Can U Waves Be “Notched”?

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Abnormal U waves (unduly larger or misshapen) are associated with several conditions such as hypokalemia, arrhythmias, cardiac ischemia, ventricular hypertrophy, and hypertension. Abnormal U waves have also been linked to certain cardiac medications, predominantly antiarrhythmics. However, mechanisms of U-wave abnormality remains debated and perhaps elusive with the true U-wave relationship to T waves still being investigated. While there have indeed been reports of bifid (“notched”) T waves, such comparably described U waves have escaped us thus far. We present a case of possibly bifid U waves that persisted over the course of 10 years in the setting of repeated negative Holter monitor findings and clinical absence of atrial tachyarrhythmias. We take this opportunity to briefly discuss common causes of “normal” and abnormal U waves.

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Presumably, the only surviving conundrum of the electrocardiographic wave forms, the U wave, first described and designated by Einthoven in 1903,¹ has seen its share of debate and hypotheses with respect to its genesis. Several causes such as, afterpotentials (“afterdepolarizations”) triggered from electromechanical ventricular wall myocardial effects² and repolarization of either midmyocardial M cells³ or that of Purkinje fibers⁴ have all been cited for its elusive origin, not one postulate conclusively gaining general acceptance over the other.⁵ Prominent U waves may be present with hypokalemia and with use of certain cardiac medications (Table 1). They have often been implicated clinically at both ends of the arrhythmia spectrum, more so with tachyarrhythmias which when present, could indicate an increased susceptibility to torsades de pointes.^{2–5} While “normal” U waves, best noted in leads V₂ and V₃, are usually upright, <1 mm, and often one-third of their concordant (similar polarity) T-wave amplitude, inverted (negative) U waves have low sensitivity but high specificity for ischemia, particularly when discordant with their corresponding T waves.⁶ Such U waves could indicate coronary artery disease mainly that

of the left main, left anterior descending, or left circumflex arteries.⁷ Interestingly, although T-wave bifidity has been described,⁸ there have been no reports of such unique U-wave morphology.

CASE SCENARIO

An 81-year-old female with a history of coronary artery disease, hypertension, hypercholesterolemia, osteoporosis, and spinal stenosis who had been on aspirin, isosorbide dinitrate, metoprolol, lisinopril, nifedipine, simvastatin, and estrogen replacement therapy, presented with acute angina and dyspnea on numerous occasions over a span of 10 years. She had been ruled out for myocardial infarction with electrocardiogram (ECG) as well as laboratory investigations and had undergone exercise tolerance tests with nuclear persantine assessment that were negative. No changes in the U wave were reported with these evaluations. A recent coronary angiogram for a similar acute presentation had revealed 70% and 50% stenoses of the left anterior descending (LAD) and circumflex coronary arteries, respectively, but stent angioplasty could not be accomplished. Transthoracic

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Table 1. Common Causes of U Waves

A. Prominent upright/Accentuated U waves
Sinus bradycardia
Increased tendency for torsades de pointes
Hypokalemia
Class IA antiarrhythmics (mainly quinidine)
Class III antiarrhythmics (sotalol, amiodarone)
Digoxin
CNS dysfunction with long QT intervals (T and U waves may fuse to form giant “TU fusion waves”)
Atrioventricular valvular disease (commonly of mitral valve)
Hyperthyroidism
B. Inverted U waves (Especially with T-wave discordance)
Myocardial infarction (commonly anterior > inferior infarction; in leads with pathologic Q waves)
Angina (or exercise-induced ischemia)
Post extrasystolic in patients with coronary heart disease
During coronary artery spasm (Prinzmetal’s angina)
Atrioventricular valvular disease
Hypertension (commonly when severe or poorly controlled)
Right ventricular hypertrophy
Left ventricular hypertrophy
Long QT syndrome (TU fusion waves)

echocardiogram had revealed moderate-to-severe left atrial dilatation (59 mm in two-dimensional parasternal long-axis view) and mild left ventricular (LV) hypertrophy with preserved LV function (LV end-diastolic diameter 55 mm and LV end-systolic diameter 28 mm in two-dimensional parasternal long-axis view) without atrioventricular valvular involvement. There was also mild LV diastolic dysfunction ($E/A < 1$, E-wave inflow deceleration time 288 ms, $E/E' > 9$). The right ventricle was not enlarged (mean right ventricular systolic pressure was 18 mmHg). Frequent serial telemetry evaluations did not reveal atrial flutter or fibrillation and subsequent Holter monitoring on three separate occasions over 10 years from two different hospitals did not reveal any aberrant atrial activity. Throughout the course of presentations, complete blood count, basic metabolic panel (including potassium), and thyroid function tests had been normal. No major changes have been made with her medications except a change from nifedipine to diltiazem. However, almost all of her ECGs showed bifid U waves with intermittent nonspecific T-wave changes and interatrial conduction delay (P waves ≥ 110 ms) (Figs. 1 and 2 compare ECGs 10 years apart), a correlate of atrial tachyarrhythmias.⁹ There were

Figure 1. Magnified segment of patient’s ECG on initial presentation (Lead V₂ shown).

no recent ECGs available from when the patient was completely symptom-free for objective comparison. (The patient has since declined further testing.)

DISCUSSION AND CONCLUSION

This is the first known case of, quite possibly, bifid U waves. As these changes have reemerged over time on separate ECG leads, they are most unlikely to be artifactual. While the mere presence of U waves, be it with a nonacute or acute presentation, as in this patient, could in fact be related to her coronary artery occlusions^{6,7} or symptoms of angina and dyspnea, the bifidity of these complexes, if indeed these are truly U waves, remains an enigma. In the absence of electrophysiologic studies on this patient, the possibility of atrial flutter and/or fibrillation, or ectopic P waves cannot be ruled out. However, there has been neither Holter monitor nor telemetry evidence of such events and comparison of her past ECGs over 10 years, specifically of the T-P isoelectric line, have repeatedly revealed distinct notches on deflections consistent

Figure 2. Magnified segment of patient’s ECG during a similar presentation almost 10 years later (Lead V₃ shown).

with U waves in different precordial leads. There has also been no accountable, constant relationship between these peculiar U waves and the following QRS complexes to substantiate possible atrial activity and that these could actually be P waves, apart from the incidental-widened P waves. Nevertheless, an electrophysiologic study, besides further focused assessment, would be most helpful to help ascertain this and perhaps, identify repolarization abnormalities.

Previously unreported adverse effects of medications should also be considered in the absence of other common causes of U-wave aberrancies (Table 1). However, this patient had been stable on her medications except for a change from one type of calcium channel blocker to another while these notched U waves persisted. Furthermore, although estrogen replacement could be associated with increased QT intervals (implying prolonged myocardial repolarization), its effects specifically on the U wave or T-U complex have not been described.¹⁰ It is also uncertain what the role of intratrial conduction delay in this scenario is, if not merely an incidental finding. While severe LV diastolic dysfunction can be associated depolarization and repolarization alterations, it is difficult to assess the exact contribution of the patient's diastolic dysfunction in this scenario, particularly as it is only mild. This case of bifid deflections, if indeed that of U waves, is not only perplexing but most importantly, encourages us to carefully scrutinize serial

ECGs and perhaps rethink traditional concepts on U-wave genesis.

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