Myalgia with lymphadenopathy

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J R Soc Med 2001;**94**:521–522

Myalgia has a broad differential diagnosis (Box 1). Sometimes the patient's country of origin is relevant.

Box 1 **Causes of myopathy/myalgia**

- Mechanical
  - Muscle strain/overuse
- Localized muscle infection (pyomyositis)
  - *Staphylococcus aureus*
  - *M. tuberculosis*
- Generalized infection
  - Influenza
  - Rheumatic fever
  - HTLV-1/HIV
- Idiopathic
  - Polymyositis
  - Dermatomyositis
  - Polymyalgia rheumatica
  - Fibromyalgia
- Drugs
  - Statins and fibrates
  - Chloroquine
- Metabolic
  - Hypokalaemia
  - Hypocalcaemia
- Dystrophinopathies

CASE HISTORY

An Angolan man aged 23, resident in the UK for 7 years, developed a 'flu-like illness with dry cough, coryza, headache, fever and myalgia. Symptoms resolved but returned a few weeks later, this time with night sweats, weight loss, severe myalgia in the forearms, thighs and calves and difficulty walking due to pain and weakness. He was unable to climb stairs or walk more than 10 metres. He also complained of morning stiffness in his knees and ankles. His general practitioner prescribed oral cefalexin, amoxicillin and indometacin with some improvement.

On referral he was afebrile. Painless lymph nodes were palpable in the right axilla. Muscle bulk was reduced in his arms and legs, and the biceps, forearm extensors, quadriceps and calves were tender to palpation. Power was normal in the upper limbs and MRC grade 3–4/5 in the lower limbs, though difficult to assess because of pain. Reflexes and sensation were normal.

Initial investigations showed a normocytic anaemia with haemoglobin 9.0 g/dL (normal range 13.0–17.0), total white cell count 4.8×10^9/L (3.0–10.0), lymphocytes 0.6×10^9/L (1.5–4.0), platelets 303×10^9/L (150–400), erythrocyte sedimentation rate 93 mm/h (0–20) and C-reactive protein 138 mg/L (0–5); albumin was 30 g/L (35–50), alkaline phosphatase 154 U/L (45–122), alanine aminotransferase 109 U/L (7–63), creatine kinase 243 U/L (24–195). Further normal or negative investigations were: plasma urea, electrolytes and calcium; immunological investigations (antinuclear antibody, rheumatoid factor, extractable nuclear antigen); an infection screen (urine culture, blood cultures, urine/blood mycobacterial culture); viral serology (Epstein–Barr, cytomegalovirus, HTLV-1, HIV); X-ray and chest abdominal/pelvic ultrasound; nerve conduction studies and electromyography.

A CT scan showed a mass in the right axilla, and at operation several large axillary lymph nodes were found. The biggest was excised and acid-fast bacilli were seen on smear staining. Caseating necrosis and epithelioid granulomata were seen on histological examination. Tuberculous lymphadenitis was diagnosed and the patient was started on rifampicin, isoniazid and pyrazinamide. When fully sensitive *Mycobacterium tuberculosis* was grown on culture, the pyrazinamide was stopped. Treatment was continued for 6 months and the patient made a speedy recovery, with total resolution of his debilitating myalgia and weight loss. The creatine kinase returned to normal.

COMMENT

Muscle involvement in tuberculosis, first reported in 1886, is rare. Only twelve other cases have been reported; and all but two presented with a focal muscle mass, abscess formation or pulmonary disease. To our knowledge there are only two case reports of proximal weakness as the predominant complaint in the absence of a muscle mass[1,2]. All previous reports describe pulmonary or direct muscle infection with *M. tuberculosis*. Our case is unusual in that there was no evidence of pulmonary or disseminated infection yet the symptoms were severe.

M. tuberculosis can cause a myopathy in several ways. There may be direct extension of infection into neighbouring muscle, as in psoas abscess formation, or haematogenous spread. An ill-defined polymyositis may also be seen, of unknown aetiology[2]. A muscle biopsy might have been of interest in our patient but was not done because his symptoms improved rapidly with treatment.

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Giant vaginal stone with embedded contraceptive device

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Vaginal stones and lost intrauterine contraceptive devices (IUCDs) are commonly reported separately but seldom together.

