The baffling issues of Brugada electrocardiogram pattern for anaesthesiologist!

Sir,
Recently, we had a 64 year 50 kg old male patient who was posted for Laparoscopic low anterior resection. ECG [Figure 1] showed J point elevation in leads V1 to V3, described as Brugada pattern. Echocardiogram showed mild aortic regurgitation with normal systolic function and no regional wall motion abnormalities.

Brugada syndrome is an autosomal dominant genetic disorder affecting ion channel of the heart. Patients with Brugada syndrome are at risk for fatal ventricular arrhythmias. Characteristic ECG abnormalities are incomplete right bundle block with ST-segment elevations in the anterior precordial leads. The ECG abnormalities may be missing in 30% of patients[1] Obvious causes of ST segment elevation in anterior chest leads like an acute myocardial syndrome should be eliminated before contemplating the possibility of Brugada syndrome.[2] Clinical presentation of patients with Brugada ECG pattern includes patients with life-threatening arrhythmias as well as patients without any major cardiac events.[2]
Many patients come for surgery with an automated implantable cardioverter defibrillator (AICD) in situ. Usually, patients become symptomatic around 40 years of age. There is no consensus of opinion regarding the feasibility of elective ICD insertion in asymptomatic patients. Some authors advocate that such patients should be ideally subjected to electrophysiological studies and to have an AICD if there is a high probability of inducible ventricular fibrillation or ventricular tachycardia. On the contrary, there are case reports wherein known Brugada syndrome patients had an uneventful perioperative course without ICD in situ.

We planned general anaesthesia for our patient. An external cardioverter-defibrillator and antiarrhythmic drugs were kept standby. He was given pre-medication with Midazolam 2 mg, Ondansetron 4 mg and ranitidine 50 mg intravenously (IV). Pre-induction monitors are non-invasive blood pressure, electrocardiogram (lead 11 and V5), pulse oximetry, and left radial artery cannulation under local anaesthesia. General anaesthesia was induced with thiopentone sodium 250 mg and fentanyl 100 microgram, and endotracheal intubation facilitated with vecuronium 6 mg. Anaesthesia was maintained with oxygen, air and sevoflurane (MAC 0.8-1) along with Fentanyl infusion at 50 µg/h. Right subclavian vein was cannulated. Temperature was monitored and fluid warming and body warmers were used to prevent hypothermia.

Continuous monitoring of ECG peroperatively showed no fresh changes. After adequate recovery of neuromuscular blockade shown by train of four responses, residual neuromuscular blockade was reversed with neostigmine 2.5 mg and glycopyrolate 0.4 mg.

Major issues for anaesthesiologists managing patients with the Brugada syndrome are avoiding drugs which can potentially inhibit cardiac sodium channels, and managing AICD, if present, in the perioperative period. It is important to maintain adequate depth of anaesthesia since autonomic changes can precipitate arrhythmias. ECG with specific lead II helps in arrhythmia detection.

Selection of acceptable drugs is crucial in Brugada syndrome patients. For quick reference, one can search the website Brugada Drugs.org developed by Amsterdam academic medical centre department of cardiology. Beta blockers and alpha agonists should be used with extreme caution in the perioperative period since they can unmask Brugada ECG pattern. We have used thiopentone sodium for induction as we were worried about the propensity of propofol to cause ion channel dysfunction. Safety of sevoflurane as an anaesthetic agent is well documented since it does not prolong the QT interval. Vecuronium bromide was used because of its known cardiac stability, and has been shown to be safe in Brugada patients. It must be recognised that local anaesthetics block sodium channels and can be lethal in Brugada patients. However no changes were observed on local infiltration with ropivacaine 0.2% in our patient.

Interestingly in a recent retrospective clinical observational study involving 12-year case series, no arrhythmogenic activity was observed with the use of drugs which are recommended to be avoided. However, the theoretical risk is very serious and warrants precaution in using drugs which can potentially cause ECG changes.

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Conflicts of interest
There are no conflicts of interest.
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