[C-29] A Case of Torsade de Pointes Associated with Dexmedetomidine Bolus

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Case:
A 7 year-old boy with extracardiac Fontan for hypoplastic left heart syndrome presented for heart catheterization and electrophysiology (EP) study for episodes of chest pain and presyncope. EKG showed sinus bradycardia and borderline prolonged QTc interval. Holter monitor revealed occasional junctional rhythm at less than 75 bpm. Exercise stress test produced ST depression and mild hypoxemia that resolved with rest. Echocardiogram revealed normal RV function with mild outflow tract obstruction (peak gradient 20mmhg)
Anesthesia consisted of inhalation induction, endotracheal intubation and maintenance with isoflurane in air plus fentanyl and rocuronium. Hemodynamic measurements were fairly unremarkable. Rapid atrial pacing induced inferior ST depression, but no conduction abnormality was discovered during the EP study. During arterial and venous sheath removal, ondansetron, glycopyrrolate, and neostigmine were administered. Just prior to extubation, dexmedetomidine (0.8 mcg/kg) was given as a bolus and the patient immediately developed junctional bradycardia at 40 bpm. Frequent ventricular ectopy occurred, with pause-dependent prolongation of the QT, leading to torsade de pointes (TdP) after R-on-T phenomenon (Fig.). The TdP stopped spontaneously, but recurred on a background of junctional bradycardia with frequent ventricular ectopy. Treatment with an isoproterenol drip and magnesium converted the rhythm to sinus. The patient remained intubated and was transferred to ICU. An automatic implanted cardioverter defibrillator was placed, as this event was thought to correlate with his episodic symptoms.
Discussion:
TdP is a polymorphic ventricular tachycardia caused by a ventricular reentrant phenomenon associated with underlying prolonged QT interval. Acquired QT prolongation can occur in the setting of many common anesthetic drugs – indeed, in the majority of patients after general anesthesia(1). One study of children with LQTS observed 3 arrhythmias out of 114 anesthetics in 76 patients, but only 1 was clearly TdP. All events occurred during emergence from volatile anesthesia with reversal of neuromuscular blockade and ondansetron administration(2). Our case included similar medication risk factors in a patient with prior cardiac surgery and a structurally abnormal heart. Dexmedetomidine has not been shown to prolong the QT interval in previous studies(3). Nonetheless, we feel that it played a critical role in this case by producing severe bradycardia and pause dependent QT prolongation leading to R-on-T and TdP.
References:
1. Nagele P, et al. Anesthesiology. August 2012;117(2):321-328.
2. Nathan AT, et al. Anesth Analg. May 2011;112(5):1163-1168.
3. Chrysostomou C, et al. Intensive Care Med. May 2010;36(5):836-842.