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Case Report

Disseminated Gonococcal Infection: An emerging challenge: A case report

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Abstract

Globally, gonorrhoea is the second commonest bacterial sexually transmitted infection. In 2023, reported gonorrhoeae cases in Sri Lanka have increased accounting for 10% from total reported STDs. Delayed diagnosis or inadequate treatment may cause systemic complications like disseminated gonococcal infections (DGI). Though known DGI prevalence is <1%, per estimations it is 0.5% - 3% among untreated cases. We report a 28-year-old unmarried male, initially treated for smear positive, culture negative urethral gonorrhoea with oral cefixime and azithromycin, presented one week after treatment with atypical skin lesions. This case highlights diagnostic and therapeutic challenges in the current context of rising gonococcal incidence and emerging antibiotic resistance. Atypical presentation can delay the diagnosis and treatment. Improved awareness among clinicians, improving advanced diagnostic facilities availability, effective partner notification and national surveillance are the key to prevent emerging threat of DGI.

Key words: *Neisseriae gonorrhoeae*, pathogenesis, Disseminated gonococcal infection, atypical presentation, arthritis-dermatitis syndrome

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Introduction

Neisseria gonorrhoeae, an obligate human pathogen and gram negative diplococcus causes gonorrhoeae.(1) It is the world's second commonest bacterial sexually transmitted infection (STI). The world health organization (WHO), estimated 82.4 million new gonorrhoeae cases in 2020 among individuals aged 15 – 49 years.(2) In Sri Lanka, reported gonorrhoeae cases number rose from 754 in 2022 to 1128 in 2023, with predominant contribution by men over 25 years.(3)

Typically, *N.gonorrhoeae* causes localized infections at exposure site, uro-genital, rectal, oro-pharyngeal or ocular. Delayed diagnosis or inadequate treatment in infected men produce local complications like tysonitis, cowperitis, epididymitis, prostatitis, penile lymphangitis and systemic complications like disseminated gonococcal infections (DGI), gonococcal endocarditis or meningitis.(1)

Though DGI is the commonest systemic complication in gonorrhoeae, it remains rare with <1% prevalence among gonorrhoeae cases.

Per estimations, 0.5% - 3% untreated gonococcal infections progress to DGI. It, also known as acute arthritis-dermatitis syndrome is characterized by arthritis, tenosynovitis or dermatitis or combination as a result of bacteremia. (1,4)

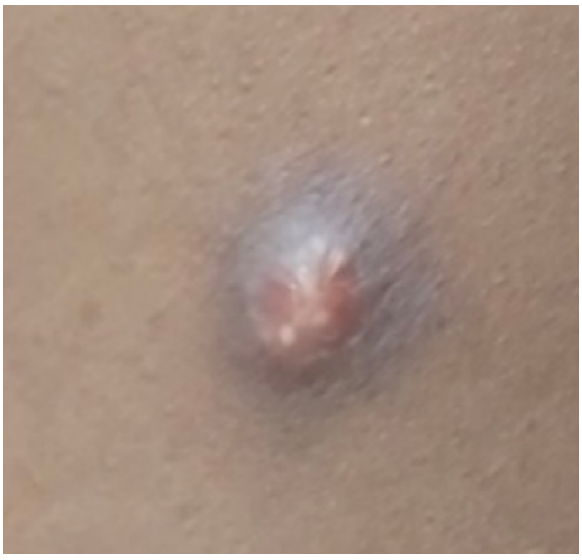
Case presentation

A 28-year-old unmarried male, injecting drug user (DU) with a history of multiple contacts with female commercial sex workers (FSW) was referred from Welikada prison hospital to the Central STD clinic, Colombo. Initially, he presented with purulent urethral discharge, was diagnosed as having smear positive, culture negative urethral gonorrhoeae which was treated with oral cefixime and azithromycin.

A week later at follow-up visit his urethral discharge had resolved, but had developed painless, pustular lesions over trunk for three days. His last sexual exposure was four months prior with a FSW. He also reported a history of urethral discharge and scrotal swelling which was treated 18 months ago, but further details were unavailable.

