Lessons from practice

Acute abdominal pain from a zoonosis in Australia

Clinical record

A 70-year-old man presented to the emergency department with abdominal pain, vomiting, diarrhoea and shortness of breath on the background of a one-week history of fevers, dysuria and headaches. His past medical history included hypercholesterolaemia and gastro-oesophageal reflux disease. He had no history of alcohol excess and lived on a cattle farm in rural Victoria.

On presentation he had a temperature of 38.2°C, heart rate of 120 beats per minute, respiratory rate of 30 breaths per minute, a blood pressure of 96/70 mmHg and abdominal tenderness with guarding. Differential diagnoses included an acute abdominal pathology, chest sepsis, urinary sepsis or a viral infection. Intravenous fluids, analgesia and broad spectrum antibiotics (piperacillin–tazobactam and metronidazole) were commenced promptly. Blood tests (Box 1) revealed an acute kidney injury, elevated lipase levels, raised inflammatory markers and deranged liver function. Blood cultures were sterile. Urinalysis was positive for protein, and urinary microscopy demonstrated microscopic haematuria. A computed tomography scan of the abdomen and pelvis showed uncomplicated interstitial pancreatitis with secondary duodenitis (Box 2). An abdominal ultrasound did not show cholelithiasis. He was subsequently transferred to a tertiary hospital for ongoing management of pancreatitis and investigation of alternative causes of sepsis. Further history revealed recent exposure to rodents and investigations for zoonotic infections were therefore performed. *Leptospira* IgM antibodies were detected in serum using an enzyme-linked immunosorbent assay, and leptospirosis was considered to be the most likely unifying diagnosis. He was commenced on a 7-day course of doxycycline and made an uneventful clinical recovery.

Subsequent acute and convalescent *Leptospira* microscopic agglutination testing (MAT) revealed a greater than fourfold increase in titre (Box 1), confirming acute *Leptospira borgpetersenii* serovar Arborea infection.

Discussion

Leptospirosis is a zoonotic disease that commonly occurs in tropical regions.[1] Infections in humans occur as a result of contact with infected animal tissue or exposure to soil or water contaminated with urine of infected animals. Entry sites are via mucosa (eyes, nose and mouth), skin or conjunctivae.[1] In Australia, one person per 100 000 population per year is diagnosed with leptospirosis, with the greatest prevalence being in Queensland (1.85 cases per 100 000 population per year).[1] In Queensland, infections peak in the wet summer months with occupational exposure being the leading source (eg, abattoir and meat workers, dairy farmers).[1] Other groups at risk of leptospirosis include watersport enthusiasts and people living in areas with livestock, rodent infestations, inadequate sewage drainage and flooding.[1] An estimated one million cases with 60 000 associated deaths occur worldwide every year.[1]

Cases of leptospirosis doubled in most Australian states in 2021 (personal communication, Paul Collins, Data Manager, National Interoperable Notifiable Disease Surveillance System, Public Health and Surveillance Branch, Australian Government Department of Health, 23 Nov 2021), potentially because of increased rodent activity secondary to climatic conditions. Our patient was infected with *L. borgpetersenii* serovar Arborea, which is associated with exposure to rodents and cattle.[3] The first human infection with *L. borgpetersenii* serovar Arborea in Australia occurred in 1998 in northern New South Wales.[3]

Leptospirosis can present with a wide range of clinical manifestations.[1] The incubation period ranges from 2 to 30 days and classically manifests as a biphasic illness. The acute bacteraemic phase lasting 2 to 9 days presents as an influenza-like illness (fevers, chills, headache, conjunctival suffusion and lethargy).[1] The subsequent latent phase lasting 4 to 30 days results in immune-mediated damage of organs which may cause hepatic and renal impairment, pulmonary haemorrhage, myocarditis, arrythmias and meningitis.[1] Common laboratory findings in leptospirosis include thrombocytopenia, lymphopenia, renal impairment and hyponatraemia.[1] A urinalysis may show microscopic haematuria.[1] Our patient demonstrated some of these findings.

Microbiological confirmation of leptospirosis is challenging. A confirmed case requires the isolation of a pathogenic *Leptospira* species or serological evidence of recent infection (Box 3).[4] Our patient demonstrated a fourfold increase in MAT titre for *L. borgpetersenii* serovar Arborea.

Acute pancreatitis in leptospirosis has previously been described[2] but is not a well recognised complication. Leptospirosis may result in pancreatitis due to immune-mediated mechanisms such as toll-like receptor 2 activation and leptospirosis causing small vessel vasculitis in the pancreas, resulting in the release of proteolytic enzymes causing autodigestion.[2] Patients with leptospirosis and pancreatitis can become critically unwell.[2]

The treatment of leptospirosis is a 7-day course of either oral doxycycline or intravenous ceftriaxone or benzylpenicillin.

In summary, we describe a case of acute pancreatitis in the setting of leptospirosis. To our knowledge, this is the first such case described in Australia. Clinicians

Ajinkya Bhonsle[1]
Mithun Nambiar[1,2]
Michael Swan[1]
Ralph Junckerstorff[1]

