SELF ASSESSMENT ANSWERS

Purple skin and a swollen thigh in an alcoholic

Q1: How would you describe these skin lesions? What is the differential diagnoses?

Figure 1 (see p 430) shows confluent ecchymosis in the background of icteric skin, mainly involving the left thigh. The differential diagnoses of non-palpable purpura/ecchymosis include primary skin conditions (trauma, solar purpura, steroid purpura, capillaritis, and lividoid vasculitis), clotting disorders (thrombocytopenia, clotting factor deficiency, abnormal platelet function), conditions characterised by vascular fragility (amyloidosis, Ehlers-Danlos syndrome, and scurvy), conditions such as monoclonal cryoglobulinaemia, warfarin therapy, cholesterol and fat embolism, autoerythrocytic sensitivity, and Waldenstrom's macroglobulinaemia.[1]

Q2: What is the most likely cause of this patient's skin lesions and thigh abnormality? What features of the skin lesion (fig 1, inset; see p 430) suggests this diagnosis? How will you confirm the diagnosis?

The principal diagnosis to consider in this patient is scurvy (vitamin C deficiency). As is evident, the haemorrhagic lesions are perifollicular, with hyperkeratotic papules (fig 1, inset; see p 430). This patient had mild thrombocytopenia and a mildly prolonged INR, neither of which is likely to produce ecchymosis and haematoma in a single anatomic area in the absence of significant local trauma. As laboratory testing suggested cholestasis, this patient received empirical vitamin K injections (10 mg) subcutaneously for three days, but without clinical improvement. Indeed, spontaneous bleeding into thigh muscle is rare, even in patients with INR values >2.0, such as those on therapeutic anticoagulation with warfarin (coumadin). Sreenivas *et al* reported two patients on warfarin who had spontaneous iliopsoas haematomas resulting in compressive femoral neuropathy,[2] as have others, but this patient did not have evidence of iliopsoas bleeding on computed tomography of the abdomen.

Follicular hyperkeratosis and perifollicular haemorrhages are such a distinctive occurrence in scurvy that they are almost pathognomonic.[3] Also, scurvy skin lesions tend to preferentially involve the legs. The various clinical and radiological manifestations of scurvy are listed in box 1. A useful aide memoire about scurvy manifestations is to remember "the four Hs"—*namely, haemorrhage, hyperkeratosis, hypochondriasis, and haematological manifestations*.[4] The diagnosis of scurvy needs a high index of clinical suspicion. It is facilitated by obtaining a good dietary history and is confirmed by plasma and leucocyte ascorbic acid levels. This patient had erratic food habits, and his diet did not include fresh fruits/fruit juices and vegetables. His plasma ascorbate level was 5.68 µmol/l (laboratory normal range 23–57 µmol/l). In eumetabolic individuals, plasma ascorbate levels should be more than 35.2 µmol/l. The normal body pool of ascorbic acid is about 1500 mg (85 mmol) and 3% of this body pool turns over each day, resulting in a half life of about 18 days.[1] Scorbutic manifestations tend to occur at levels below 300 mg (17 mmol).[1 3] Whole body ascorbate level estimation is impractical, and hence the need for plasma and leucocyte ascorbate levels. Plasma values are affected by recent dietary intake, while leucocyte levels, which changes more slowly, better indicate tissue and total body content.[3] Ascorbic acid ($C_6H_8O_8$), a ketolactone with a molecular weight of 176.1, is required for the peptidyl hydroxylation of procollagen, and its deficiency results in abnormal and unstable triple helical structure of collagen.[4] This leads to defective perivascular supportive tissues resulting in capillary fragility and poor wound healing. Ascorbic acid is synthesised by most animals from glucoronic or galactonic acid, which in turn are synthesised from glucose.[4] Only a few species, including humans, non-human primates, the Indian fruit bat, monkeys, several species of bulbuls, and guinea pigs lack the enzyme system required for the production of L-ascorbate from glucose. In these species, ascorbic acid must be obtained from exogenous sources.[3 4]

