present with a metabolic 'flap'. There were no stigmata of chronic liver disease. Pupil reaction was noted as 'sluggish', and nystagmus was present, more marked in the horizontal plane, the remainder of the cranial nerves were normal. The ankle reflexes were absent, but the physical examination was otherwise normal. Investigations, including a clotting screen, were normal except urea 19.3 mmol/l (normal 3–7 mmol/l), bilirubin 75 mmol/l (normal 3–17 mmol/l), haemoglobin 9.3 g/dl. Ultrasound confirmed live twin pregnancy and the mother's liver and renal tract were normal.

She received intravenous rehydration and high dose vitamin supplementation including thiamine. Anti-emetic drugs failed to stop the vomiting. Thyroid function tests subsequently returned with a TSH 0.08 mU/l (normal 0.32–5.0) and free thyroxine 38.7 pmol/l (normal 9–24). She received propranolol for three days and propylthiouracil. After a further week the vomiting desisted.

During recovery she was vague, forgetful and ignored personal hygiene. Her relatives confirmed a change from her pre-morbid state. A mental state examination revealed loss of short-term memory, confabulation, and poor orientation in time only. Truncal ataxia was noted, but her neurology was otherwise normal. A computed tomography (CT) scan of the brain showed only mild ventricular dilatation. The haematology, biochemistry (except thyroid function), microbiology and virology screen, electroencephalogram, and cerebrospinal fluid were normal or negative.

Vitamin supplementation continued and the nystagmus disappeared by day 5 and the vomiting by day 7. The twins were born at 29 weeks gestation with severe hyaline membrane disease but three years later both are well. After childbirth, the mother's thyroid function normalised and treatment stopped. Her CT scan was normal 8 months later and her only residual symptom is impaired short-term memory.

Wernicke's encephalopathy complicating hyperemesis gravidarum was first reported in 1914 and since then approximately 14 cases have appeared in the literature.^{2–4} The mean age of the patients was 26 years (range 18 to 35 years) and their symptoms occurred between the 14th and 20th week of pregnancy (median 15 weeks) after at least three weeks of persistent vomiting. Only half of these pregnancies resulted in normal healthy children, and symptoms persisted in three although the follow-up interval was variable. A single report describes a 61-year-old lady with thyrotoxicosis who developed Wernicke's encephalopathy after excessive vomiting.⁵ We believe our patient is the first to be described with hyperemesis gravidarum aggravated by twin pregnancy and thyrotoxicosis that subsequently developed Wernicke's encephalopathy.

CE MILLSON
K HARDING
RM HILLSON

Department of Medicine and Obstetrics,
The Hillingdon Hospital,
Pield Heath Road, Uxbridge,
Middlesex, UK

Correspondence to Dr CE Millson, National Medical Laser Centre, University College London Medical School, Rayne Institute, 5 University Street, London WC1 6JJ, UK

Wernicke's encephalopathy: classical features

- confusion
- nystagmus
- ataxia

- 1 Wernicke C. In: *Lehrbuch der Gehirnkrankheiten für Ärzte und Studieren*. Kassel: Theodore Fischer, 1881, vol 2, p 229.
- 2 Reuler JB, Girard DE, Cooney TG. Current concepts. Wernicke's encephalopathy. *N Engl J Med* 1985; 312: 1035–9.
- 3 Flannery G. Persistent hyperemesis gravidarum complicated by Wernicke's encephalopathy. *Ir J Med Sci* 1990; 159: 82.
- 4 Galloway PJ. Wernicke's encephalopathy and hyperemesis gravidarum [letter, comment]. *BMJ* 1992; 305: 1096.
- 5 Enoch BA, Williams DM. An association between Wernicke's encephalopathy and thyrotoxicosis. *Postgrad Med J* 1968; 44: 923–30.

Peritonitis complicating leptospirosis

Sir

Leptospirosis is an acute generalised infectious disease, characterised by extensive vasculitis. All organ systems may be involved. The rare complications include acute cholecystitis, myocarditis, pancreatitis, parotitis, and epididymo-orchitis. To the best of our knowledge, peritonitis complicating the course of leptospirosis has not been reported so far. Serum aminotransferase elevation in leptospirosis is rarely more than five-fold, regardless of the degree of hyperbilirubinemia – bilirubin levels may be as high as 1111.5 µmol/l.¹ We report here a patient with leptospirosis and peritonitis with marked elevation of aminotransferases.

