Lymphogranuloma venereum presenting as genital ulceration and inguinal syndrome in men who have sex with men in London, United Kingdom

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Key Words: lymphogranuloma venereum, bubo, inguinal, ulcer

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Key Messages

- Genital ulcers and inguinal node disease caused by LGV have been observed in the current LGV epidemic in London men who have sex with men (MSM).

- *C. trachomatis* nucleic acid tests performed on swabs, urine and lymph node aspirates provide suitable diagnostic specimens that allow confirmation by LGV-specific molecular analysis.

- LGV should be included in the differential diagnosis of anogenital ulceration in all MSM.
Abstract

Objectives: To describe a series of lymphogranuloma venereum (LGV) cases presenting as inguinal syndrome and/or genital ulceration, seen among men who have sex with men (MSM) in London, UK.

Methods: Collaborative retrospective case note review. Clinicians from three London Genitourinary medicine (GUM) clinics accessed by large populations of MSM within the current LGV outbreak collected clinical data from confirmed cases of LGV inguinal syndrome or genital ulcer. LGV was confirmed by the detection of LGV-specific DNA from specimens such as bubo aspirates, ulcer swabs, urethral swabs, first void urine and rectal biopsy material.

Results: There were thirteen cases detected overall: five cases of urethral LGV infection with inguinal adenopathy, three cases of genital ulcer with LGV inguinal adenopathy, three cases of isolated LGV-associated inguinal buboes, one case of a solitary LGV penile ulcer and another case with a penile ulcer and bubonulus. Only six of the thirteen were HIV-positive and all tested negative for hepatitis C. The majority of cases reported few sexual contacts in the three months preceding their diagnosis.

Conclusions: Clinical manifestations of LGV have not been confined to proctitis in the current MSM outbreak in the UK, and a small but significant number of inguinogenital cases of LGV have been observed. Epidemiologically many of the cases described seem to have occurred at the periphery of the current MSM LGV epidemic. Clinicians need to be vigilant for these less common presentations of LGV among MSM and specific diagnostic tests should be attempted from the relevant lesions.
Introduction

Lymphogranuloma venereum (LGV) is caused by Chlamydia trachomatis serovars L1-L3. These serovars display tropism for the lymphatic system, in contrast to serovars A-K that affect mucocutaneous tissue. Endemic in Africa, the Caribbean and parts of Asia, the classical presentation of LGV is inguinal lymphadenitis and abscess formation, sometimes preceded by anogenital ulceration. Since 2004 there has been a resurgence of LGV proctitis affecting predominantly HIV-positive men who have sex with men (MSM) in the United Kingdom, Western Europe and the USA. Detected cases have had high rates of co-infection with other STIs including hepatitis C. Anogenital ulceration and/or lymphadenitis have been reported rarely in the current MSM LGV epidemic.

We describe thirteen recent cases of LGV that presented with “classical” inguino-genital manifestations. LGV was detected, usually in the absence of other sexually transmitted infections (STIs), and symptoms resolved after treatment with long-course regimens of doxycycline or azithromycin.

Methods

Clinicians from three inner London GUM clinics servicing large populations of MSM collected clinical data from confirmed cases of LGV inguinal syndrome or genital ulcer. Case histories were anonymised and relevant data were collated and compared. Written informed consent was obtained from all cases that provided clinical images for this paper. As this was a retrospective anonymised case note review, ethics committee approval was not sought from the participating centres.
Initial tests for *C. trachomatis* and other STIs were performed at the respective hospital laboratories using commercially available assays performed according to the instructions of the manufacturer. All thirteen cases described had specimens confirmed as LGV using molecular tests performed at the Sexually Transmitted Bacteria Reference Laboratory (STBRL), Health Protection Agency, Colindale, UK.

