Meningitis four years after treatment of macroprolactinoma

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After treatment of macroprolactinoma, patients are advised to look out for cerebrospinal fluid (CSF) rhinorrhoea. But meningitis can develop without this warning sign.

CASE HISTORY

A man aged 33 was admitted with pneumococcal meningitis. Four years previously the occurrence of three generalized convulsions had led to a diagnosis of macroprolactinoma. An MRI of his brain at that time showed hydrocephalus and a large heterogeneously enhancing pituitary mass with suprasellar expansion and extension into the sphenoid sinus (Figure 1). His serum prolactin was 100 000 U/L. Further pituitary testing indicated that he had secondary hypothyroidism and hypogonadism, for which thyroxine and testosterone were prescribed.

For the prolactinoma he was treated for one month with bromocriptine 7.5 mg daily and subsequently with cabergoline 0.5 mg twice weekly. After three months on this regimen, serum prolactin was 1800 U/L (normal <450), so the dose of cabergoline was increased to 0.5 mg three times weekly. The dose of cabergoline was finally increased to 1 mg twice weekly twelve months after diagnosis, and serum prolactin then became normal. A repeat MRI scan at thirty months showed a large reduction in tumour size. It was noted that the sphenoid sinus was destroyed and occupied by CSF. He never required neurosurgical intervention.

During the two weeks before his current admission he developed flu-like symptoms and occasional mild headache. On the day of admission, he woke with a severe frontal headache, which was not relieved by simple analgesia and was associated with nausea, dizziness and mild neck stiffness. Glasgow coma score was 15 and there were no signs of meningism. His temperature was 37.1°C. No abnormalities in his cranial or peripheral nerve examination were found. Initial blood testing revealed a neutrophilia—white cells 15.3^9/L, neutrophils 13.7^9/L. After treatment with intravenous morphine in the accident and emergency department the symptoms completely resolved. He was admitted for observation with a working diagnosis of postviral headache, but in view of his pituitary tumour the possibilities of a pituitary haemorrhage or infarction were considered. Later that evening he had a generalized convulsion, after which he was drowsy and sweaty. He became hypotensive and tachycardic. A CT scan showed a 3.5×7 cm mass in the pituitary fossa, with calcification of the superior aspect and no haemorrhage. Cerebrospinal fluid protein was 5.3 g/L, white cells 147/μL, glucose <0.5 mmol/L; *Streptococcus pneumoniae* was subsequently cultured from cerebrospinal fluid and blood. It was a penicillin-insensitive strain with penicillin minimum inhibitory concentration 0.75 mg/L and cefotaxime 0.19 mg/L. He was treated with 3 g cefotaxime 4-hourly and rifampicin 600 mg once daily for 14 days and made a full recovery. Review of his CT scans showed extensive bony erosion of the sphenoid sinus and it was felt that the source of his infection was a sinus connecting his pituitary fossa with the sphenoid sinus (Figure 2). There had never been any rhinorrhoea.

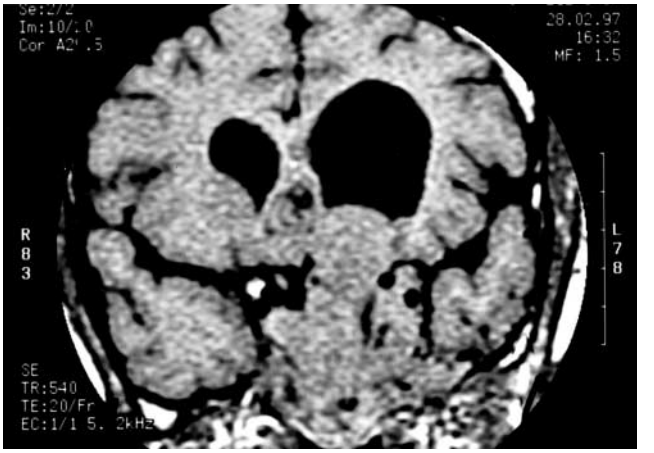

Figure 1 **MRI coronal view of brain showing hydrocephalus and a large heterogeneously enhancing pituitary mass with suprasellar expansion and extension into sphenoid sinus**

COMMENT

The pneumococcal meningitis in this patient was probably caused by a defect in the pituitary sella floor. The case highlights several issues that are important in the management of patients with macroprolactinomas. First, the complication of meningitis occurred several years after the start of treatment of his prolactinoma. This, together

