epidemic areas of the disease or not. [35] For the purpose of notification, all probable and confirmed cases must be notified to the nearest Health District Office within a week of the date of diagnosis.

Treatment and management of Leptospirosis

Leptospirosis can be treated only if it is diagnosed early to avoid complication. Untreated patient can lead to a more severe and potentially fatal stage. [38] It is treated by primarily using antimicrobial therapy. Treatment with antibiotics is started before confirming if a patient has leptospirosis. This is because test and diagnosis may take a long time to process and the condition of the patient can become more serious if they continue to wait. Several antibiotics that are used to treat this disease are ampicillin, ceftriazone, doxycycline and penicillin. Doxycycline is an excellent choice of initial antimicrobial treatment for an individual. [39] Oral doxycycline is used if the infections do not require hospitalization. The use of antibiotics decreases the duration of fever and most symptoms. However, there is considerable doubt as to whether antibiotic therapy is efficacious for leptospirosis. Nevertheless, a significant improvement is observed if the treatment is started on the fifth day or earlier. [32] For mild illnesses, which include suspected cases, the amount of doxycycline needed is 100mg twice daily for seven days, while 2g is administered daily for seven days if amoxicillin or ampicillin is chosen. For more severe cases, 1 gm of ceftriaxone is given intravenously for seven days. However, if there is indication of organ dysfunction such as renal, hepatic, pulmonary, haemorrhagic and neurological to the patients, the patient should be transferred to referral healthcare. [40]

For hospitalized patients, intravenous penicillin G is given. [41] Any organ system can be affected in severe leptospirosis leading to multiple organ failure. It is important for patients to be managed in a monitored setting because their condition can rapidly progress to cardiovascular collapse and shock. [41] For patients with kidney failure, dialysis should be carried out. [38] Even though there are antibiotics that can be used to treat the disease, taking steps to prevent infection is still the best option. After all, prevention is better than cure. Since there is currently no vaccine that is effective for any of the leptospirosis disease yet, avoiding activities that expose to the bacteria is the way to go. If an individual who frequently comes into contact with potentially contaminated water or soil, protective clothing should be worn.

Prevention and control

According to the Guidelines for the Diagnosis, Management, Prevention and Control of leptospirosis in Malaysia, prevention and control should be targeted at 3 different sources, which are the infection source, routes of transmission between the infection source and human host, and prompt and proper treatment of infection.

The Malaysian government has taken the first step in order to create awareness among the public regarding the leptospirosis disease and its prevention. The Ministry of Health has organized health education activities to motivate the public about taking preventive actions. Eradication of leptospirosis is not the best treatment after the occurrence of disease, but the most important step is the preventive action taken before the infection itself. [22] The prevention of leptospirosis must be tackled at the source of the infection. [42] Exposure through water contaminated by urine from infected animals is the most common route of infection and rodents are animals that are recognized as the major reservoirs in the transmission of leptospirosis. Therefore, establishing rodent control programs is one of the essential components in prevention and control of this disease. [3]

The leptospirosis bacteria commonly infect animals such as rodents. Hence, in order to diminish the incidence of leptospirosis infection, the public must practice some preventive actions such as practicing good personal hygiene. Some practical steps that can be taken include wearing of footwear to prevent the transmission of leptospirosis bacteria through feet. Besides that, elimination of infected animal reservoirs at home and occupational site are steps of practicing good sanitation. [22] The use of rodenticides is also
successful in minimizing the risk of disease transmission. [3]
Since soil and water are easily contaminated with infected animal urine such as rats, it is best to avoid any direct contact with this soil and water. [42] As rats are the major hosts of these bacteria, one of the steps to control this disease is by controlling the rat population. Therefore, practicing good sanitation can control the rat population. For high risk groups including farmers, standard occupational hygiene such as wearing special protective attire, shoes and gloves at cattle farms should be practiced. [22, 43]
Another preventive action is the use of chemoprophylaxis measure. For leptospirosis, doxycycline is normally used as a prophylactic measure. [42] Doxycycline is an antibiotic that is used to diminish the infection for short term protection in high-risk environments among people who have a strong likelihood of exposure. [3, 4] It is more effective as chemoprophylaxis if doxycycline is given 200mg once a week. [4] It was found that administration of doxycycline can reduce the rate of symptomatic leptospirosis from 6.8% to 3.1% and the percentage of preventive efficacy is about 54%. However, this treatment did not reduce the rate of infection. [44]
Vaccination of domestic animals is another control measure that can be used to prevent the transmission of leptospirosis to human. This type of prevention provides certain protection, but it is not fully effective because some vaccinated animals that have been infected may excrete the bacteria in their urine. [43] Even though vaccination cannot eradicate leptospirosis, it is still useful in minimizing the transmission of leptospirosis infection from animals to humans. After decades of research, there is still no effective vaccine for human use. [4]

Conclusion

In summary, leptospirosis is not a new disease Malaysia but has been around since 1925. Its recurrence in the recent years proves to be a source for concern. Therefore, the Malaysian government should continue to work out measures to tackle this infection so that its incidence does not continue to rise. Although there are antibacterial treatments available, there is yet to be a vaccine available for prophylaxis treatment of leptospirosis in humans. Thus, it is essential for the general public to be informed of steps to take to prevent the spread of leptospirosis in the country.

Acknowledgement

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Illustrations

Illustration 1

Table 1

Table 1: Cases of Leptospirosis in Ministry of Health Hospitals, Malaysia from Year 2004 to 2009

<table>
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<tr>
<th>No</th>
<th>State</th>
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<th>2008</th>
<th>2009</th>
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<td>143 (3)</td>
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<td>280 (19)</td>
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</tbody>
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(1) indicates number of death
Source: Guidelines for the Diagnosis, Management, Prevention and Control of Leptospirosis in Malaysia. Ministry of Health, Malaysia 2011 [1]:3

Illustration 2

Table 2

Table 2: Pathogenic leptospires isolated from Malaysian waters and wet soils

<table>
<thead>
<tr>
<th>Serogroup</th>
<th>Serovar</th>
<th>Isolates within locations (%)</th>
<th>Selangor jungle</th>
<th>Selangor ricefield</th>
<th>Selangor mining pool</th>
<th>Pahang jungle</th>
<th>Sabah jungle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Icterohaemorrhagiae</td>
<td>mansanoi*</td>
<td>18.3</td>
<td>23.2</td>
<td>2.9</td>
<td>8.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>smithii</td>
<td>2</td>
<td>1.1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>brnk</td>
<td>1.1</td>
<td>1.1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Austriais</td>
<td>australis*</td>
<td>13.5</td>
<td>7.1</td>
<td>94.1</td>
<td>6.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Betaviae</td>
<td>paidan</td>
<td>11.6</td>
<td>7.1</td>
<td>5.9</td>
<td>1.9</td>
<td>58.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>bataeviae</td>
<td>3.7</td>
<td>5.9</td>
<td>9.7</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Canicola</td>
<td>jeness</td>
<td>8.1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td>shuffneri</td>
<td>4.6</td>
<td>1.8</td>
<td>31.7</td>
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<tr>
<td></td>
<td>malayai*</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>sumneri</td>
<td>0.8</td>
<td>3.8</td>
<td>8.7</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*The identity of representative strain was confirmed by agglutination adsorption tests
Illustration 3

Figure 1

Figure 1: Incidence and case fatality rates of Leptospirosis in Malaysia
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