DNA was extracted from clinical specimens (ulcer swabs, urethral swabs, first void urine) using the MagNA Pure total NA kit (Roche Diagnostics GmbH, D-68305 Mannheim, Germany), according to the manufacturer’s instructions. In instances where limited clinical specimen was available (bubo aspirates and rectal biopsies), manual DNA extractions were performed using the QIAamp Viral Mini Kit (Qiagen, UK). The Chlamydia status of all referred specimens was confirmed initially using a plasmid based real-time PCR method\(^6\). The presence of LGV DNA was then detected using an LGV-specific *pmpH* real-time PCR assay\(^7\).

**Results**

**Clinical records**

The characteristics of the thirteen cases are summarised in Table 1.

All were MSM with a median age of 36 years (range 21 to 61). Ten were of white ethnicity, only six were HIV-infected and none tested positive for hepatitis C antibodies. Twelve cases presented with unilateral or bilateral inguinal adenopathy, four of whom had concurrent genital ulceration, and one patient presented with penile ulceration only. Five of the twelve men with inguinal adenopathy appear to have had urethral infection as their primary LGV lesion and none of the five reported any preceding anogenital ulceration, though such symptoms might have been
transient and unnoticed. Six of the men reported only solitary sexual contacts in the previous 3 months, three of whom were their HIV-positive regular male partners.

**Notable features of cases**

Of note, Case 1 was first seen in December 2003 when he developed a tender right inguinal swelling. He reported regular unprotected insertive and receptive anal sex with anonymous male partners in London in the preceding months. On examination there was a single fluctuant 4cm by 5cm right inguinal swelling. Fine needle aspiration biopsy showed reactive lymphoid hyperplasia; Gram stain, microscopy and culture of the aspirate were negative for bacterial pathogens including mycobacteria. Oral amoxicillin 500mg tds and flucloxacillin 500mg qds were prescribed with no improvement in the mass, which ruptured spontaneously and discharged pus periodically for several months. The patient then began to pass red blood per rectum, developing tenesmus and weight loss. Colonoscopy in October 2004 showed an “unusual, chronic-looking fissure” in the anal canal and a purulent proctitis, biopsies of which showed acute and chronic inflammation with crypt abscesses and focal granuloma formation, consistent with LGV. A rectal swab specimen (ProbeTec, Becton Dickinson, Sparks, MD) tested positive for C. trachomatis by strand displacement amplification (SDA). The inguinal and rectal symptoms resolved completely after 3 weeks of doxycycline therapy. LGV-specific DNA was detected retrospectively from rectal biopsy material using techniques described previously.

Case 2 also had a complicated clinical course after first presenting in September 2005 with a week’s history of a 5mm tender ulcer over the right hemiscrotum and a
healing perianal ulcer. He was prescribed a course of aciclovir and flucloxacillin but a swab from the ulcer tested negative for both herpes simplex virus (HSV) and C. trachomatis (ulcer swab and urethra). The patient re-presented two weeks later with worsening of the ulcer that was now indurated with granulation tissue evident in the base (see Figure 1). This time an ulcer swab specimen tested positive for C. trachomatis by SDA, subsequently confirmed to be LGV. Five days after testing the patient was recalled and by this time had developed an enlarged firm right inguinal node and was thus commenced on doxycycline 100mg twice daily for presumed LGV. After ten days he had developed further lymphadenopathy above the inguinal ligament despite compliance with therapy. After 17 days of doxycycline, the swelling had progressed to a hot, fluctuant 4cm by 3 cm bubo, confirmed with ultrasound. Treatment was switched to azithromycin 1g daily but despite the change in antibiotic therapy the bubo continued to enlarge. Surgeons were reluctant to incise the lesion due to extensive overlying cellulitis but the abscess ruptured spontaneously 2 weeks after commencing azithromycin. A sample of fluid draining from the sinus also tested positive for C. trachomatis, and was later confirmed to be LGV. Symptoms improved on azithromycin, reduced to 500mg daily for the last 5 days. The lesions showed complete resolution after a total of five weeks therapy (18 days azithromycin and 17 days doxycycline) though some residual skin induration remained.

Case 4 had a 2-month history of a solitary, weeping, painless, indurated 0.7 cm ulcer in the dorsal coronal sulcus yet never developed inguinal lymphadenopathy.

