Abstract:

Leptospirosis is a widespread and prevalent zoonotic disease. It occurs in both temperate and tropical regions; the incidence in the tropics is approximately 10 times higher than in temperate regions.In the tropics, leptospirosis is mainly a disease of poverty (including low education, poor housing, absence of sanitation, and poor income) [5]. It is acquired through occupational exposure (subsistence farming) and living in rodent-infested, flood-prone urban slums [6]. We report an unusual presentation of leptospirosis in a patient who presented to the Emergency Department (ED) with yellowish discoloration of the body and decreased urine output.

Introduction:

Leptospirosis is a widespread and prevalent zoonotic disease. It occurs in both temperate and tropical regions; the incidence in the tropics is approximately 10 times higher than in temperate regions [1]. Leptospirosis is an underreported disease, and there are no reliable global incidence figures. A modeling exercise by the World Health Organization's (WHO's) Leptospirosis Burden Epidemiology Group estimated that there were 873,000 cases worldwide annually with 48,600 deaths [2]. The organism infects a variety of both wild and domestic mammals, especially rodents, cattle, swine, dogs, horses, sheep, and goats. Rodents are the most important reservoirs for maintaining transmission in most settings.[3]. Human infection usually results from exposure to environmental sources, such as animal urine, contaminated water or soil, or infected animal tissue. Portals of entry include cuts or abraded skin, mucous membranes, or conjunctivae. The infection may rarely be acquired by ingestion of food contaminated with urine or via aerosols. Controversy exists as to whether *Leptospira* can penetrate the intact skin. [4]. We report an unusual presentation of leptospirosis in a patient who presented to the Emergency Department (ED) with yellowish discoration of the body and decreased urine output.

Case report:

A 35 years old male patient, farmer by occupation, presented in the ED with complaint of fever, nausea and vomiting for 6 days which was associated with history of yellowish discoloration of the body and decreased urine output. History of alcohol intake for the past 15 years was also present. The blood pressure was

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130/80 mm of hg and PR was 110 bpm. General physical examination was remarkable for icterus and conjunctival suffusion. Systemic examination was apparently within normal limits(WNL). ABG was suggestive of metabolic acidosis (Ph: 7.15, HCO3: 7, Pco2: 21, Na: 120, K: 2.8). ECG in the ED was WNL. Provisional diagnosis of leptospirosis, malaria and dengue was kept with due consideration to other atypical infections like scrub typhus and chikungunea fever. Patient was given empirical antibiotics (including coverage for leptospira) and antimalarials. Initial lab reports were suggestive of severe sepsis and hepatorenal dysfunction. (Refer to table 1). As per the lab reports the antibiotic support was escalated. Dengue (NS-1 antigen and Ig M) and malaria tests (Antigen and peripheral blood smear) were negative on day 2 of admission. Antimalarials were stopped. Howeveer despite antibiotic escalation there was progressive worsening of sepsis, renal and heatic dysfunction, although patient was hemodynamically stable. Serum procalcitonin was more than 75 IU/ml. In view of deranged renal function patient was given 2 sessions of hemodialysis. Final Blood and urine cultures reports did not show any growth of bacteria. In the mean time USG and later on CT abdomen (non contrast) reports were s/o acute pancreatitis. Conservative approach was followed.An upper GI endoscopy was done which revealed oesophageal candidiasis. Considering the critical state of the patient and non recovery of sepsis, a possibility of systemic fungal infection was kept and parentral antifungal agent (Mikafungin) was started. On day 5 leptospira Ig M report came to be positive. Chikungunea and scrub typhus (Weil Felix test) tests were negative. Gradually sepsis, renal and liver functions started resolving and patient was discharged with normal renal function and near normal liver function.

Table 1

	29/10/2017	30/10/2017	31/10/2017	1/11/2017	2/11/2017	3/11/2017	4/11/2017	6/11/2017	8/11/2017
Hb	11.87	10.58	10.06	9.2	8.6		7.9	6	7.5
TLC	41.31	45.87	47.53	55.25	52.2	43.3	33.2	33.4	26.4
Creatinine	5.38	3.59	2.62	3.67	4.8	3.97	3.85	2.32	1.21
Bilirubin (T)	26.66	24.19		24.94	23.19	22.04	23.25	21.7	11.26
Bilirubin (D)	24.34	20.29			23.12	21.16	22.9	20.58	10.18
Malaria Ag		negative							
Dengue NS1		negative							
Dengue Ig M									
Chikungunea Ig M					negative				
Weil Felix					negative				
Leptospirosis Ig M					Positive				

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Discussion:

In the tropics, leptospirosis is mainly a disease of poverty (including low education, poor housing, absence of sanitation, and poor income) [5]. It is acquired through occupational exposure (subsistence farming) and living in rodent-infested, flood-prone urban slums [6]. These are often associated with increased rainfall or flooding, which presumably increased the risk of exposure to contaminated water [7-11]. This case of leptospirosis was unusual because though the diagnosis of leptospirosis was straight forward clinically, the recovery was delayed because of underlying fungal infection. Leptospirosis presenting along with fungal infection is an unusual presentation.

REFERENCES:

1. Hartskeerl RA, Collares-Pereira M, Ellis WA. Emergence, control and re-emerging leptospirosis: dynamics of infection in the changing world. ClinMicrobiol Infect 2011; 17:494.
2. World Health Organization. Global burden of human leptospirosis and cross-sectoral interventions for its prevention and control. http://www.pmaconference.mahidol.ac.th/dmdocuments/2013-PMAC-Poster-P9-Bernadette%20Abela-Ridder.pdf (Accessed on August 13, 2013).
3. Ko AI, Goarant C, Picardeau M. Leptospira: the dawn of the molecular genetics era for an emerging zoonotic pathogen. Nat Rev Microbiol 2009; 7:736.
4. Stern EJ, Galloway R, Shadomy SV, et al. Outbreak of leptospirosis among Adventure Race participants in Florida, 2005. Clin Infect Dis 2010; 50:843.
5. Jesus MS, Silva LA, Lima KM, Fernandes OC. Cases distribution of leptospirosis in City of Manaus, State of Amazonas, Brazil, 2000-2010. Rev Soc Bras Med Trop 2012; 45:713.
6. Reis RB, Ribeiro GS, Felzemburgh RD, et al. Impact of environment and social gradient on Leptospira infection in urban slums. PLoSNegl Trop Dis 2008; 2:e228.
7. Kawaguchi L, Sengkeopraseuth B, Tsuyuoka R, et al. Seroprevalence of leptospirosis and risk factor analysis in flood-prone rural areas in Lao PDR. Am J Trop Med Hyg 2008; 78:957.

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