Box 1: Manifestations of scurvy*

1. Constitutional: lassitude, diminished appetite and fatigue. Perifollicular skin haemorrhage and hyperkeratosis, splinter haemorrhages of nails.
2. Leg oedema.
3. Hair abnormalities: alopecia; "swan neck" deformity—hair bent in many places; "corkscrew" deformity—coiled, fractured hair.
4. Red, smooth, shiny, and swollen gums: especially the interdental and marginal gingivae.
5. Haemorrhage: into muscles and soft tissues (swelling, myalgia, pseudoparalysis); into subperiosteal bone (bone pain) and, rarely, fractures; in the eye—subconjunctival, periorbital, and intraretinal haemorrhages; gastrointestinal bleeding.
6. Haematological: anaemia (normocytic and normochromic, can be macrocytic and/or megaloblastic in 20% cases); leucopenia and thrombocytopenia rarely; reticulocytosis and reduced haptoglobulin levels.
7. Others: irritability and cognitive impairment; dyspnoea and cardiac failure in severe deficiency states.
8. Radiographic changes (children and adults): onset six months to two years; earliest signs in knees; osteoporosis, loss of epiphyseal density with a pencil thin cortex (Wimberger sign), dense zone of provisional calcification (excess osteoid calcification), metaphyseal lucency (Trummerfeld zone), metaphyseal corner fractures (pelkan spurs), and periosteal reaction due to subperiosteal haemorrhage.

* Adapted from references 1, 3 , 4, and 5.

Q3: How is this condition treated?

Very small quantities of ascorbic acid—as little as 6.5 mg daily—can result in a clinical cure.[1 3] However, the dose that corrects the deficit and repletes body stores expeditiously is 100 mg thrice daily.[3] Improvement can be dramatic, and sometimes within days, and usually within a few weeks. A subjective sense of wellbeing is often apparent within the first 24 hours, and the lethargy, pain, and anorexia diminish in two to three days. When joint symptoms occur, improvement is seen within a few days. The skin lesions become purplish, may become pigmented, and resolve eventually in a few weeks. Hyperkeratosis decreases in about two weeks. Except for lost teeth, permanent tooth damage from scurvy does not occur.[3] Dietary advice is mandatory. More than 90% of vitamin C in western diets comes from fruits and vegetables, including potatoes, tomatoes, berries, green vegetables, and citrus fruits. Liver and kidney contain ample amounts, but most other meat, poultry, dairy products, and grain contain little or no ascorbic acid unless fortified.[1 3] This patient's symptoms, skin lesions, and muscle swelling improved dramatically with ascorbic acid repletion.

In summary, this patient had scurvy, alcoholic liver disease, and gallstones with extrahepatic biliary dilatation. He was advised to undergo endoscopic retrograde cholangiopancreatography as further work-up, but he refused and was discharged home.

Final diagnosis

Scurvy (hypovitaminosis C) with skin and soft tissue haemorrhage.

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An unusual case of multiple infarcts

Q1: What are the differential diagnoses before imaging?

Conditions which may present as a simultaneous stroke and apparent myocardial infarction include subarachnoid haemorrhage, a vasculitis (such as polyarteritis nodosa), drug abuse (for example, cocaine, ecstasy), infective endocarditis, mechanical valve failure, and cardiac tumours. Although coronary arterial embolism is a recognised complication of left sided cardiac tumours simultaneous myocardial and cerebral infarction from such lesions is very rare.[1 2]

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Q2: What does the echocardiogram show (see p 431)?

Echocardiography revealed a large pedunculated tumour arising from the left atrium. The typical features can be seen of the tumour obstructing the mitral orifice at diastole. Doppler echocardiography demonstrated a degree of associated mitral incompetence.

Q3: What is the final diagnosis?

Cerebral and myocardial infarcts due to emboli (either thrombus or tumour fragments) arising from a large left sided cardiac tumour—an atrial myxoma.

Atrial myxoma is the commonest primary cardiac tumour. It may occur at any age but is most common in middle age. The prevalence is estimated at up to five per 10 000 from autopsy series, with a 2:1 female preponderance. Much fewer are recognised during life with an estimated prevalence of two per 100 000 in the general population. They are usually solitary, pedunculated, and typically occur in the left atrium, arising from the interatrial septum near the fossa ovalis.

Myxomata may present with obstructive, embolic, or constitutional manifestations. Most commonly it may mimic mitral valve disease, both stenosis (due to tumour obstruction of valve orifice) and regurgitation (due to valve trauma). The symptoms and signs may be positional in nature, and classically a "tumour plop" (a low pitched sound during early/mid-diastole) is heard. It should be noted, however, that as a cause of left atrial obstruction, myxomata are approximately 200 times less common than mitral stenosis. Embolic phenomena occur in 40% and may be simply thrombus or contain tumour. Constitutional effects include fever, weight loss, Raynaud's phenomenon, finger clubbing, anaemia, polycythaemia, thrombocytopenia, leucocytosis, a raised erythrocyte sedimentation rate, and raised immunoglobulin levels. It is therefore often misdiagnosed as conditions such as endocarditis, connective tissue disorders, and non-cardiac malignancy.

