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Travel-related sexually transmitted infections

Krzysztof Korzeniewski[1], Dariusz Juszczak[2]

[1]Head of Department of Epidemiology and Tropical Medicine, Military Institute of Medicine, Warsaw, Poland
[2]Commander of the 7th Navy Hospital, Gdansk, Poland

ABSTRACT

Sexually transmitted infections (STIs) are among the most common notifiable health problems worldwide, with particularly high rates in developing countries. Men and women with multiple sexual partners at home or a previous history of STIs are more likely to have casual sexual exposure (CSE) while travelling. Over the last several decades 5% to even 50% of short-term travellers engaged in CSE during foreign trips. It is estimated that only 50% of travellers use condoms during casual sex abroad. Sexual contact with commercial sex workers is an exceptionally high-risk behaviour. The common risk factor is also young age. Adolescents and young adults constitute 25% of the sexually active population, but represent almost 50% of all new acquired STIs. Many STIs are asymptomatic and therefore can be difficult to identify and control. The clinical manifestation of STIs can be grouped into a number of syndromes, such as genital ulcer or erosion, urethral or vaginal discharge, pelvic inflammatory disease. STIs are divided into curable infections caused by bacteria (gonorrhoea, chlamydiasis, syphilis, chancroid, lymphogranuloma venereum, granuloma inguinale) or protozoa (trichomoniasis) and incurable viral infections (genital herpes, genital warts, HIV). STIs are not only a cause of acute morbidity, but may result in complications including male and female infertility, ectopic pregnancy, cervical cancer, premature mortality or miscarriage. Monogamous sex with a stable, uninfected partner or sexual abstinence remains the only way to avoid the risk of becoming infected with STIs.

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Key words: sexually transmitted infections, travellers, epidemiology, clinical symptoms, prevention

INTRODUCTION

Travel is one of the essential risk factors in the spread of sexually transmitted infections (STIs) [1, 2]. This is the time when travellers may break social norms that normally restrict their sexual behaviour [3]. The most predictive factor is travelling without a steady partner. Men and women with multiple sexual partners at home or a previous history of STIs are more likely to have casual sexual exposure (CSE) while travelling [4]. Over the last decades, 5% to even 50% of short-term travellers engaged in CSE during foreign trips. The risk of developing STIs may be up to 3 times higher in people who experience casual travel sex, especially in travellers who take advantage of commercial sex in various destinations [3]. Commercial sex workers (CSWs) run a particularly high risk for an STI infection. Prevalence rates among CSWs are remarkably high in major centres around the world: Nairobi, 81%; Kinshasa, 35%; Bangkok, 44%; and Port au Prince, 69%. The median HIV prevalence rate in female prostitutes in Thailand is 15% [5]. Long-term overseas workers are more likely to engage in sexual activity while abroad than other types of travellers. The studies of Peace Corps volunteers and European expatriates showed that 13% to 60% reported at least one CSE while living overseas [6]. Seamen and military personnel are also reported to have high rates of sexual contact with overseas nationals, ranging from 45% to 56% [7, 8]. United Kingdom and Polish troops deployed in the tropics show similar rates of casual sex with local partners [9, 10]. In the last 30 years sex tourism has become more popular [11]. The contribution of commercial sexual relationships between tourists and hosts to a country's rise in STIs has been alerted in Thailand in the 1980s [12], and lately in Trinidad and Tobago in 2012 [13].

Krzysztof Korzeniewski, MD, PhD, Professor at Military Institute of Medicine, Department of Epidemiology and Tropical Medicine, ul. Grudzińskiego 4, 81–103 Gdynia, Poland, e-mail: kktropmed@wp.pl

Sex tourism is defined as an organised holiday for men whose main objective is sex with local women (men, children). Nowadays, sex tourism also applies to women looking for sex with local men (e.g. in eastern Africa). In his studies Bauer [14] characterised women travelling for sexual relationships dividing them into two groups: the older ones (lonely, disillusioned, not seen as attractive at home, bored in current home relationships, often retired, able to return frequently, seeking 'different' kind of attention, focused on relaxation, thrill, free from social constraints) and younger (adventurous, not seen as attractive at home, bored in current home relationships, focused on sex, alcohol, drugs, free from social constraints). de Graaf et al. [15] divided travellers of both sexes into four groups with regard to their CSE overseas: the 'unprepared' (who are surprised when sex happens), the 'fanatical' (who must have sex to have a successful vacation), the 'unaffected' (who feel that sex abroad is the same as sex at home), and the 'slightly accessible' (who feel that sex abroad is different and come prepared).

The risk of acquiring STIs by travellers depends on the number of sexual partners, the use of condoms, and the prevalence of STIs in other travellers and among the local population. It is estimated that only 50% of travellers use condoms during casual sex abroad [16]. Even if they use condoms, there may be greater risks of failure because of the poor quality of locally purchased products [17]. Latex condoms manufactured in the developed world are generally more effective against viral pathogens spread by semen, vaginal secretions, and blood than bacterial agents associated with lesions around the genitalia. The most common factors involved in condom breakage include inappropriate application, repeated or prolonged use, and anal intercourse [18]. The prevalence of STIs in the destination country is affected by the uneven distribution of STIs worldwide. Male sex, travelling to visit friends or relatives, not having a pre-travel consultation, and duration of travel less than 30 days were independently significantly associated with diagnosis of an STI in multivariate analysis [19]. Many international travellers incubating the STIs transmit their infections unknowingly to their sexual partners on returning home, while they are still asymptomatic. Hepatitis B, HIV and syphilis may also be transmitted by contaminated blood and blood products, contaminated syringes and needles used for injections, and potentially by unsterilised instruments used for acupuncture, piercing and tattooing [5]. The common risk factor of many STIs is young age. Adolescents and young adults (15–24 years old) make up 25% of the sexually active population, but represent almost 50% of all newly acquired STIs [20]. This may be explained by the fact that this age group is more likely to engage in high-risk sexual behaviour than the older population. Other groups known to engage in high-risk sexual behaviour (such as sex with multiple partners and unprotected sex) include CSWs, intravenous drug users, and prison inmates. Additional risk factors for STIs include lack of male circumcision, low socioeconomic status, poor hygiene, and the route of transmission [21]. There is clear evidence that circumcised men are at a significantly lower risk of acquiring HIV infection and other STIs [22]. The risk of acquiring HIV, HBV, or HCV from a percutaneous injury is, on average, 0.5%, 4–30%, and 3–10%, respectively [23]. The risks which may arise from a single, unprotected, heterosexual intercourse are thought to be much lower: 0.001% for HIV [24] and 0–0.6% for HCV [25]. The presence of genital lesions can dramatically increase the risk of acquiring HIV and other STIs [26]. A single episode of vaginal intercourse incurs a 20% to 50% risk of acquiring gonorrhoea [27]. The transmission efficiencies for open syphilis and chancroid lesions are probably at least as high. *Chlamydia trachomatis* is transmitted heterosexually with only slightly lower efficiency than gonorrhoea (0.8–8%/episode) [28].

