Leptospirosis: A case study

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Abstract

Leptospirosis is rare and potentially fatal and can be confused with meningitis and hepatitis due to similar symptoms. Most patients experience fever, myalgias and general weakness. A small proportion develop multiple organ failure in severe cases. We report a case of leptospirosis infection that was encountered in a 58 year old man who acquired this infection from an unidentified source in a non-endemic leptospirosis region in Australia. He had a delayed diagnosis and further manifestation of acute symptoms. Although he recovered, he required admission to intensive care and haemodialysis support. This case highlights the difficulties of diagnosis with non-specific symptoms such as persistent rigors, headaches and lethargy, and the importance of considering leptospirosis in the clinical evaluation by medical, support staff and nephrology clinicians.

Key Words

Leptospirosis, acute kidney injury, Weil's disease, nursing

Introduction

Human leptospirosis is an acute febrile illness found around the world. Infections primarily occur in tropical and sub-tropical climates (Kobayashi 2005). Rodents, especially rats due to their alkaline urine, are the most common reservoir of leptospiras (Visith & Kearkiat 2005). Certain occupational groups such as vetinaranians, farmers, fisherman and plumbers can be considered at increased risk. (Durmaz Cetin et al 2004).

People suffering from leptospirosis are known to experience a rapid deterioration in their clinical course as most serological tests for this disease do not yield a positive result for about a week from the onset of illness (Kobayashi 2005). This highlights the importance of ruling out all other possibilities for infection, obtaining a good clinical history and the provision of rapid medical intervention. As the disease is not common, especially in non-endemic regions, the diagnosis may be missed (Bal 2005).

The severe form of leptospirosis is also known as Weil's disease. It is characterised by a rapid onset of liver and kidney failure resulting in a high mortality rate. Common clinical features include fever, headache, myalgia and vomiting. Leptospirosis is a biphasic illness which means after the initial symptoms there is a brief period where the patient is afebrile. Fever returns usually after 12 -48 hours in the second phase. This is then accompanied by jaundice and renal failure (Bal 2005).

Case Report

A 58 year old male, Mr D, was admitted to the medical ward via his general practitioner (GP) after 5 days of lethargy and fevers. During this time Mr D suffered from lower abdominal pain, 24 hrs of myalgia and back ache, right sided headache, persistent vomiting and high fevers. All symptoms spontaneously resolved until a subsequent admission 24 hours later when Mr D presented with further high fevers (39.9 C), lethargy and vomiting. His condition rapidly deteriorated with rapid on set of oliguria and abnormal liver function tests and was subsequently admitted to the Intensive Care Unit (ICU).

On admission to ICU he remained febrile at 38.8 C with a blood pressure of 120/65, P86. Blood cultures, serology, electrolytes and liver function tests (ELFT) and full blood count (FBC), airborne virus screen and chest x-ray and abdominal ultrasound were also taken. A nephrology review was requested by the intensive care team 48 hours after initial admission. This was due to the decreasing renal function and ongoing septic symptoms; following this review by the nephrologist a Leptospirosis test was ordered.

Leptospirosis was suspected due to the combination of hepatic and renal dysfunction with thrombocytopenia and generalised septic symptoms. Obstructive jaundice and sepsis were suspected at this early stage. Four antibiotics (gentamicin, vancomycin and flagyl) and fluids were also ordered.

During the next 24 hours renal failure and marked jaundice with high bilirubin levels continued with a marked fall in platelets (Table 1). The patient's febrile episodes continued. Mr D became hypotensive 95/55. Following further nephrology review dialysis was to commence following insertion of a vascular catheter. Mr D was subsequently

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dialysed daily for 5 days up to 4 hours a session. Packed cells and platelets were administered each session. It was during this period his initial Leptospirosis test returned a negative result. Mr D remained on antibiotics, intravenous fluids in ICU. His urine output improved (50 mls per hour) after day four. At day six he received his final dialysis treatment as his urine output rapidly returned (100 - 150 mls per hour). His blood results also showed marked improvement as shown in Table 1.

Mr D was discharged home after 16 days in hospital under the care of the treating nephrologist and his GP

Discussion

Leptospirosis is rare but can be noted in tropical regions. Infected mammalian hosts such as rodents, swine, and household pets can excrete the leptospirosis in their urine (Theilen et al 2002). They infect humans by invading through cut or abraded skin, mucus membranes or the conjunctivae. In this case there is only speculation as to where the initial infection occurred. Occupational exposure, recent travel and infected pets were all ruled out.

