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Clickbait: A Rare Case of Monomorphic Ventricular Tachycardia and Clicking Sensation without Infectious Presentation Revealed as Infective Endocarditis with Aortic Root Abscess

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Abstract Presenting symptoms of infective endocarditis (IE) typically includes infectious signs such as diaphoresis, fever, and malaise. Pathogenic microorganisms implicated in IE may form vegetations, collection of debris that may subsequently embolize. Notable complications and concomitant sequelae of systemic embolization from resulting from valvular vegetations include stroke, acute renal failure, and cutaneous manifestations. Valvular dysfunction warranting surgical repair does not preclude redo-repairs from subsequent emboli, highlighting the extensive propensity of IE to cause acute decompensation across multiple domains. A presenting feature of arrhythmia stemming from valvular dysfunction have been appreciated in the literature. However, literary accounts of ventricular tachycardia (VT) as the resultant arrhythmia are scant. Moreover, these accounts usually include concomitant infectious presentation. We report the case of a 36-year-old male with a past medical history of *Bartonella spp.* endocarditis and aortic valve repair who appeared to the emergency department with acute onset monomorphic VT. Complicating his presentation was a reported sensation of clicking in his surgically repaired valve while lacking infectious symptoms. Arrhythmia was treated with further workup delineating *S. epidermidis* vegetations and aortic root abscess successfully treated with antibiotics and ICD placement.

Keywords: *ventricular, tachycardia, endocarditis, bartonella, valvular, Vtach*

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1. Introduction

The appraisal of morphological features in ventricular tachycardia offers insight into the pathophysiology underlying this pathological circuit. Subsequently, this divulges clinical etiologies that may be targeted to guide therapeutic options. Of interest is monomorphic ventricular tachycardia (MVT), whose namesake derives from features noted on electrocardiograms (ECG)- namely, heterogenous QRS complex waveforms (three or more consecutive beats) and a ventricular rate greater than 100 beats per minute [1]. Concurrent reproducibility of the waveform along the rhythm strip in space and time may indicate a pathologic regional automaticity within the heart that may be localized or the presence of discrete ventricular reentry circuits [2]. The classic presentation of the latter is myocardial fibrosis resulting in a scar that is unable to conduct electricity, but generates a zone of electrical potential surrounding the scar that is amenable to depolarization [3]. While traditionally described in patients with ischemia from complications of coronary artery disease and hypertension, recent findings have suggested presentations of ischemia from inflammatory cardiac disease [4,5]. MVT may also occur due to left ventricular outflow tract (LVOT) abnormalities, such as anatomical obstruction (i.e, aortic stenosis, aortic root abscess) that modulate pathways of electrical conduction that predispose to arrhythmias [6].

Mindfulness of these associations can be paramount in managing long-term sequelae after stabilization of the

patient who initially presents with classic symptoms of MVT such as dizziness, malaise, and chest discomfort but with equivocal past medical history. We report the case of atypical infective endocarditis presenting as MVT and left bundle branch block (LBBB) in an adult male with a previous history of endocarditis and aortic valve repair, later worked up for active vegetations and development of aortic root abscess successfully treated with pharmacotherapy. Given the paucity of reports in the literature characterizing endocarditis presenting without infectious symptoms but with electrophysiologic considerations of atypical MVT, encounters such as these hold value in informing clinicians with utility in guiding prospective workup of arrhythmia with complicated medical history. The proclivity to commentate on such a phenomenon serves as the aim for our case report. Our objectives are to share to introduce IE but given the novel aspect of this rare case report, to shift the perspective of providers to give viability to IE as part of a differential diagnosis despite the lack of hemodynamic instability associated with illness. Moreover, a secondary objective would be to introduce the concept of electrophysiologic sequelae as an association with IE given the association of MVT with this case.