EPIDEMIOLOGY OF TRAVEL-RELATED STIs

Worldwide, international tourist arrivals have increased from 278 million in 1980 to 1,133 million in 2014, potentially enhancing the interaction between travel and the spread of STIs [29]. According to the World Health Organisation (WHO) more than 1 million people acquire a sexually transmitted infection every day; more than 530 million people are infected with *Herpes simplex* virus which causes genital herpes (HSV-2); more than 290 million women have *Human Papillomavirus* (HPV), an etiological factor for genital warts. The majority of STIs are present without any symptoms; some STIs may increase the risk of HIV acquisition 3-fold or more; drug resistance, especially for gonorrhoea, is a major threat to reducing the impact of STIs worldwide [30]. Large population-based prevalence surveys undertaken in some African countries have confirmed high prevalence of STIs, even in rural populations. Syphilis was found in 5% to 10% of adults, trichomoniasis in 20% to 30% of women and 10% of men, and bacterial vaginosis in up to 50% of women [31]. The impact of HIV/AIDS has been catastrophic in many developing countries; more than 20% of adults are infected in some parts of Africa [32]. An estimated 500 million cases of the most common STIs (chlamydiasis, gonorrhoea, syphilis, trichomoniasis) occur in the world each year [30]. Many STIs are asymptomatic and therefore can be difficult to identify and control. Thus, the worldwide incidence of new STIs may be even higher. For example, it is estimated that actually reported cases represent only 50% to 80% of reportable STIs in the United States, reflecting limited screening and low disease reporting [20]. STIs are not only a cause of acute morbidity in adults, but may result in complications

including male and female infertility, ectopic pregnancy, cervical cancer, premature mortality or miscarriage [33].

The spectrum of STIs presumably acquired during travel has been found to be broad [34]. Some infections are more prevalent in developing countries (chancroid, lymphogranuloma venereum (LGV), granuloma inguinale) or in specific regions (treatment-resistant gonorrhoea and gonorrhoea with decreased susceptibility to cephalosporins in Asia), and may be imported into other countries by travellers returning from such destinations [35]. Matteelli et al. [36] described the range of diseases and the demographic and geographical factors associated with the acquisition of travel-related STIs through the analysis of the data gathered by the GeoSentinel travel medicine clinics worldwide in the period between June 1996 and November 2010. STIs were identified in three clinical settings: after travel, during travel, and immigration travel. In total, 112,180 ill travellers were seen. 974 (0.9%) patients had diagnoses of STIs, and 1,001 STIs were diagnosed. The proportionate STI morbidities were 6.6, 10.2, and 16.8 per 1,000 travellers in the three groups, respectively. The most common STI diagnoses were non-gonococcal or unspecified urethritis (30.2%), acute HIV infection (27.6%), and syphilis (22.2%) in patients seen after travel; non-gonococcal or unspecified urethritis (21.1%), epididymitis (15.2%), and cervicitis (12.3%) in patients seen during travel (travellers visited GeoSentinel clinics in Kathmandu, Singapore, Beijing, Hong Kong, Ho Chi Minh City, and Peekshill, United States); and syphilis in immigrant travellers (67.8%), who visited GeoSentinel clinics in western Europe, Canada, United States, and Australia. In ill travellers seen after travel, significant associations were noted between diagnosis of STIs and male sex, travelling to visit friends or relatives, travel duration of less than 1 month, and not having pre-travel health consultations. The high frequency of epididymitis, cervicitis, and acute pelvic inflammatory disease (PID) in travellers seen during travel suggests that STI-related acute complications in the urogenital tract might be an underestimated risk in travellers.

STIs can be divided into curable infections caused by bacteria (gonorrhoea, chlamydiasis, syphilis, chancroid, LGV, granuloma inguinale) or protozoa (trichomoniasis) and incurable viral infections (genital herpes, genital warts, HIV) that are often of lifelong duration [21, 32]. The epidemiology of the most common travel-related STIs (excluding HBV and HCV infections) is discussed below.

Gonorrhoea. Estimated prevalence rates for gonorrhoea range from 5.7% to 17% in sub-Saharan Africa and 0.1% to 3.5% in Asia and Western Pacific countries. Although the number of reported cases increased in some European countries during the 1990s, the overall incidence of gonorrhoea urethritis in the developed world remains at quite a low level (2.5–125 per 100,000) [37]. Gonorrhoea is more likely to be transmitted from asymptomatic carriers than from symptomatic ones. Over 62 million people become infected worldwide annually [38]. Centres for Disease Control and Prevention estimate that only 50% of gonorrhoeal infections are actually reported since the disease often presents with few or no symptoms. 50% of women contract gonorrhoea after a single exposure with infected males and 20% of men contract infection after a single sexual contact with infected females [39].