Mild hypotension during the initial phase of the infection is not unusual. This could be attributed to decreased vascular resistance by a higher bacterial load. Then hypotension is usually followed by renal failure then pulmonary involvement subsequently follows (Niwattayakul 2002). However these symptoms follow most other septic symptoms in severe cases. Renal Failure is observed in 44 – 67% of patients with Leptospirosis (Durmaz Cetin 2004). When the disease is identified and treated quickly renal function resolves completely.

The symptoms of Leptospirosis are non-specific and laboratory tests can (be) in some cases be negative. It is therefore important to recognise clinical symptoms during initial diagnosis. The association of acute renal failure and Jaundice should lead to the clinician suspecting Leptospirosis (Durmaz Cetin 2004).

Severe Leptospirosis may carry a high mortality if not treated early. Early markers include Hypotension, oliguria, hyperkalemia, hemoptysis, metabolic acidosis and thrombocytopenia (Bal 2005). Treatment includes the use of antibiotics (cefotaxime, doxycycline, ampicillin, and amoxicillin). There is no vaccine for this disease. Clinical recovery happens in a relative short time. It has been noted that fever was only present on 17% of patients beyond 5 days and normal levels of platelets and bilirubin or at 1/3 of its maximum value occurred within 2 weeks (De Francesco 2003). It must also be noted that mortality can reach almost 50% when complications such as acute respiratory failure and multiple organ failure complicate recovery (Covic 2003).

Upon discharge Mr D returned to the care of his GP with regular consultation with his nephrologist. The patient recovered fully from this episode and has returned to normal activities without any lasting effects. It is interesting to note that his initial leptospirosis test turned out negative thus leaving the clinicians to be guided by the patient's physical symptoms. It was a subsequent test after discharge that showed a positive result.

Conclusion

Early clinical intervention and appropriate evaluation of the affected patient need to be considered when Leptospirosis is suspected. The case study highlights the importance of a sound clinical diagnosis taking into account the region and environmental factors as well as the need to treat the acute symptoms with a combination of antibiotics and haemodialysis in the more severe cases.

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Table 1

Laboratory Data obtained after hospitalisation.													
Date	2/03/05	3/03/05	4/03/05	5/03/05	6/03/05	7/03/05	8/03/05	9/03/05	10/03/05	12/03/05	13/03/05	15/03/05	18/03/05
Haemoglobin (135-180 g/L)	130	121	109	113	136	126	120	111	119	94	93	87	92
Hematocrit (0.38 - 0.52)	0.4	0.35						0.32	0.35	0.29		0.26	0.27
White Blood Cells (4.0 - 11.0)	5		8.4	12.6	10.4	9.5	9.6	9.5	11.5	9.8	9.6	8.7	8
Platlets (150 -450)	70	42	24	25	45	49	64	73	142	207	249	270	255
Bilirubin (<20umol/L)	86	101	119	167	174	64	44	39	41	27	26	24	21
GGT (<70)	257	254	191	327	444	354	271	226	197	117	105	83	71
ALT (45 units/L)	93	79	76	112	154	275	276	194	152	82	68	48	32
ALP (30 - 115 u/L)	166	165	126	239	282	208	156	132	135	95	89	80	76
LDH (80 -250 units/L)	413	394	352	352	442	401	388	314	405	320	322	257	220
Corrected Calcium (2.25 -2.65 mol/L)	2.12	2.27	2.11	2.27	2.25	2.25	2.12	2.14	1.88	1.96	2.16	2.11	2.27
Phosphate (0.8 - 1.5 mmol/L)	1.1	0.8	1.3	1.6	1.7	1.8	2.4	2.5	2.3	2.6	2.3	2.2	1.9
Creatinine (0.04-0.14 mmol/L)	0.62	0.78	0.92	0.77	0.75	0.76	0.73	0.77	0.75	0.81	0.78	0.69	0.48
Urea (2.5 - 7.9 mmol/L)	20	26.8	30.3	27.6	28.4	32.2	32.6	31.3	30.5	33.9	33.1	30.5	22.2
Albumin (35 -50 g/L)	33	31	22	23	28	24	23	23	29	26	24	28	32

Shaded area indicates laboratory results at discharge