Extra-articular bloody tophi in pacemaker pocket: A case report



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Introduction

Complications arising from pacemaker implantation may occur during the procedure, particularly in high-risk patients with conditions such as coagulopathy and heart failure. However, there is limited available data on the association between inflammatory disorders and pacemaker complications. In this report, we present a case of pacemaker-related complications in a patient with gouty arthritis.

Case report

A 64-year-old Thai man with a medical history that includes mitral valve replacement, sick sinus syndrome evolved into permanent atrial fibrillation receiving warfarin therapy, chronic illnesses (including hypertension and chronic kidney disease), and gouty arthritis underwent successful pacemaker implantation in April 2020.

In April 2022, the patient visited the hospital owing to progressive shortness of breath. His electrocardiogram showed a pacing stimulus followed by a wide QRS complex compatible with paced rhythm. Echocardiography revealed left ventricular dysfunction, with the ejection fraction decreasing from 53% in April 2020 to 40%. The patient was diagnosed with pacemaker-induced cardiomyopathy. The patient was scheduled for an upgrade to cardiac resynchronization therapy in May 2022. Unfortunately, owing to the absence of a suitable coronary vein for lead placement, the procedure was unsuccessful (Supplemental Figure 1).

One month later, the patient returned to our clinic with swelling at the pacemaker wound site. He was apyretic, normotensive, without any pain at the pacemaker pocket site. Upon examination, a painless swelling was observed

KEYWORDS Permanent pacemaker; Gouty arthritis; Pacemaker pocket complication; Cystic gout; Hematoma (Heart Rhythm Case Reports 2023;9:701–703)

KEY TEACHING POINTS

- Holistic and multispecialty care of patients with pacemaker is critical.
- Hematoma is one of the most common pacemaker complications, which can occur up to 3% of cases and increase the risk of other adverse side effects.
- Poorly controlled gout could affect pacemaker wound healing, which may result in cystic gout inside the pocket.

at the pacemaker site. The patient also had gouty tophi on both first metatarsophalangeal joints and both ankles (Figure 1). The rest of the physical examinations yielded unremarkable results. The pacemaker was interrogated and showed 90% ventricular pacing. Other parameters exhibited normal function without any abnormal events. An ultrasound revealed a 4-cm-diameter fluid collection in the pacemaker pocket. Despite 2 weeks of antibiotic treatment, the pacemaker wound continued to swell. After a thorough discussion of the risks and benefits, a wound revision operation was scheduled.

A chest radiograph showed soft tissue swelling at the pacemaker pocket site (Figure 2). Laboratory results indicated normal platelet counts without leukocytosis, but elevated serum creatinine levels of 2.09 mg/dL (reference range: 0.67–1.17 mg/dL) and an elevated international normalized ratio level of 3.43 were observed. Additional laboratory data are presented in Table 1. The patient underwent pacemaker pocket revision, which revealed bloody content with chalk-like material. Following wound exploration and evacuation, the patient's symptoms resolved. Analysis of the content revealed clotted blood without evidence of bacterial infection. Polarized light microscopy demonstrated needle-shaped crystals with negative birefringence, consistent with uric acid crystals (Figure 3). After 2 months of

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Figure 1 A physical examination of the patient showed a swelling pacemaker wound without tenderness. The lower extremities showed gouty tophi on both first metatarsophalangeal joints and ankles.

intensive serum uric acid control, the wound healed without recurrent pocket swelling.

Discussion

The implantation of pacemakers involves creating a space between the fascia or muscle, known as the pacemaker pocket, and the transvenous insertion of pacing wires. Complications can arise after pacemaker insertion, occurring in up to $6\%^1$ of all implantation procedures, ranging from superficial bleeding to severe systemic infection.

Hematoma is among the most common complications, occurring in up to 3% of pacemaker cases and increasing



Figure 2 The chest radiograph showed cardiomegaly with sternal wire present. A prosthetic mitral valve was seen. The pacemaker generator and lead were in position. Enlargement and cloudiness of the pacemaker wound were noted.

the risk of other adverse effects,² including pacemaker infection.³ The use of anticoagulation therapy further elevates the risk of hematoma following pacemaker or intracardiac defibrillator procedures.^{1,4} Additionally, patients who experience hematoma complications have a slightly higher in-hospital mortality rate (1.3%) and incur significantly increased hospital costs of over \$14,000 (US dollars).⁵

This particular patient presents a higher risk for pocket hematomas owing to factors such as oral anticoagulation therapy, heart failure, and chronic kidney disease. However, there may be other risk factors contributing to adverse outcomes in this patient.

