ROYAL COLLEGE of PHYSICIANS of EDINBURGH

Evening Medical Update: Cardiology Conundrums
January 2022

Acute Management of life threatening arrhythmias – Dr Anna Maria Choy, Consultant in Cardiovascular Medicine, University of Dundee & Ninewells Hospital

In patients who have successful DCCV in the ED, would you recommend they are given an antiarrhythmic post-DCCV to prevent recurrence?

Not usually, treat the cause first and only consider AAD only if it recurs, and the patient needs to be stabilised acutely. If the ventricular arrhythmia occurs in the setting of an acute MI (up to 48h), the trigger is the acute MI/ischaemia, and an antiarrhythmic drug (AAD) is not usually required following optimal ACS treatment (including betablockers antiplatelet therapy, primary PCI or thrombolysis as indicated). Ventricular arrhythmia occurring in this setting is unlikely to be recurrent in the long term, unless the MI is complicated by LVSD, with LVEF <40%, or LV aneurysm where the first line treatment of the VA in these situations would be an ICD and not an AAD. The only time an AAD is to be used in the ER is if the VA is recurrent and the patient need to be stabilised. The management to stabilise the patient includes treatment with beta-blockers and either amiodarone or lignocaine in ischaemia driven VAs (avoid in heart failure because of increased risk of toxicity) and treating the underlying cause. As a general rule, AADs may be used as adjunctive therapy in the management of ICD patients with recurrent VA to reduce ICD shocks but must be used with caution because of potential adverse effects.

Is it ever a bad idea to give Mg in a broad complex tachy? Or just always give as a low risk intervention that might help?

Magnesium is generally safe (if administered properly by infusion) and does not adversely impact cardiac function. It is recommended by guidelines for specific arrhythmia namely for the prevention and management of perioperative atrial fibrillation and flutter for thoracic surgical procedures (J Thorac Cardiovasc Surg, 2014;148:e153–e193), and in TdP (doi: 10.1093/europace/euv319. Epub 2015 Aug 29.). There is some evidence that is can increase the threshold for VF and can supress ventricular ectopy but the strongest evidence if for polymorphic VT (TdP). Nevertheless, as many patients with sustained VA (up to almost 40%) have low magnesium, which is associated with diuretic use, and because of its low risk, giving Mg in a WCT can be considered to be helpful.

Could magnesium be administered even if QT is not prolonged?

Yes, see previous answer

Is the 2% Lignocaine local anaesthetic vial regularly available on the ward licensed for IV use in an emergency?

Technically yes, but you need to know the volume to inject the correct dose. Better to use the preloaded syringes in the resuscitation trolley.

What is the role of 8.4% Sodium bicarbonate?

To correct metabolic acidosis from prolonged cardiac arrest hypoxia. Acidosis can reduce chances of spontaneous resumptions of circulation, and is arrhythmogenic.

How do you select patients for sotalol?

Sotalol is used as adjuvant therapy in patients with ICDs to reduce VA burden and ICD therapies, especially as an alternative in patients not suitable for amiodarone. It does not have long term AEs unlike amiodarone, so should be considered in younger patients. It is safe in impaired LVSF and HF, and structural heart disease, especially arrhythmogenic right ventricular cardiomyopathy.

When would you consider over drive pacing. Is that something you would want to be called overnight for by cardio/ med registrar?
Generally, overdrive pacing is considered when the arrhythmia is incessant, or incessantly recurring when other measures are not effective. In the scenarios below:

1. If in the setting of bradycardia induced TdP, try correction of electrolytes, and isoprenaline.
2. If not related to bradycardia, try Mg and K+ infusion, to increase serum K to upper limit of normal range, maximise betablockers

Where ECG shows polymorphic VT in cardiac arrest would you presume the cause of arrest is MI and treat for this?
Acute MI is the most common cause of CA and so should be the top of the differential diagnosis, and a need to be ruled out before another cause is considered. After resuscitation of the patient, the ECG in sinus rhythm, serum troponins, history of CVD, FH suggestive of a channelopathy, current medication, and echo for regional wall motion abnormalities suggestive of MI can help clarify the underlying cause.

How can you assess the QT interval in arrhythmias? Won't it be unclear? Thanks.
The QT should not be measured in the QRS complex during VA, unless there are intermittent beats of SR, or after SR is restored. Review of historic ECGS when available can be helpful if a genetic channelopathy is suspected.
In AF, the QT tends to shorten, and usually the average QT of several consecutive beats is measured

What is the need for B-blocker after pacing for bradycardia LQTS?
Beta blockers counteract the sympathetically mediated triggering mechanisms, including suppression of ventricular ectopics, an important trigger for TdP.

Would you still consider potassium infusion in LQTS even if serum potassium levels are normal?
If the QT is still significantly prolonged and arrhythmia is recurring, particularly if drug induced, infusion to increase serum K to the upper limit of the normal range should be considered, even if baseline K+ is normal.

How would you test for other channelopathies apart from long QT?
Other channelopathies are quite rare and include Brugada syndrome and Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT). Generally clinical diagnosis is by provocation testing, e.g., Ajmaline infusion in Brugada syndrome, exercise testing, or adrenaline provocation testing in CPVT. Genetic testing is not essential to make a diagnosis and should only be considered when the clinical diagnosis is highly likely. In most of these cases genetic testing may not reveal a mutation in the panel of currently known culprit genes, but it does not exclude the diagnosis, or a genetic cause, merely that the mutation may be elsewhere in an unknown gene.

With sudden cardiac death due to myocarditis is there a typical arrhythmia which precipitates SCD?
In acute myocarditis, with the underlying pathology being acute myocardial inflammation and necrosis, the VA is usually polymorphic ventricular ectopy, VT, and even in some cases asystole when the myocarditis is so widespread, and the cardiac conduction system is involved. In survivors of myocarditis, the resultant fibrosis caused by the inflammation and necrosis can cause re-entrant ventricular arrhythmia (seen as monomorphic VA).

Recommended resource:
The ESC guidelines from 2015 in the management of ventricular arrhythmia - EP Europace, Volume 17, Issue 11, November 2015, Pages 1601–1687, https://doi.org/10.1093/europace/euv319