Chlamydiasis. *Chlamydia trachomatis* is responsible for more cases of STIs than any other bacterial pathogen. Since asymptomatic infection is common, it can easily be passed unknowingly between sexual partners. The WHO estimates that over 90 million new cases of chlamydia are diagnosed each year [40]. Worldwide, prevalence rates are similar to those reported in the United States, ranging between 5% and 15% [41, 42]. Prevalence rates for chlamydia infection in family planning clinics range from 1.9% to 12.2% in Latin America, 6% to 13% in African countries, and 5.7% to 26% in South/Southeast Asia [37]. The highest rates have been consistently found among young sexually active women, particularly adolescents [43]. Prevalence among females appears to be up to three or four times higher than in males. This may be due to anatomical factors, as the cervix of adolescent females is not sufficiently developed and is therefore particularly susceptible to STIs [44]. Worldwide, the greatest number of chlamydia infections were detected among individuals of the black Caribbean race [40]. High prevalence of chlamydia was also observed among female sex workers in Central Africa (38.3%). Chlamydia is associated with increased susceptibility to other STIs and transmission of HIV infection as well [45].

Syphilis. The prevalence of syphilis decreased to low levels in high-resource settings over the last decades, mainly as a consequence of an expanded coverage of health systems and the availability of effective diagnostic and treatment tools. In this scenario, individuals who acquire the infection during international travel have the potential to act as infectious foci and start epidemic outbreaks [46]. In most developed countries, syphilis incidence rates remained well below 5 per 100,000 through the 1990s. In contrast, there has been a striking increase in the reported rates of new syphilis cases during this period in Eastern Europe, in Russia and Ukraine (120–170 per 100,000). The reported seroprevalence of syphilis is generally higher in developing countries ranging from 3.5–8% in South/Southeast Asia and the Western Pacific, 5–6% in Latin America, and to 2.5–17% in sub-Saharan Africa [37].

Chancroid. This bacterial STI is endemic in many developing and resource-poor countries. Most infections are clinically apparent (few asymptomatic carriers). Chancroid is easily spread; it is estimated that the risk of transmission

from an infected to an uninfected person during a single sexual exposure is between 35% and 70%. Worldwide estimates range between 6 and 7 million new cases of illness each year. However, due to the lack of availability of diagnostic tests, these numbers are often approximate. The prevalence of chancroid in cases of genital ulcerative disease ranged between 23% and 56% in endemic areas of Asia, Africa, and the Caribbean. This compares to approx. 1% of the ulcers from STI clinics in European countries [47].

Lymphogranuloma venereum. The epidemiology of LGV is not well-defined owing to the lack of sensitive and specific diagnostic tests. The classical form of LGV is largely confined to the tropics, where in most places it accounts for only a small proportion of patients with STIs. The disease is seen more often in men than women. Since 2004, there has been a dramatic rise in LGV presenting with proctitis among homosexual HIV-positive males in Europe [48].

Granuloma inguinale. Prior to the antibiotic era, granuloma inguinale (donovanosis) was prevalent worldwide, nowadays, however, significant numbers are found only in a few developing countries, with the main foci being Papua New Guinea, Zimbabwe, parts of Republic of South Africa, India and Brazil, and among the Aboriginal population of Australia [49, 50].

Trichomoniasis. The WHO estimates an incidence of 170–190 million new cases of *Trichomonas vaginalis* infection worldwide each year. An estimated 7.4 million new cases of *T. vaginalis* infection are reported every year in the United States. Prevalence ranges between 2.2% for young women (< 20 years) compared with 6.1% in women > 25 years. Male prevalence is lower for both age categories (0.8% among men < 20 years and 2.8% in males > 25 years) [51]. Among pregnant women in tropical countries trichomoniasis prevalence rates ranged from 2.1% in Brazil [52] to 9.9% in the Central African Republic [53], and 41.4% in South Africa [54]. Trichomoniasis is frequently asymptomatic in men, or may cause a short-lived course of nongonococcal urethritis. Women infected with *T. vaginalis* may also be asymptomatic (up to 30% of cases), but the majority experience vaginitis [51].

Genital herpes. The prevalence of HSV-2 seropositivity varies widely with generally higher rates in developing countries (from 2% to 74% according to the country, age, gender, and urban vs. rural areas). In sub-Saharan Africa, 30–80% of women and 10–50% of men were found to be seropositive for HSV-2. Central and South America had rates from 20% to 40% [55]. A population-based study in rural Tanzania found that 50% of women and 25% of men were infected with HSV-2 by the age of 20 [56]. A very high prevalence of HSV-2 infection has been found in several other African countries [57]. The proportion of genital ulcers caused by HSV-2 has increased greatly in populations with a high HIV prevalence. In the United States, the overall prevalence of genital herpes is higher in women (25.6%) compared with men (17.8%) and in blacks (45.9%) compared to non-Hispanic whites (17.6%) [58].

Genital warts. At present, HPV infections are commonly diagnosed viral STI worldwide. It is estimated that around 290 million women are infected with HPV. Africa had the highest (31.6%), while Southeast Asia (6.2%) and southern Europe (6.8%) the lowest estimated HPV prevalence [59]. The prevalence of HPV infections is believed to be lower in men than in women. Circumcision is an independent protective factor in men. HPV is recognised as a causal agent in the pathogenesis of cervical cancer, the second most common malignancy in women, representing 9.8% of all female cancers [60].

HIV/AIDS. The WHO estimates that 70 million people have been infected with HIV since the start of the epidemic, 35 million of whom have died. 35.3 million people were living with HIV (two-thirds of them in sub-Saharan Africa), and 1.6 million people died of AIDS-related conditions in 2012 [61]. The highest prevalence of HIV is in Botswana, Lesotho, Swaziland, Zimbabwe and the Republic of South Africa. According to the UNAIDS statistics, the highest number of HIV infected is reported in Republic of South Africa (5.5 million people). The second highest prevalence of HIV infections is seen in Southeast and South Asia, India alone has 5.2 million inhabitants infected HIV. Currently, there has been a surge in newly reported HIV infections and AIDS cases in the post-Soviet countries. In Russia, HIV prevalence has been estimated at 2% of the country's population (UNAIDS estimates that there are 500,000 people living with HIV in the Moscow metropolitan area alone). The World Bank analyses show that there will have been approx. Five million HIV-infected individuals in Russia by 2020. The HIV prevalence in Ukraine is probably 1.5% of the country's population [62].