Case 6 presented with bilateral 2cm by 3cm fluctuant lymph node abscesses that had ruptured spontaneously. The aspirated pus tested positive for C. trachomatis by SDA, confirmed to be LGV.
Case 7 presented with striking clinical signs of a left-sided 12cm by 6cm inguinal mass and a smaller 4cm by 2cm mass on the right. An ultrasound study arranged by his GP had shown multiple enlarged pathological-appearing lymph nodes bilaterally. A urethral swab specimen tested positive for *C. trachomatis* by chlamydial cell culture and Roche Cobas Amplicor PCR (Roche Diagnostics Systems, Branchburg, NJ) and extracts from both were confirmed to be LGV. The lesions resolved completely without rupture after a three week course of doxycycline.

Case 8 was admitted to hospital for investigation of bilateral inguinal lymphadenopathy with a differential diagnosis including lymphoma. His urine had tested positive for *C. trachomatis* by SDA four days before and he was reviewed in the GUM clinic where a diagnostic aspirate was performed from the non-fluctuant lymph node mass using 0.5ml normal saline (see Figure 2) and the blood-stained material obtained was deposited onto a ProbeTec female swab. *C. trachomatis* was detected using SDA, and was confirmed to be LGV, as was the DNA extract from his initial *Chlamydia*-positive urine specimen. His symptoms and lesions resolved after 3 weeks of doxycycline therapy.

Case 9 also had a diagnostic aspirate performed from non-fluctuant inguinal lymphadenopathy and was commenced on doxycycline therapy. He returned the following day after the mass had doubled in size to 8cm by 4cm but it was still non-fluctuant. Urgent ultrasound assessment showed a cluster of enlarged lymph nodes up to 3cm each in size with surrounding cellulitis and oedema but only a small 1.4cm by 0.5 cm abscess situated deeply and not amenable to drainage. Doxycycline therapy was continued for three weeks and the symptoms and mass resolved without further suppuration.
Case 11 presented with a non-tender, non-fluctuant lymph node swelling yet 1ml of frank pus was aspirated from the node and this tested positive for *C. trachomatis*, confirmed to be LGV (Figure 3).

Case 12 presented with a 3cm by 5cm right inguinal lymph node mass and asymptomatic LGV urethritis yet the non-purulent lymph node aspirate tested negative for *C. trachomatis* by SDA.

Case 13 presented with a penile ulcer and subsequently developed unilateral inguinal lymphadenopathy as well as a penile bubonulus, only the second case to be described in the recent MSM LGV epidemic. His new male partner was asymptomatic but had LGV detected from a rectal swab specimen.

**DISCUSSION**

The cases described in this series differ epidemiologically from the typical LGV proctitis cases seen thus far in the UK MSM epidemic. Less than half was HIV-positive compared to 74% of proctitis cases and other concurrent STIs were not detected. Half of the cases appear to have contracted LGV from relatively isolated episodes of sexual risk with far fewer recent sexual contacts than reported from most proctitis cases, particularly those seen early in the epidemic. Notably, most of the present cases reported no downstream sexual contacts following the onset of their symptoms and thus were unlikely to have transmitted LGV to subsequent partners.

The prompt symptomatology and management seen in cases 2 to 13 suggests that they are unlikely to have been a source of onward transmission of LGV. In case 13, the index case’s asymptomatic partner was diagnosed subsequently with rectal LGV, reminding us that asymptomatic LGV exists in the MSM population. This is in
accordance with findings from the Netherlands where 40% of men with LGV proctitis reported few complaints and/or had no physical abnormality\(^ {10} \). Nevertheless a recent UK case finding exercise failed to demonstrate a significant reservoir of asymptomatic infection to explain persistent transmission (Ward H, in press) and further work is needed to define true differences in LGV epidemiology between Dutch and British MSM.

Whilst Patient 1 was not diagnosed with LGV inguinal syndrome contemporaneously his inclusion in the present series serves to demonstrate the consequences of missed diagnosis. He presented in an era when LGV was not recognised as a prevalent pathogen in the UK MSM population and much morbidity and onward transmission could have been averted by early diagnosis and treatment. It is not possible to determine if his eventual LGV proctitis was due to progression of his inguinal syndrome or from newly-acquired anorectal infection.