2. Case Description

A 36-year-old Hispanic male with a past medical history significant for end-stage renal disease (ESRD) secondary to focal segmental glomerulosclerosis (FSGS) on home hemodialysis and aortic valve endocarditis due to *Bartonella spp.* status post bioprosthetic valve replacement with subsequent redo presented to the emergency room with complaints of lightheadedness and palpitations. Moreover, the patient endorsed feelings of chest discomfort and the sensation of rapid valvular "clicking". Presenting vital signs indicated a low index of suspicion for toxic appearance (temperature of 36.8°C, respiratory rate of 18 breaths per minute, a blood pressure of 133/80, saO_2 of 100% on room air). Initial ECG showed atrioventricular discordance consistent with wide-complex sustained monomorphic ventricular tachycardia with a ventricular rate of 170 BPM and LBBB with negative inferior axes, suggestive of a septal or inferior wall origin (Figure 1). Serum electrolytes returned normal and mild troponin elevation consistent with ESRD warranted another source for rhythm control and diagnosis. The patient was subsequently started on an IV amiodarone drip for MVT with hemodynamically stable status unable to restore neither heart rate nor rhythm. Subsequent elective cardioversion with one synchronized 200-J shock administration resulted in immediate conversion to normal sinus rhythm (NSR) with 1^0 degree AV block with a PR interval of >400ms (Figure 2). Given history of endocarditis, blood cultures were sent and returned positive for gram-positive cocci, verified upon recurrent studies. Polymerase chain reaction testing was negative for methicillin-resistant *S. aureus* but was able to designate *S. epidermidis* as the infectious agent, with confirmed culture sample from the tunneled catheter used for home dialysis, strongly suggesting a line-associated infection. Subsequent nuclear stress testing (Figure 3) and transesophageal echocardiogram (Figure 4) were conducted to confirm the diagnosis, revealing a vegetation in the process. Nuclear perfusion scans showed a dilated LV with EF = 30% and some fixed inferolateral defects. The TEE was notable for otherwise normal chamber sizes, dilated LV, and preserved RV systolic function. No thrombus can be seen in the LA or LAA. There is bileaflet mechanical aortic valve, which is well-positioned and mobile echodensities on both leaflets of the mechanical valve. The larger one measures 0.9 x 0.9 cm and the smaller one measures 0.5 x 0.4 cm. Both are best seen in view 16 (second top left window in Figure 4). There is also significant edema and thickening of the aortic root extending into the aortic annulus consistent with early abscess formation and trace aortic regurgitation.