CLINICAL SYMPTOMS OF TRAVEL-RELATED STIs

The clinical manifestation of STIs can be grouped into a number of syndromes, such as genital ulcer or erosion, urethral or vaginal discharge, PID. However, many infections are asymptomatic in the early stages and the infection can be transmitted during this period without knowing it. STIs can lead to a variety of acute and chronic illnesses, such as infertility or ectopic pregnancy, genital ulcerations and mutilation, urethritis followed by urethral strictures, genital cancer caused by HPV, and premature death from HIV infection [63]. More than 20 different infectious agents can be acquired and/or spread by various types of sexual contact (e.g. vaginal sex, anal sex, oral-genital/oral-anal contact) [64]. The correct diagnosis of an STI is based on the clinical symptoms and suitably selected laboratory

tests. Examination of genital skin lesions (ulcer, erosion) should be supported by serologic tests for syphilis and a polymerase chain reaction (PCR) testing for genital herpes. If exposure occurred in areas where chancroid is common (Africa, Asia, Latin America), a test for *Haemophilus ducreyi* should be performed. Lymphadenopathy can accompany genital ulceration with chancroid infections, as well as with LGV and granuloma inguinale. If painful perianal or mucosal ulcers are detected, presumptive therapy should include a regimen for anogenital herpes. LGV should be suspected in a traveller with tender unilateral inguinal or femoral lymphadenopathy or proctocolitis. Testing specimens from the anatomic site of exposure with a PCR technique can detect *Chlamydia trachomatis* and *Neisseria gonorrhoea*. Anyone who is diagnosed with an STI should be screened for a HIV infection. The incubation period of STIs is important for the diagnostic purposes. For HIV, it is accepted practice to conduct a control antibody test 3 months after exposure, but some individuals take longer to seroconvert, and therefore a second test should be performed after 6 months. In the case of syphilis, it can take some time after disappearance of the primary chancre, which may go totally unnoticed, before the basic screening tests (VDRL) turn positive. Therefore, it is mandatory to conduct a control examination at least 3 months after exposure. To prevent transmission during the period before seroconversion, safe sexual practices, foremost through condom use, need to be proposed [65]. The clinical symptoms of the common travel-related STIs are presented below.

Gonorrhoea. In females, the illness is frequently asymptomatic, often misdiagnosed as bladder or vaginal infection. Males experience epididymitis, urethritis, and penile discharge [66]. Typically men develop symptoms after a 2–5-day incubation period, with 90% of symptomatic infections manifesting within 14 days. Asymptomatic infections in women (up to 80% of cases) are detected in contacts of symptomatic partners. Symptomatic uncomplicated infections in males typically manifest as a thick, yellow urethral discharge. In symptomatic females, vaginal discharge or dysuria are the major symptoms. Accompanying symptoms include a variable degree of meatal itching, burning, dysuria, pollakiuria. Infections of the pharynx and rectum (mostly asymptomatic) can result from orogenital and genito-anal sexual contact. In females the rectum is easily infected by contamination from an infected vaginal discharge, common are also local infections of the Skene's and Bartholin's glands. Further spread of infection in women may lead to acute salpingitis, abscess formation or long-term problems of chronic PID, and increased risk of ectopic pregnancy [67].

Chlamydiasis. The incubation period of infection may range from days to months (usually 1–3 weeks). In males, chlamydial infection causes urethritis and, in a proportion of cases, epididymo-orchitis. In females, chlamydial cervicitis is often asymptomatic. Sometimes patients will complain of vaginal discharge, and the finding of a mucopurulent discharge at the cervical os is suggestive of chlamydial or gonococcal cervicitis. Ascending infection of the female genital tract may lead to endometritis, salpingitis or PID. Because the symptoms of chlamydial PID are often mild, patients may present only when the sequelae of irreversible damage to the Fallopian tubes, leading to infertility, ectopic pregnancy, become apparent [67].

Syphilis. After an incubation period of 10–70 days (range 3–90 days), a primary chancre develops at the site of inoculation (primary syphilis). The chancre is typically painless, indurated, with a clean base and a raised edge, and does not bleed on contact. There is usually only a single lesion; in males it is most commonly on the glans, the foreskin, the coronal sulcus or the shaft of the penis, and in the female on the cervix or vulva. The primary chancre is often accompanied by inguinal lymphadenopathy; the glands are characteristically hard and painless. Secondary syphilis presents with an array of dermatological lesions and eruptions that can occur 4–10 weeks after exposure. Other symptoms include fever, meningismus, myalgias, weight loss, anorexia, hair loss, arthralgias, mucous patches, and condylomata lata. The tertiary stage involves other organ systems and may lead to devastating cardiovascular and neurological complications [68]. Notable variations in the clinical expression of syphilis have been described in patients co-infected with HIV. These include multiple primary chancres, overlap of primary and secondary phases and early development of gummatous lesions [67].

Chancroid. After an incubation period of 3–7 days (range 1–35 days), a papule appears at the site of inoculation which soon ulcerates. The typical ulcer of chancroid is painful and soft, has a purulent base with an undermined edge, and bleeds on contact. Multiple ulcers are commonly present, and there is painful inguinal lymphadenopathy in 50% of cases, often unilateral. Atypical presentations are common, which makes chancroid difficult to reliably distinguish from primary syphilis on clinical grounds. Chancroid may cause extensive local destruction, particularly in HIV-infected individuals who may fail to respond to antimicrobial treatment [67].

