SELF ASSESSMENT QUESTIONS

Intermittent jaundice and rigors in a patient with longstanding ulcerative colitis

N D Hawkes, D Mutimer, G A O Thomas

Answers on p 412.

A 44 year old man was admitted with a history of intermittent jaundice and rigors. Three months previously he had been admitted to another hospital with similar symptoms and half a stone (3.2 kg) weight loss. At this time his problems were thought to be secondary to choledocholithiasis. At operation, gallstones were seen in the common bile duct with an associated lower common bile duct stricture. He underwent cholecystectomy and choledochoduodenostomy. The symptoms initially resolved postoperatively but subsequently recurred on an intermittent basis. He was transferred to our hospital for further management of his problems.

He had a 28 year history of ulcerative colitis, based on barium enema, which had remained quiescent for many years on salazopyrin maintenance treatment. His past medical history included segmental bronchiectasis secondary to childhood whooping cough. He was a life long non-smoker and did not drink alcohol.

He was jaundiced but apart from a right Dupuytren's contracture and his cholecystectomy scar, clinical examination was normal.

His liver function tests were abnormal. Serum alkaline phosphatase was 1020 IU/l, aspartate transaminase 128 IU/l, total bilirubin 113 µmol/l. Serum albumin was 33 g/l and the international normalised ratio mildly raised at 1.3. His full blood count and urea and electrolytes were normal. He was started on antibiotics for possible ascending cholangitis. An abdominal ultrasound confirmed gas in the biliary tree consistent with his previous surgery, but there was no evidence of duct dilatation or recurrent stones in the common bile duct. Endoscopic retrograde cholangiopancreatography was attempted. The common bile duct and common hepatic duct were of small calibre but the intrahepatic ducts were not seen, most of the contrast emptying via the choledochoduodenostomy. A percutaneous transhepatic cholangiogram (PTC) was performed (fig 1).

His symptoms settled on cephadrine and he was discharged. He was followed up in the outpatient clinic and remained reasonably well apart from suffering episodic attacks of jaundice and rigors, which responded quickly to courses of antibiotics. Twelve months after admission his albumin and bilirubin were normal and his alkaline phosphatase and aspartate transaminase were stable at 561 IU/l and 151 IU/l respectively. In view of his longstanding pancolitis surveillance colonoscopy was offered but at this time he declined any further investigation of the colon.

The pattern of intermittent jaundice and rigors continued, although between these episodes he remained well. Five years after his initial presentation he agreed to colonoscopic surveillance. Four pedunculated polyps, each between 1 and 2 cm in size, were removed from the mid-ascending and descending colon. Histological review of these specimens revealed low grade dysplasia. His liver function over this period had deteriorated (table 1).

The prothrombin time was prolonged at 17.3 seconds. Full blood count, urea, electrolytes, and glucose were normal. Repeat abdominal ultrasound showed a complex cystic lesion in the left lobe of the liver, free

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Figure 1 Percutaneous transhepatic cholangiogram outlining the intrahepatic biliary tree which endoscopic retrograde cholangiopancreatography had failed to demonstrate in this patient with a past history of choledochoduodenostomy.

Table 1 Serial liver function tests 1991–1996

Date	Albumin (g/l)	Bilirubin (µmol/l)	Alkaline phosphatase (IU/l)	Aspartate transaminase (IU/l)	Prothrombin time (sec>control)
May 1991	39	50	990	120	1
Feb 1992	48	22	561	151	2
Nov 1993	42	32	602	185	2
Nov 1994	41	40	599	224	3
Jan 1996	36	64	652	213	2
Aug 1996	29	101	562	201	3
Dec 1996	28	172	399	172	16

intraperitoneal fluid, and marked splenic enlargement. Computed tomography confirmed the cystic abnormality in the left lobe to be in continuity with the left main hepatic duct. The appearance of the liver suggested macronodular cirrhosis with associated portal hypertension. Hydatid serology was negative and serum tumour markers (α -fetoprotein, CA 19-9) were normal. His liver function tests had worsened despite there being no clinical evidence of ongoing sepsis (table 1).

Questions

- (1) What does the PTC (fig 1) show and given the history of ulcerative colitis what is the most likely diagnosis?
- (2) What is the connection between this hepatological diagnosis and the findings of dysplasia at colonoscopy?
- (3) Given the deteriorating liver function tests, computed tomography findings, and colonoscopic changes how would you manage this case?

An elderly man with pleural effusion and abnormal behaviour

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Answers on p 413.

