

Case Report

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Fulminant Disseminated Gonococcal Infection Complicated by Meningitis, Endocarditis, and Septic Shock in an Immunocompetent Young Woman: A Case Report

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ABSTRACT

Disseminated gonococcal infection (DGI) is an uncommon but potentially life-threatening complication of *Neisseria gonorrhoeae* infection caused by hematogenous dissemination from mucosal sites. The classical manifestations of DGI include dermatitis, tenosynovitis, and migratory polyarthritides. Severe invasive complications such as meningitis, endocarditis, septic shock, and multiorgan failure are rare.

An 18-year-old immunocompetent woman with no significant past medical history presented to the infectious diseases ward with fever and severe suprapubic pain. On admission, her temperature was 38.2°C, blood pressure was 130/85 mmHg, heart rate was 80 beats/min, respiratory rate was 18 breaths/min, and oxygen saturation was 98% on room air. Physical examination revealed multiple painful pustular and hemorrhagic bullous lesions measuring approximately 1 mm over the elbows, along with clinical evidence of tenosynovitis.

Microbiological investigations confirmed disseminated gonococcal infection, with *N. gonorrhoeae* isolated from vaginal swab, blood, and synovial fluid cultures. Concurrent *Chlamydia trachomatis* infection was identified. Treatment was initiated with intravenous ceftriaxone (1 g once daily) and oral doxycycline (100 mg twice daily). Despite antimicrobial therapy, the patient developed polyarthritides on hospital day 5, mitral valve endocarditis on day 7, severe hepatic dysfunction on day 8, and acute kidney injury and meningitis on day 9. Cerebrospinal fluid culture subsequently confirmed *N. gonorrhoeae*. On day 10, she developed septic shock requiring intensive care admission and norepinephrine support. Seizures developed on day 11, and the patient died on day 12 from septic shock secondary to disseminated gonococcal infection with multiorgan failure.

This case illustrates a rare, fulminant presentation of DGI with progressive involvement of the skin, musculoskeletal system, cardiovascular system, central nervous system, and multiple organs. Early recognition of characteristic DGI manifestations, prompt microbiological investigation, and timely initiation of appropriate antimicrobial therapy are essential components of management.

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Introduction

Neisseria gonorrhoeae is a sexually transmitted bacterial pathogen responsible for gonorrhea worldwide. Although gonococcal infection commonly remains localized to mucosal surfaces, hematogenous dissemination may occur, resulting in disseminated gonococcal infection (DGI).

DGI is an uncommon but clinically important complication of gonococcal infection. Its characteristic manifestations include dermatitis, tenosynovitis, migratory polyarthralgia, and septic arthritis. The classical clinical presentation comprises a combination of skin lesions, tendon sheath inflammation, and joint involvement. Recognition of this pattern is important because early diagnosis and appropriate antimicrobial treatment may reduce

morbidity and prevent progression to severe invasive disease [1,2].

Diagnosis can be challenging because mucosal symptoms may be absent or inconspicuous, and the systemic manifestations can resemble other infectious, inflammatory, or rheumatological conditions. Although many patients respond to appropriate antimicrobial therapy, DGI can rarely progress to life-threatening complications, including infective endocarditis, meningitis, septic shock, and multiorgan failure [3-5].

This report describes a rare, fulminant case of disseminated gonococcal infection in an immunocompetent young woman. The infection was microbiologically confirmed at multiple sites and was complicated by mitral valve endocarditis, gonococcal meningitis, septic shock, and progressive multiorgan dysfunction, ultimately resulting in death despite antimicrobial treatment.

Case Presentation

An 18-year-old immunocompetent woman with no significant past medical history was admitted to the infectious diseases ward with fever and severe suprapubic pain.

On admission, her temperature was 38.2°C, blood pressure was 130/85 mmHg, heart rate was 80 beats/min, respiratory rate was 18 breaths/min, and oxygen saturation was 98% on room air. Physical examination revealed multiple painful pustular lesions with hemorrhagic bullous characteristics, measuring approximately 1 mm and localized over the elbows. Clinical examination also demonstrated tenosynovitis involving the tendon sheaths.

Based on the initial presentation, the differential diagnoses included pneumococcal meningitis, septic arthritis, pelvic inflammatory disease, and *Staphylococcus aureus* bacteremia with infective endocarditis. The combination of fever, characteristic skin lesions, and tenosynovitis raised suspicion for disseminated gonococcal infection (DGI).

Laboratory Investigations

Initial laboratory investigations demonstrated leukocytosis, neutrophilia, thrombocytopenia, and elevated inflammatory markers. The complete blood count showed a hemoglobin concentration of 11 g/dL, a white blood cell count of 19,000/ μ L with 81% neutrophils and 8% lymphocytes, and a platelet count of 80,000/ μ L. Inflammatory markers were elevated, with a C-reactive protein (CRP) level of 60, an erythrocyte sedimentation rate (ESR) of 55, and a procalcitonin level of 2.1. The units for these inflammatory markers should be confirmed against the original laboratory reports.