Diagnosis of LGV is dependent on the detection of an LGV-associated serovar of *C. trachomatis* from the site of pathology, however serology may be helpful should this fail and the clinical suspicion is high. Only three of five cases in our series showed typical serological responses and more work is needed to assess the diagnostic value of serology in this population.

The LGV genital ulcers seen in this series showed non-specific clinical features and clinicians should consider obtaining suitable specimens for *C. trachomatis*/LGV when assessing anogenital ulceration in MSM. Swabs from ulcers and diagnostic aspirates of scant material from non-fluctuant inguinal lesions produced suitable specimens for the successful detection and typing of *C. trachomatis* using standard nucleic acid
amplification assays and we recommend this approach for investigation of
suspicious lesions.
Urethral *C. trachomatis* appears to have been the primary LGV lesion in five of the
present cases. Variability in symptomatology and urethral smear microscopy findings
in the present cases suggest that this might not simply represent LGV-associated
urethritis and that other primary endourethral lesions such as ulceration may occur.
We do not believe that routine LGV typing of *C. trachomatis*-positive urethral isolates
from MSM is currently indicated, based on the rarity of urethral LGV seen in the UK
case-finding exercise (Ward H, in press). Nevertheless, in the presence of additional
clinical signs such as ulceration or lymphadenopathy, or in LGV contacts, then
referral of *C. trachomatis*-positive specimens for detection of LGV serovars is
warranted.
The presence of persistent anogenital ulceration and severe proctitis can enhance
HIV11 and possibly hepatitis C transmission12. Although no new diagnoses were
made within the present case series, incident HIV and hepatitis C infection may not
have been detected by testing performed at the time of LGV diagnosis and follow-up
serology beyond the relevant window periods is indicated.
Treatment with doxycycline for three weeks achieved resolution of symptoms and
signs in eleven of twelve patients and azithromycin in two patients using multiple-
dose regimens. As demonstrated in Patient 2, diagnosis of LGV can be challenging
and patients with large buboes may require longer courses of treatment than the
recommended three weeks13. Adjuvant drainage of fluctuant abscesses may hasten
the resolution of such lesions and prevent spontaneous rupture and sinus formation.
There were no signs of tertiary sequelae such as chronic lymphoedema reported in any of the present cases though some local scarring was seen. The present cases demonstrate that clinical manifestations of LGV have not been confined to proctitis in the current MSM outbreak in the UK, despite the predominance of anorectal disease reported. Clinicians who see MSM patients should familiarise themselves with the clinical features and diagnostic pathways illustrated in this series. The striking clinical signs seen in some of these cases, especially in those who were systemically unwell, led to provisional diagnoses of incarcerated herniae and lymphoma. In addition there has been a recent report of two heterosexual LGV cases, further evidence that suggests the epidemic is spreading beyond its initial core group. Other relevant clinicians, including surgeons, microbiologists and histopathologists should be alerted to the current epidemiology of LGV in the UK, Europe and the US and it should be considered in the differential diagnoses of proctitis, anogenital ulceration and inguinal lymphadenopathy, particularly in MSM.
Acknowledgments

The authors would like to thank the laboratory staff at STH, MMC and C&W for their assistance in analysing specimens; HPA Bristol for performing the Chlamydial serology in case 4; Professor Cathy Ison for helpful comments on the manuscript.

Contributions

GS and JW conceived the paper, collected data on cases seen at STH, co-ordinated the multicentre collaboration and wrote the final draft; EA-J, JR, NTA and DH each contributed cases from their respective clinics and edited the manuscript; AE wrote the early first draft; SA was responsible for the molecular typing and verification of LGV results as well as editing the manuscript.