A 76 year old man, who had been an asbestos insulation worker, presented with a four month history of right sided severe diffuse chest pain, breathlessness on exertion, and weight loss of 10 kg. He had recurrent episodes of lethargy, agitation, and abnormal behaviour. These episodes were partially relieved by eating a high carbohydrate diet. On admission he was found to be unrousable. His Glasgow coma scale score was 7/15 with no focal neurological deficit. He had clubbing and right sided massive pleural effusion and contracted right hemithorax with mediastinal shift to same side. Full blood counts, urea, electrolytes, liver function tests, and serum calcium were normal. Plasma glucose concentrations were 1.5 mmol/l. He was given 50 ml of 50% dextrose intravenous with correction of his hypoglycaemia and he became conscious. A chest radiograph (posteroanterior) showed complete opacification of right hemithorax (fig 1). Computer tomography of his thorax revealed right pleural thickening encasing the collapsed right lung and contraction of right hemithorax. Pleural aspiration revealed a viscous jelly-like exudate with "atypical" mesothelial cells and high hyaluronidase levels. Pleural biopsy revealed a malignant sarcomatoid mesothelioma. The subsequent week was characterised by rapid reaccumulation of pleural effusion in spite of repeated aspirations. The hypoglycaemic episodes increased in frequency though he was on approximately 380 g of glucose intravenously per day with intramuscular glucagon intermittently.

Plasma glucose concentrations were 0.8 mmol/l, insulin (<25 pmol/l), C peptide (<75 pmol/l), and β -hydroxybutyrate (<20 mmol/l) were undetectable, serum growth hormone was 0.9 mU/l. Insulin-like growth factor-II (IGF-I) concentration was 0.16 U/ml (reference range 0.4–2.0), serum IGF-II concentration was raised at 2.0 U/ml, and ratio

Figure 1 Chest radiograph (posteroanterior) showing complete opacification of right hemithorax.

of IGF-I: IGF-II was 0.08 (normal >0.2). "Big" IGF-II concentration was 20.8 nmol/l (0–14.4) and IGF binding protein-3 (IGFBP-3) concentrations were 2.2 mg/l (2.0–4.8). A short tetracosactrin (Synacthen) test was normal.

Questions

- (1) What is the cause of this patient's hypoglycaemia?
- (2) What pharmacological agents may alleviate his hypoglycaemia?
- (3) What is the role of surgery in management of this patient?

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A difficult case of gastrointestinal haemorrhage

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Answers on p 414.

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A 64 year old man presented to the accident and emergency department, having been found collapsed in the street. He gave a four day history of melaena. On examination he was shocked, with a pulse of 140 beats/min and a blood pressure of 70/40 mm Hg. Initial haematological assessment revealed a haemoglobin concentration of 65 g/l, platelet count $68 \times 10^9/l$, and international normalised ratio 1.3. After resuscitation with intravenous colloid fluids, blood, fresh frozen plasma and platelets, an emergency upper gastrointestinal endoscopy was performed. This showed four large oesophageal varices, two of which were actively bleeding. Endoscopic sclerotherapy was attempted, but haemostasis was not achieved, and a Sengstaken tube was sited.

The next day the Sengstaken tube was removed and endoscopic band ligation of the oesophageal varices performed. The patient remained stable for 24 hours until there was evidence of further gastrointestinal bleeding, with passage of melaena, and haemodynamic compromise. A further upper gastrointestinal endoscopy was performed, which revealed a large quantity of blood in the stomach; the oesophagus was well visualised and confirmed not to be bleeding. A therapeutic procedure was performed (fig 1).

The procedure successfully stopped further gastrointestinal haemorrhage. However, two days later, he became drowsy and disoriented.

Figure 1 Therapeutic procedure performed on patient.

Questions

- (1) Following the insertion of the Sengstaken tube what additional management would you have instigated?
- (2) What was the cause of the rebleed and what procedure was performed?
- (3) What is the likely cause for the patient becoming drowsy, and how would you manage this?

Recurrent pulmonary oedema in a 53 year old woman

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Answers on p 416.

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A 53 year old woman presented with pulmonary oedema after an inferior myocardial infarction three months earlier. Blood pressure on admission was 210/120 mm Hg. Initial treatment included diuretics, nitrates, and oxygen and her symptoms settled. Echocardiography before discharge showed mild impairment of left ventricular contraction. Coronary angiography performed several months later showed an occluded right coronary artery, with a 50% stenosis of the left anterior descending vessel. Overall left ventricular function was good (ejection fraction of 58%) with some regional inferior hypokinesia. Over

the next two years, the patient was admitted on numerous occasions with hypertensive pulmonary oedema and was treated medically. Between admissions she remained symptom free although blood pressure control was difficult.

