CASE REPORTS

Sleeping with the enemy: *Pasteurella multocida* infection of a hip replacement

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Arthroplasty infection has been attributed to transmission from spouse to spouse[1]. Transmission from an animal has not previously been recorded.

CASE HISTORY

A man aged sixty-nine was referred with a loose, infected, total hip replacement. 4 years earlier he had received a right cemented total hip replacement for an intracapsular fracture of the femoral neck. After a year, this had become loose, necessitating revision, at which time microbiological cultures were negative. Two years later he fell and sustained a periprosthetic fracture, which was managed with an acetabular allograft and an uncemented Wagner femoral prosthesis. The prosthesis dislocated soon after operation and a sinus developed three months later (Figure 1). After the first part of a two-stage procedure, multiple tissue samples were sent for culture and *Pasteurella multocida* was the only organism identified (in enrichment cultures from femoral canal and acetabular specimens). Infections with this organism are usually caused by animal bites or scratches, but the patient strongly denied any such injuries. He did, however, indicate that his pet dog, a sheltie, had shared his bed both before and after his operations, sleeping under the covers on the side of the affected leg, as it had done every night for the past ten years.

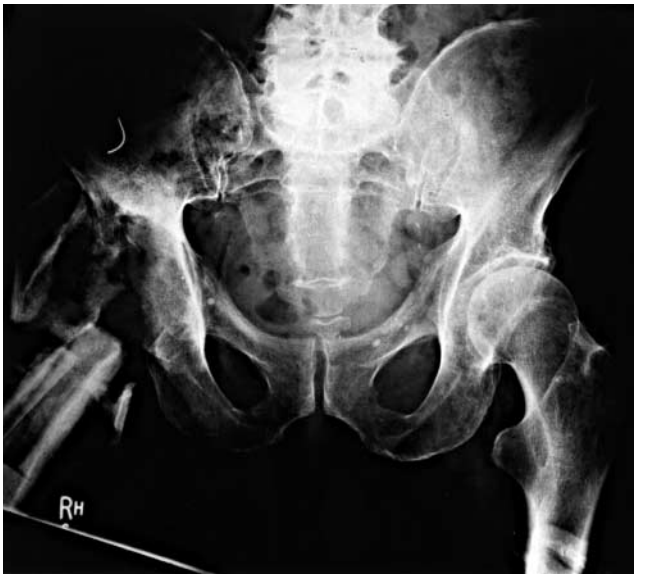

Figure 1 **Anteroposterior radiograph of pelvis after removal of infected total hip replacement.** There is a sinus in the proximal epiphysis and metaphysis of the right femur with cortical destruction.

COMMENT

When a total hip replacement becomes infected, definitive organism identification is important to management but is often difficult. *P. multocida* is a small bipolar Gram-negative coccobacillus carried in the oropharynx of a range of wild and domestic animals. When human beings become infected, cats are the source in 60–80%, dogs in most of the remainder. Local infections are characterized by the rapid onset of warmth and erythema[2,3]; serious local complications include septic arthritis and osteomyelitis, commonly in the hand. There are only two recorded cases of *P. multocida* transmission to man by dog lick[2,4].

How might this patient have acquired his infection? *P. multocida* could have been a skin commensal at the time of one or more of his operations, because of the prolonged contact with his dog at night. Despite preoperative skin preparation it might then have been inoculated directly into his deep tissue. Another possible route was bone allograft, which has been associated with bacterial infection in 1.2% of recipients[5]. The bone tissue for allografts originates mainly from femoral heads obtained at primary hip replacement and cadaver material. *P. multocida* has not been reported as a bacterial contaminant of bone allograft and this route of transmission is highly unlikely.

Haematogenous spread from a small injury, seeding to the total hip replacement postoperatively, is another possibility. Although the patient had no recollection of a bite from his dog, a scratch might have been sufficient inoculation.