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Competing Interest: None declared.
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<th>Case number</th>
<th>Date &amp; Clinic</th>
<th>Ethnicity &amp; Age (years)</th>
<th>Clinical Presentation</th>
<th>Urethritis symptoms</th>
<th>HIV IgG Status</th>
<th>Number of sexual contacts last 3 months</th>
<th>Negative STI tests performed</th>
<th>LGV-specific PCR result</th>
<th>LGV serology</th>
<th>Treatment</th>
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<td>1* Dec 2003 STH</td>
<td>White British 54</td>
<td>Tender unilateral 4cm by 5cm inguinal lymphadenopathy, subsequent rupture bubo</td>
<td>Nil</td>
<td>Positive CD4 615 VL&lt;50</td>
<td>&gt;5</td>
<td>Urethral, rectal and pharyngeal <em>Neisseria gonorrhoeae</em> (GC) culture; <em>Ureaplasma urealyticum</em> (UU) and <em>Chlamydia trachomatis</em> (CT) SDA; RPR (past history of treated syphilis)</td>
<td>Positive (rectal biopsy specimen from subsequent LGV proctitis)</td>
<td>CGAb CFT negative (titre &lt;20 in Feb 2005)</td>
<td>Doxycycline 100 mg twice daily for 3 weeks</td>
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<tr>
<td>2 Sept 2005 C&amp;W</td>
<td>White British 56</td>
<td>0.5cm scrotal and perianal ulcers with subsequent unilateral inguinal lymphadenopathy and 4cm by 3 cm bubo formation</td>
<td>Nil</td>
<td>Positive CD4 523 VL&lt;50</td>
<td>1</td>
<td>Urethral swab and rectal swab GC/CT (SDA) Urethral, rectal, and pharyngeal GC culture</td>
<td>Positive (scrotal ulcer swab and swab of fluid from ruptured bubo)</td>
<td>Psittacosis/LGV CFT negative (titres 1:20 twice, 2 weeks apart)</td>
<td>Doxycycline 100 mg twice daily for 17 days then azithromycin 1g daily for 12 days then azithromycin 500mg daily for 5 days</td>
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<td>3 Jan 2006 C&amp;W</td>
<td>White British 46</td>
<td>Penile ulcer and unilateral inguinal lymphadenopathy</td>
<td>Nil</td>
<td>Positive CD4 470 VL&lt;50</td>
<td>10</td>
<td>Urethral swab and rectal swab GC/CT (SDA) Urethral, rectal and pharyngeal GC culture</td>
<td>Positive (penile ulcer swab)</td>
<td>Not done</td>
<td>Doxycycline 100 mg twice daily for 3 weeks</td>
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<tr>
<td>4 April 2006 STH</td>
<td>White British 61</td>
<td>0.7cm painless solitary penile ulcer. No lymphadenopathy</td>
<td>Nil</td>
<td>Negative</td>
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<td>Rectal and pharyngeal GC culture Syphilis IgM and RPR (past history of treated syphilis) HSV and <em>Treponema pallidum</em> PCR (in-house assay) from ulcer swab</td>
<td>Positive (penile ulcer swab)</td>
<td>Positive: CGAb titre 1:320 (STH); CGAb /LVG CFT titre 1:512 (HPA); CT WIF (L2 strain) titre 1:3000 (HPA, Bristol)</td>
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<td>5 Dec 2006 STH</td>
<td>White Irish 24</td>
<td>Bilateral inguinal buboes and preputial ulcer</td>
<td>Nil</td>
<td>Negative</td>
<td></td>
<td>Preputial ulcer swab darkground microscopy Syphilis IgM and RPR (past history of treated syphilis)</td>
<td>Positive (bubo aspirate)</td>
<td>Positive CGAb CFT (titres 1:320 and 1:1280 two weeks apart)</td>
<td>Doxycycline 100 mg twice daily for 3 weeks</td>
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<td>6 April 2007 STH</td>
<td>White British 34</td>
<td>Bilateral tender 2cm by 3cm ruptured buboes</td>
<td>Nil</td>
<td>Positive CD4 787 VL&lt;40</td>
<td>1</td>
<td>Rectal and pharyngeal GC culture</td>
<td>Positive (swab of bubo pus)</td>
<td>Not done</td>
<td>Doxycycline 100 mg twice daily for 3 weeks</td>
<td></td>
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<tr>
<td>7 July 2007 MMC</td>
<td>White other 36</td>
<td>Tender right 12cm by 6cm and left 4cm by 2cm inguinal lymphadenopathy</td>
<td>Nil</td>
<td>Negative</td>
<td></td>
<td>Rectal swab CT (PCR) Urethral, rectal and pharyngeal GC culture</td>
<td>Positive (urethral swab PCR and chlamydial culture)</td>
<td>Positive CGAb (1:400)</td>
<td>Doxycycline 100 mg twice daily for 3 weeks</td>
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<tr>
<td>8 Sept 2007 STH</td>
<td>Brazilian 21</td>
<td>Bilateral tender inguinal lymphadenopathy</td>
<td>Dysuria</td>
<td>Negative</td>
<td></td>
<td>Rectal swab GC/CT (SDA) Urethral, rectal, and pharyngeal GC culture</td>
<td>Positive (FU and lymph node aspirate)</td>
<td>Not done</td>
<td>Doxycycline 100 mg twice daily for 3 weeks</td>
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<td>Symptom Description</td>
<td>Staging</td>
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<td>Treatment</td>
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<td>9 Sept 2007 STH</td>
<td>British Indian</td>
<td>35</td>
<td>Tender 3cm by 2cm unilateral inguinal lymphadenopathy</td>
<td>Nil</td>
<td>Rectal swab GC/CT (SDA) Urethral, rectal, and pharyngeal GC culture Syphilis EIA</td>
<td>Positive (lymph node aspirate)</td>
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<td>Urethral smear 5-10 PMNLs/hpf *FVU specimen was C. trachomatis positive (SDA) but specimen was discarded prior to typing</td>
<td>Nil done</td>
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<td>White British</td>
<td>36</td>
<td>Tender 4cm by 4cm unilateral inguinal lymphadenopathy</td>
<td>Nil</td>
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<td>Positive (urethral swab)</td>
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<td>Nil done</td>
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<td>11 April 2008 STH</td>
<td>White Dutch</td>
<td>37</td>
<td>Non-tender 2cm by 3cm unilateral inguinal lymphadenopathy</td>
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<td>CD4 519 VL 35814</td>
<td>Doxycycline 100 mg twice daily for 3 weeks</td>
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<td>Positive (lymph node aspirate)</td>
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<td>12 May 2008 STH</td>
<td>Argentinian</td>
<td>35</td>
<td>Painful 5cm by 3cm unilateral inguinal lymphadenopathy</td>
<td>Nil</td>
<td>Urethral and pharyngeal GC culture Rectal swab and lymph node aspirate GC/CT (SDA)</td>
<td>Doxycycline 100 mg twice daily for 3 weeks</td>
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<td>13 July 2008 STH</td>
<td>White British</td>
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<td>Painful single 1cm penile subpreputial ulcer, subsequent tender unilateral inguinal lymphadenopathy and tender 1cm dorsal penile bubonulus formation with penile lymphoedema</td>
<td>Nil</td>
<td>Penile ulcer swab darkground microscopy and HSV PCR Syphilis EIA IgM and RPR (past history of treated syphilis)</td>
<td>Doxycycline 100 mg twice daily for 3 weeks</td>
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<td>FVU GC/CT (SDA) and rectal swab CT (SDA)</td>
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<td>*FVU CT (SDA) equivocal</td>
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<td>*Rectal swab GC (SDA) detected</td>
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Table 1: Characteristics of thirteen confirmed cases of lymphogranuloma venereum (LGV) presenting with inguinal syndrome and/or genital ulceration