Questions

- (1) What is the most likely cause of this patient's recurrent pulmonary oedema?
- (2) What investigations should be performed next?
- (3) What treatment options are available to this patient?

A case of unusual septicaemia

R Sivakumar, E Roche, R M Faizallah

Answers on p 417.

A 74 year old man was admitted with lethargy. There was no significant past medical history. General examination was unremarkable. Investigations revealed iron deficiency anaemia with a haemoglobin concentration of 90 g/l. He developed swinging pyrexia and blood cultures grew a Gram positive organism in the anaerobic bottle. Barium enema was performed and shown in fig 1.

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Questions

- (1) What is the finding in the barium enema?
- (2) What is the association between the barium enema finding and septicaemia referred to?

Figure 1 Barium enema.

Hydronephrosis and a hard neck swelling

T A Chowdhury, C E Ellis, R Jaganathan, P M Dodson

Answers on p 418.

A 45 year old woman presented to a surgical clinic with an eight month history of intermittent colicky right loin and suprapubic pain associated with nausea, with no exacerbating or relieving factors. She had lost 1 stone (8 kg) in weight over that period. She had no bowel or menstrual disturbance, but did complain of polyuria and nocturia. One year earlier she had been diagnosed as hypothyroid by her general practitioner when she presented with a thyroid swelling and tiredness. Biochemistry at that time confirmed borderline hypothyroidism (free thyroxine 9.8 pmol/l (normal range 10.0–24.0), thyroid stimulating hormone 13.2 mU/l (0.3–4.6), thyroid microsomal antibodies positive, titre 1:1200). She was treated with thyroxine 100 µg daily, with no improvement in her tiredness. She had no other past medical history of note. Cardiorespiratory examination was normal. Examination of the neck showed a stony hard diffusely enlarged thyroid gland, although she was clinically euthyroid. Examination of the abdomen showed tenderness to deep palpation and the suggestion of a mass in the right loin. Biochemical profile showed mild renal impairment with a serum creatinine of 143 µmol/l (50–100), but was otherwise normal. Amylase, thyroid function test, and full blood count were all normal. Erythrocyte sedimentation rate was raised at 72 mm/hour, as was C reactive protein at 28 mg/l (0–10).

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Figure 1 Computed tomogram of the abdomen and pelvis.

A renal ultrasound scan was performed, and then subsequently computed tomography of the abdomen and pelvis (fig 1). The latter showed a markedly hydronephrotic right kidney, and a periaortic soft tissue mass, encasing the aorta, inferior vena cava, and iliac arteries.

Questions

- (1) What is the likely diagnosis based on the computed tomogram appearance?
- (2) What is the link with the thyroid abnormality?

A woman with an abnormal mass in the thyroid gland

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Answers on p 419.

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A 69 year old woman presented with a sudden history of dysphagia and stridor. A month earlier she had noticed neck swelling and hoarseness of voice after a bout of flu. Her appetite was fine, weight steady, and there was no history of fever or night sweats.

Past history included a hysterectomy, and she had had insulin dependent diabetes for the past 10 years. She did not have any family history of diabetes or thyroid disease and her medications included insulin and hormone replacement therapy. On examination she was acutely short of breath with cyanosis and frank stridor. There were no signs of thyroid overactivity but she had obvious vitiligo in her neck and shoulder area. Neck examination revealed a hard and fixed non-tender swelling mainly in the midline with some prominence on the left side. The rest of her systemic examination was unremarkable.

She was given intravenous hydrocortisone and started on insulin infusion and regular oral steroids. Blood tests showed a normal full blood count, erythrocyte sedimentation rate,

calcium, and renal and liver function. A thyroid profile showed a free thyroxine concentration of 22.4 pmol/l (normal range 9.6–26.5 pmol/l) and thyroid stimulating hormone of 6.6 mU/l (normal range 0.6–4.8 mU/l) indicative of “compensated euthyroidism”. The thyroperoxidase antibody level was strongly positive.

She was started on thyroxine and gradually she began to feel a lot better. Fine needle aspiration/tru-cut biopsy of her neck mass was attempted but was non-conclusive on two successive occasions.

Questions

- (1) What other investigations would you consider?
- (2) What is the diagnosis and what is it commonly mistaken for?
- (3) What is a common predisposing factor?
- (4) What is the treatment and prognosis of this condition?

A hungry baby fails to thrive

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Answers on p 420.