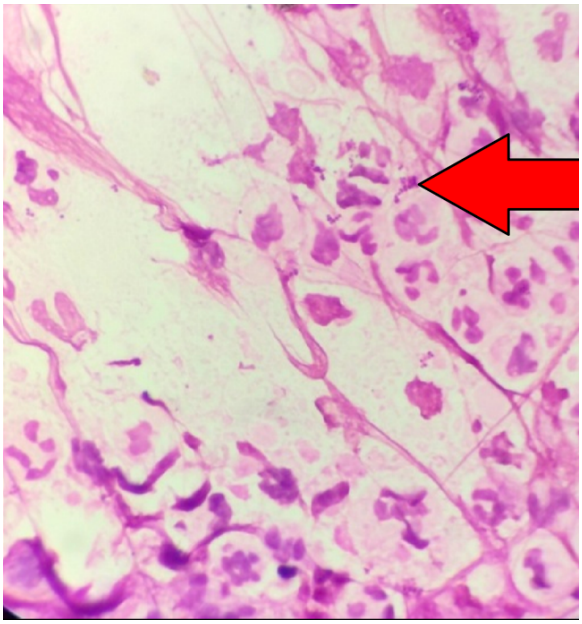
Examination revealed multiple (10 – 15), discrete, non-tender pustules 1 – 2cm in diameter asymmetrically distributed over his trunk (Figure 1). There were no signs of fever, arthritis, tenosynovitis, conjunctivitis or petechia. Extremities and head were spared. Cardiovascular, neurological and genital examinations were unremarkable.

Figure 1. Skin lesion on the trunk



Gram stained smear from pustules showed intracellular (IC) and extracellular (EC) gram negative diplococci (GNDC) (Figure 2). But, culture on Modified Thayer Martin (MTM) Agar was negative.

Figure 2. Intracellular GNDC with typical morphology within polymorpho-nuclear leucocytes with few extra cellular organisms



Based on patient's history, clinical features and smear results presumptively DGI was diagnosed. Patient was treated with intramuscular ceftriaxone 1g daily for three days followed by oral cefixime 400mg twice daily for one week per national STI management guideline.(5) Dermatitis completely resolved with treatment. He was tested negative for HIV, syphilis and hepatitis B but was positive for hepatitis C, hence, referred for further care. Partner tracing was attempted but was unsuccessful.

Discussion

DGI presents typically with the "classical triad" dermatitis, tenosynovitis, arthralgia or arthritis often accompanied by fever.(6) In this patient only dermatitis was present; non-tender, necrotic pustules on an erythematous base, fewer than 30, asymmetrically distributed on trunk rather than over extremities where lesions commonly occur.(1,4) Absence of fever, arthritis or tenosynovitis initially obscured the diagnosis. Similarly, in 2016, in a 41-year-old female

presented with lower back pain, DGI was not suspected initially until pyomyositis and spinal lesions developed due to bacteriemia. (7)

Though, there's no proper surveillance for DGI in Sri Lanka, the prevalence is assumed to be < 1%.(5) Initially, if this patient had no uro-genital features and presented only with dermatitis, the diagnosis could have been easily missed. This is because DGI is a rare condition, with a diverse clinical presentation and there is a misleading expectation that DGI always present with uro-genital features. (8) This can be why the numbers of reported DGI are less in number. In Germany, DGI in a 38-year-old male was misdiagnosed as rheumatoid arthritis as he lacked genitor-urinary symptoms. (9) DGI misdiagnosis can be overcome by low-diagnostic threshold, robust history and by improving awareness on diverse clinical presentation. (7,9)

Based on auxotyping, protein-I (PI) serotyping and coagulation patterns gonococci strains vary. Core groups (FSWs/ DUs), where gonorrhoea transmission and acquisition prevalence is higher is an epidemiological factor which determines the predominant strain. Mostly females with gonorrhoea are asymptomatic and act as a reservoir. (1,4) As gonorrhoea acquisition in this patient is likely from an asymptomatic FSW, there is higher possibility of acquiring a DGI producing strain. Partner management which is essential to prevent onward transmission was unsuccessful in this case.

Protein-IA-serotype and AHU-auxotype strains are more invasive and produce DGI. Though, gonorrhoea usually remains localized due to host responses, certain virulence factors evade defenses resulting dissemination. Only the gonococci phagocytosed by neutrophils get killed. As DGI causing strains lack stimulating neutrophil chemotaxis, gonococci engulfed by monocytes survive facilitating dissemination. Phase and antigenic variations also facilitate dissemination. Strains with sialylated lipo-oligosaccharide (LOS) mimic host cells and become serum resistant causing DGI.(4)

Natural immunoglobulins (Ig) and complements are responsible for human serum bactericidal activity. AHU/1A-1 and AHU/1A-2 strains producing DGI mostly are resistant to complement-mediated bactericidal activity.(1,4) Gonococcal or meningococcal bacteremia or DGI develop in terminal complement (C5 – C8) deficiencies.(10) Rarely, DGI occur in C2 deficiency also.(6) Complement deficiency can be inherited or episodical in association with clinical flares of diseases like SLE.(1,10) In 2020, a 23-year-old Japanese man with congenital C7 deficiency was diagnosed with DGI and recurrent Neisseria infections.(10) Recommendation is to screen patients presenting with two or more episodes of gonococcal or meningococcal bacteremia for complement deficiency (especially C6-C8).(1,4) Though this patient recall treatment for urethral discharge and scrotal swelling 18 months ago, complement deficiency screening was lacking.