**Abbreviations:** HIV, human immunodeficiency virus; STH, St Thomas’ Hospital; VL, HIV viral load; GC, Neisseria gonorrhoeae; CT, Chlamydia trachomatis; FVU, first void urine; SDA, strand displacement amplification; RPR, rapid plasma reagin; CGAb, Chlamydia Group Antibody; CFT, complement fixation test; C&W, Chelsea and Westminster; EIA, enzyme immunoassay; TPPA, Treponema pallidum particle agglutination; VDRL, Venereal Disease Research Laboratory; HSV, herpes simplex virus; PCR, polymerase chain reaction; WIF, whole immunofluorescence; MMC, Mortimer Market Centre; HPA, Health Protection Agency; PMNLs/hpf, polymorphonuclear leucocytes per high power field (x100).
*Case 1 also reported as part of case series in “Twelve men with proctitis: a case series of rectal biopsies from HIV-positive men diagnosed subsequently with lymphogranuloma venereum proctitis” – Soni S et al, submitted for publication, November 2008.
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**Figure 1.**
Clinical images from Case 2 showing a small tender indurated scrotal ulcer (inset) and early right-sided inguinal lymphadenopathy that progressed to 3cm by 4cm bubo formation prior to spontaneous rupture. LGV DNA was isolated from both ulcer swab and bubo pus.