The organism might have reached the joint cavity postoperatively by retrograde passage from the wound, having been present as a commensal or acquired through a dog lick when the patient returned to his canine sleeping partner. Finally, it might have been a secondary infection, gaining access to the deep fascia via the open sinus produced by another organism such as *Staphylococcus aureus*. Whatever the mechanism, this case illustrates the importance of involving patients actively in their postoperative wound care.

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Facial pain with intracranial aneurysm

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Facial pain due to compression or irritation of the trigeminal nerve can mimic true trigeminal neuralgia. The possible causes are many.

CASE HISTORY

A woman aged 52 was referred to an ear, nose and throat department with a two-month history of pain over the left cheek and side of the nose. She had no other symptoms of note except for some nasal blockage. Left maxillary sinusitis was diagnosed, a left intranasal antrostomy was performed and the pain disappeared. However, two months later she was complaining bitterly of pain over the left temple and zygomatic arch. Over the next eight months the pain returned to the left cheek and side of the nose. It fluctuated, becoming more severe over a few days and then gradually decreasing, but never disappearing completely. She had no other symptoms and cranial nerve testing showed no abnormalities.

Although the pain was not typical of trigeminal neuralgia she was treated with carbamazepine. Her pain remained intractable. A magnetic resonance scan of the head was performed and, fortuitously, the most posterior coronal slices revealed a vascular abnormality within the left cavernous sinus. Angiography showed this to be a giant (2.5 cm) aneurysm arising from the posterior communicating artery and extending into the left cavernous sinus. At craniotomy, the aneurysm was clipped and aspirated and she immediately became pain-free. 5 years later there has been no recurrence.

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COMMENT

The distinction between atypical facial pain and trigeminal neuralgia is not always clear. True trigeminal neuralgia is characterized by recurrent brief but intense spasms that may include more than one division of the trigeminal nerve. It can occur at any age and in either sex but is most frequently seen in women aged 50–60 years[1]. Often a trigger point can be localized and the patient will vigorously resist attempts to elicit the pain. Whilst our patient's pain was not typical of trigeminal neuralgia (being permanent) its episodic increase in intensity was suggestive.

Trigeminal neuralgia can arise from neurological diseases such as multiple sclerosis and vascular disorders such as cross compression. It can also be mimicked by nasopharyngeal tumours, dental abscesses and temporomandibular joint dysfunction. Facial pain in patients with intracranial aneurysms is sometimes associated with other cranial nerve palsies. An expanding posterior communicating artery aneurysm is a well-documented neurosurgical syndrome, causing facial pain and an oculomotor nerve palsy. Treatment by clipping and aspiration is reported to give relief[2]. An occasional patient is unsuitable for craniotomy and clipping because of a severe intercurrent medical condition and percutaneous trigeminal rhizotomy has then given impressive results[3]; this treatment, however, should be regarded as palliative. Cranial nerve palsy may also be seen in patients with an intracranial internal carotid artery aneurysm, arising from pressure on the nerve as it passes through the cavernous sinus[4,5]. Posterior circulation aneurysms have caused progressive motor weakness by compressing the brainstem[6].

The symptoms of facial pain in our patient stopped immediately after clipping and aspiration of the aneurysm. We cannot say why a posterior communicating artery aneurysm should affect only the maxillary division of the trigeminal nerve whilst sparing the upper and lower divisions. In an earlier reported case of facial neuralgia with aneurysm of the posterior communicating artery the maxillary division was the only division of this nerve involved, although there were other cranial nerve palsies[2]. Why should clipping of the aneurysm put an end to the pain? A possible explanation is that, by stopping the

transmitted pulsation, it prevents the intermittent stretching of the nerve that is thought to be the cause[7].

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Lemierre's syndrome

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The salient features of Lemierre's syndrome are septicaemia preceded by oropharyngeal infection, lateral neck tenderness or swelling due to internal jugular thrombophlebitis and metastatic abscesses, especially to lungs but also to liver and kidneys.

