with greater testicular size, less severe gynaecomastia and fewer secondary sexual characteristic changes, often with normal mental function (though character or personality disorders are common) (Paulsen *et al.* 1968). The normal HCG levels in 1968 and the presence of elastic fibres around the tunica propria (suggesting post-pubertal onset of the seminiferous tubule failure (Ferguson-Smith 1959, de la Balze *et al.* 1954) were features indicative of late-onset gonadal failure in our patient. Clinically, his gynaecomastia, evident only in 1979, was mild and although his job as an archivist suggests above-average intelligence, psychiatric opinions have suggested that he does have a personality disorder. Sclerosed acellular tubules and Leydig cell hyperplasia have been well described in Klinefelter's syndrome (Sniffen *et al.*, 1951, Heller & Nelson 1945). Although varying degrees of partial seminiferous tubule dysgenesis are more commonly seen in the chromatin-negative type (Gordon *et al.* 1972), the tubules have also been reported to be hyalinized and acellular with a reduced number of ghost tubules lined with elastic fibres (Ferguson-Smith *et al.* 1960), as in our patient.

The juvenile presentation of congenital adrenal hyperplasia in this case modified some aspects of the presentation (by masking eunuchoid signs and preventing the increase in height commonly associated with the XYY karyotype) but its contribution to the other atypical aspects of this case is not known. Since cell lines with an abnormal sex chromosome complement may only become apparent when 100–500 cells are examined (often from multiple tissue biopsies), we may not yet have adequately characterized the sex chromosomal mosaicism in this patient on the material available so far. Cytogenetic persistence and time are required in such cases to support the clinical and histological features.

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# Pelvic actinomycosis presenting with rectal stricture[1]

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A case is reported of pelvic abscess due to actinomycosis presenting with a rectal stricture. Such a presentation has been reported only once previously.

*Case report*

A 19-year-old girl presented as an emergency with left iliac fossa pain associated with nausea and vomiting. She dated the start of her problems to the insertion of an intrauterine contraceptive device (IUCD) 20 months previously, although this had been removed 9 months prior to

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admission. She complained of constipation alternating with diarrhoea and of the loss of 3 stone (19 kg) in weight over the preceding 9 months. Prior to admission she had had some dysuria but no haematuria.

On examination she was tender in both iliac fossae but more so on the left and a mass could be palpated in the left iliac fossa. Vaginal examination was normal but on rectal examination her general practitioner had felt a stricture. Sigmoidoscopy showed a normal mucosa with a stricture at 10 cm which was biopsied on two separate occasions. The histology of the first biopsy was inconclusive but the second showed a submucosal lymphocytic infiltrate, compatible with an inflammatory mass outside the bowel wall; there was no evidence of Crohn's disease. Her haemoglobin was 10 g/dl and her ESR 100/h. Other biochemical investigations were normal. A barium meal and follow through showed some local duodenitis, compatible with Crohn's disease, and suspicious features in the pelvis. Barium enema showed a 5 cm long stricture at the rectosigmoid junction (Figure 1).

She was transfused. A laparotomy was performed and on opening the abdomen dense, hard adhesions were encountered and an inflammatory mass in the left iliac fossa was noted. The small bowel and omentum were adherent to the inflammatory mass. The left broad ligament was indurated but the other pelvic organs appeared normal. When the adhesions were separated an old abscess cavity was entered which contained inspissated pus; the edge of the cavity was biopsied and a drain inserted. The histology showed subacute and chronic inflammation with colonies of Actinomyces (Figure 2). An endometrial biopsy at the time of laparotomy failed to show any Actinomyces.

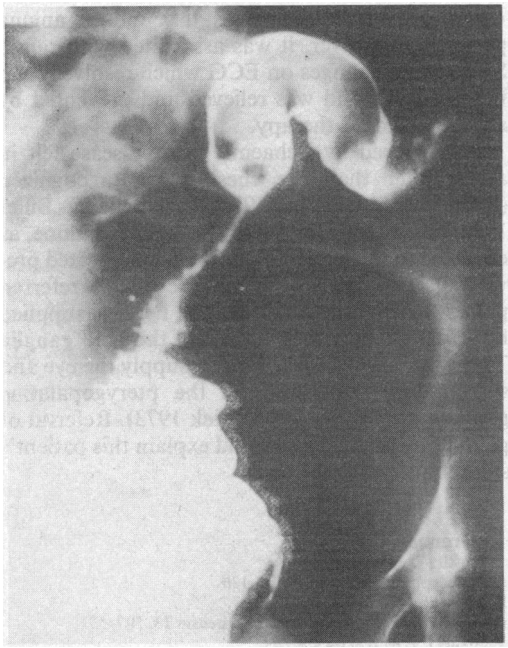

Figure 1. Barium enema showing stricture at rectosigmoid junction due to actinomycosis