A 32-year-old male scientist working with laboratory animals presented with a 10-day history of high fever associated with myalgias. He developed progressive deterioration in sensorium three days prior to admission. On presentation the patient was febrile, comatose, and jaundiced. The conjunctivae were suffused. Examination of the heart and chest was unremarkable. Meningeal signs were absent and no focal neurological deficit was noticed. The patient had splenomegaly of 2 cm. There was no clinical evidence of free fluid in the abdomen. Investigations on admission revealed a haemoglobin of 91 g/l, total leucocyte count of $12.2 \times 10^9/l$ (polymorphonuclear neutrophils 63%, lymphocytes 37%) and a platelet count of $123 \times 10^9/l$. Blood sugar and electrolytes were within normal limits. Blood urea nitrogen was 13.57 mmol/l and serum creatinine 344 µmol/l. Urinalysis showed proteinuria of 2+ (24-h excretion 0.8 g) and 1–2 red blood cells per high-powered field. Liver function tests revealed a serum bilirubin of 53.01 µmol/l and serum alkaline phosphatase of 236 IU/l. Aspartate transaminase and alanine transaminase were 4918 IU/l and 2309 IU/l, respectively, almost a 100-fold elevation. Total serum proteins, albumin, and globulin were 47, 23, and 24 g/l, respectively. Prothrombin time was 21 s (control 12 s). Serum creatine phosphokinase was 440 IU/l. CSF examination was unremarkable. Arterial blood

gas analysis showed mild metabolic acidosis. Chest X-ray and electrocardiogram were normal. Blood and urine cultures were sterile. A provisional diagnosis of leptospirosis was made and the patient was started on intravenous penicillin G (1.5 million units six hourly) along with supportive treatment. On admission leptospiral serology done by indirect haemagglutination and microscopic agglutination using pooled antigen was negative. Leptospiral serology repeated 10 days after admission showed a four-fold rise in titres. Serotype determination could not be done.

On the third day of hospitalisation the patient developed progressive distension of abdomen with rigidity and rebound tenderness. Plain X-rays of the abdomen did not show any air under the diaphragm. Ultrasound and contrast-enhanced CT scan of the abdomen showed ascites and splenomegaly. A litre of straw-coloured ascitic fluid was tapped. It had a protein content of 26 g/l with a white blood cell count of $0.5 \times 10^9/l$, mainly polymorphonuclear leucocytes. Cultures of ascitic fluid were sterile. During his hospital stay the patient's renal parameters deteriorated and he was given hemodialysis three times. After a week the patient's clinical condition gradually improved with reduction in ascites and progressive improvement in biochemical parameters. The renal and liver function tests returned to normal at the end of seven weeks.

Leptospirosis presents in various ways, from inapparent to fulminating and fatal infection. Confirmation of diagnosis is either by isolating the organism or more commonly, by detecting antibodies in the patient's blood.² Peritonitis in leptospirosis may be explained on the basis of immuno-inflammation due to leptospiral antigens during the immune phase. Since culture of the ascitic fluid was sterile a superimposed infection seems unlikely in our patient. Liver involvement in leptospirosis is usually associated with mild elevation of aminotransferases.³ Our patient exhibited more than 100-fold elevation in aspartate transaminase and alanine transferase. This is an unusual feature. However, as expected, the abnormalities in the liver function tests returned to normal with no evidence of residual hepatic damage.

R HANDA

A SOOD

JP WALI

Department of Medicine,
All India Institute of Medical Sciences,
New Delhi-110029, India

- 1 Johnson WD Jr, Silva IC, Rocha H. Serum creatine phosphokinase in leptospirosis. *JAMA* 1975; 233: 981–2.
- 2 Ferguson IR. Leptospirosis update. *BMJ* 1991; 302: 128–9.
- 3 Heath CW, Alexander AD, Galton MM. Leptospirosis in the United States – analysis of 483 cases in man, 1949–1961. *N Engl J Med* 1965; 273: 857–64, 915–22.