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A 6 week old boy, who had been well at birth after a normal delivery at term, was admitted to the local children's hospital in Belfast with a two day history of irritability and poor colour. He had been feeding well and always appeared hungry, taking excessive quantities of formula milk (up to 265 ml/kg/day). He had no vomiting or diarrhoea, but had not regained his birth weight of 2270 g (<3rd centile).

On admission he was extremely restless, with a depressed fontanelle, alert eyes and pale mottled skin. He showed an unusually high level of consciousness of such a severely dehydrated infant. A ketotic smell was detectable on his breath. He had a marked increase in respiratory effort, with a respiratory rate 65 breaths/min, heart rate 200 beats/min, and temperature 38°C. He passed large volumes of very dilute urine. Urinalysis showed glucose +++++, protein ++, blood +, and a large amount of ketones. He developed generalised seizure activity during his initial rehydration. The initial laboratory findings are summarised in the box.

Blood results on admission

- pH = 7.162
- Oxygen pressure = 15.0 kPa
- Carbon dioxide pressure = 1.41 kPa
- Bicarbonate = 3.7 mmol/l
- Base excess = -22.7 mmol/l
- Haemoglobin = 107 g/l
- White cell count = $19.4 \times 10^9/l$
- Platelet count = $737 \times 10^9/l$
- Potassium = 5.8 mmol/l
- Sodium = 154 mmol/l
- Urea = 15.1 mmol/l
- Creatinine = 62 $\mu\text{mol/l}$
- Blood glucose = 73.2 mmol/l

Questions

- (1) What is the diagnosis?
- (2) What is the likely long term outcome?
- (3) What is the genetic linkage to this condition?

An unusual case of diarrhoea and weight loss

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Answers on p 421.

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Table 1 Laboratory investigations

Test	Result	Reference range
Sodium (mmol/l)	136	135–145
Potassium (mmol/l)	4.3	3.8–5.0
Urea (mmol/l)	5.4	2.5–7.1
Creatinine (μmol/l)	63	71–133
Glucose (mmol/l)	3.6	
Albumin (g/l)	34	35–50
Calcium (mmol/l)	2.29	2.10–2.55
Thyroid stimulating hormone (mU/l)	3.5	0.5–4.5
Prolactin (mU/l)	336	39–450
Testosterone (nmol/l)	22	9–24
Luteinising hormone (U/l)	4.5	2.1–9.3
Follicle stimulating hormone (U/l)	4.3	0.9–8.3
Haemoglobin (g/l)	105	130–180
Mean corpuscular volume (fl)	86	80–100
Erythrocyte sedimentation rate (mm/hour)	107	<20
Antithyroid microsomal antibody	Positive (titre 1:18432)	
Adrenal antibody	Negative	

A 70 year old man had been attending an out-patient clinic for investigation of diarrhoea, loss of appetite and weight loss, for which full clinical investigation (including duodenal and colonic biopsy series) had revealed no clear cause. Over a period of 10 years his weight had fallen from 82 kg to 64 kg. There was no history of vomiting. On this occasion he complained of dizziness, especially when he stood up. A slight postural fall in blood pressure was noted (120/70 to 105/70 mm Hg on standing) and a random serum cortisol was requested. This was reported as 27 nmol/l and he was admitted as an emergency for investigation.

He had been diagnosed as hypothyroid several years previously when his thyroid stimulating hormone (thyrotrophin) concentration had been 47 mU/l. Thyroxine replacement (150 μg/daily) had resulted in some amelioration of his diarrhoea (although he still required occasional loperamide) consistent

with this being an uncommon manifestation of his hypothyroidism.¹ He had also been investigated for a long standing (four year) normochromic normocytic anaemia and raised erythrocyte sedimentation rate for which no cause had been found. Ten years previously he had had problems with erectile failure which had not responded to papaverine and he had accepted that he would no longer have erections; testosterone and gonadotrophins at that time had been normal. He did not smoke or drink and had no cough or wheeze. There was no drug history other than those noted above.

On examination he was pale and thin with no goitre. There were no signs of hyperpigmentation. He was afebrile and some peripheral oedema was noted. His pulse was 74 beats/min and a postural fall was again observed (140/80 to 110/80 mm Hg). Laboratory investigations are shown in table 1.

A short tetracosactrin (as Synacthen; Novartis) test (250 μg tetracosactrin given intramuscularly) was undertaken; serum cortisol concentrations at 0, 30, and 60 mins after injection were 28, 87, and 120 nmol/l respectively.

Questions

- (1) What is the differential diagnosis?
- (2) What further investigations would you request?
- (3) How would you manage the patient?

¹ Donowitz M, Kokke FT, Saidi R. Evaluation of patients with chronic diarrhea. *N Engl J Med* 1995; 32:725–9.