Q4: What is the treatment?

Surgical resection is almost mandatory. Ideally this should be an urgent elective procedure. This case necessitated emergency resection within 24 hours of presentation. At surgery a very large gelatinous tumour mass ($7 \times 6 \times 5$ cm) was removed from the inferior margin of the fossa ovalis and the inferior wall of the left atrium. Histology showed an extensively myxoid stroma (fig 3; see p 431). Immunohistochemistry was positive for CD34, S100, neuron specific enolase, and vimentin consistent with a diagnosis of atrial myxoma.

Q5: What is the prognosis?

Surgical resection is usually successful, with a mortality rate <3% for elective procedures, but higher for emergency resections. The main perioperative risks are embolisation to the cardiac or cerebral circulation. Rarely the tumours recur.[3] The patient described improved rapidly after surgery and made an excellent neurological and cardiological recovery.

Q6: Is there any risk to her children?

Autosomal dominant inheritance of atrial myxomata may occur as part of Carney's syndrome, characterised by multiple myxomata (from anywhere in the heart, and elsewhere), lentiginosis, and various endocrine hyperactivity.[4] In contrast the patient described here had a typical sporadic myxoma.

Q7: How could the diagnosis have been made earlier?

In this case the degree of haemodynamic embarrassment pointed towards the need for emergency reperfusion of the heart by angioplasty. Cardiac catheterisation and coronary angiography were therefore performed and provided the diagnosis almost by serendipity. However, in the context of a simultaneous stroke and myocardial infarction, early transthoracic echocardiography should be performed since this has a high degree of sensitivity and specificity, and is applicable even in critically ill patients. Coronary angiography would still be required in the work up for surgical resection if the presence of a left atrial myxoma was confirmed.

Final diagnosis

Atrial myxoma.

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Abdominal distension

Q1: What does the plain abdominal radiograph (fig 1; see p 432) show? What was the subsequent investigation (fig 2; see p 432) and what does this show?

Pneumoperitoneum and small bowel fluid levels are shown. The subsequent investigation is a small bowel barium meal, and this shows jejunal diverticulosis. The presence of small bowel fluid levels on the plain abdominal radiograph suggests that jejunal diverticulosis is the most likely underlying cause.

Q2: How would you manage this patient?

Exploratory laparotomy is mandatory if the patient presents with clinical stigmata of peritonitis. However, spontaneous pneumoperitoneum can occur without peritonitis or perforation of a viscus. Jejunal diverticulosis and pneumatosis cystoides intestinalis are the most common gastrointestinal causes of this condition. Where a non-surgical cause of pneumoperitoneum can be discerned and there are no associated findings to suggest peritonitis or a perforated viscus, then continued observation should avoid an unnecessary laparotomy. This was the case in our patient whose distention improved with conservative management. In instances where aetiology of the pneumoperitoneum remains unclear, a diagnostic peritoneal lavage may obviate the need for laparotomy.

Q3: What mechanism results in the development of the radiological abnormality shown in fig 1 (see p 432)

The mechanism of pneumoperitoneum in jejunal diverticulosis is relatively unclear. It is thought to result from the passage of intraluminal gas, without gross faecal contamination, into the peritoneal cavity through perforations in the wall of the thin walled diverticula. Hyperactive peristaltic activity and fermentation in the diverticula may also contribute. Our patient subsequently developed diarrhoea and underwent a hydrogen breath test, which was markedly abnormal, indicating bacterial overgrowth in the small bowel.

Discussion

Diverticulosis of small intestine is a relatively unusual finding and the cause of significant symptoms in less than 50% of patients in whom diverticula are found.[1] The most frequent symptoms are a result of low grade intestinal obstruction. These consist of upper abdominal discomfort, and fullness and vomiting after meals. Other presentations include acute obstruction, inflammation, bleeding, perforation, inspissation with enterolith formation, and macrocytic anaemia. Spontaneous pneumoperitoneum unassociated with signs or symptoms of peritoneal irritation is an uncommon presentation of small intestinal diverticulosis. Since the vast majority of these patients are asymptomatic and remain so, conservative management is indicated.

