Acquired causes of Short QT interval 2021

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Hyperkalemia (mild elevations of K^+<6.5 mEq/L), consequence of narrow-based, peaked T waves. T waves with short duration, »150 to 250 msec. (**Ivana P Dewi et al. Short QT syndrome: The current evidences of diagnosis and management. J Arrhythm. 2020 Oct 6;36(6):962-966. doi: 10.1002/joa3.12439**.)

Hypercalcemia (**Yingwei Liu et al. Severe hypercalcemia from multiple myeloma as an acquired cause of short QT. J Electrocardiol. Nov-Dec 2018;51(6):939-940. doi: 10.1016/j.jelectrocard.2018.07.020**.)

Hyperthermia (**Fichet, J., Genee, O., Pierre, B., & Babuty, D. (2008). Fatal QT interval. The American Journal of Emergency Medicine, 26(6), 739.e5–739.e6. doi:10.1016/j.ajem.2007.11.039**)

Acidosis (**Fichet, J., Genee, O., Pierre, B., & Babuty, D. (2008). Fatal QT interval. The American Journal of Emergency Medicine, 26(6), 739.e5–739.e6. doi:10.1016/j.ajem.2007.11.039**)

Effect of catecholamine's (**Masaomi Chinushi 1, Exercise-related QT interval shortening with a peaked T wave in a healthy boy with a family history of sudden cardiac death. Pacing Clin Electrophysiol. 2012 Aug;35(8):e239-42. doi: 10.1111/j.1540-8159.2012.03363.x**)
Toxicity and digitalis effect. PR prolongation is a commonly present. Additionally, characteristic sagging, "coved," or "scooped" appearance of the asymmetric and downsloping ST depression, which resembles a reversed check mark (**Cheng TO. Digitalis administration: an underappreciated but common cause of short QT interval. Circulation. 2004 Mar 9;109(9):e152**).
In response to atropine (**Preben Bjerregaard et al. Short QT interval in clinical practice. J Electrocardiol. Sep-Oct 2010;43(5):390-5. doi: 10.1016/j.jelectrocard.2010.06.004**.)
Dysautonomia of Chronic Fatigue Syndrome with QTc mean values of 371 a 384 ms (**Naschitz J, Fields M, Isseroff H, Sharif D, Sabo E, Rosner I. Shortened QT interval: a distinctive feature of the dysautonomia of chronic fatigue syndrome. J Electrocardiol. 2006 Oct;39(4):389-94**.).

Selective K^{+}_{ATP} channel activation.* ATP-dependent potassium channel openers such as pinacidil and levcromakalim have long been known to shorten action potential duration and to be profibrillatory in non-clinical models.(**A. Jahangir et al. KATP channel therapeutics at the bedside. J Mol Cell Cardiol. 2005 Jul; 39(1): 99–112.doi: 10.1016/ j.yjmcc.2005.04.006**)

Activation of K_{Ach} caused by strong parasympathetic stimuli to the heart. **(Joachim Behar,The Autonomic Nervous System Regulates the Heart Rate through cAMP-PKA Dependent and Independent Coupled-Clock Pacemaker Cell Mechanisms. Front Physiol. 2016; 7: 419.doi: 10.3389/fphys.2016.00419**)

Klinefelter syndrome (KS). It is a sex chromosomal aneuploidy (47,XXY) affecting 1/660 males. QTc was shortest among testosterone treated males with KS, while untreated and thus hypogonadal KS had QTc interval comparable to controls (**Jørgensen IN, Skakkebaek A, Andersen NH, Pedersen LN, Hougaard DM, Bojesen A, Trolle C, Gravholt CH. Short QTc interval in males with klinefelter syndrome-influence of CAG repeat length, body composition, and testosterone replacement therapy. Pacing Clin Electrophysiol. 2015 Apr; 38(4):472-82**.).

Rufinamide, an approved anticonvulsant, illustrates the current regulatory approach to drugs that shorten QT interval (**Schimpf R, Veltmann C, Papavassiliu T, Rudic B, Göksu T, Kuschyk J, Wolpert C, Antzelevitch C, Ebner A, Borggrefe M, Brandt C. Drug-induced QT interval shortening following antiepileptic treatment with oral rufinamide. Heart Rhythm. 2012 May;9(5): 776-81**.)