Lymphogranuloma venereum. The initial manifestation, occurring 10–14 days (range 3–21 days) after exposure, is typically a small, painless, usually herpetiform ulcer of the genitalia, which may pass unrecognised and resolve spontaneously. It is thought likely that some patients develop asymptomatic infections of the urethra and cervix. The second phase of the illness is the development of increasingly painful lymphangitis and lymphadenitis, accompanied by fever and malaise. The infected nodes (bilateral in a third

of cases) coalesce into a matted mass which may project outwards below or above the inguinal ligament to give the classical 'groove sign'. The nodes are liable to rupture, forming multiple sinuses. Untreated, the disease may cause extensive lymphatic damage, resulting in elephantiasis of the genitalia. In women and homosexual men, the disease may present as an acute proctocolitis, which leads to abscess formation, fibrosis, fistula and rectal stricture [67].

Granuloma inguinale. The first manifestation, appearing after a 3–40-day incubation period, is usually a small papule, which ruptures to form a granulomatous, painless lesion, bleeding readily on contact and often elevated above the level of the surrounding skin. The lesion has to be differentiated from other forms of genital ulcer. Untreated, the ulcers slowly extend, particularly along skin-folds towards the groins and anus. Special features are extragenital lesions (mostly neck and mouth); cervical lesions (resembling carcinoma or tuberculous cervicitis); involvement of uterus; tubes and ovaries (hard masses, abscesses, 'frozen pelvis', hydronephrosis) [67]. Granuloma inguinale usually affects the genital (90% of cases) or inguinal region (10%). There are four classic presentations of the disease: ulcerogranulomatous, hypertrophic or verrucous ulcers, necrotic deep ulcers, and cicatricial lesions [69].

Trichomoniasis. Incubation period of infection lasts 5–28 days. Up to 75% of infected women complain of vaginal, profuse yellow-green frothy discharge. Pruritus vulvae, dyspareunia and dysuria are also common symptoms. The vulva and vaginal walls may be excoriated and erythematous in severe cases, and punctate haemorrhages may be seen on the cervix. In men yellow urethral discharge is observed [67].

Genital herpes. The clinical picture is highly characteristic (in the absence of immune suppression), with its localised clusters of vesicles, which break down to form ulcers, crust over and then resolve. Sites of involvement include the external genitalia, neighbouring skin, the urethra and cervix, pharynx and rectum. Tender lymphadenopathy may occur. The complications of genital herpes include a sacral radiculomyelopathy which may manifest with constipation and retention of urine as well as shooting pains down the legs. Other complications include aseptic meningitis, extragenital lesions and disseminated herpes. In pregnant women, recurrences and dissemination are more frequent and premature delivery may complicate primary attacks. Severe and intractable ulceration due to HSV-2 occurs in patients immunosuppressed by HIV [67].

Genital warts. The genital, painless lesions induced by HPV vary from the soft, fleshy, vascular condylomata acuminata to papular warts. In pregnancy, in immunosuppressed patients and in the presence of genital discharge, there is a tendency for warts to grow rapidly [67]. Viral transmission may occur even in the absence of visible lesions and many patients may be unaware that they are infected. The infection may be obvious clinically or completely asymptomatic [70].

HIV/AIDS. The most common routes of HIV transmission include (an estimated number of infections per 10,000 exposures is given inside the brackets): blood and blood products transfusion (9,000); the use of contaminated needles or syringes (67); direct contact with contaminated blood in work settings, e.g. a health care worker pricked by a needle (30); transplacental mother-to-child transmission (2,500); sexual intercourse — homosexual, bisexual or heterosexual sexual contacts (without the use of condoms): receptive anal intercourse (50), insertive anal intercourse (6.5), vaginal intercourse — a female (10), vaginal intercourse — a male (5), oral sex — the performing person (1), oral sex — the receptive person (0.5); other routes of transmission: infected human milk (breastfeeding), artificial insemination (semen from infected men), organ transplants. HIV infection may first present with flu-like symptoms several weeks after exposure. This is followed by an asymptomatic period ranging between several and nearly 20 years depending on the immune response of an infected individual. As the impairment progresses, CD4 lymphocyte levels are reduced while HIV viruses multiply and eventually the disease becomes fully symptomatic. In 1992, Centres for Disease Control and Prevention classified symptoms associated with HIV/AIDS (the classification has been valid since 1993). The classification system involves both clinical (A, B and C) as well as immunological criteria. In Europe the diagnostic criteria for identifying AIDS include only conditions listed in the clinical category C (stages C1, C2, C3), i.e. the occurrence of infections indicative of AIDS. In the Unites States, AIDS is also identified based on the immune-response criteria (CD4 + lymphocyte count < 200/mm^3) regardless of clinical symptoms [62]. The opportunistic infections in AIDS-infected people include bacterial, protozoal, fungal, and viral diseases in addition to HIV-associated malignancies. Persistent generalised lymphadenopathy occurs in Stage C1. Weight loss, respiratory tract infections, herpes zoster, oral ulcerations, and pruritic eruptions are evident in Stage C2. Severe weight loss, chronic diarrhoea, persistent fever, oral candidiasis, tuberculosis, and oral hairy leucoplakia occur in Stage C3. Pneumonia, empyema, meningitis, acute necrotising stomatitis, and bacteraemia are also characteristic in this stage. The progression from HIV to AIDS is associated with chronic herpes simplex infection, Kaposi sarcoma, central nervous system toxoplasmosis, disseminated mycosis, recurrent septicaemia, and nephropathy [71]. The fungal infection *Pneumocystis jiroveci* that causes pneumonia is the most prevalent opportunistic infection in persons with AIDS and the leading cause of death [72].

