CASE REPORTS

Progressive subcutaneous emphysema and respiratory arrest

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J R Soc Med 2002;**95**:90–91

Subcutaneous emphysema is often seen after thoracic surgical procedures. In most cases it is due to a leak from the lung parenchyma and is self-limiting, requiring no specific treatment. Massive subcutaneous emphysema, however, should be treated both to reduce discomfort and to prevent respiratory embarrassment.

CASE HISTORY

A man aged 71 with severe chronic obstructive pulmonary disease who had suffered recurrent right-sided pneumothoraces was admitted to a district general hospital for elective pleurectomy and apical bullectomy. The procedure was performed thoracoscopically and was uneventful; an area of bullous emphysema was identified in the right upper lobe and was stapled and excised. Postoperatively, the patient was noted to have a continuous air-leak and was maintained on 5 kPa of thoracic suction via apical and basal intercostal drains. Chest radiography showed a 10% pneumothorax. Subcutaneous emphysema of the thorax and neck was noted, but it was causing no symptoms. However, over the next two days, the subcutaneous emphysema worsened, involving the face, arms and abdomen. Despite an increase in the suction to 10 kPa, the patient was uncomfortable and had an obvious rise in the pitch of his voice. The pneumothorax was unaltered. On the third postoperative night he became acutely distressed and had first a respiratory arrest then a cardiac arrest. Initial attempts at intubation failed because of vocal cord and soft-tissue swelling (seen at laryngoscopy), but cricothyroidotomy allowed ventilation to be established. After resuscitation he was successfully intubated by the orotracheal route. At this point he had massive subcutaneous emphysema extending from his face to his lower extremities and scrotum (Figure 1). The pneumothorax remained small. Two further intercostal drains were inserted and connected to suction. A substantial air-leak continued for three days and the subcutaneous emphysema persisted. The patient was transferred to the regional cardiothoracic unit. At right thoracotomy, an air-leak was identified from the staple-line on the lung, which was considerably diseased. A right upper lobectomy was performed. Postoperatively there was no further air-leak and the lung expanded well. The subcutaneous emphysema resolved over several days and the patient recovered well.

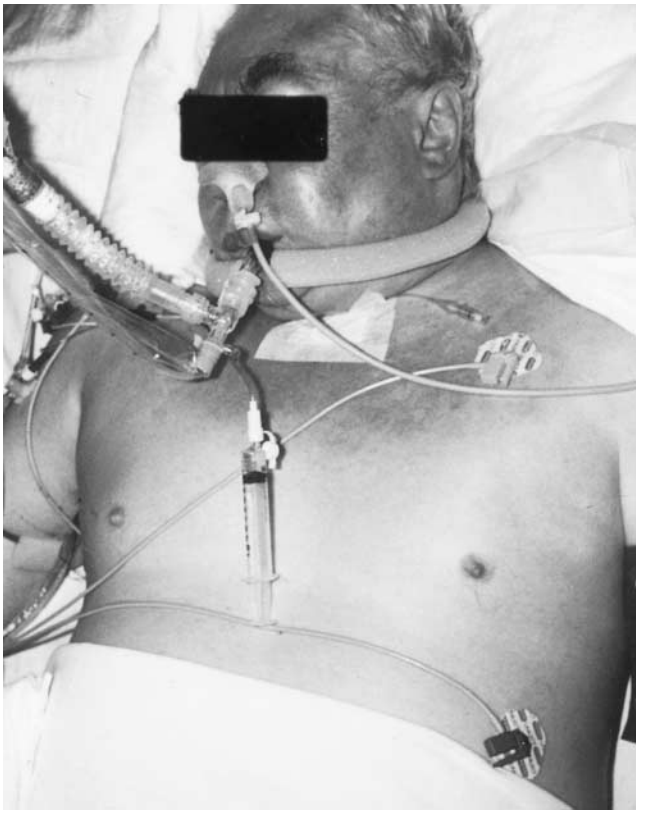

Figure 1 **Massive subcutaneous emphysema involving the face and trunk.** The lower limbs were also affected