CASE HISTORY

A woman of 63 had a radiograph taken to evaluate hip pain and was discovered to have an ovoid calcified mass measuring 9 × 5 cm in her pelvis (Figure 1). She was suspected of having a giant bladder stone and was referred by her general practitioner to a urologist.

The patient, who had three children, had been well until the age of 41 when she had become decerebrate after a 'flu-like illness. Brain biopsy showed probable acute allergic leukoencephalitis and for the next three months she was in a vegetative state. Much to the surprise of her carers, she gradually improved and was discharged home after 10 months. Her indwelling catheter had been removed and apart from occasional urinary accidents she was managed satisfactorily with 'toileting' and precautionary incontinence pads. Until the present admission she lived at home or in residential care. Characteristically when approached she would subject her family and the medical staff to a colourful stream of invective interspersed with occasional elements of normal speech. She declined examination of any sort. However, if the verbal tirade was ignored, she was entirely cooperative. Occasionally she had needed treatment for urinary tract infections, and these had become chronic in the months before admission.

A pelvic ultrasound scan indicated that the mass might not be in the bladder but in the pouch of Douglas. At cystoscopy and examination under anaesthesia by the urologist the calculus was found to be in the vagina, but it could not be removed digitally. The patient was referred to the gynaecology department. At the time of a second anaesthetic, despite use of Wrigley's forceps[1] and much lubrication, it was still not possible to dislodge the mass intact. The rough surface would not slide over the vaginal mucous membrane without causing soft-tissue damage. It was removed by partial morcellation after episiotomy. In the centre of the laminated structure was a Lippes loop IUCD (Figure 2). Postoperatively the patient received intravenous antibiotics for a *Klebsiella* urinary tract infection. Six weeks later she had healed and was continent.

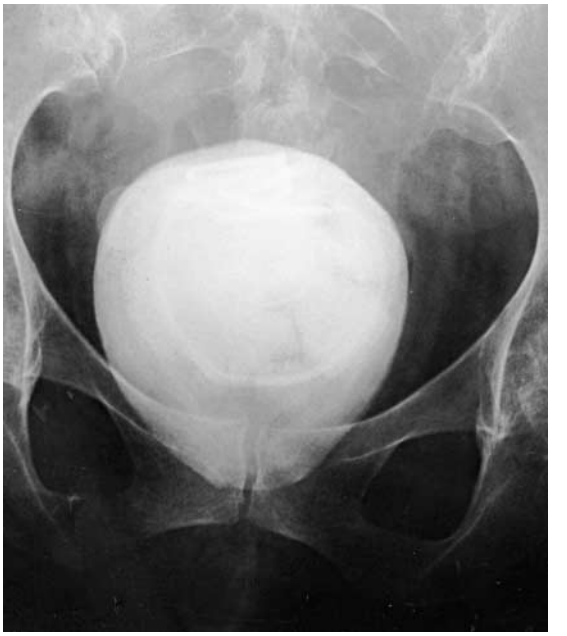

Figure 1 **Radiograph of pelvis showing calcified mass**

Figure 2 **Vaginal stone with embedded device**

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Probably the Lippes loop had been inserted about 23 years previously after a miscarriage. Since her encephalitis no one had been prepared to do a pelvic examination or had attempted a cervical smear, because of her abusiveness. Analysis of the stone showed it to consist of calcium carbapatite (calcium carbonate and phosphate).

COMMENT

Primary vaginal stones occur where there is stasis of urine and infection, associated with a congenital abnormality such as an ectopic ureter or meningomyelocele[2]. Secondary stones can arise after surgical damage and may be associated with surgical sutures or other forgotten material placed in the vagina[3].

Intrauterine contraceptive devices, even when normally located, tend to form calcareous deposits on their surfaces. These deposits differ between devices. Lippes loops characteristically have 'mud-cracked incrustations' and 'rounded masses', which form in the surface microenvironment. The masses are rosettes of euhedral crystals with porous tips that under high magnification look like city tower-blocks and are similar in composition to the apatite of dental calculus. Different patterns are observed with copper-bearing devices[4].

