PostScript

LETTERS

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Perforating chancre: any cause-effect relation with HIV infection?

Variation in clinical pictures of syphilis, when co-infected with HIV are well known.[1] Normally, a classic Hunterian chancre heals within 1–2 weeks of treatment without scarring.[2] Primary chancre, healing with perforation of the site, does not commonly occur.[3] Here we report four patients with primary syphilis, in whom the chancres healed with perforation of the genitalia. Concomitant infection with HIV is presumed to be responsible for this destructive sequela.

Case 1

A 21 year old woman presented with a painless, indurated ulcer on the inner aspect of the left labia majora, along with same sided inguinal lymphadenopathy of 1 week's duration. Dark ground microscopy (DGI) was positive for *Treponema pallidum* and VDRL titre was 1:64. Following treatment with penicillin, the ulcer healed slowly, leaving a perforation on the labia majora.

Case 2

A 20 year old unmarried male patient with high risk behaviour presented with a painless indurated ulcer over the dorsal aspect of the prepuce and unilateral inguinal lymphadenopathy. DGI was positive for *T pallidum* and VDRL titre was 1:32. He had a history of genital ulceration and was treated for suspected lymphogranuloma venereum. Following treatment with penicillin, the ulcer healed at a slower pace leaving a large perforation on the prepuce (fig 1).

Case 3

A 23 year old unmarried man, with a history of repeated unprotected exposure to commercial sex workers, presented with a painless, indurated ulcer on the dorsal prepuce, multiple genital mollusca contagiosa, and genital warts.

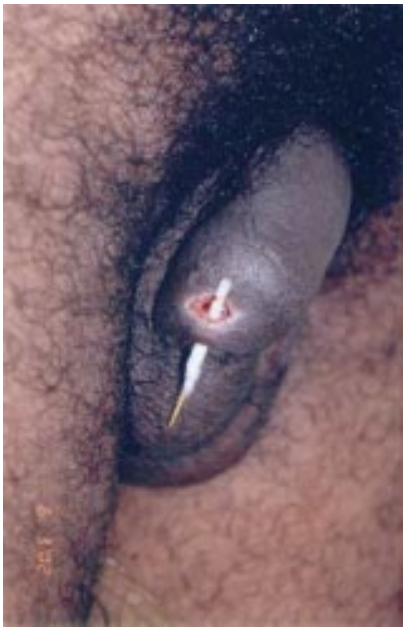

Figure 1 Perforation of prepuce.

Bilateral inguinal lymphadenopathy was present. DGI from the ulcer was negative and VDRL was 1:64. Following penicillin therapy, it healed with perforation of the prepuce.

Case 4

A 45 year old married man with high risk behaviour presented with a large perforation on the lateral side of the shaft of the penis. He gave a history of a painless ulcer on the same site about 1 month earlier. At presentation, his VDRL was 1:32. He was treated with penicillin.

Comment

Gram stained smears from the ulcers and culture for aerobic and anaerobic organisms were negative in first three cases. In all the four patients, ELISA for HIV was positive.

Immune response to *T pallidum* is primarily cell mediated.[2] In an immunocompetent host with primary syphilis, CD4+:CD8+ T lymphocyte ratio is high at the site of the chancre,[2] which possibly prevents local multiplication of the organism. Consequent to the loss of local cellular immunity as a result of HIV infection there may be an enhanced ability of the organism to multiply locally, giving rise to larger and deeper ulcers which are slower to heal. This fact has been demonstrated experimentally in animal models.[4]

Studies exploring the correlation of CD4+ T cell count and stage of HIV infection with this altered manifestation of primary syphilis should be undertaken. This might show the impact of HIV infection on the clinical severity of primary chancre.