1 Monash Health, Melbourne, VIC.
2 Monash University, Melbourne, VIC.

ajinkya.bhonsle@monashhealth.org

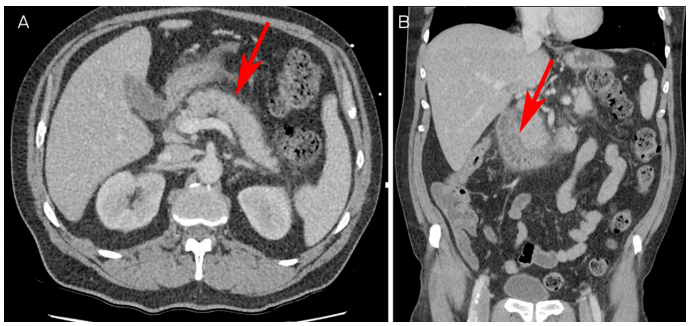
doi: 10.5694/mja2.51547

1 Pathology

Variable	Admission (27/5/21)	After discharge from hospital (18/6/21)	Reference interval
Haemoglobin (g/L)	134	139	125–175 g/L
Platelet count (× 10^9/L)	115	209	150–450 × 10^9/L
White cell count (× 10^9/L)	7.1	6.7	4–11 × 10^9/L
Neutrophil count (× 10^9/L)	6.3	2.9	2–8 × 10^9/L
Lymphocyte count (× 10^9/L)	0.4	2.6	1–4 × 10^9/L
INR	1.2	n/a	< 1.3
Prothromboplastin time (seconds)	12.6	n/a	11–13.5 s
Fibrinogen (g/L)	> 4.5	n/a	1.5–4.0 g/L
APTT (seconds)	28	n/a	24–34 s
D-dimer (µg/ml)	> 4.5	n/a	< 0.5 µg/ml
Sodium (mmol/L)	131	140	135–145 mmol/L
Potassium (mmol/L)	3.5	4.1	3.5–5.2 mmol/L
Bicarbonate (mmol/L)	26	29	22–32 mmol/L
Urea (mmol/L)	11.2	6.8	3–10 mmol/L
Creatinine (µmol/L)	122	93	60–110 µmol/L
eGFR (mL/min/1.73 m^2)	51	71	> 90 mL/min/1.73 m^2
Albumin (g/L)	26	37	34–47 g/L
Bilirubin (µmol/L)	68	10	0–20 µmol/L
ALP (U/L)	271	113	30–110 U/L
GGT (U/L)	600	147	0–50 U/L
AST (U/L)	88	35	0–35 U/L
ALT (U/L)	94	49	0–35 U/L
Lipase (U/L)	1443	91	0–60 U/L
C-reactive protein (mg/L)	246	n/a	0–10 mg/L
Leptospira			
IgM	Detected (30/5/21)	Detected (30/6/21)	
Micro-agglutination titre (*L. borgpetersenii* serovar Arborea)	Acute phase (30/5/21): < 50	Convalescent phase (30/6/21): 1600	

ALP = alkaline phosphatase; ALT = alanine aminotransferase; APPT = activated partial thromboplastin time; AST = aspartate aminostransferase; eGFR = estimated glomerular filtration rate; GGT = γ-glutamyltransferase; INR = international normalised ratio; n/a = not applicable. ◆

2 Computed tomography scan of the abdomen and pelvis



A: Axial view showing fat stranding around the head, body and tail of the pancreas (arrow). **B:** Coronal view showing inflammatory change around the pancreatic head and associated secondary duodenitis of the second part of the duodenum (arrow).

3 Leptospirosis: case definition[4]

A confirmed case requires definitive laboratory evidence of leptospirosis infection by one of the below:

- Isolation of a pathogenic *Leptospira* species
- A fourfold or greater rise in *Leptospira* microscopic agglutination test titre between the acute and convalescent-phase sera; these must be obtained at least 2 weeks apart, and ideally should be conducted at the same laboratory
- A single *Leptospira* microscopic agglutination test titre ≥ 400 which is also supported by a positive enzyme-linked immunosorbent assay IgM result

should consider a diagnosis of leptospirosis in patients with acute pancreatitis without traditional risk factors such as alcohol and cholelithiasis. Microbiological testing should be reserved for patients with significant epidemiological risk, laboratory findings and a clinical syndrome consistent with leptospirosis.

Lessons from practice

- Leptospirosis classically causes a biphasic illness; an acute bacteraemic phase and a subsequent immune mediated phase.
- Ask about animal exposure in patients presenting with an undifferentiated febrile illness to help rule out zoonotic diseases as a cause.
- Leptospirosis should be considered as a differential diagnosis in patients who present with pancreatitis without traditional risk factors (such as cholelithiasis or alcohol intake) and with laboratory findings and epidemiological risk factors consistent with leptospirosis.
- Microbiological diagnosis of leptospirosis can be confirmed either by the isolation of a pathogenic *Leptospira* species, a fourfold or greater rise in *Leptospira* MAT titre between the acute and convalescent phase sera, or a single *Leptospira* MAT titre ≥ 400 which is also supported by a positive enzyme-linked immunosorbent assay IgM result.

Acknowledgements: We thank Maryza Graham (medical microbiologist and infectious diseases physician) for her advice regarding laboratory testing techniques for *Leptospira* at Monash Health.

Competing interests: No relevant disclosures.

Provenance: Not commissioned; externally peer reviewed. ■

1 Lau C, Townell N, Stephenson E, et al. Leptospirosis: an important zoonosis acquired through work, play and travel. *Aust J Gen Pract* 2018; 47: 105-110.

2 Herath N, Kamburapola C, Agampodi S. Severe leptospirosis and pancreatitis: a case series from a leptospirosis outbreak in Anuradhapura district, Sri Lanka. *BMC Infect Dis* 2016; 16: 644.

3 Lau CL, Skelly C, Dohnt M, Smythe LD. The emergence of *Leptospira borgpetersenii* serovar Arborea in Queensland, Australia, 2001 to 2013. *BMC Infect Dis* 2015; 15: 230.

4 Australian Government Department of Health. Australian national notifiable diseases case definitions – Leptospirosis. https://www1.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-nndss-casedefs-cd_lepto.htm (viewed Aug 2021). ■