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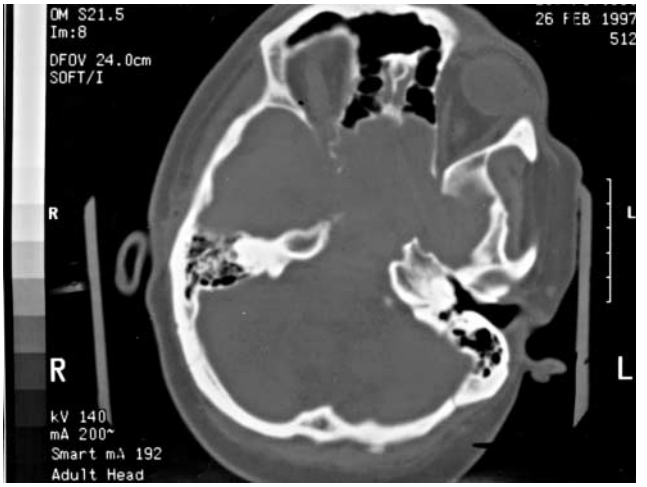

Figure 2 **CT transverse view showing destruction of the sphenoid sinus**

with the insidious nature of the presentation followed by a rapid decline, meant that the cause of his illness was not immediately apparent. Secondly, although CSF rhinorrhoea is a recognized complication after neurosurgery or dopamine agonist therapy, the absence of a history of rhinorrhoea did not exclude a defect in the skull base. Finally, it is unclear what measures should be taken to prevent meningitis in patients with macroprolactinomas.

The dopamine agonist cabergoline is an effective and well-tolerated treatment for macroprolactinoma. It is now regarded as first-line therapy but may also be useful in bromocriptine-resistant tumours. CSF rhinorrhoea is a rare but potentially lethal complication following primary dopamine agonist treatment with bromocriptine[1] or cabergoline[2]. Dopamine agonist therapy causes a rapid shrinkage of the tumour and may produce a defect in the sella floor, particularly when dopamine agonists are used to treat invasive prolactinomas with skull-base destruction. These defects predispose to bacterial meningitis. In a review of 15 patients with bromocriptine-induced CSF leaks[3], 8 patients had developed leaks within one month of the start of treatment and the remainder occurred by seventeen months. 4 individuals developed meningitis. The most likely organisms to cause meningitis in this setting are upper respiratory tract pathogens—typically *S. pneumoniae*, *Neisseria meningitidis* and *Haemophilus influenzae*. Consequently, patients with large macroprolactinomas under treatment with dopamine agonists should be made aware of the symptom of CSF rhinorrhoea and the risk of meningitis.

The absence of a known CSF leak does not exclude the diagnosis of a skull-base defect. This has been noted before[4]. Although our patient had never reported rhinorrhoea, a skull-base defect was presumed to be the portal of bacterial entry. The interval of 4 years between the start of treatment and the development of meningitis is the longest yet reported. What preventive measures can be adopted? Where a CSF leak is proved and surgical repair is not feasible, vaccination against *S. pneumoniae*, *H. influenzae* and meningococci has been recommended. We suggest that this strategy might be extended to all patients treated for invasive macroprolactinoma. As regards treatment for meningitis in patients with a known CSF leak, the recommendation is to start with a third-generation cephalosporin and vancomycin pending the results of culture, because of the increasing prevalence of penicillin-resistant pneumococci, as seen in this case.

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Hyperpnoea and emesis in a diabetic man

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In diabetic ketoacidosis (DKA), breathlessness reflects the underlying respiratory alkalosis in response to metabolic acidosis. However, it can also be a manifestation of a rare respiratory complication.