COMMENT

The simplest explanation for the respiratory compromise would be restriction of thoracic expansion by a large volume of air in the subcutaneous tissues[1]. An alternative explanation would be direct compression of the airway itself. Air in the subcutaneous tissues, originating from the lung, may get there by two routes. First, if the parietal pleura is torn, air which has entered the pleural space may pass directly into the chest wall and subcutaneous tissues. Alternatively, alveolar air may track proximally within the bronchovascular sheath towards the hilum of the lungs where it may pass superficial to the endothoracic fascia, producing subcutaneous emphysema[2]. It may also pass into the mediastinum and then into the cervical visceral space which invests the trachea and oesophagus[3]. Whether air in this fascial compartment can actually compress the airway is debatable. The exact nature of the airway compression in

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this case is largely speculation based on laryngoscopic findings at the time of respiratory arrest—bulging vocal cords occluding the airway. Air in the cervical visceral space may have entered the submucosa of the trachea. The laryngeal sinus is the site of loosest attachment to the surrounding skeletal tissues and may therefore bulge into the airway. Certainly, a rise in the pitch of the voice is frequently seen in patients with subcutaneous emphysema originating in the lungs, pointing to laryngeal disturbance. The presence of subcutaneous emphysema in the cervical region is usually taken as evidence of decompression of the mediastinal and cervical visceral spaces. However, in this case the pressure was clearly sufficient to cause dissection throughout the body, right to the toes. The presence of a one-way valve in the pathway of tracking air could allow a substantial volume to accumulate with each breath, leading to progressive build-up of pressure beyond the valve. Air in the mediastinum, accumulating by a similar mechanism, has been associated with a picture of cardiac tamponade[4], and several case reports describe respiratory distress with pneumomediastinum although the mechanism is difficult to ascertain.

Whatever the exact mechanism of airway compression in this case, the patient would clearly have benefited from earlier decompression of the subcutaneous tissues. Various approaches have been described, including the use of subcutaneous incisions, needles or drains[5–7]. Cervical mediastinotomy is an option when these interventions do not relieve increasing respiratory distress[8].

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Hypocalcaemia during fusidic acid therapy

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J R Soc Med 2002;**95**:91–93

Fusidic acid is an antistaphylococcal agent widely used in the treatment of osteomyelitis. Hepatotoxicity is a well recognized side effect but renal failure is rare. Severe hypocalcaemia and acute renal failure developed in two diabetic patients treated with oral fusidic acid.

CASE HISTORIES

Case 1

A man aged 53, obese, with type 2 diabetes, peripheral vascular disease, retinopathy and hypertension, developed osteomyelitis of the left distal tibia. Routine biochemical tests were normal. He was treated with intravenous flucloxacillin 1 g four times daily and fusidic acid 500 mg three times daily. After three weeks urea was 4.7 mmol/L (reference range 2.5–8.0), creatinine 80 mmol/L (40–135), bilirubin 37 μmol/L (<25) and alkaline phosphatase (ALP) 132 U/L (30–250). Corrected serum calcium was 2.35 mmol/L (2.15–2.6). Intravenous flucloxacillin was stopped and fusidic acid was continued orally. Two months after presentation he became jaundiced and was readmitted to hospital. Drug therapy on admission included metformin, orlistat, lisinopril, aspirin, ciprofloxacin and fusidic acid. Urea was then 21.4 mmol/L, creatinine 346 mmol/L, bilirubin 125 μmol/L, ALP 183 U/L, alanine amino-transferase (AST) 227 U/L (normal <40), gamma-glutamyltranspeptidase (GGT) 52 U/L (<55). Corrected serum calcium was 1.68 mmol/L and phosphate 0.98 mmol/L (0.7–1.1). Parathyroid hormone was appropriately raised at 79 ng/L (10–50) and 25 hydroxy-cholecalciferol was 7.0 μg/L (8–50). Hepatitis A and B serology and blood and urine cultures were negative. Metformin, orlistat and ciprofloxacin were discontinued and 4 L intravenous fluids was given over the two days after admission. Biochemical findings further deteriorated over these two days—urea 26.6 mmol/L, creatinine 404 mmol/L, corrected calcium 1.52 mmol/L, bilirubin 169 μmol/L. There were no clinical or electrocardiographic (ECG)

