Tuberculosis of the cervix: case presentation and a review of the literature

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CASE REPORT

A Kenyan woman attended a genitourinary medicine (GUM) clinic, complaining of a 4 year history of amenorrhoea. She was aged 29 years, and had been studying in London for the past 20 months. She had lost 5 kg in weight over the previous 6 months. She denied any fever, cough, or abdominal pain. An antibody test for HIV infection had been negative 4 years previously. Subsequently, her only HIV risk factor was unprotected vaginal intercourse with three Kenyan male partners. Six months before attending, she had an abnormal cervical smear which was reported as showing mild dyskaryosis. She had been in close contact with an index case of pulmonary tuberculosis at around the time her menstruation ceased.

On examination she had an abnormal cervix, with ulceration, bleeding, and a friable papillary growth covering almost the entire ectocervix. Tests for Chlamydia trachomatis, Neisseria gonorrhoeae, Trichomonas vaginalis, and herpes simplex virus were negative. A hormone profile was normal. A repeat smear was taken. The patient was referred for investigation to exclude carcinoma.

A cervical punch biopsy was taken. Histological examination showed ulcerated fragments of cervix with severe chronic active inflammation and granuloma formation and numerous giant cells. The repeat cytology confirmed the presence of koilocytes and mild dyskaryosis. There were no malignant cells seen and stains for mycobacteria and fungi were negative. Despite the negative auramine stain, tuberculosis was considered the most probable diagnosis.

A wedge biopsy was taken for further histological evaluation and to provide specimens for culture. The original histological appearance was confirmed. A chest radiograph was normal. A Heaf test gave a grade 4 response. Three urine samples were negative for acid fast bacilli and failed to culture mycobacteria. The biopsy specimen also failed to culture mycobacteria and fungi.

Other causes of granulomatous cervicitis were excluded as follows: three urine samples were negative for schistosoma ovum; schistosomal, amoebic, and brucella antibodies were negative; and a serum level of angiotensin converting enzyme was normal.

Magnetic resonance imaging (MRI) revealed lymphadenopathy along the pelvic walls, with an abnormal signal to the entire body of the uterus. The endometrial cavity, myometrium, and junctional zone could not be differentiated and thus the radiological appearance was consistent with Asherman’s syndrome.

A full blood count showed lymphopenia. After counselling, the patient tested positive for HIV-1 infection. The CD4 count was 200 cells ×10³/l. A viral load was ascertained using Chiron 3.0 and Roche 1.5 assays. The results were 1052 and 7600 RNA copies/ml respectively.

Anti-tuberculous quadruple therapy was instigated. Chemophylaxis against Pneumocystis carinii was also initiated. Antiretroviral therapy was deferred until her treatment for TB was completed.

At 4 weeks, the cervix had an almost normal macroscopic and colposcopic appearance. At 6 months, after completion of TB treatment, MRI showed normal signal differentiation within the uterus making the myometrium, junctional zone, and endometrium distinguishable. Enlarged lymph nodes were still noted in the pelvic and internal iliac areas and probably relate to the HIV infection.

Twelve months after completion of anti-tuberculous therapy menstruation resumed.

DISCUSSION

Symptomatic genital tract TB usually presents with abnormal vaginal bleeding, menstrual irregularities, abdominal pain, and constitutional symptoms. 1–8

Pelvic organs are infected from a primary focus, usually the chest, by haematogenous spread. 2,4,10–12 The cervix is infected, as part of this process, by lymphatic spread or by direct extension. The primary lesion is often healed at presentation. 1,11 In rare cases, cervical TB may be a primary infection. 2,4,5,10,13 introduced by a partner with tuberculous epididymitis or other genitourinary disease. Chowdhury 7 has suggested that sputum, used as a sexual lubricant, may also be a route of transmission. There may be hormone dependence of infection, 1,5 given that 80% of cases occur in the reproductive age.

The macroscopic findings of cervical TB are illustrated by this case. There may be papillary or vegetative growths, a milky appearance, and/or ulceration present thus simulating invasive cervical cancer. 2,4,5,10

Microscopically there are (casing) granulomata. These are not diagnostic. The differential diagnoses for granulomatous disease of the cervix include amoebiasis, schistosomiasis, brucellosis, tularaemia, sarcoidosis, and foreign body reaction.

The diagnosis of cervical TB is usually made by histological examination of a cervical biopsy specimen. 1–13 In one study, staining for acid fast bacilli was not found to be very useful in making the diagnosis. 13 A retrospective review found that ulcerative lesions usually are auramine negative. The detection of granulomata on cervical cytology specimens 14,15 has been documented. Isolation of the mycobacterium is the gold
standard for diagnosis. A third of cases are culture negative. Therefore, the presence of typical granulomata is sufficient for diagnosis if other causes of granulomatous cervicitis are excluded or a primary focus identified. Molecular probes may be more sensitive than culture but also have reduced specificity. The lesion should respond to 6 months of standard therapy.

A lesion on the cervix provides a marker to assess response to therapy. Histological examination of serial biopsy specimens can similarly confirm a therapeutic response. Lastly, radiological abnormalities can be followed.

Fertility is generally poor even after treatment, owing to endometrial and tubal involvement at presentation and subsequent healing by fibrosis.10–12

The incidence of TB has increased recently and is partly attributable to the HIV pandemic. There should be a high index of suspicion of tuberculosis in women, with an abnormal cervical appearance, especially from areas where HIV and TB are common.

CONTRIBUTORS
HL, main author and directly involved in care of the patient as an inpatient and outpatient; MB, co-author and directly involved in the outpatient care of the patient; RG, co-author and examined the histological specimens provided; CJ, co-author and directly involved in the inpatient care of the patient.

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