CASE HISTORY

A man aged 44, diagnosed with type 1 diabetes at the age of 20, was admitted as an emergency. Nausea and malaise had

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begun four days earlier; on the day before admission this had progressed to severe retching and vomiting and he missed his insulin injections (Human Mixtard 30/70, 40 and 20 units daily). There was a history of recurrent prolonged hypoglycaemic episodes leading to mild to moderate cognitive impairment. Clinically he was very dehydrated with Kussmaul respiration. He was afebrile, with blood pressure 148/89 mmHg and heart rate 140 per minute. Subcutaneous emphysema was palpable in the supraclavicular fossae. Biochemical investigations confirmed DKA with plasma glucose 43.5 mmol/L, arterial blood pH 7.23, bicarbonate 9 mmol/L, $PaCO_2$ 3.3 kPa and PaO_2 16.8 kPa. Urine dipstix showed heavy ketonuria (3+). He also had compensatory hyponatraemia (Na^+ 125 mmol/L) and mild renal impairment (K^+ 4.6 mmol/L, urea 18.5 mmol/L, creatinine 141 μmol/L). A chest radiograph confirmed interstitial emphysema and pneumomediastinum. There was no pneumothorax. Treatment was with intravenous fluid, insulin and a course of antibiotics (cefuroxime and metronidazole) to cover possible oesophageal rupture. A water-soluble contrast swallow study later excluded oesophageal perforation. He gradually improved over the next 48 hours, and the interstitial emphysema resolved over four days. A chest radiograph 2 weeks after admission showed complete resolution of the pneumomediastinum.

COMMENT

The incidence of pneumomediastinum complicating DKA is likely to be underestimated because the resultant breathlessness tends to be overshadowed by hyperventilation due to DKA. Symptoms such as retrosternal pain are uncommon. Subcutaneous emphysema is present in about half the cases. Occasionally, a crackling or crunching sound synchronous with the heart beat (Hamman's sign) is audible over the left sternal edge[1]. The major differential diagnosis is oesophageal rupture (Boerhaave's syndrome), which should be excluded by contrast study or endoscopy.

In DKA, several factors may contribute to the development of pneumomediastinum[2]. Severe vomiting in DKA produces a Valsalva-like effect with large momentary swings in intrathoracic pressure which can lead to alveolar rupture[3]. This is much more likely to occur when alveoli are already susceptible due to over-distension during Kussmaul respiration. Rupture of the alveolar wall introduces air into the perivascular adventitia, leading to interstitial emphysema. Interestingly, however, pneumomediastinum in association with severe DKA has been described in the absence of cough and vomiting[4]. This prompts the speculation that in some instances the alveolar wall is weakened by the metabolic derangement of ketoacidosis. In the published work there are indications that rupture may be due to constitutional weakness. Males outnumber females and the typical patient is young[4,5]. Most are under 20, and before the present case the oldest reported was 29[5].

The prognosis of pneumomediastinum complicating DKA is excellent without special interventions other than management of DKA.

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Bilateral neck swelling in an elderly man

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Swellings of the neck can present a difficult diagnostic challenge, especially when bilateral.

CASE HISTORY

A domiciliary visit was requested on an 81-year-old male smoker because of weight loss, shortness of breath and general weakness. He had lately been in hospital elsewhere with pneumonia. The most striking feature on physical examination was the massive swelling on either side of his neck (Figure 1). For 25 years he had had swelling of the right cheek and for 9 years swelling of the left cheek. These caused little discomfort; both had become progressively larger over the past year. The swellings were cystic, with a little tenderness to pressure on the right. A core biopsy was attempted on both lesions, but only purulent fluid was

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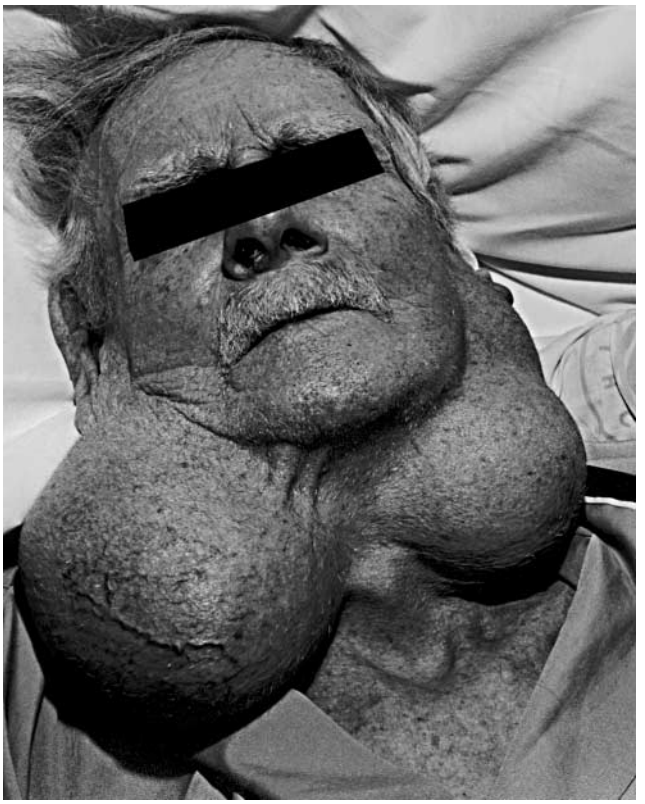