CASE HISTORY

A girl aged 15 was admitted as an emergency after experiencing pleuritic pains for four days. Twelve days earlier she had had a sore throat; otherwise she had been in good health. On examination she was clearly ill, with temperature 38.6°C, pulse 130/min and respiration 34/min. Pleural rubs were audible bilaterally; her abdomen was held rigidly but there was no overt peritonism. Arterial blood showed a partly compensated respiratory alkalosis, with pO_2 9 kPa (patient breathing air). There was a moderate leukocytosis (white cell count 13.4×10/L) with evidence of slight dehydration (urea 10.3 μmol/L, creatinine normal). Liver function tests subsequently showed modest increases in transaminases and bilirubin. On chest radiography there was a left-sided pleural collection with an opacity in the right upper zone; a computed tomographic scan of the abdomen revealed nothing of note.

The working diagnosis was septicaemia originating from the chest, and she was started on intravenous co-amoxiclav and flucloxacillin, with a single dose of intravenous gentamicin (160 mg). Nevertheless her clinical and respiratory state continued to worsen rapidly. On repeat chest radiography at 24 hours the left pleural collection had greatly increased and there was now a collection on the right; the opacity in the right upper zone had cavitated (Figure 1). The left-sided pleural collection was drained and the aspirate contained numerous Gram-negative rods. At 48 hours her condition was so poor as to demand admission to the intensive care unit, where she was ventilated and received inotropic support; a flow-guided balloon catheter was inserted. The diagnosis was finally achieved 56 hours after admission, when *Fusobacterium necrophorum* was isolated from a blood culture. This, with the original feature of sore throat, pointed to Lemierre's syndrome.

After bacterial sensitivities became known, she was switched to intravenous flucloxacillin (1 g four times daily) and metronidazole (500 mg three times daily). Her clinical condition continued to fluctuate. Four days after arrival,

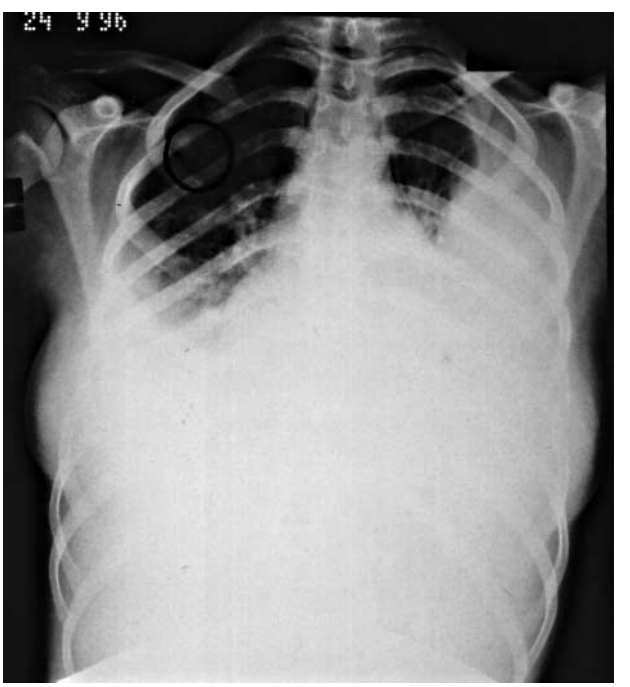

Figure 1 **Radiograph showing bilateral empyema and cavitating lesion in right upper zone (circled)**

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suppurative arthritis developed in the left hip. She was weaned from inotropic agents and extubated by the fifth day but the chest drain continued to discharge. Intrapleural fibrinolysis was attempted with streptokinase but there was no improvement; ultimately she required thoracoscopic drainage of a multiloculated empyema, a large-bore tube being left *in situ* until the exudate ceased. She was deemed fit for discharge eleven weeks after admission. On clinic review six months later, no important sequelae were evident.