Initial renal function was relatively preserved, with a blood urea nitrogen level of 15 mg/dL, serum creatinine of 1 mg/dL, and a reported estimated glomerular filtration rate of 98 mL/min/1.73 m². Initial liver function tests showed mild aminotransferase elevations, with an aspartate aminotransferase (AST) level of 35 U/L and an alanine aminotransferase (ALT) level of 50 U/L. Initial coagulation studies showed a prothrombin time of 12 seconds, a partial thromboplastin time of 27 seconds, and an international normalized ratio (INR) of 1.

Repeat liver function testing on hospital day 8 demonstrated substantial biochemical deterioration. The ALT level was 600 U/L, AST was 650 U/L, bilirubin was 110 μ mol/L, serum albumin was 22 g/L, and alkaline phosphatase (ALP) was 450 U/L. These findings indicated marked hepatic biochemical abnormalities during the patient's clinical deterioration.

Imaging and Cardiac Investigations

Computed tomography of the pelvis demonstrated an adnexal abscess. Imaging of the affected joints also revealed joint effusion.

On hospital day 7, transthoracic echocardiography demonstrated a 6 mm vegetation involving the mitral valve, accompanied by mitral regurgitation. In the context of *Neisseria gonorrhoeae*-positive blood cultures, these findings supported the diagnosis of gonococcal infective endocarditis.

Microbiological Investigations

Microbiological investigations confirmed *Neisseria gonorrhoeae* infection at multiple sites. Cultures of vaginal swab, blood, adnexal abscess and synovial fluid all yielded *N. Gonorrhoeae*. Antimicrobial susceptibility testing reportedly demonstrated susceptibility without evidence of resistance. Concurrent

Chlamydia trachomatis infection was also identified.

Synovial fluid analysis demonstrated a high neutrophil count, and Gram staining revealed Gram-negative organisms, supporting the diagnosis of gonococcal septic arthritis in the setting of disseminated infection.

Cerebrospinal fluid (CSF) examination was performed following neurological deterioration on hospital day 9. Lumbar puncture revealed cloudy CSF with an opening pressure of 38 cmH₂O. The CSF white blood cell count was 2,200 cells/ μ L, with 92% neutrophils. The protein concentration was 84 mg/dL, and the glucose concentration was less than 10% of the simultaneous serum glucose concentration. Gram staining revealed Gram-negative diplococci, and subsequent CSF culture confirmed *N. Gonorrhoeae*, establishing the diagnosis of gonococcal meningitis.

Treatment and Clinical Course

Following microbiological confirmation of disseminated gonococcal infection, the patient was treated with intravenous ceftriaxone at a dose of 1 g once daily and oral doxycycline at a dose of 100 mg twice daily. Despite antimicrobial treatment, her clinical condition progressively deteriorated, with sequential involvement of multiple organ systems.

On hospital day 5, the patient developed polyarthritis involving the knee, hip, shoulder, elbow, and temporomandibular joint. This progression was consistent with extensive musculoskeletal involvement in the setting of disseminated infection.

On day 7, transthoracic echocardiography revealed a 6 mm vegetation on the mitral valve, accompanied by mitral regurgitation. Together with the positive blood culture for *Neisseria gonorrhoeae*, these findings supported the diagnosis of gonococcal infective endocarditis.

By hospital day 8, the patient had developed substantial biochemical abnormalities in liver function, including an alanine aminotransferase level of 600 U/L, an aspartate aminotransferase level of 650 U/L, a bilirubin level of 110 μ mol/L, a serum albumin level of 22 g/L, and an alkaline phosphatase level of 450 U/L. Her deterioration prompted escalation of care.

On day 9, the patient developed acute kidney injury, with a reported serum creatinine level of 2.5 mg/dL and blood urea nitrogen level of 30 mg/dL. She also experienced neurological deterioration, prompting lumbar puncture. The cerebrospinal fluid opening pressure was 38 cmH₂O. Cerebrospinal fluid analysis showed a white blood cell count of 2,200/ μ L, with 92% neutrophils, protein of 84 mg/dL, and glucose below 10% of the simultaneous serum glucose concentration. Gram-negative diplococci were observed, and subsequent cerebrospinal fluid culture confirmed *N. Gonorrhoeae*, establishing gonococcal meningitis.

On hospital day 10, the patient developed septic shock, with a blood pressure of 70/40 mmHg and a lactate level of 3 mmol/L. She required admission to the intensive care unit and norepinephrine support. Despite ongoing management, her condition continued to worsen. Seizures occurred on day 11.

