1. Introduction

The use of cardiovascular implantable electronic devices (CIEDs) has increased significantly for the prevention of life-threatening arrhythmias in coronary artery disease and congestive heart failure [1]. CIEDs comprise implantable cardioverter-defibrillators (ICDs), pacemakers, and cardiac resynchronization therapy devices. Concordant with this trend is a rising incidence of CIED-related infections, with ICDs contributing to a lifetime risk of 1.9% [2–4]. A number of host, procedure, and device-related risk factors have been studied, with recent device manipulation or exchange being the most significant [5]. Staphylococcus species (Staphylococcus aureus and coagulase-negative Staphylococci) have been the usual culprit in infectious complications [6]. We discuss the first reported case of a pocket-site and lead infection secondary to N. gonorrhoeae bacteremia in an individual with a history of high-risk sexual behavior.

2. Case Presentation

A 56-year-old African-American male with past medical history of paroxysmal atrial fibrillation (on apixaban), chronic obstructive pulmonary disease (COPD), chronic...
kidney disease (CKD) stage III, and heart failure with reduced ejection fraction (EF: 25–30%) status post-ICD placement for primary prevention presented to the arrhythmia clinic with a pocket-site swelling. The patient reported that the swelling began recently a few days after lifting a heavy object at home. He had previously undergone complete ICD device and lead extraction and subsequent reimplantation 2 years prior due to a pocket-site infection which was secondary to methicillin-resistant *Staphylococcus aureus* (MRSA). Pocket-site ultrasound in clinic revealed a 4.5 cm × 2 cm × 2 cm fluid collection concerning for hematoma, prompting discontinuation of apixaban. The swelling progressively increased in size on a subsequent clinic visit. This, along with the development of pocket-site pain, erythema, and tenderness, in association with generalized malaise and fatigue prompted admission.

On presentation, he was afebrile (98.3°F) and hemodynamically stable (pulse 93 bpm and blood pressure 115/82 mm Hg) with oxygen saturations of 96% on room air. Physical examination revealed a 5 cm × 6 cm precordial swelling exhibiting localized warmth and redness. A new-onset II/V systolic murmur was auscultated in the tricuspid area. Relevant laboratory workup showed no leukocytosis on blood cultures, with the patient divulging any urogenital symptoms during the course of his illness. Of note, he did not report any high-risk sexual behavior (unprotected, multiple sexual partners) in the recent past. Of note, he did not report any urogenital symptoms during the course of his illness.

Further clarification of his penicillin allergy, which was limited to hives, allowed for transition to intravenous (IV) ceftriaxone on the second day of antibiotic therapy. Repeat blood cultures 24 hours later also grew gonococci but cleared thereafter. The patient underwent surgical debridement and complete device and lead extraction a week later which revealed infected and necrotic tissue at the pocket-site with drainage of serosanguineous fluid. The pocket site was washed with a triple-antibiotic solution and closed. Cultures from the tissue, fluid drained from the pocket, and the device failed to grow any organisms while Gram stain was negative for any pathogens as well. Infectious disease (ID) was consulted. Given the isolation of *N. gonorrhoeae* alone on blood cultures and clinical evidence of pocket-site infection, a diagnosis of device and pocket-site infection secondary to gonococcal bacteremia was made. In the absence of prior exposure to antibiotics with Gram-positive coverage, pocket-site infection with Gram-positive organisms, including the commonly implicated *Staphylococcus* or *Streptococcus* species, was effectively ruled out as the etiology of the infection. In accordance with ID recommendations, he continued to receive IV ceftriaxone for 4 weeks with gradual resolution of the pocket-site swelling. Since the ICD was implanted for primary prevention of ventricular arrhythmias, it was deemed prudent to wait until the patient had completed an appropriate course of antibiotics. He responded well to this course of therapy and was successfully discharged. He continued to follow with ID and cardiology as outpatient, eventually undergoing successful reimplantation of an ICD 1 month later at a different site without complications.

### 3. Discussion

CIED infections encompass pocket-site and systemic (infective endocarditis or lead infection) subtypes. These subtypes are not mutually exclusive, as demonstrated in our patient who exhibited both a pocket-site infection and infective endocarditis (IE). Infections may also be classified as primary or secondary; our case exhibited CIED infection secondary to gonococcal bacteremia acquired as a sexually transmitted disease. *S. aureus* and coagulase-negative *Staphylococci* contribute to a majority (70–90%) of cases, with Gram-negative bacilli and fungal pathogens causing the remainder [6, 7]. Cultures from pocket site, lead tips, and blood samples may be, respectively, positive only in 61%, 79%, and 77% of all patients according to one study [8]. In addition to a previous episode of device revision/replace-ment and pocket reoperation, our patient also exhibited some other significant risk factors for infection. These risk factors included CKD, COPD, congestive heart failure, and anticoagulant use [8, 9].