Recognition of this entity is important if unnecessary surgery for a suspected perforation of the gastrointestinal tract is to be avoided.[2]

Pneumoperitoneum is usually the result of hollow viscus perforation with associated peritonitis.[3] Common causes are perforated duodenal or gastric ulcers, perforation of colon diverticula or appendix, and perforated ulcerative colitis or amoebic colitis.[4] Spontaneous pneumoperitoneum consequent upon intrathoracic, intra-abdominal, gynaecological, iatrogenic, and other miscellaneous conditions not associated with perforated viscus have been documented in the literature. In some instances no cause for pneumoperitoneum is evident.

Final diagnosis

Spontaneous pneumoperitoneum secondary to jejunal diverticulosis.

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Prolonged fever with recurrent diarrhoea

Q1: What is the single most important investigation?

Multiple blood cultures. Blood cultures grew *Salmonella enteritidis* in this patient.

Q2: What is the diagnosis?

Mycotic aneurysm of the common iliac artery. The history of self limiting episodes of diarrhoea and vomiting should make one to suspect salmonella mycotic aneurysm.

Q3: What are the risk factors for this complication?

The commonest risk factors for salmonella mycotic aneurysms are age greater than 50 years, atherosclerosis, diabetes mellitus, and immunocompromised states especially AIDS. Besides the age, there were no other risk factors in this patient.

Discussion

Salmonellae are non-encapsulated, non-sporeforming Gram negative rods whose classification continues to evolve. All salmonellae but one (*Salmonella bongori*) are widely believed to be members of a single species *S enterica* which is further divided into seven groups considered as subspecies encompassing on a whole at least 2324 serovars.[1]

Salmonellosis may manifest in five different clinical forms including asymptomatic chronic carrier state, gastroenteritis, enteric fever, bacteraemia, and extraintestinal localised complications of which endovascular infection is one of the most serious.

Though *S typhi* and *S paratyphi* infect only humans and cause enteric fever and chronic carrier state, non-typhoidal salmonella are widely spread in nature and affect all age groups causing gastroenteritis, bacteraemia, and extraintestinal localised complications. Despite high standards in food processing, contaminated food products remain the main source. In a recent study of non-typhoidal bacteraemia, adults were more likely to have predisposing factors and a high incidence of extraintestinal organ involvement and a high mortality.[2]

Mycotic aneurysms are localised dilatations of arterial wall that develop secondary to an infective process spreading either contiguously from an adjacent source of infection or more commonly haematagenously. The incidence of 10% for endovascular complications in salmonella bacteraemia in patients over 50 years is consistent across three studies.[2–4] The commonest predisposing factors for salmonella mycotic aneurysms are age greater than 50 years, atherosclerosis, diabetes mellitus, and immunocompromised states especially AIDS. The clinical course can be acute, subacute, or chronic and cases of aneurysm have been reported even after six months of primary infection. Almost every arterial site in the body may be involved; however, infections of the aorta especially the infrarenal segment appear to be the most frequent. Nearly all instances of salmonella aortitis result in aneurysm or more rarely enlargement of a previously existing aneurysm. The diagnosis of vascular infection due to salmonella requires a high index of suspicion, and the important clues are listed in the box 1.

The assessment should be done urgently in order to reduce morbidity and mortality as the aneurysms may rapidly expand and rupture. The method of choice for diagnosing infected aneurysms appears to be computed tomography of the chest and abdomen. Surgical resection should soon follow the start of effective antimicrobial therapy, which is mainly with quinolones or third generation cephalosporins. Although in situ repair has been reported as successful in some patients,[5] restoration of blood flow by extra-anatomical bypass with or without subsequent reconstruction seems to lead to improved short and long term prognosis. Although no consensus exists on the length of postoperative antibiotic treatment, it should be for at least six weeks and may be for life in immunocompromised individuals.[6]

Box 1: Clinical clues for vascular infection with salmonella

- Prolonged fever after an episode of gastroenteritis.
- Recurence of salmonella bacteraemia during or after adequate treatment.
- Pain in the back, abdomen, or chest accompanied by salmonella bacteraemia.
- Vertebral spinal involvement with salmonella bacteraemia.
- Salmonella bacteraemia in patients with prosthetic vascular grafts.

Learning points

- The diagnosis of vascular infection due to salmonella requires a high index of suspicion.
- Assessment should be done urgently as resultant aneurysms may rapidly expand and rupture.