The combination of a displaced IUCD some time previously, personal neglect and lack of regular pelvic examination permitted the development of the stone in this case. From reports of large calculi in children it is clear that the speed of encrustation can be rapid. Therefore, if the IUCD had been expelled soon after its insertion we would expect the stone to have become apparent sooner. Records made in the recovery period from her encephalitis 22 years earlier indicate that, although she expressed interactive distress very forcefully, she did not show any sign of pain. This lack of awareness of pain probably contributed to the late discovery and large size of the mass.

There have been reports of traumatic erosion of the bladder wall by a bladder calculus[5] and of the vaginal wall by a vaginal calculus, both leading to fistula formation[6]. Cystoscopy and careful vaginal examination under anaesthesia did not reveal such damage in our patient, and her return to near-normal bladder function was impressive. Fortunately there had been only superficial infection and there was no sign of pelvic actinomycosis.

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Primary pulmonary hypertension in pregnancy

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The diagnosis of pulmonary hypertension in pregnancy requires early investigation and difficult judgments.

CASE HISTORY

A woman aged 31, in the 24th week of her third pregnancy, reported haemoptysis for the preceding day. Before this pregnancy she had smoked 20 cigarettes a day; she had noticed mild dyspnoea on exertion over the preceding 12 months. 8 years earlier she had used three different slimming pills over a period of 3 months. Her two previous pregnancies, 14 and 11 years previously, had been uncomplicated. There was no personal or family history of thromboembolic disease. On physical examination there was central cyanosis, pulse 97/min, blood pressure 131/68 mmHg, normal jugular venous pulse. The precordial right ventricular impulse was exaggerated with a prominent pulmonary second heart sound. The lung fields were clear. There was no ascites or peripheral oedema, and signs of deep venous thrombosis were absent. Haematological and biochemical indices were normal apart from a mildly raised white cell count consistent with pregnancy (14×10^9/L, neutrophils 73%, lymphocytes 19%, eosinophils 1%). The C-reactive protein was normal. The 12-lead electrocardiogram showed right axis deviation, right ventricular hypertrophy and strain with an incomplete right bundle branch block. On chest radiography there was bilateral proximal enlargement of the pulmonary vasculature and peripheral vascular pruning (Figure 1).

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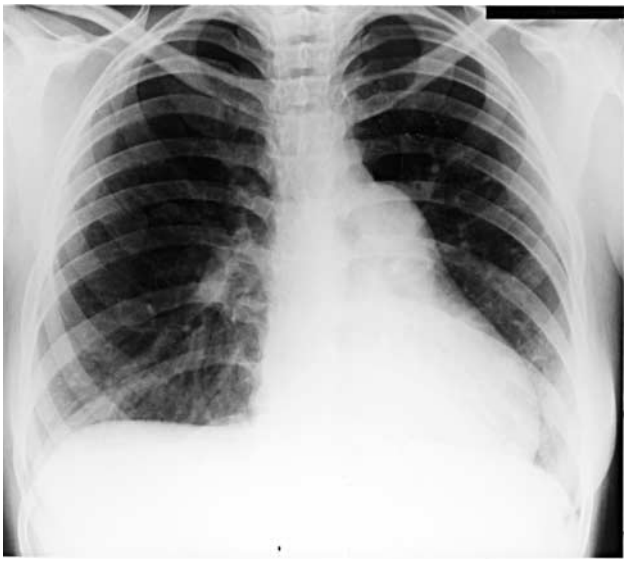

Figure 1 **Radiograph showing bilateral proximal enlargement of pulmonary vasculature and peripheral pruning**

Pulmonary hypertension was diagnosed. Arterial blood gases, with the patient breathing room air, were PO_2 8.4 kPa and PCO_2 3.2 kPa; pH was 7.4, bicarbonate 17.7 mmol/L and base excess −3.5. Pulmonary embolism became an unlikely explanation when a radionuclide ventilation perfusion lung scan (VQ) was reported normal together with a fibrin degradation product D-dimer of 0.1 mg/L (normal < 0.3). Transthoracic echocardiography showed a dilated and hypertrophied right atrium and right ventricle (Figure 2), without septal defect or thrombus. The peak velocity of tricuspid regurgitation was 4.3 m/s with a calculated peak pulmonary artery (PA) pressure of 74 mmHg (normal 15–25). After careful consideration of the possible causes of pulmonary hypertension (Box 1), primary pulmonary hypertension was felt the most likely diagnosis. This was supported by findings at right-heart catheterization, when the absence of a step-up O_2 saturation excluded a significant left to right shunt; moreover, the PA pressure was 94/44 mmHg (normal systolic 15–25, diastolic 8–15) and pulmonary capillary wedge pressure was 8 mmHg (normal 4–12). The response of the pulmonary vasculature to O_2 and nifedipine was tested and there was no significant fall in the PA pressure.

