Ophthalmic zoster sine herpete

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Herpes zoster ophthalmicus is usually an obvious clinical diagnosis. When, however, the classic rash is absent, conclusive diagnosis may demand identification of the virus by invasive methods. We have instead used nasal and conjunctival swabs.

CASE HISTORY

A woman aged 95 took an overdose of paracetamol after three days of severe and unremitting pain in her left eye. Her general practitioner had diagnosed conjunctivitis, prescribing paracetamol and chloramphenicol ointment. On examination, there was left-sided conjunctival erythema and injection with slight erythema of the upper and lower palpebrae and obvious lacrimation. No vesicles or rash were visible. Visual acuity was unaffected and pupillary reactions were normal. The anterior chamber was quiet, with no cells seen and no flare. There was no tenderness or thickening of the temporary artery, which was pulsatile. Differential diagnosis was zoster sine herpete or temporal arteritis.

The patient was treated with intravenous *N*-acetylcysteine despite having a serum paracetamol below the threshold, because of her advanced age and potentially diminished reserve of liver function. Liver function tests remained normal, as was the erythrocyte sedimentation rate. Clinically she was well but she needed an opioid (morphine sulphate) to control her left eye pain. She continued to express suicidal intent. Nasal and conjunctival swabs were sent in viral transport medium for viral isolation and polymerase chain reaction (PCR) tests for varicella zoster and herpes simplex DNA. PCR for varicella zoster DNA was positive in both nasal and conjunctival samples, thus confirming the diagnosis of zoster sine herpete; no herpes simplex DNA was found. On serological testing varicella zoster IgG was positive and IgM negative by enzyme-linked immunosorbent assay (herpes simplex IgM and IgG not detected). No viruses were isolated in standard cell cultures after 14 days. Meanwhile, treatment with intravenous acyclovir (10 mg/kg three times daily) had been started and the eye pain was diminishing. After 3 days of intravenous treatment, oral acyclovir was substituted at 800 mg five times a day, continuing for 14 days. The patient's mental state and requirement for analgesia improved greatly and she was discharged home a few days later.

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COMMENT

In a typical case of herpes zoster ophthalmicus the dermatological manifestations in the area innervated by the ophthalmic branch of the Vth cranial nerve will indicate the diagnosis. But in zoster sine herpete[1] this clue is absent. We are unaware of any previous report of the molecular diagnosis of ophthalmic zoster sine herpete obtained through non-invasive sampling methods though such methods have been used successfully in cases of acute peripheral facial palsy associated with varicella zoster[2,3]. Various groups have reported PCR testing of intraocular samples[4–6]. Experience in the present case indicates that early and definitive diagnosis of ophthalmic zoster sine herpete is possible with non-invasive samples. This is important since prompt treatment with acyclovir in herpes zoster ophthalmicus reduces eye damage and pain[7]. Newer agents such as valaciclovir and famciclovir may also prove useful[8,9].

PCR testing needs to be done in a laboratory where carryover contamination by amplified products is avoided by strict physical separation of pre-amplification and post-amplification processes and where the clinical material is handled with barrier pipette tips. These are generally regarded as sufficient precautions if multiple negative controls are incorporated into every assay batch. The PCR assay employed has proved specific for varicella zoster virus.

The PCR test can be completed in about an hour, allowing early and appropriate treatment, and use of swabs avoids the risk associated with aqueous tap.