A C Inamadar, A Palit

Department of Dermatology,Venereology and Leprology, BLDEA'S Shri BM Patil Medical College, Hospital and Research Centre, Bijapur, Karnataka, India

Correspondence to: Dr Arun C Inamadar; Aparuna1@rediffmail.com

References

1 **Musher DM**, Hamill RJ, Raughn RE. Effect of human immunodeficiency virus (HIV) infection on the course of syphilis and on the response to treatment. *Ann Intern Med* 1990;**113**:872–81.
2 **Sanchez MR**. Syphilis. In: Freedberg IM, Eisen AZ, Wolff KW, *et al*, eds. *Fitzpatrick's dermatology in general medicine*. Vol 2. 5th ed. New York: McGraw-Hill, 1999:2551–80.
3 **Pavithran K**. Perforating syplilitic chancre. *Indian J Dermatol Venereol Leprol* 1987;**53**:352–4.
4 **Marra CM**, Handsfield NH, Kuller L, *et al*. Alterations in the course of experimental syphilis associated with concurrent simian immunodeficiency virus infection. *J Infect Dis* 1992;**165**:1020–5.

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Superior mesenteric artery syndrome in an HIV positive patient

A 27 year old HIV positive man with a CD4+ lymphocyte count of 26 cells $\times 10^6$/l presented with a 2 week history of progressive left sided weakness, vomiting, and weight loss. A computed tomograph (CT) brain scan demonstrated ring lesions bilaterally in the basal ganglia. Toxoplasma serology was positive at a titre of 1:256 and treatment for cerebral toxoplasmosis commenced. His weakness responded to therapy but vomiting continued despite antiemetics. An ultrasound scan demonstrated an enlarged, dilated stomach, dilated first and second parts of the duodenum, and an obstruction at the level of the third. Barium studies confirmed these findings but also demonstrated prominent peristalsis in the second part of the duodenum and an abrupt cessation of flow to barium in the middle of the third (fig 1). Some flow of barium into the jejunum was noted when the patient was turned prone. An abdominal CT scan demonstrated a reduction in the angle between the superior mesenteric artery and the aorta (fig 2). A diagnosis of superior mesenteric artery (SMA) syndrome was considered. Two litres of bile were aspirated per nasogastric tube daily and he continued to lose weight. His body mass index (BMI) fell to

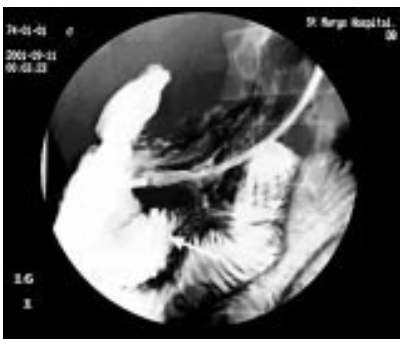

Figure 1 Image from barium meal series. The proximal duodenum is dilated. There is an abrupt calibre change (arrow) in the third part where the superior mesenteric artery crosses. Distinct peristalsis was seen in this region during the study.

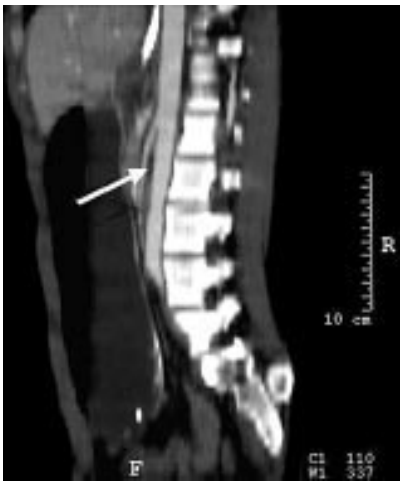

Figure 2 Multislice CT with intravenous contrast medium: sagittal reconstruction through mid-abdomen. The angle between the superior mesenteric artery and the aorta is reduced causing compression of the duodenum (arrow). Note grossly dilated stomach anteriorly.

12 and total parenteral nutrition was introduced for 6 weeks after which an exploratory laparotomy was performed. An anterior gastrojejunostomy was made and a jejunal feeding tube inserted into the collapsed proximal small bowel. The patient recovered postoperatively but continued to vomit after meals. After 4 weeks his BMI increased to 15, vomiting stopped, and he demanded food. At the time of writing he is well, independent, and on antiretroviral therapy.

