Purple skin and a swollen thigh in an alcoholic

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

The principal diagnosis to consider in this patient is scurvy (vitamin C deficiency). As is evident, the haemorrhagic lesions are perifollicular, with prominent keratotic papules (fig 1, inset; see p 430). This patient had mild thrombocytopenia and a mildly prolonged prothrombin time and was being treated with warfarin. He also had a history of recent dental surgery, which might account for the haemorrhagic lesions.

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?

Q2: The most likely cause of this patient’s skin lesions is scurvy (vitamin C deficiency). As is evident, the haemorrhagic lesions are perifollicular, with prominent keratotic papules. This patient had mild thrombocytopenia and a mildly prolonged prothrombin time and was being treated with warfarin. He also had a history of recent dental surgery, which might account for the haemorrhagic lesions.

Q3: How is this condition treated?

Vitamin C is rapidly repleted and symptoms resolve; however, replacement therapy is necessary in cases of chronic deficiency. Ascorbic acid is usually given as a daily supplement of 10 mg/kg or 100 mg thrice daily. In acute cases of scurvy, intravenous ascorbic acid may be required. The skin lesions become purplish, may occur, improvement is seen within a few days. A subjective sense of wellbeing is expeditiously is 100 mg thrice daily.

Final diagnosis

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

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), infections (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.
Q2: What does the echocardiogram show? (See p 432)?
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—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 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.

Myxoma 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, myxoma 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, polyuria, and polydipsia, thermal hyperactivity, and vimentin consistent with a diagnosis of atrial myxoma.

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 x 6 x 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 <5% for elective procedures, but higher for emergency resections. The main perioperative risks are embolisation to but higher for emergency resections. The surgical resection is usually successful, with a mortality rate, and raised immunoglobulin levels. It is therefore often misdiagnosed as one of the features can be seen of the tumour obstructing the mitral orifice at diastole. Doppler echocardiography demonstrated a degree of associated mitral incompetence.

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 coronary angiography were therefore performed and provided the diagnosis almost by serendipity. However, in the context of a simultaneous stroke and myocardial infarction, early trans-thoracic 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.

References

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 pneumatosus 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.

References
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?
Myotic aneurysm of the common iliac artery.

Q3: What are the risk factors for this complication?
The commonest risk factors for salmonella myotic 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. Nearby 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 below.

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, 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.

Box 1: Clinical clues for vascular infection with salmonella

- Prolonged fever after an episode of gas troenteritis.
- Recurrence of salmonella bacteraemia during or after adequate treatment.
- Pain in the back, abdomen, or chest accompanied by salmonella bacteremia.
- Vertebral spinal involvement with salmonella bacteremia.
- Salmonella bacteremia in patients with prothetic 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.

Pleural 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 hyperaesthesia 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, its incidence is between 0.1% to 0.5%, and other less common causes of SRS include influenza, rubella, tuberculosis, and lymphoma. 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,
An unusual endocrine cause of hyponatraemia

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 hypo- natraemia. 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 osmolal- ity of <280 mosmol/kg and clinical dehydration. Isovolaemic hyponatraemia (common causes being the polydipsia, hypokalaemia, renal failure, hypothyroidism, and adrenal insufficiency) is associated with a serum osmolality of >280 mosmol/kg. The syn- drome 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 hyponatraemia.

Q2: What is “short bowel” syndrome and how does it cause hyponatraemia?
“Short bowel” syndrome refers to the clinical sequela as a result of excision of a substantial length of bowel. Sodium homeostasis 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. The pathophysi- ological consequences depend on extent and site of resection, adaptation and integrity of the remaining bowel, and whether the colon has been preserved or not. 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 as the colon is believed to take on some small bowel features (especially absorption of salt and water) and result in more efficient fermentation. Illium 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 intesti- nal flow rate related to delayed gastroduo- denal emptying.

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 was normal (free T3 3.9 pmol/l, free T4 14.5 pmol/l, normal 7.0–12.9 pmol/l), and a suppressed thyroid stimulating hormone of 0.01 IU/l (normal 0.35–5.1 IU/l) in keeping with thyrotoxicosis. Thyroid antibodies were undetect- able. Hyponatraemia is well recognised in pa- tients with primary hypothyroidism, especi- ally in severe forms and can also develop in patients with secondary hypothyroidism. However, hyponatraemia associated with thyrotoxicosis is rare and very few cases have been described in literature. Thyrotoxicosis is characterised by changes in the cellular con- tent of sodium and potassium while fluctua- tions in the intracellular fluid composition are less important and less stable. Diurnal urinary sodium excretion, glomerular filtration rate, and excretion of sodium and creatinine has been shown to be increased in mild to moderately severe thyrotoxicosis. Thyrotoxi- cosis is also classically known to be associated with increased intestinal motility and gastric emptying. 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 hy- pothesis 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 an associ- ated new onset thyrotoxicosis which was probably coincidental, but nevertheless in- strumental 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 hypovolaemia, and slightly abnormal electrocardio- graphy. The planned V/Q scan was not done as the patient had deteriorated. However he 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 mono- nucleosis cases with SRS is a retrospective analysis by the Mayo clinic. 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. This patient underwent emergent 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 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; 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 exacerb- ated an already ruptured or weakened spleen.

The diagnosis of SRS is usually confirmed by abdominal ultrasound or computed tomog- raphy.

Final diagnosis
Spontaneous rupture of spleen secondary to infectious mononucleosis.

References

Learning points
• Infectious mononucleosis can lead to the rare complication of SRS.
• Beware of pleuritic pain in infectious mononucleosis. Chest pain accom- panyed 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 tomogra- phy to check that there is no underlying SRS.

Although hyponatraemia has been clas- sically associated with hypothyroidism, thyrotoxicosis can also precipitate hypo- natraemia in patients with short bowel syndrome.

A variety of restorative adaptive mecha- nisms 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.

An unusual endocrine cause of hyponatraemia
hyponatraemia occurs in patients with short bowel syndromes.

**Final diagnosis**

Thyrotoxicosis precipitating hyponatraemia in short bowel syndrome.

**References**


An unusual endocrine cause of hyponatraemia

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