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Case Report
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A Case of Recurrent Ventricular Tachycardia from Gouty Cardiomyopathy

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Abstract

The major advanced clinical feature of gout is tophi, which affects the heart very rarely. We present a case of recurrent ventricular arrhythmia in a 49-year-old male who had chronic gout for 10 years. The color Doppler echocardiography was performed and found multiple hyperechoic, oval-shaped masses on the mitral annulus, left ventricle wall, and chordae tendineae, along with multiple hyperdense were found in Computed Tomography (CT) of the heart.

Keywords: Gout; Tophi; Arrhythmia; Ventricular tachycardia; Gouty cardiomyopathy

Case Presentation

A 49-year-old male with history of chronic gout for approximately 10 years, palpitation for 3 years lasting for 1 h at a time, and no medication history because of the gastric discomfort. There was no previous history of clearly diagnosed cardiac arrhythmia, and his family also had no known history of arrhythmia. Due to the reappearance of palpitation, he was admitted to the emergency room of our hospital. On physical examination during admission, the pulse was 114 beats/min, the respiratory rate was 20 breaths/min, and the Electrocardiography (ECG) showed Ventricular Tachycardia (VT) (Figure 1). The patient was immediately given amiodarone 0.15 g by emergency first responders and was referred to our cardiovascular department for further treatment and determine the cause of VT after successful cardioversion. On arrival, the blood routine examination had an increased level of WBC (21.03 × 10^9/L; normal value: [3.50-9.50] × 10^9/L), Neu (17.20 × 10^9/L; normal value: [1.80-6.30] × 10^9/L), and FR-CRP (14.67 mg/L; normal value: <4.00 mg/L). In addition, the biochemical profiles showed increased serum Uric Acid (UA) level (648.00 mmol/L; normal value: [208.00-506.00] mmol/L).

Due to the significant elevation of routine laboratory tests, we performed a sputum culture which showed the results of *Haemophilus influenzae*, and he was treated with anti-infection and anti-inflammatory therapy. In addition, given the possibility of Coronary Artery Disease (CAD) causing VT, we examined coronary Computed Tomography Angiogram (CTA). CTA showed a mixed plaque formation in the proximal segment of the left anterior descending branch with moderate stenosis. The results of CTA considered the possibility that the patient has CAD, but the lesion was not severe and the cardiac markers tests were normal, so we ruled out the diagnosis of CAD leading to VT. Then the color doppler echocardiography (Figure 2A) was also performed and found multiple hyperechoic, oval-shaped masses on the mitral annulus, left ventricle wall, and chordae tendineae, along with multiple hyperdense were found in Computed Tomography (CT) of the heart (Figure 2B). Finally, after ruling out other possible causes, we considered the diagnosis of gouty cardiomyopathy, the VT induced by community-acquired pneumonia and caused by deposition of tophi in the cardiac.

To further confirmed our diagnosis, a pathophysiological biopsy of the oval-shaped masses in cardiac of patient was required and the patient was suggested to undergo Implantable Cardioverter Defibrillator (ICD) implantation, but he declined for financial reasons, so cardioversion with amiodarone was continued, and benzbromarone and febuxostat were used to lower uric acid.

Discussion

In this report, we present a rare case of a middle-aged man with chronic gout and recurrent VT possibly due to tophi deposition in the heart. To our knowledge, the arrythmia due to tophi deposition in cardiac in patients with chronic gout have been unreported.

VT is a life-threatening arrythmia for which determining the underlying cause is often the

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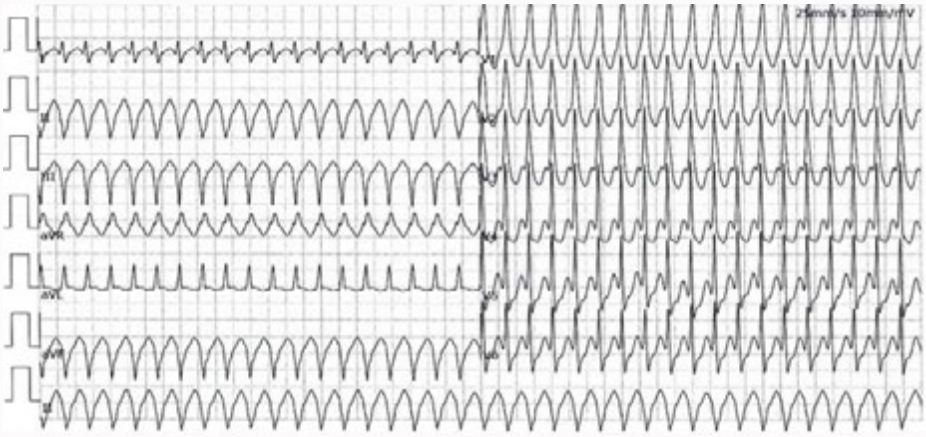

Figure 1: Twelve-lead Electrocardiogram (ECG) on the day of the emergency room. The ECG shows ventricular tachycardia. The ventricular rate was 228 bpm and the QT intervals were 240ms.