In repeated gonococcal infections IgG for reduction modifiable proteins (RMP) facilitate dissemination. These antibodies develop following cross-reactivity to enterobactericiae during anal sex. (4) This patient had practiced insertive anal sex with FSW.

N.gonorrhoeae and *N.meningitidis* are morphologically identical in gram stain. *N.meningitidis* can colonize at any mucosal site of sexual transmission and may mimic gonococcal infection.(1) Non-pathogenic commensals like *N.lactamica* may yield positive in smear but fail to grow on selective medium. *N.gonorrhoeae* and *N.meningitidis* is differentiated with superoxol and carbohydrate utilization tests.(4) In this case as there was no growth in culture within 24 hours, sub-culture or biochemical tests were not performed.

Following kidney-shaped ICGNDC detection in smear from skin lesion exudates, considering history and clinical features which were atypical but suggestive of the clinical syndrome, presumptively DGI was diagnosed. Later, as culture was negative, the diagnosis was re-formed as possible DGI. In this case, kidney-shaped ICGNDC detection in smear was crucial for diagnosis despite negative cultures as when DGI present with atypical features it could delay diagnosis.

Gonococcus is fastidious; requires strict conditions to ensure in-vitro growth. Temperature variations and logistic limitations during inoculated culture transportation affect

its viability. (1,4) Per literature, culture has limited diagnostic value in DGI.(9) MTM-Agar medium permit only gonococci and meningococci growth. DGI producing auxotype-AHU strains are inhibited by vancomycin in selective media and by anticoagulant used in blood culture media. This inhibition is greater with comparatively smaller inoculation. For DGI diagnosis, a selective culture medium containing lincomycin is recommended in parallel with non-selective culture medium. Modified New York City medium containing lincomycin ensure more gonococcal isolations. (1,4) As national reference laboratory (NRL) use MTM-Agar, if DGI in this patient is by vancomycin sensitive strain, no culture growth will be observed. Culture associated diagnostic issues could be overcome by gonococcal urine-based PCR test.(8) It was not performed in this case due to non-availability of the facility in NRL. As culture sensitivity is poor for gonococci in skin lesions, there growth is difficult.(1,4) Skin biopsy is another diagnostic option in culture negative DGI with cutaneous manifestations. (8)

As blood culture was not performed in this case the exact DGI phase at presentation; "haematogenous" or "transitional" was uncertain. However, as the patient lacked joint involvement the late "joint localization" phase was excluded.(4)

Gonococci are resistant to sulfanilamides, penicillin, tetracycline, macrolide, spectinomycin and fluoroquinolones. (11) Currently, the recommended antibiotic class for gonorrhoeae first-line treatment is ceftriaxone and other cephalosporin. Ceftriaxone is effective for gonorrhoea at all sites and any gonorrhoeae related complications. (12) Drug pharmacokinetic and gonococcal susceptibility studies recommend equal or threefold of minimal inhibitory concentration (MIC) in antibiotic serum levels to cure an uncomplicated gonococcal infection. Cefixime and ceftriaxone MICs for *N.gonorrhoeae* are same. Prophylactic use of antibiotics following risk sexual exposures has facilitated gonococcal antibiotic-resistant. Chromosome gene mutation and plasmid mediated antibiotic-resistant strains mostly produce DGI. (1,4) This patient developed dermatitis following cefixime, giving arise to a doubt on its susceptibility. Antibiotic sensitivity testing (ABST) was not performed as there was no culture growth. In 2021 and 2022 prevalence of cefixime resistant *N.gonorrhoeae* in Australia was 6.3% exceeding the 5% threshold. (12) Gonococcal antimicrobial resistance is rapidly increasing limiting the treatment; hence, strengthening ABST is important.

As there is a rising trend of gonorrhoea, the possibility of developing complications like DGI has also increased, especially due to antibiotic resistance development. In developed countries like Spain, gonococcal infections are included in "obligatory declaration diseases".(6) Establishing a surveillance system to grab all DGIs and a follow-up pathway for gonorrhoea cases is essential.

Conclusion

This case highlights the diagnostic and therapeutic challenges in the current context of rising gonococcal incidence and emerging antibiotic resistance. Atypical presentation can delay the diagnosis and treatment. Improved awareness among clinicians, improving the availability of advanced diagnostic facilities, effective partner notification and robust national surveillance are essential to prevent the emerging threat of DGI.

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