Figure 1 **Clinical photograph**

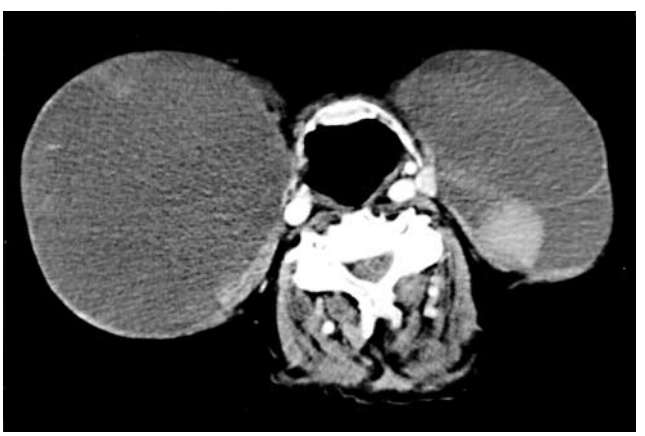

Figure 2 **Computerized tomogram showing bilateral neck cysts, the left containing slight septation and a small nodule**

obtained. Cultures were negative. Cytology showed no malignant cells; otherwise the samples were poorly cellular.

Ultrasound of the neck showed that the swellings were predominantly cystic, and uniformly low-level echoes suggested proteinaceous content. On a CT scan, well-defined cystic masses on each side of the neck were seen to arise from behind the angle of the mandible to the level of the hyoid (Figure 2). There was no cervical adenopathy. The neck swellings were thought to be longstanding branchial cysts. Unfortunately, radiological imaging showed metastatic disease within the liver from a primary bronchogenic carcinoma. Also, ultrasound suggested a lesion in one kidney. The patient's condition deteriorated rapidly and he died peacefully in hospital. At necropsy two primary tumours were found—a poorly differentiated non-small-cell bronchogenic carcinoma with hepatic metastases, and a small renal cell carcinoma. The neck cysts were confirmed as branchial, measuring 13×9×5 cm on the left and 17×10×8 cm on the right. On histological examination the walls of the cysts contained abundant lymphoid tissue with an attenuated lining of squamous epithelium; there were many cholesterol clefts.

COMMENT

There are reports of branchial cysts up to the age of 60 years[1], but we have found no record of bilateral branchial cysts in older patients. These cysts emerge in the anterior triangle of the neck under the anterior border of sternocleidomastoid where the upper third meets the middle third. They present most commonly in males, usually on the left side, after the age of 10 years and peaking in the third decade of life. The cyst fluid contains cholesterol crystals.

There are several theories about the origin of branchial cysts. They may represent remnants of pharyngeal pouches or branchial clefts or they could be due to the non-disappearance of the cervical sinus (where the second branchial arch grows down over the third and fourth). Most are lined by squamous epithelium and have lymphoid tissue in the wall—hence the notion that cyst epithelium arises from lymph node squamous epithelium[2].

A thorough history and examination will provide clues to the diagnosis but radiological imaging, fine-needle aspiration and core biopsy may be required[3]. The differential diagnosis of swelling in the neck lies between a parotid tumour, lymphadenopathy, thyroid disease, cystic hygroma, branchial cyst and a carotid body tumour. Ultrasound of a branchial cyst shows a uniformly low echogenicity[4] that distinguishes these lesions from other tumours and allows prompt surgical treatment. If a parotid mass is suspected a CT scan is advisable, to define the extent of the mass and whether there is involvement of the deep lobe or the parapharyngeal space[5]. MRI provides the best information because of greater tissue contrast resolution and multiplanar images[1]. Fine-needle aspiration and examination of the aspirate may be used to differentiate conditions that clinically mimic each other—for example, cystic nodal metastasis[6]. If an aspirate sample is insufficient or non-diagnostic, a core biopsy will be required. Treatment is by excision.