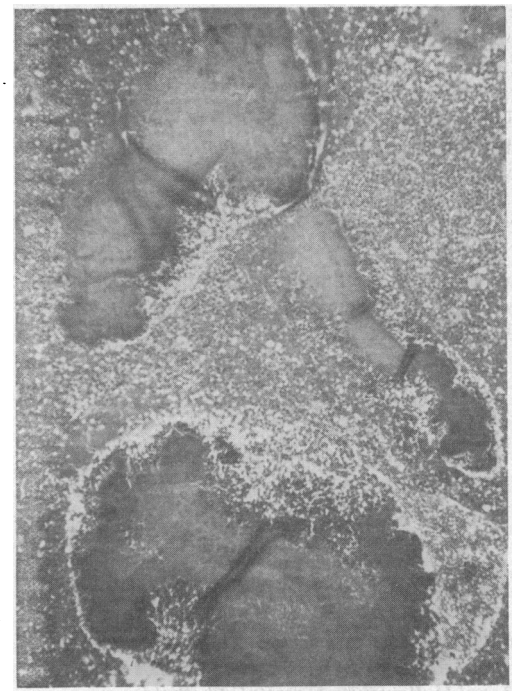

Figure 2. Histology of wall of pelvic abscess showing colonies of Actinomyces

She was treated with intravenous benzylpenicillin 3 megaunits 6 hourly for 3 weeks and with oral fusidic acid 500 mg three times daily. The mass gradually decreased in size. She was discharged home taking penicillin V 1 g five times a day and fusidic acid 500 mg three times daily, this treatment being continued for a total of 12 weeks. Follow up showed a steady recovery and by six months the mass had disappeared, the rectal stricture had disappeared and her ESR was normal. She had a further episode of right iliac fossa pain 18 months after her initial referral and was seen by a gynaecologist, but there was no evidence of recurrence of actinomycosis and her ESR was 3.

*Discussion*

Actinomycosis of the gastrointestinal tract is rare but any part may be involved. The most likely cause of this young girl's pelvic actinomycosis was the IUCD she had been using, as the relationship between the two is well established (Spence *et al.*

1978). In the only previously reported case of pelvic actinomycosis presenting with rectal stricture the patient was a middle-aged man whose rectal biopsy proved diagnostic (Morson 1961). Thus, although pelvic actinomycosis is well recognized, our case represents only the second reported case presenting as a rectal stricture. Fry *et al.* (1965) recommended high-dose penicillin or tetracycline for treating actinomycosis, and Mitchell (1983) suggested cephalosporin, erythromycin or clindamycin as alternatives for penicillin-sensitive patients. In this case, the combination of high-dose penicillin and fusidic acid given as a prolonged course was effective in eradicating the infection.

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# Headache due to ischaemic heart disease[1]

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Headache associated with ischaemic heart disease is usually caused by therapy with nitrates. A patient is reported with a headache of cardiac origin which was abolished by nitrates.

*Case report*

A 78-year-old woman was admitted to hospital with inferior myocardial infarction. She complained of tight central chest pain radiating to both arms for 4 hours, associated with a frontal headache. The diagnosis of myocardial infarction was confirmed by electrocardiograms and raised cardiac enzymes in plasma.

She gave a history of headache for about one year, occurring several times a week. The pain was frontal, continuous and radiated to the roof of the mouth and down both arms. Often it was associated with tightness in the chest, but occasionally occurred alone. It troubled her more than the chest tightness which she tended to dismiss. Walking up one flight of stairs, for several minutes uphill and from a warm room into a cold one brought on her headache.

On examination, the only abnormality was evidence of mild left ventricular failure. There was no neurological abnormality or carotid bruits and the temporal arteries were normal. We observed closely one attack of headache following a meal. There were no marked changes in blood pressure or pulse rate but her electrocardiogram showed elevation of the ST segments in leads S2, S3 and AVF which reverted to normal when the headache resolved. Her skull X-rays were normal and her erythrocyte sedimentation rate was 32/h. Treatment with nifedipine 20 mg three times daily and isosorbide dinitrate 10 mg four times daily completely prevented her headache.

*Discussion*

We consider that this patient's headache is a manifestation of myocardial ischaemia, as it occurred before or with her chest tightness and was precipitated by factors that induce angina pectoris. Moreover, it was associated with raised ST segment changes on ECG which resolved with her headache and was relieved and prevented by antianginal drug therapy.

Pain caused by ischaemic heart disease felt in areas other than the chest is well recognized (Sampson & Cheitlin 1971, Lefkowitz & Biller 1982). However pain felt in the forehead alone, as occurred in this patient, has not been reported previously. The mechanism may be due to referred pain via sympathetic fibres. The heart is supplied by sympathetic fibres from the thoracic ganglia T1-T4, and fibres from T1 also supply the eye and surrounding structures via the pterygopalatine ganglion (Williams & Warwick 1973). Referral of pain along these fibres would explain this patient's symptoms.

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