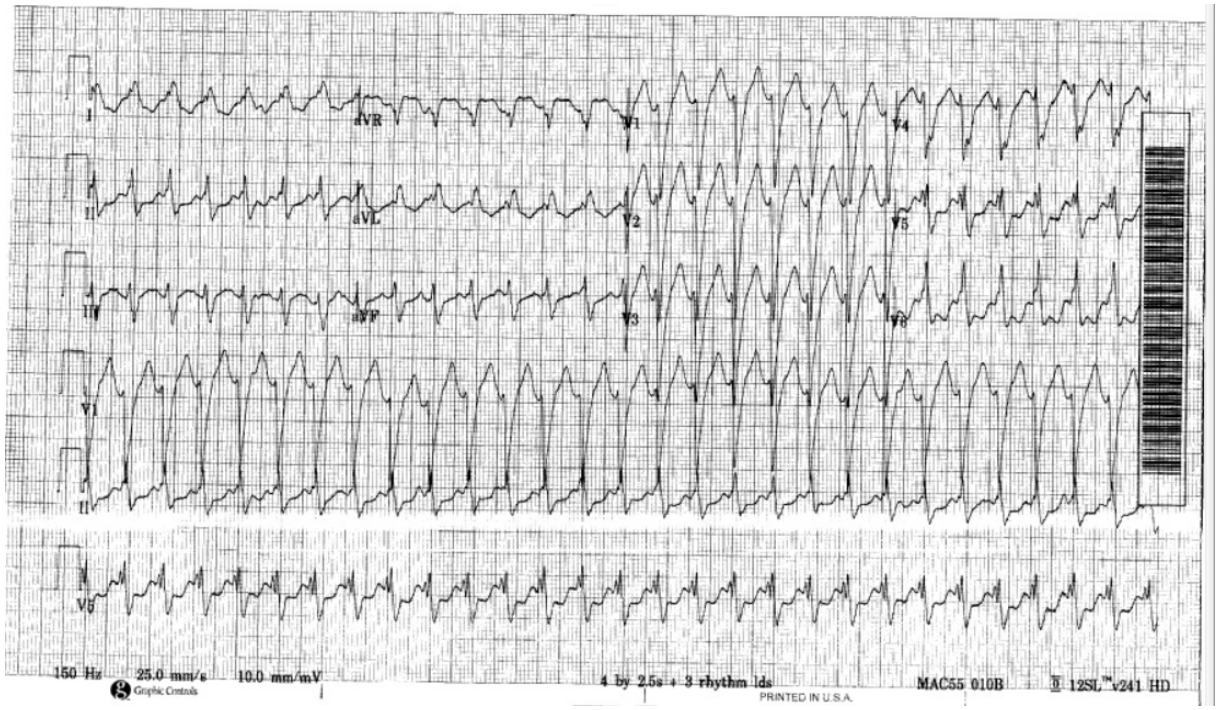

Figure 1. Initial ECG Noting Sustained Wide-Complex MVT Upon Presentation

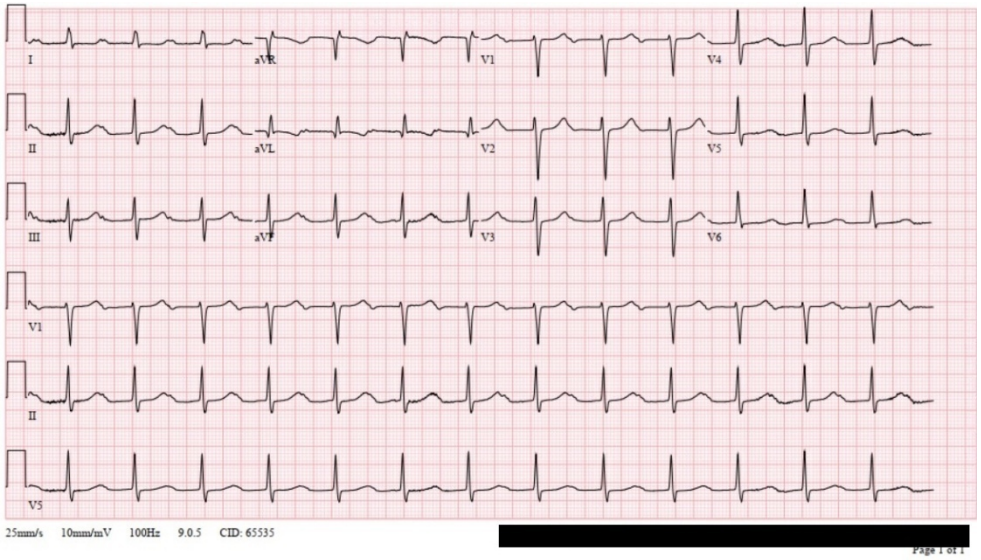

Figure 2. Post 200-J Cardioversion ECG showing transition from MVT to NSR with 1^0 AV Block