REFERENCES

1 Gilden DH, Wright R, Schneck S, *et al*. Zoster sine herpete, a clinical variant. *Ann Neurol*, 1994;**35**:530

2 Furuta Y, Fukuda S, Suzuki S, *et al*. Detection of varicella-zoster virus DNA in patients with acute peripheral facial palsy by the polymerase chain reaction, and its use for early diagnosis of zoster sine herpete. *J Med Virol* 1997;**52**:316–19

3 Murakami S, Honda N, Mizobuchi M, *et al*. Rapid diagnosis of varicella zoster virus infection in acute facial palsy. *Neurology* 1998;**51**:1202–5

4 Silverstein BE, Chandler D, Neger R, *et al*. Disciform keratitis: a case of herpes zoster sine herpete. *Am J Ophthalmol* 1997;**123**:254–5

5 Yamamoto S, Tada R, Shimomura Y, *et al*. Detecting varicella-zoster virus DNA in iridocyclitis using polymerase chain reaction: a

case of zoster sine herpete [Letter]. *Arch Ophthalmol* 1995;**113**: 1358–9

6 Stavrou P, Mitchell SM, Fox JD, *et al*. Detection of varicella-zoster virus DNA in ocular samples from patients with uveitis but no cutaneous eruption. *Eye* 1994;**8**:684–7

7 Cobo LM, Foulks GN, Liesegang TJ. Oral acyclovir in the treatment of acute herpes zoster ophthalmicus. *Ophthalmology* 1986;**93**:763

8 Wood MJ, Shukla S, Fiddian AP, Crooks RJ. Treatment of acute herpes zoster: effect of early (<48 h) versus late (48–72 h) therapy with acyclovir and valaciclovir on prolonged pain. *J Infect Dis* 1998;**178**(suppl 1):S81–4

9 Tyring S, Barbarash RA, Nahlik JE, *et al*. Famciclovir for the treatment of acute herpes zoster: effects on acute disease and postherpetic neuralgia. *Ann Intern Med* 1995;**123**:89

Appendiceal adenocarcinoma with ovarian metastases in the third trimester of pregnancy

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Primary adenocarcinoma of the appendix is a rare tumour and there seems to be only one report of its occurrence in pregnancy.

CASE HISTORY

In the 29th week of her first pregnancy a woman aged 29 reported four days of sharp lower abdominal pain associated with vomiting. A mid-stream urine specimen suggested a urinary tract infection and she was admitted and treated with antibiotics. On further questioning she said the right-sided abdominal pain had troubled her intermittently since her antenatal booking visit; she had been previously treated for urinary tract infection. Routine antenatal investigations, including ultrasound scan, had been unremarkable.

An ultrasound scan on this admission revealed a 15 × 20 cm complex mass in the left upper quadrant composed of multiple small cystic and solid areas, separate from the uterus. In addition, two hypoechoic liver lesions were seen. Fine-needle aspiration of one of the liver lesions showed numerous clusters of malignant cells. The associated cell block preparation demonstrated unequivocal adenocarcinoma. The malignant cells stained with cytokeratin and carcinoembryonic antigen, suggesting a gastrointestinal tract or ovarian primary site. In view of these findings, she had a caesarean section at 30 weeks' gestation with delivery of a live girl. At operation, a 20 cm complex mass was found involving the left ovary; there were multiple nodules in the omentum, and the appendix was adherent to the paracolic gutter with the lumen distended. Left oophorectomy, omentectomy and appendicectomy were performed.

The left ovary and fallopian tube specimen was composed of an 18 cm diameter mass of necrotic and solid yellow tissue. The omentum contained multiple firm white tumour deposits up to 3 cm in size. The appendix was irregularly thickened, measuring 6 × 2 × 2 cm, with a dilated lumen filled with mucoid material. No obvious perforation was seen macroscopically. The placenta was macroscopically normal and serial sectioning revealed no tumour deposits.

Microscopic examination of the appendix showed a moderately differentiated adenocarcinoma arising on a background of adjacent epithelial dysplasia (Figure 1), in addition to a perforation. The presence of associated dysplasia confirmed that the tumour was primary to this site. The tumour extended through the appendiceal wall to the serosal surface with vascular and lymphatic invasion present (T3, M1 stage IV). Tumour cells did not stain with antibodies to oestrogen or progesterone receptors. The ovarian and omental deposits contained necrotic moderately differentiated adenocarcinoma identical in appearance to that of the appendiceal primary tumour, with intimately associated decidual change in the desmoplastic tumour stroma (see Figure 1).