**Figure 2.**
Clinical images from Case 8 showing massive right inguinal lymphadenopathy that was tender but non-fluctuant. Inset shows aspiration of the mass with 0.5ml normal saline using a lateral approach, which obtained some blood-stained fluid that tested positive for LGV DNA.

**Figure 3.**
Clinical images from Case 11 demonstrating right-sided non-tender, non-fluctuant lymphadenopathy. Despite this, 1ml of frank pus (see inset) was aspirated from the node and this tested positive for LGV DNA.
practice it would seem logical to prescribe PDE5i in order to produce good erections that MSM can be confident will be sustained even while putting on a condom. Unfortunately, the current data do not support this contention, rather showing an association between PDE5i usage and HIV prevalence. Recent quantitative and qualitative studies strongly suggest that the likelihood of MSM having unsafe sex and having HIV is significantly higher in those with current depression, fatigue, past and current sexual assault and regular users of recreational drugs to counteract the psychological sequelae of these psychosocial issues. Many of the recreational drugs used such as crystal meth and cocaine are profound peripheral vasodilators as well as cerebral stimulants. Hence the need for PDE5i to counteract the erectile dysfunction produced by them. Furthermore, there is suggestive evidence that the use of PDE5i itself can reverse low mood, cause aggression and amnesia and enhance sensation for the receptive partner at anal sex. Enhanced engorgement of penile and anal areas with PDE5i may themselves predispose to HIV transmission. Other factors associated with erectile dysfunction in MSM with HIV are the use of antiretroviral drugs, particularly protease inhibitors and accelerated penile arterial pathology.

Most of the studies showing an association between unsafe sex, HIV and PDE5i use in MSM show that these men do not acquire PDE5i from medical practitioners, but rather via the internet or other local contacts. With this in mind it would seem prudent for all MSM who attend for STI or HIV screening or therapy to be asked about erectile dysfunction at partnered sex, as well as other relevant clinical issues, as delineated above. Management should include the whole spectrum of the clinical disorders these men present with, including the prescribing of PDE5i medication, along with advice about appropriate condom and lubrication use and the minimalisation of recreational drug use and management of other psychosocial issues. Asking about these issues in busy STI/HIV clinics may not be easy, not least because of lack of time but also because of health practitioner and patient embarrassment in bringing up these issues. Some of the health issues related to PDE5i use, such as depression, are crucial to recognise, because they are associated with poor antiretroviral compliance.

In spite of these complex interactions between PDE5i and other conditions, MSM are ethically entitled to receive these medications when there is clinical necessity.

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REFERENCES

Correction

There was an error in an article published in the June issue of the journal (Sethi G, Allason-Jones E, Richens J, et al. Lymphogranuloma venereum presenting as genital ulceration and inguinal syndrome in men who have sex with men in London, UK. Sex Transm Infect 2009;85:165–70). Fig 1 was inserted above the legend for fig 2; fig 2 above the legend of fig 3 and fig 3 should have been placed with the legend from fig 1. The correct figs and legends are available online at http://sti.bmj.com/cgi/content/full/85/si/165/DC1. The journal apologises for this error.