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Acute abdomen in Henoch–Schönlein purpura

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Patients with Henoch–Schönlein purpura are sometimes referred for a general surgical opinion because of abdominal pain or gastrointestinal bleeding. Surgical intervention is seldom needed.

CASE HISTORY

A man of 41 was admitted by the medical team with a 4-day history of a purpuric rash covering his legs and buttocks, oedema of the ankles and wrists, generalized arthralgia, abdominal pain, rectal bleeding and vomiting. He was dehydrated and tachycardic; his abdomen was distended but non-tender; bowel sounds were absent; inflamed rectal mucosa was seen by rigid sigmoidoscopy. No free intra-abdominal gas was evident on plain abdominal and chest radiography. These findings were consistent with the diagnosis of ileus secondary to a generalized vasculitis. After two days of conservative management the patient became oliguric and developed proteinuria with deteriorating renal function. Parenteral steroid therapy was instituted and renal and skin biopsies were taken which subsequently confirmed the diagnosis of Henoch–Schönlein purpura (HSP). 4 days after admission his abdominal pain worsened and he developed tenderness with guarding in the right iliac fossa. Laparotomy disclosed hyperaemic small bowel, 2 litres of ascitic fluid and 45 cm of infarcted ileum. The infarcted bowel was resected, a double-barrelled ileostomy was formed and the patient was nursed in the intensive care unit for 48 hours. The ileostomy produced up to 7 litres of fluid per day, requiring careful fluid management; over 6 months the patient had to be readmitted intermittently for rehydration. Steroids were stopped after 9 months and the stoma was closed 3 months later; he subsequently made a good recovery.

COMMENT

HSP is rare in adults; less than one-third of patients with HSP admitted to hospital are over 20. Typically, an acute phase is followed by intermittent relapses in the following months. The aetiology is unclear and bacteria, viruses, vaccines, drugs, foods and exposure to cold have all come under suspicion. The clinical manifestations are due to deposition of IgA immune complexes on the intima of small blood vessels, leading to complement activation, leukocyte recruitment and destruction of endothelial cells. Skin biopsy reveals a leukocytoclastic vasculitis with necrotic vessel walls containing neutrophils surrounded by fibrin strands.

Abdominal symptoms in HSP are caused by haemorrhage and oedema within the bowel wall and mesentery. They occur in up to 56% of adults and are the presenting feature in 15%[1]. Pain is typically colicky and poorly localized, sometimes accompanied by vomiting and bloody diarrhoea. In adults, surgical intervention is seldom necessary[2], whereas in children HSP is complicated by intussusception (2%), perforation (0.5%) and infarction (0.5%). Only one adult case of intestinal infarction secondary to HSP has been reported to our knowledge; that patient died[3].

Early involvement of the surgical team, enabling them to make frequent clinical assessments of the patient and identify deterioration, is probably the best way to avoid abdominal catastrophes in HSP.

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Discrepant thyroid function tests in a patient treated with interferon-alpha

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Interferon-alpha treatment is associated with autoimmune thyroid dysfunction[1,2]. Antibody to thyroxine (T4) can confuse the interpretation of thyroid function tests[3].

CASE HISTORY

A woman of 24 was referred with suspected hypothyroidism. For twenty months she had been receiving interferon-alpha for chronic myelogenous leukaemia. A check on thyroid function four months into treatment had shown thyroid-stimulating hormone (TSH) 7.12 mIU/L (normal range 0.3–4.0) and free T4 15.4 pmol/L (10.2–19.6). At sixteen months she noted facial puffiness and weight gain, at which time TSH was >150 mIU/L. Her symptoms partly subsided on treatment with L-thyroxine 50 μg daily. At the time of referral, TSH was >150 mIU/L but she also had raised free T4 at 20.4 pmol/L. Antimicrosomal antibody titre was 6400; antithyroglobin was negative. In view of the improvement of symptoms and the inconsistent thyroid function test results, L-thyroxine was temporarily stopped and the tests were repeated. TSH remained >150 mIU/L with a free T4 of 20.8 pmol/L. The simultaneous free T3 concentration was 2.3 pmol/L (normal range 3.5–6.5 pmol/L) and the erythrocyte zinc concentration was 334 μmol/L (155–237). Thus all biochemical results were consistent with primary hypothyroidism except for the high free T4. A two-step free T4 assay was arranged and she was given L-thyroxine 100 μg daily. On this dose the TSH fell to 52.5 mIU/L while the free T3 rose to 3.2 mIU/L. The free T4 result obtained with the two-step assay was later reported as 7.67 pmol/L, suggesting that T4 antibody had interfered with the one-step analogue assay. L-thyroxine was then gradually titrated down to 50 μg daily, on which dose TSH was 2.7 mIU/L while the free T4 result with the one-step assay was 19.5 pmol/L. Her thyroid function tests remained stable thereafter.