Final diagnosis

Salmonella mycotic aneurysm of the common iliac artery.

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Pleuritic pain and a rare complication

Q1: Given his history of haemoptysis and subsequent severe pleuritic pain, what further diagnosis would you consider and how would you treat this?

Given these symptoms, the hypoxia, and findings on electrocardiography the diagnosis of pulmonary embolism should be considered. Routinely this is treated with low molecular weight heparin and urgent V/Q scan is requested.

Q2: What condition was diagnosed from his computed tomogram (fig 2; see p 433) and what further management was indicated?

His abdominal computed tomogram appearances were in keeping with a splenic rupture with large associated haematoma. The condition he suffered was spontaneous rupture of spleen (SRS). He underwent emergency splenectomy. Operation revealed one litre of free fluid in the abdominal cavity with a fragmented spleen.

Q3: What did the laboratory find on reviewing this patient's blood film?

Atypical lymphocytes.

Q4: What follow up testing did they perform and what diagnosis was made?

A Monospot test was performed and confirmed the diagnosis of infectious mononucleosis.

Q5: What is the significance of his shoulder discomfort?

Kehr's sign is defined as pain and hyperasthesia over the left shoulder and may be due to splenic rupture.

Discussion

Spontaneous rupture of spleen is a rare complication of infectious mononucleosis with several documented cases,[1] its incidence is between 0.1% to 0.5%,[2] and other less common causes of SRS include influenza, rubella, tuberculosis, and lymphoma.[3] It is a potentially life threatening complication.

This patient had typical clinical and radiological features of pneumonia. The subsequent diagnosis of pulmonary embolism was based on the presence of significant left pleuritic chest pain, previous haemoptysis,

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Learning points

- Infectious mononucleosis can lead to the rare complication of SRS.
- Beware of pleuritic pain in infectious mononucleosis. Chest pain accompanied by Kehr's sign may be the only indication of SRS.
- If infectious mononucleosis is suspected, syncope or a falling haemoglobin level should always be investigated by urgent ultrasound/computed tomography to check that there is no underlying SRS.

hypoxia, and slightly abnormal electrocardiography. The planned V/Q scan was not done as the patient had deteriorated. However he also had manifestations of infectious mononucleosis—sore throat, fever, anorexia, malaise, and dysphagia. Confirmation of infectious mononucleosis by Monospot was established at the time of SRS.

The largest review of infectious mononucleosis cases with SRS is a retrospective analysis by the Mayo clinic.[1] The predominant features of SRS were left upper quadrant pain and tenderness, splenomegaly, and Kehr's sign. Certainly in our case, Kehr's sign was an initial complaint but did not remain a persistent feature. The Mayo clinic review also highlighted that abdominal pain may be a late presenting sign and that patients can have a low haemoglobin level or develop a marked drop in haemoglobin level.

The majority of patients with SRS undergo splenectomy.[1 2 4] This patient underwent emergency splenectomy and in our opinion surgery was mandatory because of his falling haemoglobin level, persistent symptoms, and large splenic rupture. Some authors do advocate conservative management in SRS due to infectious mononucleosis[1 2] but only with specified criteria such as haemodynamic stability and accurate transfusion assessment.

Another issue is the use of low molecular weight heparin and whether this was a precipitating factor for SRS. SRS has been reported in a patient who was on warfarin after myocardial infarction[5]; no other causal factor was identified. It is unlikely that the heparin used in this case was the main causal factor for SRS as the patient had already had symptoms and signs prior to treatment. However its use certainly would have exacerbated an already ruptured or weakened spleen.

The diagnosis of SRS is usually confirmed by abdominal ultrasound or computed tomography.

Final diagnosis

Spontaneous rupture of spleen secondary to infectious mononucleosis.

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An unusual endocrine cause of hyponatraemia

Learning points

- Although hyponatraemia has been classically associated with hypothyroidism, thyrotoxicosis can also precipitate hyponatraemia in patients with short bowel syndrome.
- A variety of restorative adaptive mechanisms occur in patients with short bowel syndrome to maintain physiological equilibrium.
- Hyponatraemia is a common in-hospital electrolyte abnormality and there should be a reasonable investigation protocol set-up for the work-up of such patients.

Q1: What further investigations would you perform and how would they help you?