When primary pulmonary hypertension was diagnosed, a multidisciplinary team comprising a cardiologist, obstetrician, intensive-care anaesthetist, paediatrician and midwife was assembled to manage the patient. Since she had reached the 26th week of gestation and was stable with no suggestion of right heart failure, pregnancy was cautiously continued with close monitoring of the maternal and fetal condition. At the 28th week she was transferred to the intensive-care unit. A pulmonary artery flotation catheter was inserted and PA pressure was 97/43 mmHg (systemic blood pressure 140/65 mmHg). Nebulized Iloprost 15 μg lowered the PA pressure to 72/33 mmHg, an effect lasting 3 hours; systemic blood pressure was unaffected. The patient was then transferred to theatre where a lumbar epidural catheter was inserted. Nebulized Iloprost 15 μg was administered and the PA fell from 100/40 to 83/33 mmHg. Epidural anaesthesia was established and a girl weighing 1.1 kg (Apgar scores 9 at 1 min and 9 at 5 min) was delivered by lower-segment caesarean section. After delivery the mother remained in the intensive-care unit for 5 days; nebulized Iloprost (20 μg 4-hourly) was continued for 16 days and gradually tailed off. The mother was discharged 20 days later accompanied by her daughter in good condition. 18 months after delivery, she was receiving warfarin and supplemental oxygen, and was being followed up by a specialized pulmonary hypertension clinic. She was dyspnoeic on mild exertion but had been able to return to work with the aid of a wheelchair.

COMMENT

Primary pulmonary hypertension is a rare disease that particularly affects women of childbearing age[1]. It is characterized histologically by the presence of medial hypertrophy, intimal fibrosis and often fibrinoid necrosis, arteritis and plexiform lesions in the pulmonary vasculature[2]. This disease can be defined clinically by a persistently raised

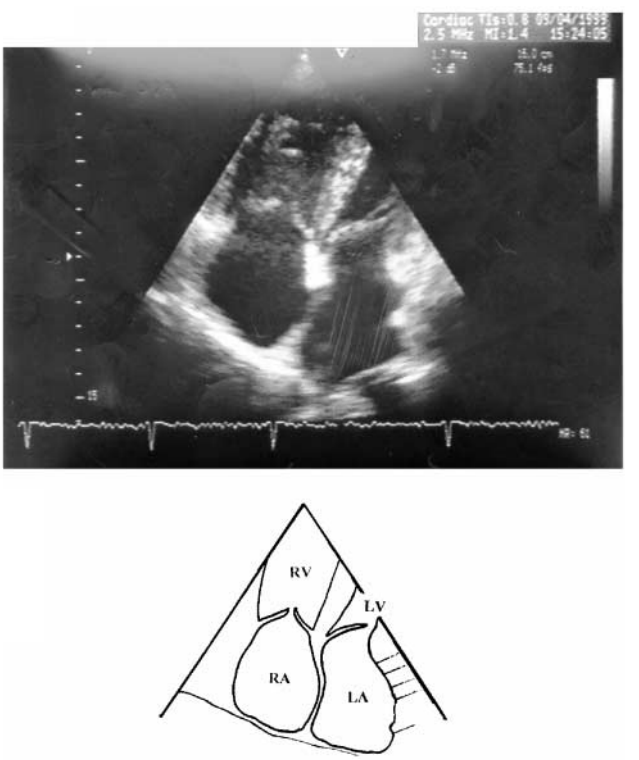

Figure 2 **Echocardiography findings of dilated and hypertrophied right atrium and ventricle** RV=right ventricle; RA=right atrium; LV=left ventricle; LA=left atrium

PA pressure (mean pressure >25 mmHg at rest or >30 mmHg during exercise) without an obvious aetiology[3]. Most of the secondary causes of pulmonary hypertension were effectively excluded in our patient, with the exception of her previous exposure to appetite suppressant drugs.