SUMMARY

In the last decades, population mobility has increased dramatically. More than 1 billion people crossed international borders for work, study, or pleasure. Most of mobile population are sexually active people who are at increased risk of acquiring STIs [73]. The anonymity of travel, the sense of isolation brought on by unfamiliar surroundings, and the desire for unique experiences may encourage sexual activity [74]. STIs are among the most common notifiable infections worldwide, and rates are particularly high in developing countries. Surveys of CSW in the developing world reveal high rates of the curable, bacterial STIs: 13–32% for chlamydia, 11–45% for gonorrhoea, and 5–55% for syphilis [75]. When prevalence rates for the non-curable viral infections are included (HIV, HSV, HPV), the risk of exposure to an STI through sexual contact with a CSW is much higher. It is certainly worth noting that the presence of one or more STIs can increase the risk of HIV transmission by 3–10 times [37]. Prevention is the best way to decrease the morbidity and mortality associated with STIs. Consistent and proper use of latex condoms during sexual activity has a protective efficacy of 40–70% against STIs and should be strongly advised by doctors in travel clinics [63]. Male travellers should always use a condom during sexual intercourse, each time, from start to finish, and female travellers should make sure that their partners use one [3]. Some spermicides, such as nonoxynol-9, which interfere with sperm viability and have shown in vitro inhibitory activity against *N. gonorrhoeae*, HSV and HIV in STIs prevention can be useful [63]. It is necessary to remember, that barrier contraceptives can provide a protection against many STIs, but 100% protection cannot be achieved even when they are carefully used. Only HBV and HPV genotypes (hepatitis B and genital warts) can currently be prevented by vaccination [76]. Rates of reported CSE during travel vary between 5% and 51% [3]. Recent studies demonstrated that male and female travellers are quite similar in their willingness to acquire new partners while abroad. However, both quantitative and qualitative behavioural differences persist between the sexes (e.g. number of partners, willingness to pay for sex, consistent condom use, and partner choice) [77]. The common risk factors for engaging in casual sex while travelling include young age, travelling alone or with the same-sex group, a history of casual sex or multiple partners at home, repeated visits to the same region, history of previous STIs, higher social status, longer duration of stay, usage of alcohol or illicit drugs [78]. The effects of sun, stimulants, and sex has been described as 'situational disinhibition' [79]. Screening for STIs is a recommended procedure for travellers who report CSE while abroad [80]. Such screening should be guided by the nature of the sexual contact and current or past symptoms and could include an examination of the genitals, a cervical/urethral/anal/pharyngeal swab and/or urine testing, and serologic tests for syphilis, HIV, and possibly HBV and HCV [4]. Monogamous sex with a stable, uninfected partner or sexual abstinence remains the only way to avoid the risk of becoming infected with STIs.

REFERENCES

1. Keystone JS, Freedman DO, Kozarsky PE, Connor BA, Nothdurft HD (eds.). Travel Medicine. 3rd Ed. Elsevier Saunders 2013, pp. 499–500.
2. Monsel G, Caumes E. Dermatologic presentations of Tropical Diseases in Travelers. Curr Infect Dis Rep 2010; 12: 186–191.
3. Matteelli A, Carosi G. Sexually transmitted diseases in travelers. Clin Infect Dis 2001; 32: 1063–1067.
4. Moore J, Beeker C, Harrison JS, Eng TR, Doll LS. HIV risk behavior among Peace Corps volunteers. AIDS 1995; 9: 795–799.
5. Committee to Advise on Tropical Medicine and Travel. Statement on travellers and sexually transmitted infections. Canada Communicable Disease Report 2006; 32: 1–24.
6. Memish ZA, Osoba AO. International travel and sexually transmitted diseases. Travel Med Infect Dis 2006; 4: 86–93.
7. Miller N, Yeager R. By virtue of their occupation, soldiers and sailors are at greater risk. Special report: The military. AIDS Anal Afr 1995; 5: 8–9.
8. Hopperus Buma AP, Veltink RL, van Ameijden EJ, Tendeloo CH, Coutinho RA. Sexual behavior and sexually transmitted diseases in Dutch marines and naval personnel on a United Nations mission in Cambodia. Genitourin Med 1995; 71: 172–175.
9. Adams EJ, Strike PW, Green AD, Masterton RG. Sexually transmitted diseases in transient British forces in the tropics. Genitourin Med 1994; 70 : 94–96.
10. Korzeniewski K, Olszański R. The incidence of sexually transmitted diseases among Polish soldiers serving in U.N. military forces in the Middle East and South-East Asia. Przedl Epidemiol 2006; 60(2): 359–366.
11. Vivancos R, Abubakar I, Hunter PR. Foreign travel, casual sex, and sexually transmitted infections: systematic review and metanalysis. Int J Infect Dis 2010; 14: e842–e851.
12. Cohen E. Tourism and AIDS in Thailand. Ann Tourism Res 1998; 15: 467–486.
13. George A, Richards D. A paradigm shift in the relationship between tourist and their hosts: the impact on the HIV/AIDS epidemic in Trinidad and Tobago. J Human Resour Hosp Tour 2012; 11: 197e209.
14. Bauer IL. Romance tourism or female sex tourism? Trav Med Infect Dis 2013; 12: 20–28.
15. de Graaf R, van Zessen G, Houweling H. Underlying reasons for sexual conduct and condom use among expatriates posted in AIDS endemic areas. AIDS Care 1998; 10: 651–665.
16. Croughs M, Van Gompel A, de Boer E, Van Den Ende J. Sexual risk behavior of travelers who consulted a pretravel clinic. J Travel Med 2008; 15: 6–12.
17. Kamenga MC, De Cock KM, St Louis ME et al. The impact of human immunodeficiency virus infection on pelvic inflammatory disease: a case-control study in Abidjan, Ivory Coast. Am J Obstet Gynecol 1995; 172: 919–925.
18. Holmes KK, Levine R, Weaver M. Effectiveness of condoms in preventing sexually transmitted infections. Bull World Health Organ 2004; 82: 454–461.
19. Gerbase AC, Rowley JT, Mertens TE. Global epidemiology of sexually transmitted diseases. Lancet 1998; 351 (suppl. 3): 2–4.
20. Da Ros CT, Schmitt S. Global epidemiology of sexually transmitted diseases. Asian J Androl 2008; 10: 110–114.