Apart from the routine investigations, serum and urinary osmolality, urinary excretion of sodium, short Synacthen test (for adrenal reserve), and thyroid function would help in distinguishing the various causes of hyponatraemia. Hypovolaemic hyponatraemia (common causes being fluid loss from skin, gastrointestinal tract, respiratory system, third space collections, renal loss, and HIV infection) is characterised by serum osmolality of <280 mosmol/kg and clinical dehydration. Isovolaemic hyponatraemia (common causes being the polydipsias, hypokalaemia, renal failure, hypothyroidism, and adrenal insufficiency) is associated with a serum osmolality of >280 mosmol/kg. The syndrome of inappropriate antidiuretic hormone secretion (SIADH) is characterised by urine osmolality greater than 200 mosmol/kg with a spot urinary sodium >20 mmol/l in the presence of normal adrenal, renal, and thyroid function. A spot urine sodium of <30 mmol/l is suggestive of an extrarenal cause of hypovolaemia.

Q2: What is "short bowel" syndrome and how does it cause hyponatraemia?

"Short bowel" syndrome refers to the clinical sequel as a result of excision of a substantial length of bowel. Sodium homoeostasis in health depends largely on renal regulation of its urinary excretion, thirst, and antidiuretic hormone secretion. Losses of sodium from bowel are small in health. The usual common sources of massive sodium losses are from the intestinal tract and kidneys.[1] The pathophysiological consequences depend on extent and site of resection, adaptation and integrity of the remaining bowel, and whether the colon has been preserved or not.[2] Patients with jejunostomy have a higher faecal output of water, sodium, and divalent cations and they often need a permanent parenteral supply of saline if their small bowel length is less than 200 cm and "parenteral" nutrition support if they retain <100 cm small bowel. In contrast, 50 cm of the jejunum often suffices for adequate oral nutrition if most of the colon is preserved[2] as the colon is believed to take on some small bowel features (especially absorption of salt and water) and result in more efficient fermentation. Ileum and colon avidly absorb sodium against a concentration gradient. In the model of short gut syndrome, the major adaptive change is said to be decreased intestinal flow rate related to delayed gastroduodenal emptying.[3]

Q3: What is the diagnosis here and how did this possibly cause hyponatraemia?

The patient described was clinically dehydrated with a reduced serum osmolality of 248 mosmol/kg and urinary excretion of sodium less than 10 mmol/l. This would be consistent with hypovolaemic hyponatraemia and extrarenal sodium loss. Synacthen test was normal but thyroid function revealed a free thyroxine of 35 pmol/l (normal 9.8–23 pmol/l), a free triiodothyronine of 9.0 pmol/l (normal 3.5–6.5 pmol/l), and a suppressed thyroid stimulating hormone of 0.01 IU/l (normal 0.35–5.5 IU/l) in keeping with thyrotoxicosis. Thyroid antibodies were undetectable.

Hyponatraemia is well recognised in patients with primary hypothyroidism, especially in severe forms[4 5] and can also develop in patients with secondary hypothyroidism.[5] However, hyponatraemia associated with thyrotoxicosois is rare and very few cases have been described in literature. Thyrotoxicosis is characterised by changes in the cellular content of sodium and potassium while fluctuations in the intracellular fluid composition are less important and less stable. Diurnal urinary excretion, glomerular filtration rate, and excretion of sodium and creatinine has been shown to be increased in mild to moderately severe thyrotoxicosis.[6] Thyrotoxicosis is also classically known to be associated with increased intestinal motility and gastric emptying.[7] This together with the fact that the patient had ileostomy (short bowel syndrome) probably led to an increase in sodium and water loss through the ileostomy resulting in hyponatraemia. Low urinary sodium excretion further supports the hypothesis that sodium loss was extrarenal, presumably gastrointestinal, and reflects increased bowel activity secondary to thyrotoxicosis (unfortunately, we were not able to quantify sodium loss through the ileostomy).

Thus, we describe a patient with long standing uncomplicated ileostomy and associated new onset thyrotoxicosis which was probably coincidental, but nevertheless instrumental in precipitating the acute onset of hyponatraemia. Hyponatraemia is a common in-hospital electrolyte abnormality. Since its pathophysiology is quite varied, accurate diagnosis of the cause of hyponatraemia is essential for the implementation of correct management. Thus, thyrotoxicosis should be considered in the differential diagnosis when

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hyponatraemia occurs in patients with short bowel syndromes.

Final diagnosis

Thyrotoxicosis precipitating hyponatraemia in short bowel syndrome.

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