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features of hypocalcaemia and the patient was not acidotic. Fusidic acid was withdrawn while lisinopril and aspirin were continued. There was an immediate improvement in renal and hepatic function. Two days after withdrawal, urea was 18.1 mmol/L, creatinine 226 mmol/L, bilirubin 30 μmol/L and corrected calcium 1.67 mmol/L. The patient was discharged four days after admission without calcium supplements or vitamin D therapy. Over the next month biochemical results gradually returned to normal—urea 5.7 mmol/L, creatinine 131 mmol/L, corrected calcium 2.38 mmol/L, bilirubin 22 μmol/L. The patient did not require surgery for his osteomyelitis and one year later was well.

Case 2

A woman of 63 with type 1 diabetes, peripheral vascular disease, ischaemic heart disease and retinopathy developed osteomyelitis of the left fourth toe and was given oral fusidic acid 500 mg three times daily. Other medication included aspirin, simvastatin, thyroxine, captopril and frusemide. Before treatment plasma glucose was 5.8 mmol/L, urea 7.2 mmol/L, creatinine 98 mmol/L, bilirubin 18 μmol/L, ALP 162 U/L, AST 30 U/L, GGT 81 U/L and corrected serum calcium 2.51 mmol/L. Thyroid function tests were normal. One month later she attended the casualty department because of chest pain and transient loss of consciousness. She was apyrexial and normotensive. ECG showed evidence of a previous inferior myocardial infarct but no QT prolongation. Plasma sodium was 143 mmol/L (133–148), potassium 3.4 mmol/L (3.4–5.2), urea 29.6 mmol/L, creatinine 146 mmol/L, bilirubin 92 μmol/L, albumin 34 g/L, AST 72 IU/L, ALP 432 IU/L, phosphate 1.3 mmol/L and corrected serum calcium 1.49 mmol/L. The patient refused admission, treatment and further investigation. Forty-eight hours later she was found dead at home. Post mortem examination was not performed.

COMMENT

Fusidic acid is an effective treatment of staphylococcal infections and is often started parenterally, then changed to the oral route. After oral administration the drug rapidly reaches inflamed bone and synovial fluid[1] and is generally well tolerated apart from mild gastrointestinal symptoms[2]. It interferes with bile salt transport and secretion[2], and hepatotoxicity (characterized by conjugated hyperbilirubinaemia and raised alkaline phosphatase) is common. Liver function tests become normal when the drug is stopped (as in case 2), but may also return to normal despite continued therapy. Jaundice is more frequently observed with intravenous administration than with oral administration[2]. Rare reported adverse drug reactions include thrombophlebitis, granulocytopenia, thrombocytopenia and rash[2].

In the above case histories we describe the development of hepatic dysfunction, acute renal failure and severe hypocalcaemia during treatment with fusidic acid. The first patient's biochemical results improved as soon as he stopped taking the drug and returned to normal within one month. There are two previous reports of renal failure in association with fusidic acid[3,4], but serum calcium levels are not mentioned. In one case[5] severe hypocalcaemia developed after intravenous administration of a high dose of fusidic acid (4 g daily) in a phosphate-citrate buffer that gave a total of 44 mmol (1.67 g) phosphate daily. The authors concluded that this dose of phosphate was sufficient to cause hypocalcaemia[5]. There are no previous reports of hypocalcaemia following oral administration.

