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Case Report Open Access

Leptospirosis in a Returning Traveller

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Abstract

Leptospirosis is a zoonotic disease which causes a variable spectrum of illness in humans, ranging from a self-limiting or subclinical illness, to the severe potentially fatal "Weil's disease," which can have a mortality of up to 50%. We present the case of a returning traveller presenting with vomiting, diarrhoea, jaundice and acute kidney injury.

Keywords: Weil's disease; Leptospirosis; Kidney injury; Bilirubin

Introduction

Leptospirosis is a zoonosis caused by spirochaetes of the genus Leptospira. It is spread from the urine of animals, and infects humans via environmental exposure through cuts, mucous membranes, and conjunctiva. It is an uncommon disease seen in Northern Ireland and the rest of the UK. According to the Leptospira Reference Unit only 15 cases were confirmed in Northern Ireland between 1998-2009 [1]. Infection in humans is usually due to environmental sources, especially contaminated water and soil. Occupational exposure to farmers and sewage workers, and recreational activities such as water sports are risk factors for contracting the disease. There was a high profile case in 2010 when British Olympic Rowing champion Andy Holmes died from Leptospirosis, presumed to be contracted by his time spent rowing in the Thames.

Case Report

A 29 year old gentleman returned from holiday in Florida, with symptoms of headache, myalgia, vomiting and diarrhoea. He did not settle with conservative treatment from his general practioner and presented with jaundice to accident and emergency.

On admission he had raised inflammatory markers with CRP of 127, and acute kidney injury with urea of 13.6 mmol/L, and creatinine of 196 umol/L. His total bilirubin was 158 umol/L with a mild rise in his ALT to

62 U/L. He was noted to be thrombocytopaenia with platelets of 43(e9/l)), and hyponatraemic with sodium of 130 mmol/L

He had recently returned from holiday in Florida, and initially appeared to have no risk factors for the derangement of his Liver Function. He had no tattoos, no alcohol or drug abuse, and no high risk sexual contact.

Ultrasound of his abdomen and renal tracts showed no abnormality, stool and blood cultures were negative. 2 days after admission he became markedly hypotensive despite adequate fluid resuscitation, with worsening liver and renal function necessitating transfer to ICU. His Bilirubin continued to rise in ICU, peaking at 655 after being in hospital for 5 days. His Creatinine rose to 236 umol/L and his urea to 17.5 mmol/L

He was treated with meropenem and teicoplanin 2 days after admission to cover for sepsis of unknown origin, and 3 days after admission the suspicion of Leptospirosis was raised and doxycycline added. On day 6 of admission the diagnosis was confirmed by Positive Leptospiral ELISA IgM and Leptospiral Microscopic Agglutination Test.

Value	Admission	Peak	Discharge
Bilirubin (Total)	158	655	76
Urea	13.6	17.5	7.8
Creatinine	196	236	98
ALT	62	362	276

Table 1: Showing Admission, Peak, and Discharge Biochemistry. Above values in SI units

After spending 5 days in intensive care he was weaned off vasopressors, and was able to return to the medical ward. He completed a one week course of oral doxycycline, and was discharged home after being in hospital for 12 days with a toatl bilirubin of 76 (Table 1).

Discussion

It transpired 2 days into his admission that this patient had risk factors for Leptospirosis in that he was a watersports enthusiast, spending a lot of his spare time on the River Bann, and had also been away on holiday in a location where there have been previous outbreaks of leptospirosis [2]. Although a thorough history had been taken pertaining to usual causes of deranged liver function in a fit and well 29 year old male, history of his watersports did not become apparent until he had developed multi-organ failure secondary to Weil's disease. It is an important diagnosis to consider in patients exposed to environmental sources, as late recognition and initiation of antibiotic therapy has been shown to correlate with development of severe Leptospirosis [3]. Thrombotic thrombocytopenic purpura and haemolytic uremic syndrome were considered but positive leptospira serology confirmed the diagnosis.

Whether this patient caught the disease on holiday in a tropical climate where he could have been exposed to leptospira, or the River Bann is uncertain. Incubation period of the disease ranges from 2-20 days so it is possible that he may have contracted it prior to going on holiday.

References

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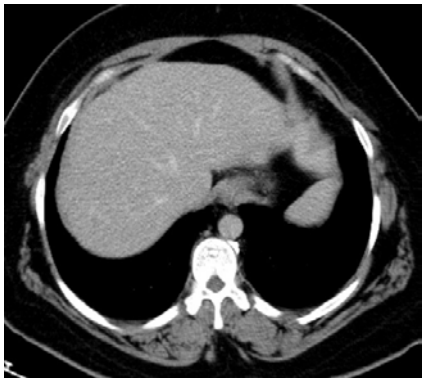

Figure 3: Thorax CT revealed the characteristic appearance of cement leakage at the level of the VP.

Authors	Patient number /Asypmtomatic PCE patients	Time between VP and diagnosis	Treatment
Grados et al. [6]	40/1	48 months	Not reported
Bernhard et al. [7]	1	6 months	Not reported
Pleser et al. [8]	1	During VP	AKG
Seo et al. [1]	1	24 months	Embolectomy
Baumann et al. [3]	1	Post-op	Embolectomy
MacTaggart et al. [11]	1	2 months	Clinical observation
Du Hwan Choe etal. [12]	64/3	3 months	Clinical observation
Quesada et al. [13]	1	1 day	Not reported
Abdul-Jalil et al. [14]	1	1 year	Clinical observation
Serra et al. [15]	175/3	Not reported	Not reported
Schneider and Plit [16]	1	2 days	Not reported
Yeo et al. [17]	75/18	Post-op	Not reported
Venmans et al. [18]	299/11	During VP	Clinical observation
Venmans et al. [19]	54/14	21 months	Clinical observation
Fornell-P´erez et al. [20]	1	Post-op	Clinical observation
Nesn´ıdal et al. [21]	1	2 days	Clinical observation
Dash and Brinster. [2]	1	Not reported	Embolectomy
Luetmer et al. [23]	244/22	Post-op	Clinical observation
Tourtier et al. [24]	1	Post-op	Clinical observation
Walter et al. [25]	1	During VP	Clinical observation
Girolamo et al. [26]	1	Post-op	AKG
Lee et al. [27]	1	3 years	Clinical observation
	87		

Table 1: Case reports and series of patients with asymtomatic pulmonary embolism after percutaneous vertebroplasty. Data was collected from PubMed database, with the queries: ''complication of vertebroplasty'', "bone cement pulmonary embolism''.

VP: Vertebroplasty; PCE: Pulmonary Cement Embolism; AKG: Anticoagulant