Brugada Syndrome: A Sudden Cardiac Death Nightmare

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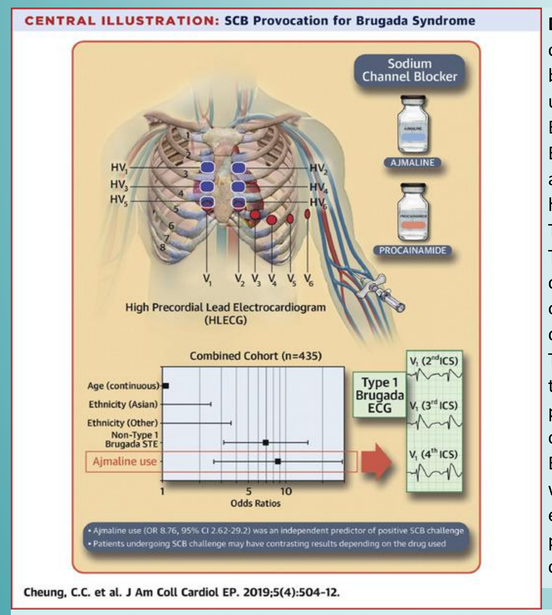
Learning Objectives:

1. Understand the pathophysiology of Brugada Syndrome and associated Brugada Patterns.
2. Learn anesthetic considerations for a patient with Brugada Syndrome.
3. Understand a pre-operative "drug challenge" for Brugada detection.

Introduction: Brugada electrocardiogram (EKG) pattern is a rare rhythym prevalent in 0.1-1% of the population, with Brugada Syndrome (BrS) being present in 10% of those patients.[2] A prospective review of 1545 patients revealed 10% had a negative outcome event and presented that men and Asian populations are at a higher risk.[7] BrS is an autosomal dominant inherited disorder of absent or abnormal ion channel function that commonly presents as sudden cardiac death (SCD).[2]

Classic Brugada pattern shows ST segment elevation and a pseudo right bundle branch block pattern in leads V1, V2, V3. There are 3 types of Brugada pattern, as shown in Figure 3., with Type 1 being the most common and diagnostic.[5]

Common causes of BrS are heart abnormalities, cocaine use, and electrolyte imbalances specifically epicardial surface of R ventricular outflow tract (RVOT) . Symptoms often include dizziness, fainting, stridor, especially at night, irregular heartrate, tachycardia, and seizures. There are many common anesthetic drugs that must be avoided in this patient population, leading to an interesting change in routine anesthetic management.[4]



Drug Challenge: Diagnosis of BrS can be done by a sodium-channel blocker (SCB) challenge. SCBs are used to unmask Type 1 on an EKG. Commonly used SCBs for Brugada Drug Challenge are ajmaline and procainamide, with higher rates of success inducing Type 1 Brugada with ajmaline.[1] The drug challenge creates conduction delays between parts of the ventricles and the RVOT, due to the depolarization theory. The challenge can also induce the transmural gradient of the action potential by shortening it, which can manifest in Brugada pattern EKG.[2] Diagnosis of BrS is achieved when a Type 1 Brugada electrocardiographic pattern is produced from the SCB drug challenge.[1]

Figure 1. Figure 1 shows a standard 12-lead ECG and high precordial lead electrocardiography. While the infusions were running, these tests are performed at baseline and repeated at 10-min intervals. Post infusion completion, they are repeated at 30-min intervals for 1 hour.[1]

Case Presentation: A 29 yo Caucasian male p/w hearing loss for a Tympanoplasty under GETA.

PMH: Hx of cocaine + ketamine abuse, denies drug use for 6 months. Has had several ER visits for "heart problems while intoxicated," and has seen a cardiologist once who diagnosed possible BrS.

Labs: preoperative electrolyte panel WNL, 12 lead EKG revealed significant ST elevations, as seen in Figure 4.