21. Gewirtzman A, Bobrick L, Conner K, Tyring SK. Epidemiology of sexually transmitted infections. In: Gross G, Tyring SK (eds.). Sexually transmitted infections and sexually transmitted diseases. Springer -Verlag, Berlin Heidelberg 2011, pp. 13–34.
22. Siegfried N, Muller M, Volmink J et al. Male circumcision for prevention of heterosexual acquisition of HIV in men. Cochrane Database Syst Rev 2003: CD003362.
23. Puro V, De Carli G, Scognamiglio P, Porcasi R, Ippolito G; Studio Italiano Rischio Occupazionale HIV. Risk of HIV and other blood-borne infections in the cardiac setting: patient-to-provider and provider -to-patient transmission. Ann NY Acad Sci 2001; 946: 291–309.
24. Pinkerton SD, Martin JN, Roland ME, Katz MH, Coates TJ, Kahn JO. Cost-effectiveness of postexposure prophylaxis after sexual or injection-drug exposure to human immunodeficiency virus. Arch Intern Med 2004; 164: 46–54.
25. Terrault NA. Sexual activity as a risk factor for hepatitis C. Hepatology 2002; 36 (5 Suppl. 1): S99–S105.
26. Macaluso M, Lawson ML, Hortin G et al. Efficacy of the female condom as a barrier to semen during intercourse. Am J Epidemiol 2003; 157: 289–297.
27. Sparling PF, Handsfield HH. Neisseria gonorrheae. In: Mandel G, Bennet JE, Dolin R (eds.). Principles and practice of infectious diseases. Churchill Livingstone, Philadelphia 2000; pp. 2242–2258.
28. Warner L, Newman DR, Austin HD et al.; Project RESPECT Study Group. Condom effectiveness for reducing transmission of gonorrhea and chlamydia: The importance of assessing partner infection status. Am J Epidemiol 2004; 159: 242–251.
29. United Nations World Tourism Organization. UNWTO Tourism Highlights 2015. Available at: www.unwto.org/annualreports. Accessed: 20 Aug 2015.
30. World Health Organization. Sexually transmitted infections. Available at: http://www.who.int/topics/sexually_transmitted_infections/en/. Accessed: 12 Oct 2015.
31. Orroth KK, Korenromp EL, White RG et al. Comparison of STD prevalences in the Mwanza, Rakai, and Masaka trial populations: the role of selection bias and diagnostic errors. Sex Transm Infect 2003; 79: 98–105.
32. Mabey D. Epidemiology of sexually transmitted infections: worldwide. Medicine 2014; 42: 287–290.
33. WHO/UNAIDS. Sexually transmitted diseases: policies and principles for prevention and care. Available at: www.who.int/hiv/pub/sti/en/prev_care_en.pdf. Accessed: 12 Oct 2015.
34. Ansart S, Hochedez P, Perez L, Bricaire F, Caumes E. Sexually transmitted diseases diagnosed among travelers returning from the tropics. J Travel Med 2009; 16: 79–83.
35. Unemo M, Nicholas RA. Emergence of multidrug-resistant, extensively drug-resistant and untreatable gonorrhea. Future Microbiol 2012; 7: 1401–1422.
36. Matteelli A, Schlagenhauf P, Carvalho AC et al.; for the GeoSentinel Surveillance Network. Travel-associated sexually transmitted infections: an observational cross-sectional study of the GeoSentinel surveillance database. Lancet Infect Dis 2013; 13: 205–213.
37. World Health Organization. Global prevalence and incidence of selected curable sexually transmitted infections: overview and estimates. WHO/CDS/CSR/EDC/2001.10. Geneva 2001; 1–54.
38. De Schryver A, Meheus A. Epidemiology of sexually transmitted diseases: the global picture. WHO Bull OMS 1990; 68: 639–653.
39. Weinstock H, Berman S, Cates W. Sexually transmitted diseases among American youth: incidence and prevalence estimates, 2000. Perspect Sex Reprod Health 2004; 36: 6–10.
40. Kucinskiene V, Sutaite I, Valiukeviciene S, Milasauskiene Z, Domeika M. Prevalence and risk factors of genital Chlamydia trachomatis infection. Medicina (Kaunas) 2006; 42: 885–894.
41. Buve A, Weiss HA, Laga M et al. The epidemiology of gonorrhoea, chlamydial infection and syphilis in four African cities. AIDS 2001; 15 (Suppl. 4): S79–S88.
42. Williams H, Tabrizi SN, Lee W, Kovacs GT, Garland S. Adolescence and other risk factors for Chlamydia trachomatis genitourinary infection in women in Melbourne, Australia. Sex Transm Infect 2003; 79: 31–34.
43. Wilson JS, Honey E, Templeton A, Paavonen J, Mardh PA, Stray-Pedersen B. A systematic review of the prevalence of Chlamydia trachomatis among European women. Hum Reprod Update 2002; 8: 385–394.
44. Sedlecki, K, Markovic M, Rajic G. Risk factors for Chlamydia infections of the genital organs in adolescent females. Srp Arh Celok Lek 2001; 129: 169–174.
45. Eron JJ, Gilliam B, Fiscus S, Dyer J, Cohen MS. HIV-1 shedding and chlamydial urethritis. JAMA 1996; 275: 36.
46. Matteelli A, Odolini S. Travel, syphilis and HIV. Travel Med Infect Dis 2014; 12: 5–6.
47. Al-Tawfiq JA, Spinola SM. Haemophilus ducreyi: clinical disease and pathogenesis. Curr Opin Infect Dis 2002; 15: 43–47.
48. Van der Bij AK, Spaargaren J, Morré SA et al. Diagnostic and clinical implications of anorectal lymphogranuloma venereum in men who have sex with men: a retrospective case-control study. Clin Infect Dis 2006; 42: 186–194.
49. O'Farrell N. Donovanosis. Sex Transm Infect 2002; 78: 452–457.
50. Bowden FJ. Donovanosis in Australia: going, going. Sex Transm Infect 2005; 81: 365–366.
51. Johnston VJ, Mabey DC. Global epidemiology and control of Trichomonas vaginalis. Curr Opin Infect Dis 2008; 21: 56–64.
52. Simoes JA, Giraldo PC, Ribeiro Filho AD et al. Prevalencia e fatores de risco associados as infeccoes cervico-vaginais durante a gestacao. Rev Bras Ginecol Obstet 1996; 18: 459–467.
53. Blankhart D, Muller O, Gresenguet G, Weis P. Sexually transmitted infections in young pregnant women in Bangui, Central African Republic. Int J STD AIDS 1999; 10: 609–614.
54. Sturm AW, Wilkinson D, Ndovela N, Bowen S, Connolly C. Pregnant women as a reservoir of undetected sexually transmitted diseases in rural South Africa: implications for disease control. Am J Publ Health 1998; 88: 1243–1245.
55. Weiss H. Epidemiology of herpes simplex virus type 2 infection in the developing world. Herpes 2004; 11 (Suppl. 1): 24A–35A.
56. Obasi A, Mosha F, Quigley M et al. Antibody to herpes simplex virus type 2 as a marker of sexual risk behaviour in rural Tanzania. J Infect Dis 1999; 179: 16–24.
57. Weiss HA, Buve A, Robinson NJ et al. The epidemiology of HSV-2 infection and its association with HIV infection in four urban African populations. AIDS 2001; 15 (Suppl. 4): S97–S108.
58. Malkin JE. Epidemiology of genital herpes simplex virus infection in developed countries. Herpes 2004; 11 (Suppl. 1): 2A–23A.
59. de Sanjose S, Diaz M, Castellsague X et al. Worldwide prevalence and genotype distribution of cervical human papillomavirus DNA in women with normal cytology: a meta-analysis. Lancet Infect Dis 2007; 7: 453–459.
60. Parkin DM, Ferlay J, Bray F, Pisani P. Global cancer statistics, 2002. CA Cancer J Clin 2005; 55: 74–108.
61. Joint United Nations Programme on HIV/AIDS. Global report: UNAIDS report on the global AIDS epidemic 2013. Available at: http://