After an uncomplicated postoperative recovery the patient received a full course of infusional 5-fluorouracil, epirubicin and carboplatin. At 6 months she was clinically well but a post-treatment computed tomographic scan shows a residual 5 cm lesion in the liver and a multicystic mass in the right iliac fossa.

COMMENT

Primary adenocarcinoma of the appendix is rare; Nielsen *et al.*[1] reported seven cases of adenocarcinoma of the vermiform appendix occurring in Iceland during 1974–1989 (incidence 1 in 500 000/year). The patients ranged in age from 25 to 83 years, average 55 years. Just over half the patients presented with symptoms and signs of acute appendicitis and all these had surgically resectable disease. In the remainder of the cases, the clinical presentation was

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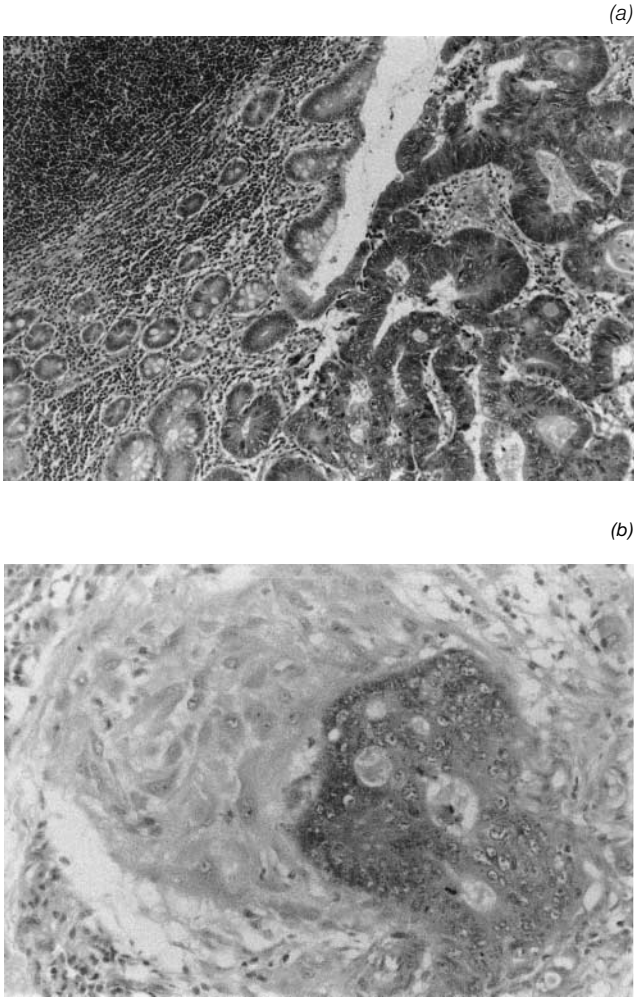

Figure 1 **(a) Photomicrograph of appendix showing adenocarcinoma with adjacent dysplastic epithelium (original magnification ×90); (b) omental deposits of metastatic adenocarcinoma with decidual stromal reaction (original magnification ×180)**

that of metastatic adenocarcinoma of unknown origin and the outcome was uniformly poor. Cortina *et al.*[2] reported on a retrospective case series over a 20-year period from 1972 to 1992; 13 patients were diagnosed with primary appendiceal adenocarcinoma, at a median age of 62 years. The disease was not suspected in any of these patients preoperatively. Three-quarters had metastatic disease at presentation and second primary malignancies were found in 15%. Furthermore, about one-third of female patients had synchronous ovarian lesions. The overall median survival was 22 months, but those with carcinomatosis died much sooner. Hananel *et al.*[3] reported on 2520 patients undergoing appendicectomy during a 14-year period, including 8 with primary adenocarcinoma (0.3%). These patients were all subsequently treated by right hemicolectomy and all were alive and disease-free after a mean follow-up of 57 months.