Superior mesenteric artery syndrome is a controversial diagnosis synonymous with vascular compression of the duodenum, arteriomesenteric duodenal compression syndrome, the cast syndrome, chronic duodenal ileus, and Wilkie's syndrome. First described by Rokitansky in 1842, frequency of reports have recently declined and its existence debated.[1] The syndrome has been ascribed to a reduction in the angle between the aorta and the superior mesenteric artery, scissoring the duodenum in its third part causing obstruction. This is often because of sudden, severe weight loss resulting in a reduction of mesenteric and retroperitoneal fat. Precipitating factors include eating disorders, severe wasting conditions, prolonged immobilisation, previous abdominal surgery, or inflammatory conditions. It has also been reported in cases of severe kyphoscoliosis.[2] It has not previously been reported in AIDS.

Characteristic symptoms, typically intermittent in nature, comprise bloating, nausea, and intractable bilious vomiting relieved by adopting the prone or knee to chest position. A barium meal is the most useful diagnostic investigation. Features of note include dilatation of the first and second parts of the duodenum and an abrupt, linear hold up of flow to barium in the third with abnormal peristalsis and even reverse peristalsis frequently observed. Relief of the obstruction can in some instances be achieved by placing the patient prone during the investigation.[1–3] CT studies can demonstrate reduction in the aortosuperior mesenteric artery angle and serve as a non-invasive diagnostic tool.[4]

Reversal of weight loss is key to resolution, by surgical means if necessary. Nutritional support should be attempted first. Endoscopic or nasogastric decompression is often difficult because of severe gastric dilatation. Duodenojejunostomy or gastrojejunostomy are the surgical procedures of choice when medical therapy fails.[2 3] Our patient did not experience immediate symptomatic relief through surgery but did achieve rapid weight gain via jejunal feeding. We report the first case of SMA syndrome in a patient with AIDS. The spread of HIV worldwide and its association with severe wasting makes this an important differential diagnosis for the clinician.

R Stümpfle, A R Wright, J Walsh
St Mary's Hospital, Praed Street, London W2 1NY, UK

Correspondence to: Dr Richard Stümpfle, Department of Anaesthetics, Northwick Park Hospital, Watford Road, Harrow, Middlesex HA1 3UJ, UK; rstumpfle@doctors.net.uk

References

1 **Anderson JR**, Earnshaw PM, Fraser GM. Extrinsic compression of the third part of the duodenum. *Clin Radiol* 1982;**33**:75–81.
2 **Hines JR**, Gore RM, Ballantyne GH. Superior mesenteric artery syndrome. Diagnostic criteria and theraputic approaches. *Am J Surg* 1984;**148**:630–2.
3 **Lundell L**, Thulin A. Wilkie's syndrome—a rarity? *Br J Surg* 1980;**67**:604–8.
4 **Konen E**, Amitai M, Apter S, *et al.* Angiography of superior mesenteric artery syndrome. *Am J Roentgenol* 1998;**171**:1279–81.

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Was the Papanicolaou smear responsible for the decline of *Trichomonas vaginalis*?

There has been a dramatic decline in the prevalence of trichomoniasis in Australia over the past 30 years. In 1979, 17.8% of women attending a Sydney STI clinic had *Trichomonas vaginalis* infection.[1] By 1998 less than 1% of non-Indigenous women presenting to family planning and STI clinics in another jurisdiction were diagnosed with the condition[2] and most Australian urban pathology laboratories do not diagnose a case from one year to the next. Similar observations have been reported elsewhere: the rate of detection of trichomoniasis in Papanicolaou (Pap) smears in Denmark fell from 19% in 1967 to <2% in 1997,[3] and a study in Brazil found similar results (a peak of 17.3% in 1978, falling to 3.4% in 1998).[4]

In the absence of any health promotional activities relating to trichomoniasis and in a setting where the prevalence of another STI, *Chlamydia trachomatis,* has shown a fourfold *increase* in notifications in the past 10 years (Communicable Diseases Network Australia, National Notifiable Diseases Surveillance System, personal communication), what can explain the decline and fall of *T vaginalis*?