Endocarditis may be reported in 1-2% of patients with disseminated gonococcal infection, making this a relatively rare entity with only a handful of cases reported in the medical literature [10, 11]. Symptoms may be subtle, requiring a high index of suspicion in patients, particularly those with CIEDs. If a diagnosis of pocket-site infection is suspected on physical examination indicating local inflammation, echocardiography and blood cultures should be performed to evaluate for lead infection and IE. No cases have been reported in the literature describing gonococcal pocket-site infections.

Corroborating prior reports, gonococcal growth on tissue and device cultures was negative in our case, likely due to prior antibiotic administration for a week, with the fastidious nature of the organism contributing to it as well. *N. gonorrhoeae* endocarditis leads to significant valvular damage, necessitating surgical intervention in up to 70% of cases, compared to 25–50% when other organisms are involved [10].

CIED infections are managed with IV antibiotics, device explantation, debridement, and reimplantation at a different site [7]. A prolonged course of antibiotics after device revision is employed depending on the etiology of the infection, with a minimum of 4 weeks reserved for IE or persistent bloodstream infection (BSI) despite device removal [12]. Device reimplantation is delayed in cases of persistent BSI until resolution of bacteremia, with successful
replacements being reported as early as 3 days or up to 2 weeks after resolution of bacteremia. Recurrent infections can be prevented by the use of prophylactic antibiotics, antibiotic pouches, or use of subcutaneous ICDs.

4. Conclusion
Gonococcal bacteremia can lead to CIED pocket-site infection necessitating device removal, surgical debridement, and systemic antibiotics. Device infections and endocarditis with *N. gonorrhoeae* should be part of the differential diagnoses in patients with high-risk sexual behavior presenting with constitutional symptoms and/or new-onset heart murmurs.

Data Availability
The data that support the findings of this study are available from the corresponding author upon reasonable request.

Consent
Informed consent was obtained from the patient for case publication.

Conflicts of Interest
The authors declare that they have no conflicts of interest.

References
[1] S. M. Kurtz, J. A. Ochoa, E. Lau et al., “Implantation trends and patient profiles for pacemakers and implantable cardioverter defibrillators in the United States: 1993-2006,” *Pacing and Clinical Electrophysiology*, vol. 33, no. 6, pp. 705–711, 2010.
[2] D. Klug, M. Balde, D. Pavin et al., “Risk factors related to infections of implanted pacemakers and cardioverter-defibrillators,” *Circulation*, vol. 116, no. 12, pp. 1349–1355, 2007.
[3] M. Dai, C. Cai, V. Vaibhav et al., “Trends of cardiovascular implantable electronic device infection in 3 decades,” *JACC: Clinical Electrophysiology*, vol. 5, no. 9, pp. 1071–1080, 2019.
[4] T. Olsen, O. D. Jørgensen, J. C. Nielsen, A. M. Thøgersen, B. T. Philbert, and J. B. Johansen, “Incidence of device-related infection in 97,750 patients: clinical data from the complete Danish device-cohort (1982-2018),” *European Heart Journal*, vol. 40, no. 23, pp. 1862–1869, 2019.
[5] F. A. Waldvogel and A. L. Bisno, *Infections Associated with Indwelling Medical Devices*, p. 247, ASM Press, Washington, D.C, USA, 2000.
[6] M. R. Sohail, D. Z. Uslan, A. H. Khan et al., “Risk factor analysis of permanent pacemaker infection,” *Clinical Infectious Diseases*, vol. 45, no. 2, pp. 166–173, 2007.
[7] J. D. Chua, B. L. Wilkoff, I. Lee, N. Jurati, D. L. Longworth, and S. M. Gordon, “Diagnosis and management of infections involving implantable electrophysiologic cardiac devices,” *Annals of Internal Medicine*, vol. 133, no. 8, pp. 604–608, 2000.
[8] M. R. Sohail, D. Z. Uslan, A. H. Khan et al., “Infective endocarditis complicating permanent pacemaker and implantable cardioverter-defibrillator infection,” *Mayo Clinic Proceedings*, vol. 83, no. 1, pp. 46–53, 2008.
[9] K. A. Polyzos, A. A. Konstantelias, and M. E. Falagas, “Risk factors for cardiac implantable electronic device infection: a systematic review and meta-analysis,” *EP Europace*, vol. 17, no. 5, pp. 767–777, 2015.
[10] A. Ramos, P. García-Pavía, B. Orden et al., “Gonococcal endocarditis: a case report and review of the literature,” *Infection*, vol. 42, no. 2, pp. 425–428, 2014.
[11] T. C. Wall, R. B. Peyton, and G. R. Corey, “Gonococcal endocarditis,” *Medicine*, vol. 68, no. 6, pp. 375–380, 1989.
[12] L. M. Baddour, A. E. Epstein, C. C. Erickson et al., “Update on cardiovascular implantable electronic device infections and their management,” *Circulation*, vol. 121, no. 3, pp. 458–477, 2010.