QTc pseudoprolongation and hypokalemia-induced T–U fusion: the authors reply

Christopher William Baugh · Andrew Tomas Reisner

Received: 21 October 2011 / Accepted: 28 November 2011 / Published online: 11 December 2011
© SIMI 2011

Dr. Omar [1] provides a valuable description of the measurement convention for the QT interval when prominent U waves are present on the ECG, as in the case presented in our paper [2]. As he correctly highlights in Lead I, there is indeed actual QT prolongation by measurement convention due to T–U fusion. However, other leads in the ECG (i.e., Lead aVF) are more suggestive of a discrete T wave, wherein a conventional QT interval can be measured. Our intention was to highlight that the presence of prominent U waves caused by hypokalemia can mimic the appearance of a markedly prolonged QT interval, but closer inspection of all leads may reveal that a normal QT interval can be calculated.

In the otherwise healthy heart, there is little evidence that hypokalemia alone causes dangerous dysrhythmias [3–5]. While Dr. Omar's references point toward a correlation between hypokalemia and QTc prolongation in special populations, those papers do not mention if T–U fusion plays a role in the measurement of the QT interval. Furthermore, the risk of Torsades de pointes (TdP) is not specifically addressed in these studies, particularly in patients with QTc prolongation secondary to T–U fusion. Indeed, from an electrophysiological standpoint, it is unclear when the risk of TdP is truly elevated, and when the prolonged QT interval is merely an artifact of the measurement convention because T–U fusion is present.

Thus, his statement that “the phenomenon of hypokalemia-induced T–U fusion...represents a form of QTc prolongation, not pseudoprolongation, and therefore can be associated with the serious form of polymorphic ventricular tachycardia” may actually be an open question [1]. Regardless, the cornerstone of clinical management of both pseudo and actual QTc prolongation due to hypokalemia are the same, which is potassium repletion.

Conflict of interest None.

References

1. Omar H (2011) Hypokalemia-induced T–U fusion. Intern Emerg Med. doi:10.1007/s11739-011-0732-2
2. Baugh CW, Reisner AT (2011) QTc pseudoprolongation. Intern Emerg Med 6:473–475
3. Haalboom JR, Bouter H, Kuyers PP, Struyvenberg A (1989) Hypokalemia in hypertensive patients treated with diuretics: no increase in cardiac arrhythmias. Neth J Med 34:81–87
4. Hirsch IA, Tomlinson DL, Slogoff S, Keats AS (1988) The overstated risk of preoperative hypokalemia. Anesth Analg 67:131–136
5. Papademetriou V (2006) Diuretics, hypokalemia, and cardiac arrhythmia: a 20-year controversy. J Clin Hypertens 8:86–92

C. W. Baugh (✉)
Department of Emergency Medicine,
Brigham and Women's Hospital, Boston, MA, USA
e-mail: cbaugh@partners.org

A. T. Reisner
Department of Emergency Medicine,
Massachusetts General Hospital, Boston, MA, USA