Quinidine: a valuable medication joins the list of ‘endangered species’

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In May 2006, AstraZeneca decided to stop the production of quinidine sulfate. Consequently, it is becoming increasingly difficult to obtain quinidine supplies in many countries. To our knowledge, neither the Heart Rhythm Society nor the European Heart Rhythm Association was informed in advance of this decision.

According to the web-page posted by AstraZeneca,1 the decision to cease production was taken ‘as the demand for their quinidine product declined considerably due to the availability of newer, more effective alternatives’. Indeed, newer class IC and class III drugs replaced quinidine in the treatment of atrial fibrillation, whereas the implantable cardioverter defibrillator (ICD) replaced practically all antiarrhythmic drugs in the treatment of ventricular arrhythmias associated with organic heart disease.

Ironically, the decision to stop quinidine production comes at a time when it is becoming increasingly evident that quinidine is the most effective—and in many cases the only effective antiarrhythmic therapy—for patients suffering from unique malignant ventricular arrhythmias including the short QT syndrome, Brugada syndrome, and idiopathic ventricular fibrillation (VF).2–4

The congenital short QT syndrome is a genetic anomaly of potassium channels. The manifestations include an abnormally short QT in the electrocardiogram, short atrial and ventricular refractory periods, and a tendency to develop spontaneous atrial fibrillation and VF. Only quinidine (but none of the ‘newer drugs’ like flecainide, sotalol, or ibutilide) significantly increases the effective ventricular refractory period and prevents provocation of VF during electrophysiological (EP) studies.5–7

The Brugada syndrome is a congenital anomaly of sodium8 or calcium channels.9 It is especially common in South East Asia, where it is the most common cause of death in young males after AIDS and road accidents. Quinidine is especially suitable to treat Brugada syndrome because: (i) it prevents phase-II re-entry and VF in the wedge preparation (animal tissue model) of Brugada syndrome;10 (ii) 76%11 to 88%3 of patients who have inducible VF at baseline EP studies are rendered non-inducible by quinidine therapy; (iii) quinidine appears to prevent spontaneous arrhythmias in high-risk patients with Brugada syndrome.3,11

Idiopathic VF is a disease of unknown aetiology that causes syncope or cardiac arrest due to VF in the absence of identifiable aetiology or electrocardiographic abnormality.12 Here too, reports of cardiac arrest survivors effectively treated with quinidine for 10 ± 5 years4,13 contrast with reports of failure with other antiarrhythmic drugs.12

It could be argued that all these patients may be treated with ICDs. In fact, the majority of them are so treated. However, quinidine is an alternative mode of therapy for those who cannot afford the expenses of ICD implantation.14 Also, the long-term risk for complications among patients undergoing ICD implantation at young age may approach 28%.15 Finally, arrhythmic storms, with multiple episodes of VF triggering multiple defibrillator shocks, may be fatal in patients with implanted ICD but are easily treated with quinidine.16–17 For these patients with short QT syndrome, Brugada syndrome, and idiopathic VF, the sudden unavailability of quinidine supplies is potentially life-threatening. It is ironic that as more editorialists endorse the use of quinidine for special indications,5,14,18 it becomes an endangered species that very soon may no longer be available.

Conflict of Interest: none declared.

References


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