Hypocalcaemia in acute renal failure may be the result of a renal calcium leak. However, impaired 25-hydroxylation of vitamin D secondary to hepatic parenchymal damage is the likely mechanism of hypocalcaemia in case 1. Parathyroid hormone levels were raised, serum phosphate was normal and 25-hydroxycholecalciferol was low, suggesting a hepatic cause for the hypocalcaemia. Ciprofloxacin may cause renal failure but hypocalcaemia and renal impairment persisted after ciprofloxacin withdrawal and improved only after discontinuation of fusidic acid. In case 2, derangement of liver function and hypocalcaemia were noted within one month of starting fusidic acid therapy. Further information was not available.

Renal failure is often associated with liver failure[3]. Both our patients had moderately raised liver enzyme and bilirubin levels. Acute tubular necrosis secondary to severe hyperbilirubinaemia (up to 739 μmol/L) after fusidic acid therapy is reported in a patient who required dialysis[3]. At the time of writing the Committee on Safety of Medicines/Medicines Control Agency has received 22 reports of renal failure and 9 reports of hypocalcaemia suspected of being associated with fusidic acid. 5 of the cases of renal failure were fatal (CSM, Personal communication).

Portier[6], in a clinical trial, continued fusidic acid when patients developed abnormal liver function tests, reducing the dose in those with hyperbilirubinaemia[6]. We suggest, however, that urea, creatinine, electrolytes, serum calcium and liver function tests should be measured before fusidic acid is given and that the drug should be avoided in those with abnormal liver function tests and impaired renal function. In patients who receive long-term fusidic acid therapy, serial monitoring of liver function, renal function and serum calcium should be considered. If abnormalities in liver function or renal function occur, the drug should be stopped.

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Suicide in a patient with symptomatic carotid occlusion

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J R Soc Med 2002;**95**:93–94

Depression and an increased risk of suicide are well recognized after stroke[1], and are more frequent than would be expected on the basis of disability alone[2]. Patients with symptomatic severe carotid stenosis or occlusion are also at high risk of depression, even in the absence of cerebral infarction[3,4]. However, the need to assess such risks in these patients is not mentioned in published guidelines[5–7].

CASE HISTORY

A man of 53 was referred to a neurology clinic with episodes of transient loss of vision in the left eye and episodes of clumsiness and weakness in his right arm. These usually lasted only a few seconds and tended to come on with exertion, suggesting low cerebral perfusion. There had been no previous cerebrovascular episodes, but he was a long-term heavy smoker, had had a myocardial infarction 5 years earlier and was on aspirin and was taking a lipid-lowering agent for hypercholesterolaemia. He lived with a partner and had no history of depression, self-harm or other psychiatric illness. However, later reports from family members suggested that he had become uncharacteristically depressed since the onset of his symptoms. Visual acuity in the left eye was 6/18, with evidence of ischaemic retinopathy. No other neurological or cardiovascular abnormality was found. Colour-flow doppler ultrasound of the internal carotid arteries showed complete occlusion on the left and 50% stenosis on the right. CT brain scan was normal.

Dipyridamole was added to the treatment and the patient was advised to stop smoking. In view of the carotid occlusion, he was not a candidate for carotid endarterectomy. The plan was to review his progress one month later and to consider anticoagulation or extracranial-intracranial bypass surgery if his symptoms persisted. Unfortunately the attacks continued and, although they did not change or increase in severity, he committed suicide by overdose before his next appointment.

Although it is impossible to be certain that this patient's medical condition led to his suicide, there seemed to be no other precipitant. According to relatives, his low mood started at around the same time as his symptoms, about three months before his death, and before any changes were made in his treatment.

COMMENT

In patients with stroke the risk of depression and suicide is high[1,8,9]. The reported frequency of depression after stroke ranges from 20% to 65%[9], and frequencies of suicide are up to 14 times greater than expected[1].

There is also evidence of an increased risk of depression in patients with symptomatic carotid artery stenosis who have had a transient ischaemic attack (TIA) rather than a stroke[3,4,10]. One study compared the frequency of symptoms of depression in elderly patients with stroke (n=25), symptomatic carotid stenosis with TIA (n=25) or non-vascular disease (n=25). Both the symptomatic carotid stenosis group and the stroke group had a significantly higher frequency of symptoms of depression than the non-vascular disease group, and there was a tendency for depression to occur more frequently in those patients with greater than 80% carotid stenosis[3]. Studies of cognitive impairment in TIA patients have also tended to show an association with severe carotid stenosis[4,8–10].