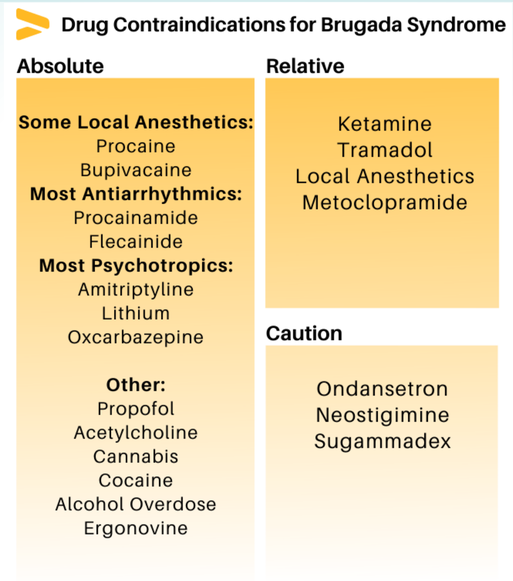


Figure 2. Drug contraindications for BrS. Most Local anesthetics (LA) should be avoided as there may be poor outcomes due to their interactions with Na+/Ca++ channels, however lidocaine used as a LA with epinephrine seems safe to use.[6] Propofol promotes arrhythmogenic effects. Patients are at a high risk for arrhythmic SCD while receiving extremely high doses of propofol, although it may be safe to use for induction and maintenance of GA. Propofol may even acutely cause a rebalancing of ion channel currents, reducing ST segment elevation.[3] Ketamine may increase ST elevation and worsen BrS. While Tramadol is not recommended, other narcotics are safe for use. Ondansetron's potential to prolonged QT may be of concern. Neostigmine is not recommended due to its parasympathetic activation. Sugammadex is relatively new and has little data to support its use.[5]

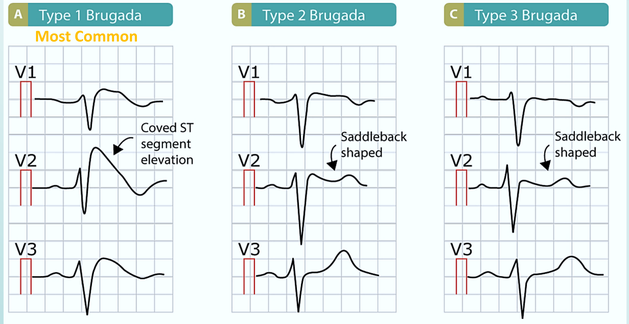


Figure 3. Types of Brugada patterns with associated characteristic ST segment elevations. Type 1 features ST segment elevation in a coved, or "shark tail", shape, a down-sloping second R wave, and an inverted T wave. Patients with Type 1 are at a higher risk for ventricular fibrillation and ventricular tachycardia that leads to SCD. Type 2 and 3 feature ST segment elevations with a saddleback shape, and Type 2 seeing a >2mm height in its elevation.[8]

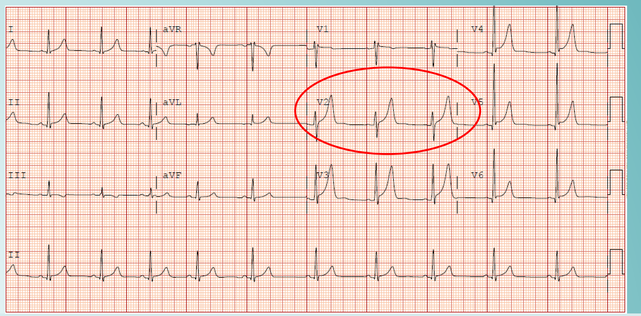


Figure 4. 12 Lead EKG findings show Sinus rhythm, LVH by voltage, ST elevation with a probable normal early repolarization pattern, Tall T waves indicative of a possible metabolic/ischemic abnormality. Unspecific changes compared to previous EKGs.

Sevoflurane	(%)	[2.3]	[2.6]
midazolam	(mg)	2	
fentanyl	(mcg)	100	
rocuronium	(mg)	35	
dexamethasone	(mg)		10
hydromorphone	(mg)		
remifentanil in…	(mcg/kg/min)	0.1	0.1
ceFAZolin IV	(g)	2	
etomidate	(mg)	10	
ePHEDrine	(mg)		
Lactated ringers	(mL)	/	
Temp			[35.9]

Figure 5. Intraoperative medications used. Known BrS exacerbating agents were avoided.

Discussion: This case illustrates the perioperative course of a rare condition that required a uniquely tailored anesthetic including monitors, medications, dosing, and other interventions. A cardio stable induction was performed, defibrillator pads were placed, and maintenance drugs were minimized. Contraindicated drugs and exacerbating agents were avoided while pain and patient comfort were adequately managed. This presentation shows a case of BrS with adequate anesthetic management that led to patient discharge with no adverse events.

References:

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