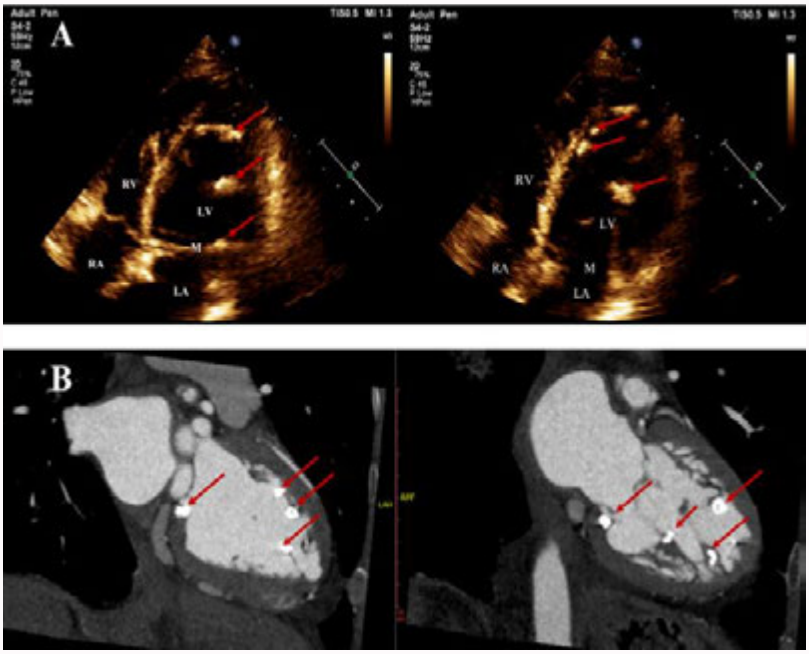

Figure 2: (A) Color Doppler Echocardiography: the apical 4-chamber view showed multiple hyperechoic, oval-shaped masses on the mitral annulus, left ventricle wall, and chordae tendineae (arrows). (B) Computed Tomography of the heart: in the sagittal plane found multiple hyperdense (arrows).

key to treatment. Clinicians need to be cautious in making the differential diagnosis when determining the cause. In this case, we also considered the possibility of hyperparathyroid cardiomyopathy in addition to ruling out CAD and found a normal serum parathyroid hormone. In fact, VT due to gout is often overlooked by clinicians, and the main reason for this outcome is that there are too few reports involving gout-induced arrhythmia to catch the attention of clinicians. However, the gold standard for this disorder still relies on pathological biopsy, which needs the detection of large amounts of deposition of urate crystals in the tophi and not just fibrosis-like changes [1].

In fact, the prognosis of this patient was found to be unsatisfactory by follow-up, which probably because the patients' cardiac discomfort did not receive sufficient attention during the last three years, and resulting in patients missing the best time for treatment. In recent years, studies found that gout and hyperuricemia are associated with the development of arrhythmias, and have demonstrated that elevated Serum Uric Acid (SUA) levels are independently associated with the recurrence of common arrhythmias [2], these mechanisms remain unclear but may be due to the local and systemic inflammatory response mediated by MSU in patients with gout, which induces a number of inflammatory cytokines, including Tumor Necrosis Factor α (TNF-α), Interleukin (IL)-1β, IL-6 and IL-8. In addition, it has been previously reported that urate crystal deposited in the myocardial leading to heart failure, then that patient was treated with allopurinol and prednisone timely and showed significant improvement in left ventricular ejection fraction [3], which suggests that the cause of chronic gout with cardiac discomfort should be promptly identified.

Conclusion

This case emphasizes that when arrhythmias occur in patients with a history of chronic gout, tophi deposits in the heart should be considered as the cause of recurrent arrhythmias, which is conducive

to clinical diagnosis and treatment. And the health education and care interventions for chronic gout patients should be enhanced for both patients and physicians.

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