The cause of psychological disturbance associated with carotid occlusive disease without stroke is not known. It is likely to be partly related to anxiety associated with symptoms and the worry about the risk of stroke, but it may also be directly related to chronic cerebral hypoperfusion. A case has been reported of a woman aged 72 presenting with severe depression who was found to have greater than 95% stenosis of her left internal carotid artery. Her depression had not responded to medical therapy but it resolved permanently and completely within four days of a left carotid endarterectomy[11].

Although evidence of a link between carotid disease and depression is mainly anecdotal, our case report illustrates the need to be aware of the possibility of depression and the risk of suicide in patients with symptomatic severe carotid

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stenosis or occlusion. Guidelines on the management of patients with carotid occlusive disease do not currently mention the assessment of depression and suicide risk[9–11]. We suggest that these patients should be asked specifically about symptoms of depression so that appropriate therapy can be given. Further research is required to investigate the relationship between carotid stenosis and depression, and to identify the best method of treatment for these patients.

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An umbilical nodule

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J R Soc Med 2002;**95**:94–95

One cause of umbilical lesions is a Meckel's diverticulum, which is subject to the ills that affect small bowel.

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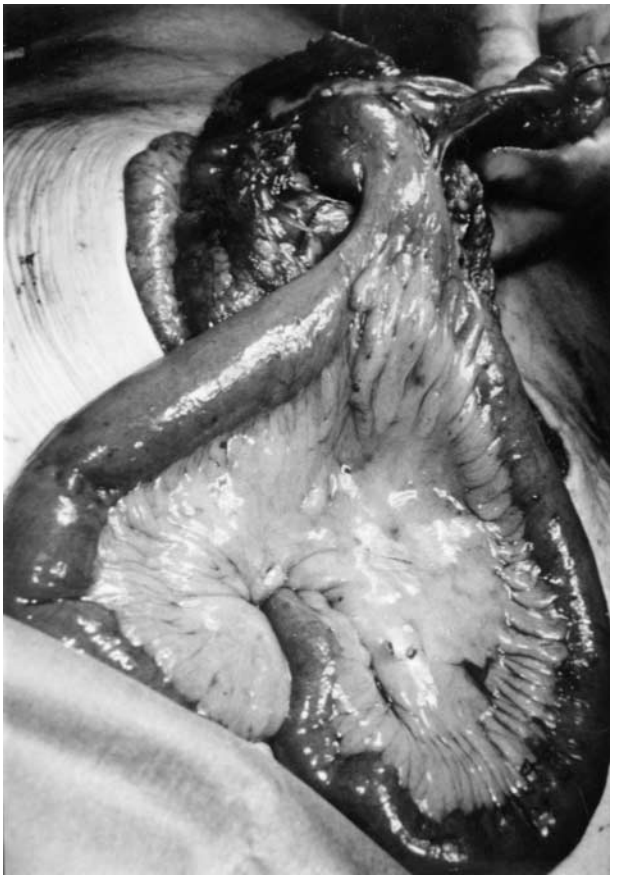

Figure 1 **Operative photograph of the mass involving tip of Meckel's diverticulum**

CASE HISTORY

A woman of 70 was referred to the gastroenterology department with abdominal distension and umbilical discomfort. For many years she had been experiencing episodes of abdominal pain and distension which had passed off after a day or two. On examination she was found to have a firm 4 cm mass at the umbilicus and was referred urgently for a surgical opinion. Immediately after the clinic, however, she left for a foreign holiday. While abroad she developed an umbilical discharge and was seen by a local surgeon, who diagnosed omphalitis with a mass and raised the possibility of a Sister Joseph's nodule. An ultrasound scan revealed 'an oval, well defined hypoechogenic lesion in the abdominal wall, possibly haematoma'. She returned to the UK and was admitted. Further ultrasound scanning showed a 6 cm soft-tissue mass abutting the small bowel and extending to the umbilicus. CT scanning confirmed this and a tumour of a urachal remnant was thought the most likely cause. Ultrasound-guided core biopsy showed poorly differentiated adenocarcinoma. No metastatic disease was seen on a chest radiograph or a CT scan of the liver.