COMMENT

André Lemierre described the syndrome caused by *Fusobacterium* infections (necrobacillosis) in 1936[1]. In the years after his description there were many reports of the syndrome, with mortality exceeding 90%. A subsequent decline in reported cases was probably due partly to the advent of antibiotics and their widespread use for sore throat. Only 36 reported cases were identified between 1974 and 1987[2]. Recent years have seen something of a resurgence[3–6], but we cannot say whether this reflects a true increase in disease incidence. In nearly all cases there is pleuropulmonary involvement. Single or multiple nodular infiltrates with pleural effusions precede cavitating abscesses and account for the presentation with high fever, chest pain and dyspnoea. Other organ involvement is variable and can lead to diagnostic confusion[7]. Most commonly affected are large joints, with suppurative arthritis and osteomyelitis. Abdominal pain on presentation is rare[8]. Clinical or subclinical icterus is probably due not to hepatic abscesses but to the cholestatic effect of the lipopolysaccharide endotoxin released by *Fusobacterium*[9]. Suppurative peritonitis, along with renal and splenic involvement, has been described[7,8].

The key to diagnosis of Lemierre's syndrome is correct interpretation of a previous sore throat in a suggestive clinical setting. Once the suspicion arises, serial chest radiography is mandatory to follow progression of the pulmonary lesions. Antibiotic therapy should clearly include anti-anaerobic cover. Most fusobacteria are penicillin-sensitive; clindamycin, cefoxitin and chloramphenicol have also been used. Lengthy treatment, for up to six weeks, is advocated. In addition, localized abscess cavities should be drained, and chest drainage is commonly indicated. There is no documented role for topical antifibrinolytic treatment, which we attempted, or for systemic anticoagulation[10,11].

Fulminant septicaemia following a sore throat in a young person should prompt antimicrobial chemotherapy for *Fusobacterium* while investigations are pursued. Under these circumstances a case fatality rate of less than 8%[12] can be achieved.

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Pericalyceal haemangioma and papillary necrosis

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Renal papillary necrosis is usually bilateral, and the most frequent clinical associations are chronic pyelonephritis, diabetes mellitus and long-term use of analgesics, especially phenacetin.

CASE HISTORY

A woman aged 45 was investigated for left ureteric colic and frank haematuria. Intravenous urography (IVU) showed a filling defect in the left renal pelvis and lower and mid calyces (Figure 1a), and on retrograde pyelography the pelvicalyceal system was distended, with filling defects as seen on IVU. Cytological examination of urine from that side showed no malignant cells. A mid-stream urine specimen grew coliforms. A computed tomographic (CT) scan revealed an area of high attenuation in the lower pole suggestive of haemorrhage within a non-enhancing mass (Figure 1b). The appearances suggested a primary renal tumour. Frank haematuria continued with falling haemoglobin; therefore radical nephrectomy was performed.

On slicing of the resected kidney, a 2.5 cm diameter rounded haemorrhagic lesion was identified in the medulla and inner cortex of the lower pole, with a 0.4 cm rim of cortex between the lesion and the renal capsule (Figure 2). The lesion appeared to communicate with the nearby calyx. The pelvis and the upper ureter seemed filled with clots. No tumour was identified in the pelvis or ureter. On microscopic examination this lesion showed papillary necrosis without features of analgesic nephropathy or diabetic nephropathy. All other papillae appeared normal. On further sectioning, areas with cystic endothelial lined spaces were identified, consistent with a pericalyceal venous haemangioma.

COMMENT

Chabrel *et al.*[1] reviewed 8 cases of recurrent haematuria associated with papillary necrosis secondary to pericalyceal haemangiomas. 5 underwent total nephrectomy and 3 partial nephrectomy. In 2 of those having a partial nephrectomy, preoperative renal angiography had raised the possibility of a vascular malformation; in the remaining 6, diagnosis emerged from examination of the resected specimen. Follow-up of 5 patients yielded no evidence of