Gout, an ancient disease, is the most common chronic inflammatory arthropathy affecting men, characterized by the deposition of monosodium urate (MSU) crystals in joints and soft tissues.⁶ Tophaceous gout can lead to the formation

Blood test	Value	Reference
BUN (mg/dL)	34	6–20
Creatinine (mg/dL)	2.09	0.67-1.17
Uric acid (mg/dL)	11.0	3.4-7.0
Calcium (mg/dL)	9.3	8.4-10.2
Magnesium (mg/dL)	2.4	1.6-2.6
Phosphorus (mg/dL)	2.7	2.3-4.7
White blood cell (/µL)	4500	4400-11,300
Hemoglobin (g/dL)	11.0	14.0-17.4
Hematocrit (%)	35.4	41.5-50.4
MCV (fL)	72.2	80.0-96.0
Platelet (/µL)	130,000	150,000-450,000
Neutrophil (%)	42.7	45-75
Lymphocyte (%)	41.9	20–45

BUN = blood urea nitrogen; MCV = mean corpuscular volume.

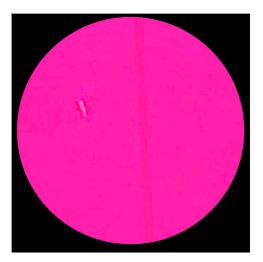


Figure 3 The polarized microscope of fluid obtained from the pacemaker pocket demonstrated a needle-shaped crystal with strong negative birefringence.

of soft tissue calcifications in the musculoskeletal system, including tendons and extremities.⁷ While cystic gout lesions are rare, they have been reported predominantly in the lower extremities.^{8–10}

Complex metabolic and inflammatory processes are responsible for development of gout. It is characterized by the intrinsic accumulation of urate to levels sufficient for the formation of monosodium urate crystals,¹¹ followed by an inflammatory response to these crystals. Various factors, including trauma, surgery, medications, and alcohol consumption, together with the appropriate intra-articular environment, such as temperature, pH of the joint, can trigger the precipitation and subsequent inflammation of MSU crystals within the joint.¹²

In our patient, elevated serum uric acid levels, bilateral tophi on the lower extremities, and bloody fluid with chalk-like content, along with monosodium urate crystals, pointed to the diagnosis of pacemaker pocket hematoma with cystic gout.

The abnormal location of MSU crystal formation in the pacemaker pocket in this patient is likely attributed to excessively high serum uric acid levels that exceed its solubility, coupled with the appropriate environment (precipitating factors such as surgery, chronic kidney disease, and anticoagulation medication). Ochoa and colleagues¹³ proposed that the presence of MSU crystals could disrupt arterial blood supply in subcutaneous tissue, leading to localized inflammation. Based on this information, cystic gout formation may occur.

The management of gout typically involves uratelowering therapy (eg, allopurinol) and lifestyle modifications.¹⁴ To the best of our knowledge, this is the first reported case associating pacemaker pocket hematoma with gout. In such situations, a holistic and multidisciplinary approach to the care of patients with pacemaker implantation is crucial.

Conclusion

In conclusion, we present the first documented case of a cystic lesion of gout occurring on top of a pacemaker pocket

hematoma in a Thai man with poorly controlled gout. This case highlights the importance for clinicians to maintain a high index of suspicion and promptly evaluate possible associated diseases that may significantly impact clinical outcomes. By recognizing and addressing these potential comorbidities, clinicians can provide optimal care and improve patient outcomes.

Acknowledgments

Verbal consent was obtained from the patient granting permission that data concerning his case could be submitted for publication.

Funding Sources: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Disclosures: None.

Availability of data and materials: The data for this case report are located at Rajavithi Hospital, Bangkok, Thailand.

Appendix Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023. 07.008.

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