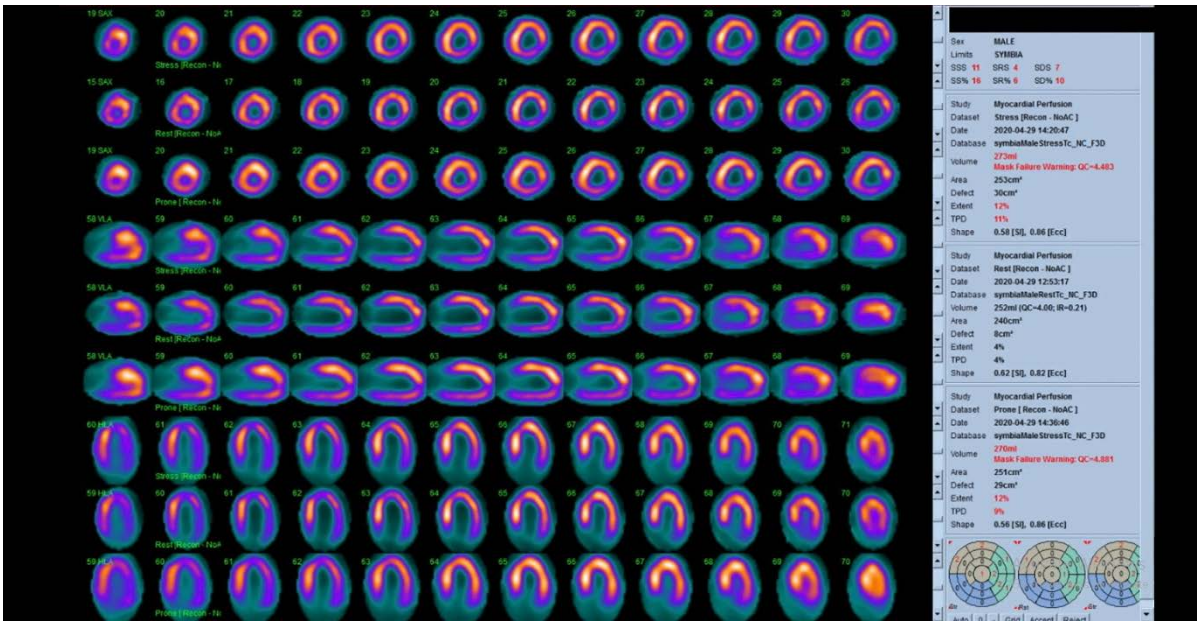

Figure 3. Nuclear Stress Test Highlighting Ischemic Tissue via Viability Study

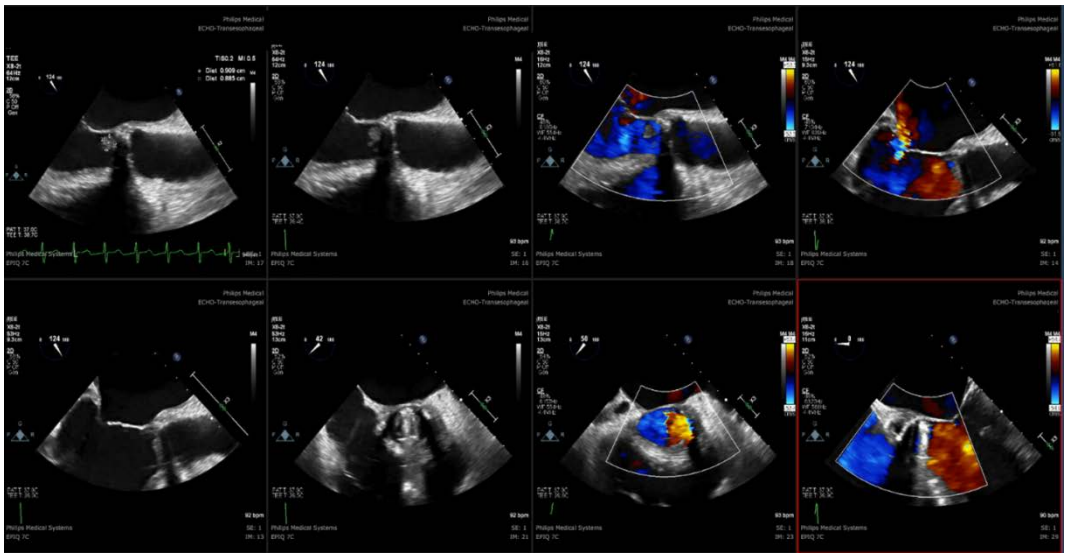

Figure 4. TEE Panel Showing Vegetations (Top right) and Cardiac Function in Context of Infective Endocarditis