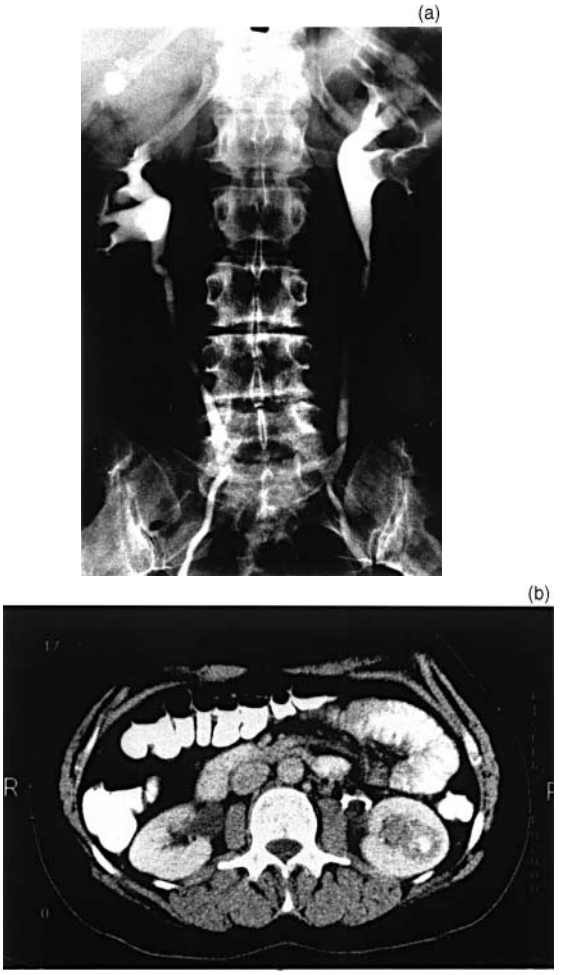

Figure 1 **Preoperative appearances.** (*a*) intravenous urogram showing filling defect in left pelvicalyceal system; (*b*) computed tomographic scan showing non-enhancing mass in left lower pole with area of high attenuation

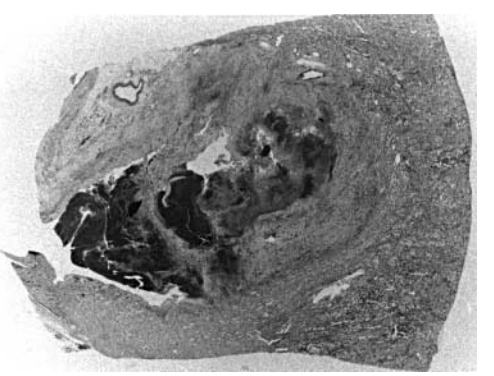

Figure 2 **Haemorrhagic papillary necrosis with adjacent normal cortex and medulla.** Haematoxylin and eosin ×10

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bilateral involvement. Histological damage to the renal papilla adjacent to the vascular abnormality was noted in 4. What is the mechanism? According to one hypothesis, pressure from an expanding haemangioma or thrombosis/haemorrhage within the haemangioma occludes the blood supply to the renal papillary tip leading to necrosis.

Symptomatic haemangiomas are pericalyceal or intrapapillary. Multicentric or diffuse haemangiomas have been reported, their incidence varying from 6% to 13%[2]. Bilateral haemangiomas are very rare but must be considered. Selective angiography is suggested as the investigation of choice, supplemented by magnification angiography and pharmaco-angiography[3]. However, in a reported series[4], preoperative diagnosis was made in only half the histologically confirmed lesions, possibly because of their small size, although even large ones may not opacify.

With hindsight, could nephrectomy have been avoided in our patient? The answer we feel is no, because continuing haematuria was producing a substantial fall in haemoglobin. The radiological appearances were dubious but did not suggest benign disease at any stage. The lesion was juxtamedullary and not cortical; therefore, although the size was < 3 cm, conservative resection was not feasible. To our knowledge there is no means of differentiating a benign lesion as small as this from a tumour and so radical nephrectomy seems to have been the treatment of choice. If recurrent haematuria had been found arising from the contralateral kidney, selective angiography and nephron sparing surgery/therapeutic embolization might have been the only possible options in such case. Angiography is still valuable as a complementary investigation to CT scan in such circumstances.

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