In a recent overview[4] the maternal mortality of primary pulmonary hypertension in pregnancy was said to be 30%, and it was as high as 56% in an earlier study[5]. Most of the deaths were in the third trimester, with the highest risk in the first 10 days postpartum. In view of the high maternal mortality, preconceptional counselling is of vital importance if feasible. In cases of unplanned pregnancy or diagnosis early in pregnancy, termination should be considered[3]. If pregnancy is to be continued, further management will require a multidisciplinary team.

Pulmonary embolism is an important differential diagnosis of pulmonary hypertension and a major cause of maternal mortality. It can be diagnosed from a positive VQ scan showing high probability (low sensitivity) and a raised D-dimer (low specificity); an unequivocally normal VQ scan and D-dimer virtually exclude the diagnosis. Alternatively, computed tomographic pulmonary angiography can be used to diagnose pulmonary embolism (sensitivity 0.8, specificity 0.9), but the radiation dose is tenfold higher. Apart from excluding the presence of a cardiac shunt, right heart catheterization can measure cardiac pressures and indicate potential benefits from various drugs (see below), and pulmonary angiography will demonstrate emboli if present. In our patient, pulmonary angiography was not performed because of severe pulmonary hypertension. It is necessary to discuss with the mother the importance of performing these investigations and the potential radiation hazards to her and the fetus. Once in possession of information on the pulmonary and systemic circulation and the state of the fetus, the multidisciplinary team can determine the optimum time for delivery.

Vasoconstriction from a reduction of nitric oxide and prostacyclin, together with an increase in endothelin and thromboxane in the vascular endothelium and smooth muscle, is important in the pathogenesis of primary pulmonary hypertension. Various vasodilator treatments have been tried in the past, and agents showing benefits include O_2, oral calcium channel blockers, continuous intravenous prostacyclin, inhaled nitric oxide, and nebulized prostacyclin or its stable analogue Iloprost. Responsiveness to these treatments can be evaluated at the time of cardiac catheterization. In our patient we chose nebulized Iloprost because of its proven efficacy, relative ease of administration and direct delivery to the lungs with few systemic effects[6].

Clinicians who encounter pregnant women with primary pulmonary hypertension would benefit from access to national and international databases. These patients should usually be followed up by specialized centres, so that their treatment and possible need for transplantation can be monitored closely.

Box 1 **Causes of pulmonary hypertension**

- Cardiac diseases
  - Congenital: left to right shunts (e.g. atrial septal defect, ventricular septal defect, persistent ductus arteriosus)
  - Acquired: left ventricular failure, mitral valve disease, left atrial thrombus or tumour
- Respiratory diseases
  - Chronic obstructive pulmonary diseases (e.g. chronic bronchitis, emphysema, asthma, bronchiectasis)
  - Chronic parenchymal lung diseases (e.g. pulmonary fibrosis, pneumoconiosis, extrinsic allergic alveolitis)
  - Cystic fibrosis
  - Obstructive sleep apnoea
  - Thoracic cage abnormalities
- Pulmonary thromboembolism
- Pulmonary vasculitides (e.g. lupus erythematosus, scleroderma, rheumatoid disease)
- Hyperviscosity syndromes (e.g. multiple myeloma)
- Infections (e.g. human immunodeficiency virus, schistosomiasis)
- Portal hypertension
- Cirrhosis
- Pulmonary veno-occlusive disease
- Primary pulmonary hypertension, including drug-related (e.g. appetite suppressants, cocaine)

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Vomiting with depression: choroid plexus papilloma

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Anorexia and vomiting in a depressed patient are not always due to depression.

CASE HISTORY

A man aged 65, depressed after the death of his wife four months earlier, had experienced vomiting and anorexia for two months. His medical history included middle-ear surgery for Menière's disease. On investigation elsewhere a month before, upper gastrointestinal endoscopy had revealed only gastritis. Anti-*Helicobacter* treatment had been given; but, when the vomiting continued, his symptoms were ascribed to bereavement and depression.