3. Discussion

Infective endocarditis, commonly associated with intravenous drug use, may also occur due to prolonged intravascular access through other modalities, such as hemodialyzed ESRD patients [7]. There have been attempts to elucidate presenting features of such patients in an attempt to gain insight into the effects of infective endocarditis in these populations. Such analyses would offer a tool for rapid diagnosis and satisfactory clinical treatment endpoints. One observational study that followed 10,612 patients (mean age 63 years, 36% female) with initiated renal replacement therapy (RRT, 7,233 hemodialyses, 3056 peritoneal dialysis, and 323 pre-emptive kidney transplantation) [8]. A hazard ratio of 5.46 regarding the risk of endocarditis in patients receiving hemodialysis (95% confidence interval [95% CI], 3.28 to 9.10) relative peritoneal dialysis was shown, highlighting the significance of prudent assessment in patients who present with signs of endocarditis with a history of hemodialysis. Such a high threshold for endocarditis predisposes patients to multiple sequelae, such as embolization. In particular, a retrospective analysis of data from a multi-center cohort of 1,345 consecutive episodes of infective endocarditis affecting the left side of the heart (as implied by the pattern of the ECG in our case), 25% of patients experienced neurological complications with significant complications in death ($P<0.01$) [9]. The predilection of *S. epidermidis* in particular to seek out lines and manifest itself into presentations consistent with the latter has been observed in other scenarios of external biomedical devices *in vivo* [10,11]. Within valvular cases, infective endocarditis has been reported in the literature to predispose to aortic root abscesses due to purulent inflammation from bacterial suppuration [12]. It is believed that by compromising the valvular architecture and the electromechanical rhythm of the heart between beats, subacute bacterial endocarditis with vegetations may incite such arrhythmias as there is no consistent cardiac motion and geometry for depolarizations to be consistently distributed across the myocardium (confirmed by TEE and nuclear perfusion in this patient) [13]. Abscesses have been associated with sepsis precluding fever, systemic symptoms such as malaise, and profound hemodynamic instability.

Nevertheless, complicating this assessment in our patient was the lack of overt signs of infectious seeding. Cases in the literature noting aortic root abscess as a complication of infective endocarditis note symptoms such as fever, malaise, and a toxic appearance- all null in our patient [14].

However, some similarity between this case and the one in the aforementioned study include aortic root abscess predisposing to PR prolongation and AV block [14]. Complicating the initial presumptive diagnosis was a seemingly novel finding of ventricular tachycardia upon introductory presentation. Ventricular tachycardia is commonly associated with structural heart disease as the dysregulation of the cardiomyocyte arrangement, metabolism, and architecture result in the scarring of the myocardium-leading to a disarray of cardiac myocytes, electrolyte defects, and gap junction deficiencies that act as the nidus for arrhythmias via aberrant channels for electrical impulses [15]. Other etiologies of ventricular tachycardia include idiopathic ventricular tachycardia, which is also known as fascicular tachycardia, a form of ventricular tachycardia that is associated with re-entry circuitry in young patients without structural heart disease. Such an etiology may be the root source of monomorphic ventricular tachycardia in our patient; however, these patients usually present with RBBB abnormalities, and not LBBB. Ventricular tachycardia may have been a component of initial disease presentation in our patient as retrograde emboli due to a component of aortic regurgitation noted on echocardiographic studies revealed retrograde ischemic emboli, subsequent micro-infarcts, myocardial fibrosis, and a circuit conducive to monomorphic ventricular tachycardia. It was only when the overlying ventricular tachycardia was managed was that underlying symptoms such as heart block and PR prolongation were appreciated, suggestive of aortic root abscess. TEE confirmed the presence of vegetations as expected in the latter scenario, with cultures verifying both patient blood and tunneled catheter as the etiology of such vegetations. The tunneled catheter was subsequently replaced. Due to high surgical risk in this case, medical management with antibiotics was approached. This strategy resulted in resolution of infection with subsequent ICD placement serving the role of an augmenting intervention to rhythm homeostasis in the event that a catheterization or subsequent hemodialysis may predispose to future infections affecting the heart, resulting in arrhythmia akin to the introductory presentation.

4. Conclusion

While an association between the two is significant for its paucity in the literature, ventricular tachyarrhythmias should be noted in patients who have a history of infective endocarditis or are at high-risk for endocarditis, such as those with valvular repair or dialysis status. Moreover, while overt signs of infection may not be present, considerable damage to the underlying structure of the heart may be involved or have already undertaken. Resolution of an abnormal heart rhythm may not be alone to heal a patient as this may only reveal a latent arrhythmia. While valve surgery is usually the choice for these patients, our case highlights that medical management with antimicrobial pharmacotherapy with subsequent aggressive electrophysiology interventions may serve paramount in these patients lives, despite scant evidence for this clinical strategy within the literature.

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