Ronnett *et al.*[4] analysed 20 cases with ovarian metastases derived from primary appendiceal adenocarcinomas and reported that the most common presentation was a pelvic mass. The appendiceal and ovarian tumours were diagnosed concurrently in 15 cases; in the remaining 5, the ovarian tumours were diagnosed before the appendiceal tumour. The ovarian tumours were bilateral in 16 cases and the appendiceal and ovarian tumours were immunophenotypically identical in each case.

To the best of our knowledge, there is only one previous reported case of appendiceal adenocarcinoma in pregnancy[5], and in this case the presentation was with acute appendicitis; adenocarcinoma was diagnosed on subsequent histological examination. In our case, the presentation was with carcinomatosis. In addition, our patient was young; the possibility of a predisposition to malignancy arises, but no family history of such is known.

The overall incidence of malignant disease is not higher in the pregnant population than in age-matched non-pregnant women, but it is possible that certain tumours may be hormone-dependent. In our patient florid decidualization of the tumour stroma in the omental metastases suggested stromal responsiveness to sex hormones. In cases of malignancy in pregnancy, there are reports of metastatic spread from mother to fetus but in most of these the tumour is melanoma or sarcoma rather than carcinoma. Furthermore, in almost all cases with fetal spread there are malignant deposits within the placenta[6]. In our patient, careful examination of the placenta revealed no malignant deposits.

REFERENCES

1 Nielsen GP, Isaksson HJ, Finnbogason H, Gunnlaugsson GH. Adenocarcinoma of the vermiform appendix—a population study. *APMIS* 1991;**99**:653–6

2 Cortina R, McCormick J, Holm P, Perry RR. Management and prognosis of adenocarcinoma of the appendix. *Dis Colon Rectum* 1995;**38**:848–52

3 Hananel N, Powsner E, Wolloch Y. Adenocarcinoma of the appendix: an unusual disease. *Eur J Surgery* 1998;**164**:859–62

4 Ronnett BM, Kurman RJ, Shmookler BM, Sugarbaker PH, Young RH. The morphologic spectrum of ovarian metastases of appendiceal adenocarcinomas—a clinicopathological and immunohistochemical analysis of tumours often misinterpreted as primary ovarian tumours or metastatic tumours from other gastrointestinal sites. *Am J Surg Pathol* 1997;**21**:1144–55

5 Donnenfeld AE, Robers NS, Losure TA, Mellen AW. Perforated adenocarcinoma of the appendix during pregnancy. *Am J Obstet Gynecol* 1986;**154**:637–8

6 Benirschke K, Kaufmann P. Maternal diseases complicating pregnancy. In: Benirschke K, Kaufmann P, eds. *Pathology of the Human Placenta*. New York: Springer-Verlag, 1995:489–93

Hereditary Madelung's disease

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SECTION OF PLASTIC SURGERY, 12 JANUARY 1999

Madelung's disease is a benign multiple symmetrical lipomatosis primarily affecting the cervical region and upper body. Occasionally it is hereditary.

CASE HISTORY

A man aged 69 was referred for further management after being treated for Madelung's disease for several years at another hospital. Since the age of 25 he had undergone nine debulking operations. There was a family history of the disease: his parents had not been affected but his 63-year-old brother and his only grandchild (a boy aged 7) had been treated for it. He had smoked for 40 years but had abstained for the last 10; his alcohol consumption was not excessive. His main complaint was attributable to the massive non-encapsulated lipomas in his cervical and shoulder region (Figure 1) in conjunction with the hard collar he wore because of cervical spondylosis. Frequently, when he wore the collar, extrinsic pressure on turning his head would compress his carotid arteries sufficiently to cause unconsciousness. On duplex imaging his internal carotids and vertebral vessels were of normal contour and lumen. In an initial debulking operation under general anaesthesia, 520 mL was removed by liposuction and a 460 g adipose mass was resected from the anterior neck. Postoperatively he was returned promptly to theatre for evacuation of a large pretracheal haematoma; the neck drain was removed after 23 days. His symptoms recurred after five months and, in a bilateral anterior neck resection, over 1 kg of adipose tissue external to the platysma was removed. There were no postoperative complications. At one-year follow-up his neck discomfort troubles him less, his blackouts are better controlled and the cosmetic appearance is improved (Figure 2).