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COMMENT

Interferon-alpha has been widely used in chronic myelogenous leukaemia[2], and various endocrine diseases have been reported in association with this treatment[4]. In a series of 581 patients with chronic myelogenous leukaemia treated with interferon-alpha, 2% became hypothyroid[2]. Development of autoantibodies alone is much more common, reaching 20% in some series[5]. Thyroid dysfunction may be more prevalent in those who are antibody-positive before treatment. On withdrawal of interferon, the antibodies sometimes disappear and sometimes persist[1].

Anti-T4 antibodies are well-known to account for a spuriously high free T4 result in the one-step analogue assay. The underlying mechanism is binding of the T4 analogue by antibody[3]. Antibodies are present in less than 1% of normal individuals[6]. Our patient had a normal free T4 three months after starting interferon, so she is unlikely to have had pre-existing T4 antibody. A causal relation is further indicated by the disappearance of T4 antibody when interferon treatment was stopped.

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An erosive pessary

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Pessaries inserted for uterine prolapse are easily forgotten in the old, and can be hazardous.

CASE HISTORY

A woman of 88 was admitted after a fall at home. Her medical history included chronic atrial fibrillation, cerebrovascular disease, osteoarthritis, and utero-vaginal prolapse. She reported a three-month history of constipation interrupted by bouts of diarrhoea, weight loss, lethargy and a decline in mobility. On examination she was frail, malnourished and dehydrated. There were hard faeces in the rectum. Respiratory and abdominal examinations were normal. Plasma urea was 13.37 mmol/L, albumin 27 g/L. Abdominal X-ray revealed faecal loading throughout the colon.

The patient was rehydrated and her constipation was treated with faecal softeners and enemas. It was then noticed that she was passing faeces per vaginam. On enquiry she said this had happened on several previous occasions. A hard rubbery object was now felt in the rectum, and a soluble-contrast enema established the presence of a rectovaginal fistula. On colonoscopy a vaginal pessary was identified eroding through the upper rectum; impacted faeces were evacuated and multiple biopsies were taken (no evidence of malignancy). A pelvic examination was performed simultaneously and a shelf pessary was removed. The cervix was normal, the uterus was small and mobile, and no adnexal masses were palpated. Closure of the fistula was attempted per rectum but this subsequently failed. Pneumonia and upper gastrointestinal bleeding complicated her postoperative recovery and she died two months later.

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COMMENT

Vaginal pessaries are devices of varying composition (rubber, clear plastic, silicone, or soft plastic with internal mouldable steel reinforcement) that serve to reposition and support prolapsed genitourinary organs[1]. Various shapes and sizes are available to provide comfortable anatomical support. In the UK, ring and shelf pessaries are commonly used.

Although surgery is the definitive treatment for severe uterine prolapse, pessaries can give satisfactory results in women who wish or need to avoid surgery[2]. Complications are usually due to inadequate hygiene—e.g. leucorrhoea, cellulitis, abscess formation. Others are incarceration, ulceration and metaplasia[3], intestinal obstruction[4,5], urosepsis and hydronephrosis[6–8]. Russell[9] reported seeing 14 patients with complications over a 4-year period: one woman had a rectovaginal fistula, the pessary having been in place for 18 years; the others had vaginal cancer (7) or chronic vaginitis (6).

In retrospect, questioning our patient about pessary use at the time of admission could have resulted in more prompt diagnosis. However, most internal physicians have little experience with these devices; furthermore, pelvic examination tends to be omitted in elderly patients unless specifically indicated. When an elderly woman is fitted with a pessary, long-term follow-up is desirable—especially if she has dementia[2,3].

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