Acute lung injury and multi-organ dysfunction; an unusual manifestation of leptospirosis

Editor—Leptospirosis is a zoonosis—an infection that can be transmitted from animals to humans—and is primarily a disease of tropical regions. It is an uncommon, but notified, disease in the UK, <1 patient million⁻¹ yr⁻¹, yet two to three people in England and Wales die every year from the disease. Severe forms are associated with a very high mortality rate. Diagnosis is based on epidemiological history, clinical features, and investigations. It is often missed and easily overlooked because of the lack of awareness and the inability to obtain early laboratory confirmation. Adequate antibody titres appear only after a period of 5–7 days and, in the UK, the specimen needs to be sent to the reference laboratory at Hereford.

Leptospirosis should be a differential diagnosis in an acutely ill patient with appropriate risk factors and symptomatology. We present a patient of an uncommon presentation of leptospirosis.

A 59-yr-old male attended the hospital with a 5-day history of fever, lethargy, body pain, headache, productive cough, and decreased appetite. He lived independently, smoked 30 cigarettes a day, and was keen on fishing in the nearby canal. He was in good health with no significant past medical or surgical history and had not recently travelled outside the UK.

On examination, he was alert, oriented, and icteric. Temperature was 37.7°C, heart rate 109 beats min⁻¹, arterial pressure 93/53 mm Hg, and well-perfused peripheries. His ventilatory frequency was 19 min⁻¹ with an oxygen saturation of 96% on room air. On auscultation, his heart sounds were normal and air entry were equal on both lungs with occasional scattered wheeze.

Initial investigations revealed a white cell count of $7.0 \times 10^9$ litre⁻¹, platelets $52 \times 10^9$ litre⁻¹, haemoglobin 13.2 g dl⁻¹, urea 22.5 mmol litre⁻¹, creatinine 262 µmol litre⁻¹, bilirubin 30 µmol litre⁻¹, GGT 83 IU litre⁻¹, and CRP of 339 mg litre⁻¹. ECG showed normal sinus tachycardia and chest X-ray revealed mildly increased bronchoalveolar markings. An initial diagnosis of sepsis with lower respiratory tract infection and acute renal impairment was made and he was transferred to a medical ward.

Within a few hours, his condition deteriorated; he became breathless, hypoxic, and hypotensive (arterial pressure 80/50 mm Hg) despite a litre of colloid and crystalloid infusion. His arterial gases were, pH 7.47, $P_{\text{aCO}_2}$ 3.3 kPa, base excess $-4$ mmol litre⁻¹, bicarbonate 17 mmol litre⁻¹, $P_{\text{aO}_2}$ 8.6 kPa, $S_{\text{aO}_2}$ 94% on oxygen 15 litre min⁻¹. He was transferred to a critical care ward.

Facial continuous positive airway pressure (CPAP) and invasive monitoring was initiated. Noradrenaline infusion was started to maintain a mean arterial pressure (MAP) of ≥70 mm Hg. Despite these measures, his condition worsened rapidly. Within 2 h, he became exhausted, more hypoxic, and acidic (blood gas, $Frac{P_{\text{aO}_2}}{P_{\text{aCO}_2}}$ 0.9, pH 7.34, $P_{\text{aCO}_2}$ 4.8 kPa, base excess $-6.4$ mmol litre⁻¹, bicarbonate 19.4 mmol litre⁻¹, $P_{\text{aO}_2}$ 7.7 kPa, $S_{\text{aO}_2}$ 84%). Investigations and chest X-ray were repeated. Repeat chest X-ray revealed an extensive bilateral alveolar shadowing (Fig. 1), simulating acute respiratory distress syndrome (ARDS). At this point, his trachea was intubated.

Repeat investigations revealed worsening of renal (creatinine 338 µmol litre⁻¹, urea 26.2 mmol litre⁻¹) and hepatic (bilirubin 114 µmol litre⁻¹, GGT 98 IU litre⁻¹), alkaline phosphatase 131 IU litre⁻¹, albumin 22 g litre⁻¹, aspartate transferase 59 IU litre⁻¹) function. Platelets fell to $23 \times 10^9$ litre⁻¹, activated partial thromboplastin time was 35.5 s, prothrombin time 10.4 s, and lactate 1.2 mmol litre⁻¹. Abdominal ultrasound revealed no abnormalities, and a cardiac echo showed a dilated and poorly contracting right ventricle with normal left

Fig 1 Acute bilateral alveolar shadowing in leptospirosis.
Effects of midazolam and dexmedetomidine on inflammatory responses and gastric intramucosal pH to sepsis, in critically ill patients

Editor—Despite advances in supportive care, the mortality rate in patients with severe sepsis continues to exceed 30%. Sedation is an important part of the therapy of critically ill patients in ICU. Although midazolam and dexmedetomidine are used for sedation in the ICU, there are limited data on its effects on inflammatory responses and gastric intramucosal pH. We studied the effect of midazolam and dexmedetomidine on the inflammatory responses (tumour necrosis factor-α (TNF-α), interleukin (IL)-1β, and IL-6) and gastric intramucosal pH in critically ill patients receiving sedation. The Regional Committee on Medical Research Ethics approved the study, and written informed consent was obtained from the patients wherever possible, or from the next of kin. Critically ill patients with bacteriologically documented infections were included in the study if they met at least two of the criteria of sepsis, defined by the American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference Committee. Exclusion criteria were known allergy to midazolam or dexmedetomidine, possible or confirmed pregnancy, haemodynamic instability, heart, liver and renal failure, and patients with known or suspected brain death. The acute physiology and chronic health evaluation (APACHE II) was employed to determine the initial severity of illness.