On day 12 of hospitalization, the patient died from septic shock and multiorgan failure in the setting of fulminant disseminated gonococcal infection complicated by infective endocarditis and meningitis.

Discussion

Disseminated gonococcal infection (DGI) is an uncommon but potentially life-threatening complication of *Neisseria gonorrhoeae* infection. Hematogenous dissemination can produce a characteristic combination of dermatitis, tenosynovitis, migratory polyarthralgia, and septic arthritis. Recognizing this clinical pattern is important because the initial manifestations may be nonspecific and can overlap with other infectious, inflammatory, and rheumatological disorders [1,2].

The present case demonstrated several characteristic features of DGI, including painful pustular and hemorrhagic skin lesions, tenosynovitis, and subsequent polyarthritis involving multiple joints. The diagnosis was supported by isolation of *N. Gonorrhoeae* from the vaginal swab, blood, synovial fluid, and cerebrospinal fluid. Recovery of the organism from multiple sites, including normally sterile sites, provided strong microbiological evidence of disseminated infection and confirmed central nervous system involvement.

Unusual Invasive Complications

The most striking feature of this case was the progression from the characteristic manifestations of DGI to severe invasive complications involving the cardiovascular and central nervous systems, followed by septic shock and multiorgan dysfunction. Although the skin and musculoskeletal manifestations are well recognized, gonococcal infective endocarditis and meningitis are uncommon complications that can make the clinical course particularly severe [3-5].

Mitral valve endocarditis was identified on hospital day 7, when transthoracic echocardiography demonstrated a 6 mm vegetation accompanied by mitral regurgitation. In the context of gonococcal bacteremia, these findings strongly supported infective endocarditis. This complication illustrates the importance of investigating persistent or worsening systemic illness for endovascular infection, particularly when blood cultures identify a pathogen capable of hematogenous dissemination.

Central nervous system involvement became apparent on hospital day 9, following neurological deterioration. The elevated cerebrospinal fluid opening pressure, marked neutrophilic pleocytosis, increased protein concentration, severely reduced glucose concentration, and observation of Gram-negative diplococci were consistent with bacterial meningitis. Subsequent cerebrospinal fluid culture confirmed *N. Gonorrhoeae*. This microbiological confirmation is particularly important because neurological manifestations in a patient with systemic infection may have several possible causes, and the responsible organism should be established whenever diagnostic testing permits.

Progressive Multiorgan Dysfunction

Despite initiation of antimicrobial treatment, the patient developed progressive organ dysfunction. Marked abnormalities in liver biochemistry were documented on hospital day 8, followed by acute kidney injury, meningitis, septic shock, and seizures over the subsequent days. The development of hypotension requiring norepinephrine support and the eventual progression to multiorgan failure demonstrate the severity of the clinical course.

Antimicrobial susceptibility testing reportedly identified a susceptible gonococcal strain without detected resistance. Nevertheless, the patient's condition deteriorated and ultimately proved fatal. This outcome illustrates that reported antimicrobial susceptibility does not, by itself, guarantee a favorable clinical outcome in severe invasive infection. Disease severity at presentation, the extent of organ involvement, and the clinical response over time must also be considered. However, the available case information does not establish why the patient deteriorated despite treatment, and the outcome should not be attributed to a single factor without further evidence.

The reported treatment regimen was intravenous ceftriaxone at 1 g once daily, together with oral doxycycline at 100 mg twice daily for concurrent *Chlamydia trachomatis* infection. Because the infection progressed to endocarditis and meningitis, the documented antimicrobial regimen, its timing, and any subsequent treatment modifications should be reviewed against the original medical records before publication. The case alone cannot establish whether treatment adequacy, timing, disease severity, or another factor accounted for the fatal outcome.

Concurrent Chlamydial Infection and Diagnostic Considerations
Concurrent *C. Trachomatis* infection was identified in this patient. This finding reinforces the importance of assessing for other sexually transmitted infections when gonococcal infection is diagnosed. Coinfection may influence treatment decisions and should be documented as part of the overall clinical assessment.

The initial combination of fever, painful pustular skin lesions, tenosynovitis, and suprapubic pain required consideration of several differential diagnoses, including septic arthritis, pelvic inflammatory disease, bacterial meningitis, and bacteremia with infective endocarditis. The subsequent recovery of *N. Gonorrhoeae* from multiple clinical specimens helped distinguish disseminated gonococcal infection from these alternative explanations and demonstrated the extent of the infection.

The computed tomography finding of an adnexal abscess is another relevant feature of the case. The culture obtained establish that the abscess itself was caused by *N. Gonorrhoeae*.