On admission direct questioning revealed that he was experiencing occasional headaches and diplopia. The ocular fundi were normal. He had nystagmus which was exaggerated on right horizontal gaze. Ocular movements seemed intact in all directions and no cranial nerve abnormalities were detected. His gait was not ataxic but past-pointing was evident on the right side. In view of the history and examination findings, a posterior fossa space-occupying lesion was suspected. A CT scan revealed a 5–6 cm area of predominantly low attenuation in the right cerebellar hemisphere with a 2–3 cm mass in the lateral aspect of this area (Figure 1). There was compression of the fourth ventricle, with hydrocephalus. The patient was transferred to the regional neurosurgical unit. Findings on cerebral angiography suggested a glomus jugulare tumour. Vertebral artery embolization of the tumour was followed by retromastoid craniectomy and excision of a tumour that proved to be a benign choroid plexus papilloma. The patient recovered without neurological deficit, his vomiting resolved and he remains well.

COMMENT

Choroid plexus papillomas account for less than 1% of all intracranial tumours in adults, though relatively more common in childhood. They are most often located in the lateral ventricle, followed by the fourth and third ventricles

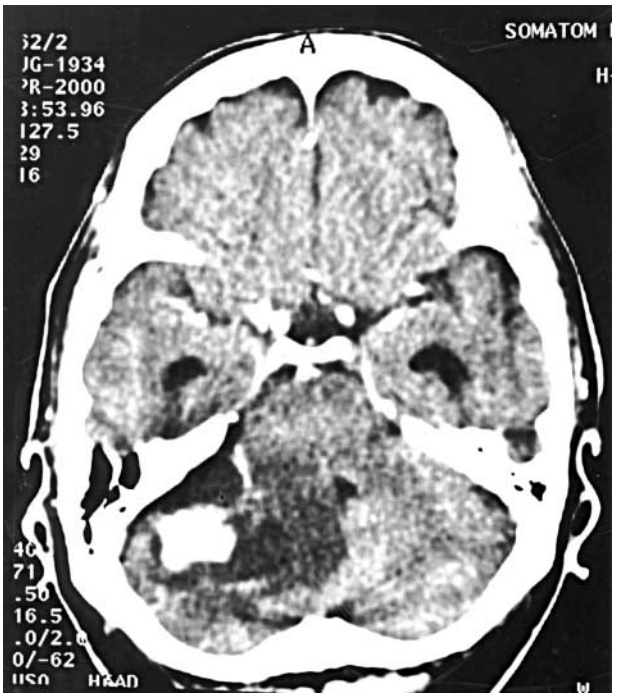

Figure 1 **CT scan showing lesion in posterior fossa**

and, rarely (as in our case), the cerebellopontine angle[1]. They are generally benign in nature with a male preponderance[2]. Derived from the neuroepithelial cells of the choroid plexus these tumours recapitulate the normal choroid plexus, often demonstrating a well preserved papillary architecture[3]. Magnetic resonance imaging is the diagnostic tool of choice[2], and total surgical excision is the optimum treatment. Radiotherapy can be used if the resection is incomplete[4].

We think this case is of interest for three reasons. First, it describes a rare tumour in an atypical place at an unusual time of life. Secondly, it illustrates the important principle that the cause of vomiting may lie beyond the gastrointestinal tract. This presentation of posterior fossa mass lesions has been reported previously[5], and caution is needed before such physical symptoms are ascribed to psychological disturbance.

The final point concerns the previous diagnosis of Menière's disease. Although we have not been able to obtain information on the neurological findings at that time or the details of the middle-ear surgery, we suspect that these earlier symptoms were due to the lesion in the posterior fossa.

Acknowledgments We thank PT van Hille and his team at the regional neurosurgical centre in Leeds for their expert help with this patient.

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Splenic rupture after vomiting

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Splenic rupture is usually secondary to trauma. Most non-traumatic ruptures are consequent on underlying disease affecting the spleen such as infection (Epstein–Barr virus, hepatitis, salmonella, malaria), neoplasia (lymphoma, leukaemia) and connective-tissue disease.

CASE HISTORY

A previously healthy man of 30 attended the accident and emergency department sixteen hours after eating a seafood meal. From four hours after the meal he had been vomiting two to four times an hour, and one hour before coming to hospital he had developed generalized abdominal pain which radiated into his left shoulder and chest. The pain was worse on movement and on lying flat. On admission he was pale, cold and clammy. His heart rate was 110 and blood pressure was 85/55 mm Hg. There was tenderness and guarding in both upper quadrants; bowel sounds were present. Haemoglobin was 10.2 g/dL, white cell count 16.2×10^9/L. Serum amylase was normal.