www.unaids.org/en/media/unaids/ contentassets/documents/epidemiology/2013/gr2013/UNAIDS_Global_Report_2013_en.pdf. Accessed: 20 Aug 2015.
62. Korzeniewski K. Travel medicine. Department of Epidemiology and Tropical Medicine. Military Institute of Medicine. Warsaw 2014; pp.206–215.
63. Memish ZA, Osoba AO. Sexually transmitted diseases and travel. Int J Antimicrob Agents 2003; 21: 132–134.
64. Brunham RC, Plummer FA. A general model of sexually transmitted disease epidemiology and its implications for control. Med Clin North Am 1990; 74: 1339–1352.
65. Kidd SE, Workowski K. Sexually transmitted diseases. In: Brunette GW, Kozarsky PE, et al. (eds.). CDC Health Information for International Travel. The Yellow Book 2016. Centers for Disease Control and Prevention. Oxford University Press, New York 2015, pp. 308–310.
66. Fox KK, Whittington WL, Levine WC, Moran JS, Zaidi AA, Nakashima AK. Gonorrhea in the United States, 1981–1996. Demographic and geographic trends. Sex Transm Dis 1998; 25, 386–393.
67. Richens J, Mayaud P, Mabey D. Sexually Transmitted Infections (Exluding HIV). In: Farrar J. Manson's Tropical Diseases. 23rd Ed. Elsevier: London, 2013; pp. 292–318.
68. Domantay-Apostol GP, Handog EB, Gabriel MT. Syphilis: the international challenge of the great imitator. Dermatol Clin 2008; 26: 191–202.
69. O'Farrell N. Donovanosis. Sex Transm Infect 2002; 78: 452–457.
70. Fazel N, Wilczynski S, Lowe L, Su LD. Clinical, histopathologic, and molecular aspects of cutaneous human papillomavirus infections. Dermatol Clin 1999; 17: 521–536.
71. Espinoza L, Hall HI, Hardnett F, Selik RM, Ling Q, Lee LM. Characteristics of persons with heterosexually acquired HIV infection, United States 1999–2004. Am J Publ Health 2007; 97: 144–149.
72. Over M, Piot P. Human immunodeficiency virus infection and other sexually transmitted diseases in developing countries: public health importance and priorities for resource allocation. J Infect Dis 1996; 174 (Suppl. 2): S162–S175.
73. Aral SO. Sexually transmitted diseases: Magnitude, determinants and consequences. Int J STI AIDS 2001; 12: 211–215.
74. Msuya W, Mayaud P, Mkanje R, Grosskurth H. Taking early action in emergencies to reduce the spread of STIs and HIV. Afr Health 1996; 18: 24.
75. Wang CC, Celum CL. Global risk of sexually transmitted diseases. Med Clin North Am 1999; 83: 975–995.
76. Ward BJ, Plourde P. Travel and Sexually Transmitted infections. J Travel Med 2006; 13: 300–317.
77. Bellis MA, Hughes K, Thomson R, Bennett A. Sexual behaviour of young people in international tourist resorts. Sex Transm Infect 2004; 80: 43–47.
78. Hawkes S, Hart GJ, Bletsoe E, Shergold C, Johnson AM. Risk behaviour and STI acquisition in genitourinary clinic attenders who have travelled. Genitourin Med 1995; 71: 351–354.
79. Apostolopoulos Y, Sonmez S, Yu CH. HIV-risk behaviours of American spring break vacationers: A case of situational disinhibition? Int J STI AIDS 2002; 13: 733–743.
80. Kemper C. Consider postexposure STI screening. AIDS Alert 2002; 17: 22.