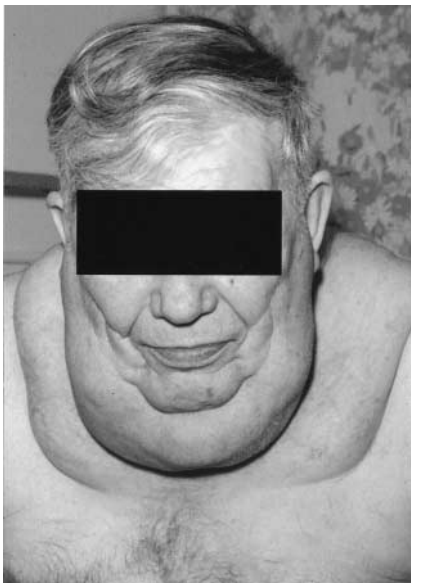

Figure 1 **Preoperative photograph**

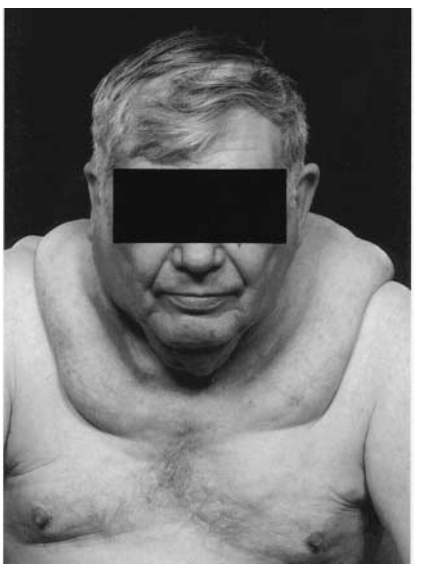

Figure 2 **Postoperative photograph (one year)**

COMMENT

Madelung's disease may be more common than is realized, sometimes being misdiagnosed as gross obesity[1]. Most cases are sporadic, and there are strong links with alcohol abuse and tobacco smoking. The family history in our patient points to a hereditary variant. It is noteworthy that both brothers were somewhat deaf in their right ears and also hypertensive. The genetics of lipomatosis show mitochondrial dysfunction due to point mutations[2] and multiple deletions in the DNA[3] giving rise to abnormalities of metabolism in adipose tissue, ragged-red muscle fibres, and peripheral and central neurons—not necessarily all present in every case. Apart from compressing arteries in the neck, Madelung's disease can result in dysphonia, dyspnoea and dysphagia[4]. A general anaesthetic in these patients usually has an American Society of Anesthesiology grade of 3, because tracheal compression and displacement make intubation difficult[5]. Since recurrence can be expected[6]

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and anaesthesia carries high risks, there is much to be said for radical debulking procedures that may not have to be repeated.

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REFERENCES

1 Stavropoulos PG, Zouboulis CC, Trautmann C, Orfanos CE. Symmetric lipomatosis in female patients. *Dermatology* 1997;**194**:26–31

2 Naumann M, Kiefer R, Toyka KV, Sommer C, Seibel P, Reichmann H. Mitochondrial dysfunction with myoclonus epilepsy and ragged-red fibers point mutation in nerve, muscle and adipose tissue of a patient with multiple symmetric lipomatosis. *Muscle Nerve*, 1997;**20**:833–9

3 Klopstock T, Naumann M, Seibel P, Shalke B, Reiners K, Reichmann H. Mitochondrial DNA mutations in multiple symmetric lipomatosis. *Mol Cell Biochem* 1997;**174**:271–5