At laparotomy the tumour was found to originate in a Meckel's diverticulum (Figure 1). The diverticulum was

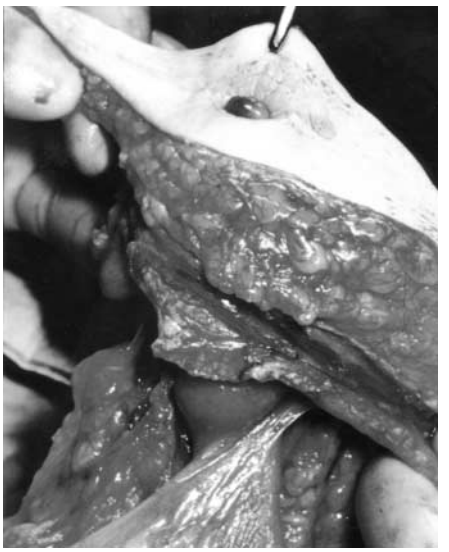

Figure 2 **Operative photograph showing umbilical nodule at apex of tumour mass**

attached to the umbilicus, and a tumour arising from within was invading the anterior abdominal wall and was attached to the transverse mesocolon (Figure 2). The mass was resected *en-bloc* with the small bowel, the transverse mesocolon and a portion of abdominal wall. The abdominal wall was repaired with mesh. She recovered without incident. The lesion was a poorly differentiated adenocarcinoma arising in a Meckel's diverticulum adherent to the umbilicus. There was a track lined by granulation tissue to the umbilicus. The resection margins were clear, but two of eleven lymph nodes contained metastatic tumour (T4, N1, M0). After discussion at the gastrointestinal multidisciplinary team meeting the patient was seen by an oncologist and despite the probable limited benefit of chemotherapy she elected to proceed with treatment with 5-fluorouracil and folinic acid. Five months after her operation she returned with a palpable groin lymph node, which on biopsy showed metastatic adenocarcinoma with the same characteristics as the Meckel's carcinoma.

COMMENT

Meckel's diverticulum is a small portion of the vitelline duct that persists into adulthood as an outpocketing of the terminal ileum. The duct is formed in the first weeks of fetal life, representing the yolk stalk. It communicates with the yolk sac, passing through the umbilicus. Failure of the duct to be broken down and absorbed leads to congenital anomalies. Commonest is the Meckel's diverticulum which lies free within the abdomen, occurring in around 2% of the population. If part of the duct persists it can remain attached to the umbilicus as in this case, either patent or as a cord. In the case presented here the Meckel's diverticulum was connected to the umbilicus at its tip. It is possible that the episodes of pain over previous years were due to twisting of small bowel around this attachment, although small-bowel obstruction secondary to a Meckel's diverticulum with a fibrous cord is rare.

There are numerous recorded tumours of Meckel's diverticula. Tumours may be benign (such as villous adenoma[1] and carcinoids[2]) but are more commonly malignant, led by adenocarcinoma[3] and malignant carcinoid[4]. Rarer tumour types include fibrosarcoma[5], leiomyosarcoma[4] and melanoma[6]. Sister Joseph's nodule is so called because it was first described (to Dr William Mayo) by Sister Mary Joseph as an indicator of metastasis from an intra-peritoneal malignancy.

Adenocarcinomas of Meckel's diverticula are biologically similar to small-bowel adenocarcinomas, which tend to respond poorly to adjuvant therapy. Because they do not usually cause small bowel obstruction until locally advanced, they seem to have an even worse prognosis[7].

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