Clinical Implications

This case highlights several important clinical lessons. First, the combination of fever, characteristic pustular skin lesions, tenosynovitis, and arthritis should prompt consideration of DGI, even when the initial presentation includes symptoms suggestive of pelvic infection. Second, microbiological sampling from relevant sites can be crucial to establishing the diagnosis and identifying the extent of dissemination. Third, deterioration despite initial treatment should prompt reassessment for complications such as endocarditis, meningitis, and evolving organ dysfunction.

The fatal outcome also underscores the importance of accurately documenting the timing of antimicrobial administration, susceptibility results, evolving organ abnormalities, and any changes in clinical management. These details are essential when interpreting an unusually severe case and determining which conclusions can reasonably be drawn from it.

Table 1: Initial Laboratory Investigations on Admission

Parameter	Result
Hemoglobin	11 g/dL
WBC	19,000/ μ L
Neutrophils	81%
Lymphocytes	8%
Platelets	80,000/ μ L
CRP	6.0 mg/dL
ESR	55 mm/hr
Procalcitonin	2.1 ng/mL
PT	12 sec
PTT	27 sec
INR	1.0
D-dimer	0.5 mg/L FEU
Fibrinogen	550 mg/dL
BUN	15 mg/dL
Creatinine	1 mg/dL
AST	35 U/L
ALT	50 U/L
ALP	127 U/L
Albumin	5 g/dL

Table 2: Microbiological Findings

Specimen or Test	Result
Vaginal swab culture	Neisseria gonorrhoeae isolated
Blood culture	Neisseria gonorrhoeae isolated
Synovial Fluid culture	Neisseria gonorrhoeae isolated
CSF culture	Neisseria gonorrhoeae isolated
Adnexal Abscess culture	Neisseria gonorrhoeae isolated
Chlamydia Testing	Chlamydia trachomatis positive
Antimicrobial susceptibility testing	No resistance detected

Table 3: Clinical Timeline and Major Events

Hospital Day	Clinical Event
Day 1	Admission with fever and severe suprapubic pain. Painful pustular and hemorrhagic bullous skin lesions and tenosynovitis were identified. Cultures were obtained, and treatment with ceftriaxone and doxycycline was initiated.
Initial Evaluation	Pelvic CT demonstrated an adnexal abscess; imaging of the affected joints demonstrated joint effusion.
Day 5	Polyarthrits developed, involving the knee, hip, shoulder, elbow, and temporomandibular joint.
Day 7	Echocardiography demonstrated a 6 mm mitral valve vegetation with associated mitral regurgitation.
Day 8	Clinical deterioration and marked abnormalities in liver biochemistry were documented; intensive care was required.
Day 9	Acute kidney injury and gonococcal meningitis were diagnosed. Cerebrospinal fluid culture confirmed N. gonorrhoeae.

Day 10	Septic shock developed, with blood pressure of 70/40 mmHg and lactate of 3 mmol/L; norepinephrine support was initiated.
Day 11	Seizures developed.
Day 12	The patient died from septic shock and multiorgan failure.

Conclusion

This report describes a fulminant presentation of disseminated gonococcal infection in an immunocompetent young woman, complicated by mitral valve endocarditis, culture-confirmed gonococcal meningitis, septic shock, and multiorgan failure, resulting in death. The case demonstrates that DGI can progress beyond its characteristic dermatological and musculoskeletal manifestations to severe invasive disease. Early recognition of the clinical pattern, prompt microbiological investigation, careful assessment for complications, and close monitoring for organ dysfunction are important components of management. The reasons for the fatal progression in this patient cannot be determined conclusively from the available information.

Learning Points

- Recognizing the clinical pattern: The combination of fever, pustular skin lesions, tenosynovitis, and arthritis should raise suspicion for disseminated gonococcal infection (DGI).
- Recognizing severe complications: Although DGI commonly presents with dermatological and musculoskeletal manifestations, clinicians should remain alert to uncommon invasive complications, including infective endocarditis and meningitis.
- Establishing microbiological confirmation: Isolation of Neisseria gonorrhoeae from multiple clinical specimens, including blood, synovial fluid, and cerebrospinal fluid, can establish the diagnosis and demonstrate the extent of dissemination.
- Monitoring for deterioration: Progressive clinical deterioration should prompt reassessment for additional infectious complications and evolving organ dysfunction.
- Considering coinfection: Testing for other sexually transmitted infections is important in patients diagnosed with gonococcal infection, as illustrated by the concurrent Chlamydia trachomatis infection in this case.

Ethical Statement

Written informed consent for publication of this case report and the accompanying clinical information was obtained from the patient's appropriate representative. Every effort was made to protect patient confidentiality and remove identifying information.

Institutional requirements for publication of this case report were addressed in accordance with the applicable institutional policies.

Conflict of Interest

The authors declare that they have no competing interests.

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