4 Baier G, Dazert S. Oropharyngeal fat hernia in Madelung's neck as a rare cause of acute dysphagia (German). *Laryngo-Rhino-Otol* 1997; **76**:425–7

5 Da Broi U, Zauli M, Bonfreschi V, *et al.* Anaethesiologic problems in patients with Launois–Bensaude–Madelung's disease (Italian). *Minerva Anestesiol* 1996;**62**:333–7

6 Kovacs A, Klein C. Madelung disease. A case report with special reference to therapy. *Chirurg* 1997;**68**:276–9

Abdominal actinomycosis presenting as psoas abscess

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CLINICAL SECTION, 15 APRIL 1999

Abdominal actinomycosis is a chronic suppurative disease due to infection by *Actinomyces israelii*. The onset tends to be insidious and it is frequently mistaken for a malignant process[1]. In 90% of cases the diagnosis is made post-operatively by the pathologist.

CASE HISTORY

A woman aged 42 was referred with a two-month history of right iliac fossa pain, fever and a worsening limp. Of particular note in the history, her mother had died of colorectal cancer aged 42, and she had not used an intrauterine contraceptive device (IUCD) for 20 years. On examination she had a low-grade pyrexia, tenderness in the right iliac fossa with erythema over the groin and a fixed flexion deformity of the right hip. The provisional diagnosis was a caecal neoplasm with associated psoas abscess, and she was started on intravenous cefuroxime and metronidazole while awaiting further investigation.

Colonoscopy revealed a polypoid lesion within the caecum, biopsies of which were inconclusive. A computed tomographic scan of the abdomen showed a mass at the caecal pole with a right iliopsoas abscess extending down to the groin. The fixed flexion deformity could be clearly seen (Figure 1). At laparotomy there was a tumour around the caecal pole, connecting to an iliopsoas abscess. A right hemicolectomy was performed and the psoas abscess was drained. Histological examination revealed chronic inflammation and microabscess formation in the caecal submucosa close to the origin of the appendix; this inflammation extended through the wall of the bowel. The microabscesses contained *Actinomyces* and there was no evidence of malignancy (Figure 2). Actinomycosis of the caecoappendiceal junction was diagnosed. The patient was discharged on a 3-month course of penicillin and was fully recovered at 6-month follow-up.

COMMENT

Actinomyces israelii is a Gram-positive, anaerobic, filamentous bacterium commensal in the oral cavity and upper intestinal tract and pathogenic only in the presence of damaged or necrotic tissue[2]. Predisposing factors include appendicitis,

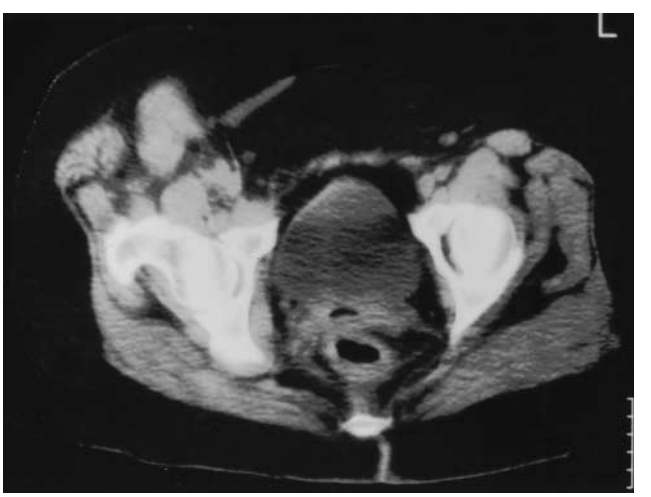

Figure 1 **Computed tomographic scan of abdomen at level of hip joint showing right psoas abscess, pointing in the groin, and flexion of right hip**