I propose that the change in prevalence is an unintended consequence of the introduction of coordinated Pap smear screening programmes in the 1970s and 1980s. As the Pap screening programmes gained momentum in the urban areas, a positive finding on the Pap smear, which has a sensitivity for the diagnosis of *T vaginalis* of around 50–60%, would have been conveyed to the referring medical practitioner who would treat the woman with metronidazole or tinidazole. In addition, the increasing use of these antibiotics for the treatment of other conditions, in particular bacterial vaginosis, may have further reduced the prevalence during the same period. As there are no cytological changes that are diagnostic of *C trachomatis*, Pap screening would be expected to have no effect on chlamydia prevalence.

In Australian urban populations the proportion of women undergoing Pap screening in the 20–40 year age group is approaching 70%. On the other hand, in some remote Aboriginal populations the introduction of coordinated screening has lagged behind urban areas[5] and trichomoniasis remains hyperendemic (prevalence of approximately 25%).[6]

(Of course these observations could be confounded by a number of factors: Pap screening rates correlate with socioeconomic status and the rate of partner change could be different between these groups. However, it has been shown that access to services is more important than differences in the rate of partner change when comparing STI rates in Indigenous and non-Indigenous populations in Australia.[2])

The Pap smear hypothesis could be tested by correlating the prevalence of trichomoniasis with the rate of cervical cancer screening in selected populations and through clinic based case-control studies. (The virtual absence of trichomoniasis in urban Australia means that this work must be performed in other populations.) If the prevalence of *T vaginalis* is related to Pap screening, a similar approach to chlamydia control—that is, routinely linking nucleic acid amplification testing for *C trachomatis* with the Pap smear, could also be considered.

Conflict of interest: None.

F J Bowden
Australian National University and Canberra Sexual Health Centre, PO Box 11, Woden ACT, Australia 2605; frank.bowden@act.gov.au

References

1 **Tapsall JW**, Puglisi J, Smith DD. Trichomonas vaginalis infections in Sydney: laboratory diagnosis and prevalence. *Med J Aust* 1979;**1**:193–4.
2 **Fairley CK**, Bowden FJ, Gay NJ, *et al.* Sexually transmitted diseases in disadvantaged Australian communities [letter]. *JAMA* 1997;**278**:117–18.
3 **Dragsted DM**, Farholt S, Lind I. Occurrence of trichomoniasis in women in Denmark, 1967–1997. *Sex Transm Dis* 2001;**28**:326–9.
4 **Adad SJ**, de Lima RV, Sawan ZT, *et al.* Frequency of Trichomonas vaginalis, Candida sp and Gardnerella vaginalis in cervical-vaginal smears in four different decades. *Sao Paulo Med J* 2001;**119**:200–5.
5 **Mak D**, Straton JA. Effects and sustainability of a cervical screening program in remote Aboriginal Australia. *Aust NZ J Public Health* 1997;**21**:67–70.
6 **Bowden FJ**, Paterson BA, Mein J, *et al.* Estimating the prevalence of Trichomonas vaginalis, Chlamydia trachomatis, Neisseria gonorrhoeae, and human papillomavirus infection in indigenous women in northern Australia. *Sex Transm Infect* 1999;**75**:431–4.

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The HIV/AIDS epidemic in Ukraine: stable or still exploding?

A recent article published in *Sexually Transmitted Infections*[1] presented evidence suggesting that the HIV/AIDS epidemic in Ukraine had peaked in 1997 and has since declined. The world has only recently awoken to the threat of a widespread HIV/AIDS epidemic in eastern Europe, including projections of an