Two hours after arrival he fainted on sitting upright. Repeat haemoglobin was 6.3 g/dL. An abdominal ultrasound scan showed fluid in the peritoneum and around the spleen and at laparotomy he was found to have a ruptured spleen with a large amount of free intraperitoneal blood. No other intra-abdominal abnormalities were detected. A splenectomy was performed and the patient recovered uneventfully. Cultures from blood, sputum, stool and urine were negative, and paired acute-phase and convalescent sera provided no evidence of acute viral infection. Screening for autoantibodies, including antinuclear cytoplasmic antibodies and rheumatoid factors, was negative. The spleen was normal on microscopic and macroscopic examination. On repeated questioning before surgery and after recovery, the patient and his family could recall no history of significant trauma, either recent or in the more distant past.

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COMMENT

True spontaneous splenic rupture was first described by Atkins in 1874 and has been a controversial diagnosis ever since. Reviewing all previously published cases, Orloff and Peskin[1] found that in only 28 out of 71 was there no alternative explanation. They identified four criteria for the diagnosis of spontaneous splenic rupture—absence of a history of trauma or of unusual effort that could injure the spleen; absence of perisplenic adhesions suggestive of previous trauma; absence of pre-existing splenic disease; and normal microscopic and macroscopic appearances of the spleen. Other workers[2] added a fifth criterion—that studies of acute-phase and convalescent sera should show no significant rise in viral antibody titres suggestive of recent infection with types associated with splenic involvement.

In view of the history of vomiting before onset of abdominal pain, we believe that the splenic rupture in our patient was not spontaneous. Rupture of a normal spleen has been reported to occur after trivial effort such as coughing[3]. To our knowledge only three cases of splenic rupture after vomiting have been published[4–6], the vomiting being attributed to ingestion of ibuprofen, constipation and a reaction to a metrizamide injection. We suspect the vomiting in our case was related to the seafood meal. A proposed mechanism for splenic rupture secondary to vomiting is that violent contraction of the diaphragm causes tractional force on the spleen via the peritoneal reflections linking the two[4].

When there is no history of trauma, splenic rupture tends not to be diagnosed until laparotomy. In Orloff and Peskin's review, only one of the 28 with genuinely spontaneous rupture had been correctly diagnosed pre-operatively. In a case such as ours diagnosis might well be delayed by false attribution of abdominal pain to the prolonged vomiting and dehydration.

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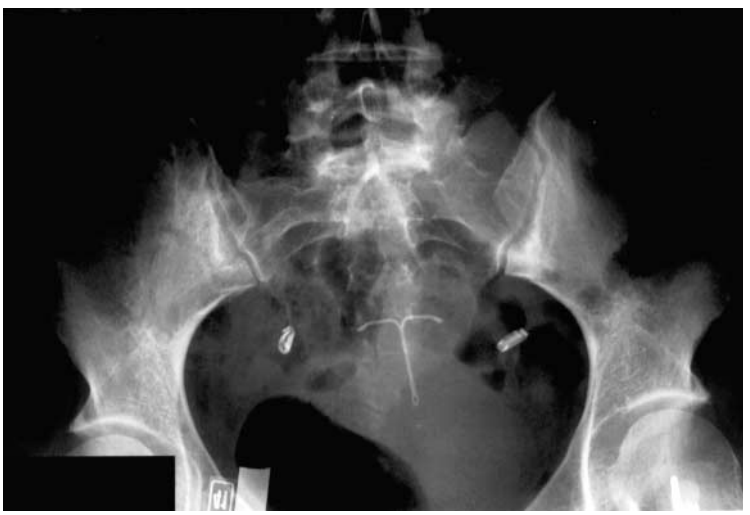

Belt and braces?

The radiograph, from a woman aged 35 with abdominal pain, shows both an intrauterine contraceptive device and sterilization clips in the pelvis. This does not reflect a clinical oversight or an unusually rigorous approach to contraception. The device is Mirena, used also for treatment of menorrhagia. By releasing the progestogen levonorgestrel directly into the uterine cavity, it reduces endometrial proliferation.

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