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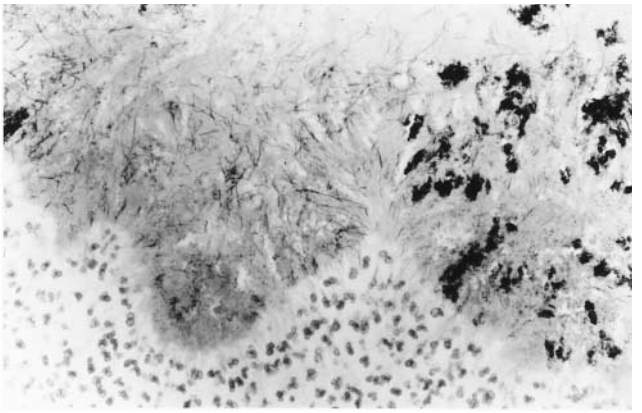

Figure 2 **Haematoxylin and eosin stained section of caecum showing pus containing a colony of *Actinomyces*, filaments (left) and clumps of other bacteria (right)**

gastrointestinal perforation, previous surgery, foreign bodies (especially IUCDs) and neoplasia. As a pathogen it causes fistulae, sinuses and abdominal masses. Actinomycosis is diagnosed preoperatively in fewer than 10% of cases[1]. Typically the pathologist detects colonies of *Actinomyces* within abscesses in resected tissue. These correspond to 'sulphur granules' that may be identified by the naked eye in pus. When actinomycotic abdominal masses and strictures are identified without surgery, long-term penicillin can avoid operation[3]. A computed tomographic scan will usually show a contrast enhancing multicystic lesion and the diagnosis can be confirmed by needle aspiration[4,5].

There are very few reports of abdominal actinomycosis presenting as psoas abscess[3,5] and none to our knowledge with all components of the classic triad—pain, fever and a limp. Needle aspiration of our patient's psoas abscess might have provided the diagnosis and obviated the need for laparotomy and bowel resection. First-line investigation of a psoas abscess should include needle aspiration, which may be both diagnostic and therapeutic[6].

REFERENCES

1 Thompson RJ, Watts R Jr, Thompson WC. Actinomycetoma masquerading as an abdominal neoplasm. *Dis Colon Rectum* 1982;**25**:368–70

2 Berardi RS. Abdominal actinomycosis. *Surg Gynecol Obstet* 1979;**149**: 257–66

3 Cintron JR, Del Pino A, Duarte B, Wood D. Abdominal actinomycosis. *Dis Colon Rectum* 1996;**39**:105–8

4 Ha HK, Lee HJ, Kim H, Ro HJ. Abdominal actinomycosis: CT findings in 10 patients. *Am J Roentgenol* 1993;**161**:791–4

5 Moosmayer S. Abdominal actinomycosis: actinomycotic abscess 10 years after appendicectomy. *Tidsskr Nor Laegeforen* 1992;**112**:2857–8

6 MacGillivray DC, Valentine RJ, Johnson JA. Strategies in the management of pyogenic psoas abscesses. *Am Surg* 1991;**57**:701–5

Isolated sarcoidosis of the breast

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Systemic sarcoidosis is a disease of unknown aetiology characterized by granulomatous lesions affecting multiple organs. Isolated breast sarcoidosis is extremely rare.

CASE HISTORY

A woman aged 53 reported right breast discomfort of recent onset. On physical examination there was localized nodularity within the lower pole of the breast and mammography revealed pronounced nodularity and architectural distortion of the breast parenchyma with benign features. Breast ultrasound was normal. Fine needle aspiration cytology from the area of localized nodularity yielded benign ductal epithelial cells and multinuclear histiocytes. A core biopsy specimen showed chronic inflammation with numerous non-caseating granulomata and Langerhans-type multinuclear giant cells. No micro-organisms were seen and breast sarcoidosis was diagnosed. On excision biopsy, performed on grounds of clinical judgment, the operative findings were of extensive white fibrous strands extending into adjacent fat tissue. The

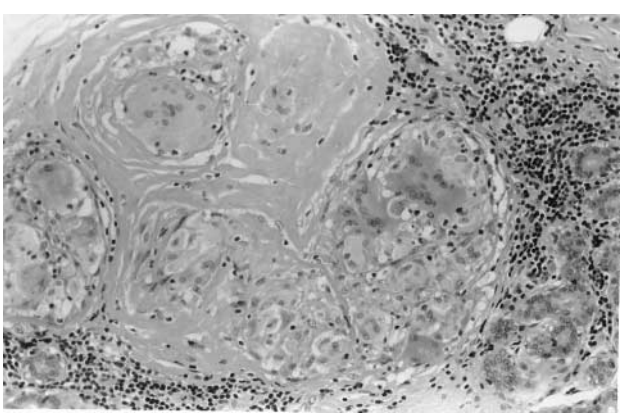

Figure 1 **Excision biopsy specimen**

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histopathological report confirmed widespread epithelioid granulomata within fibrous chronically inflamed stroma (Figure 1). There was no evidence of either *in-situ* or invasive carcinoma and all stains for microorganisms were negative. The final pathological diagnosis was granulomatous mastitis secondary to sarcoidosis. Further investigations to identify systemic disease were performed. Serum angiotensin-converting enzyme and calcium, chest radiography and abdominal ultrasound scan were all normal. Pulmonary function tests were unremarkable and a gallium scan showed no abnormal uptake. The Kveim test was negative. One year after surgery the patient remains well on no treatment, with no evidence of recurrent or systemic disease.

COMMENT

Subcutaneous sarcoidosis was first described in 1904 by Darier and Roussy[1] and the form also involving the breast by Stranberg in 1921[2]. Sarcoidosis most commonly affects the lungs but extrapulmonary involvement is seen in 40% of cases. Reviewing the published work, Donaldson[3] found 29 cases of mammary sarcoidosis, with the breast the site of primary diagnosis in only 5. Sarcoidosis of the breast may present as a persistent non-tender mobile mass or as a tender fixed mass. Since it tends to affect middle-aged women, it needs to be differentiated from a malignant lump. In all previous reports of apparently isolated breast sarcoidosis, the diagnosis was made after wide local excision or mastectomy[3,4]. Microscopic examination in all patients revealed non-caseating epithelioid granulomata with giant cells. The diagnosis of breast sarcoidosis relies on the exclusion of other causes of granulomatous mastitis such as mycobacteria, fungi and foreign-body reactions. Systemic sarcoidosis is suggested by a positive Kveim test and a raised serum angiotensin-converting enzyme or lysozyme[3]. In our patient there was no evidence of systemic disease.

Previous work has established the possible coexistence of mammary sarcoid with malignancy[4] and we should be cautious about attributing breast lumps to benign granulomatous disease. In our patient preoperative triple assessment and core biopsy had demonstrated sarcoid disease of the breast, which was subsequently confirmed at surgical excision biopsy. Although the histological diagnosis of breast sarcoidosis can be predicted from adequate core biopsy, the decision for or against surgical excision depends on clinical criteria. Mastectomy is seldom indicated.

REFERENCES

1 Darier J, Roussy G. Un cas de tumeurs benignes multiples sarcoides sous-cutaneous ou tubercuilides nodulaires hypodermiques. *Ann Dermatol Syph* 1904;**5**:144–9

2 Stranberg J. Contribution à la question de la clinique et de la pathogénie de la sarcoide de Boeck. *Acta Derm Venereol* 1921;**2**:253

3 Donaldson BA, Polynice A, Oluwole S. Sarcoidosis of the breast: case report and chart review. *Am Surg* 1995;**61**:778–80

4 Shah AK, Solomon L, Gumbs MA. Sarcoidosis of the breast coexisting with mammary carcinoma. *N Y State J Med* 1990;**6**:331–3