**Table 1**
Baseline Characteristics of the Patients in Which the GuideLiner® Was Employed and Description of the Procedure

| | Patient 1 | Patient 2 | Patient 3 | Patient 4 | Patient 5 | Patient 6 | Patient 7 |
|---|---|---|---|---|---|---|---|
| Age, years | 69 | 68 | 74 | 72 | 43 | 45 | 75 |
| Sex | M | M | F | M | M | M | M |
| Logistic EuroSCORE, % | 8 | 15 | 8 | 30[a] | 1 | 1 | 11[a] |
| Ventricular dysfunction | Yes | No | Yes | Yes | No | No | Yes |
| Clinical indication | Resting angina | Non-Q-wave infarction | Positive ischemia test | Resting angina | Resting angina | Positive ischemia test | Positive ischemia test |
| Target vessel | LMC-AD | RC | CTO RC | RC[b] | CTO RC | CTO RC | CX |
| Duration of procedure, min | 60 | 90 | 220 | 124 | 240 | 180 | 55 |
| Fluoroscopy time, min | 12.8 | 39.9 | 55 | 43 | 118 | 80 | 27.4 |
| Contrast medium volume, ml | 130 | 200 | 230 | 200 | 370 | 320 | 270 |
| Length of hospital stay following angioplasty, days | 14 | 2 | 1 | 7 | 1 | 1 | 1 |

AD, anterior descending coronary artery; CTO, chronic total occlusion; CX, circumflex coronary artery; F, female; LMC, left main coronary artery; M, male; RC, right coronary artery.
[a] Patient no. 4 had chronic renal failure requiring hemodialysis, as well as severe peripheral arterial disease. Patient no. 7 had advanced chronic obstructive pulmonary disease.
[b] Patient no. 4 had total occlusion of the right coronary artery stent; complete right coronary artery reconstruction was performed in a previous procedure.

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## Variegate Porphyria and Atrial Fibrillation: Acute Attack Induced by Propafenone

### *Porfiria variegata y fibrilación auricular: ataque agudo inducido por propafenona*

**To the Editor,**

Porphyrias are metabolic bone diseases caused by deficiencies of enzymes involved in heme biosynthesis. Acute hepatic porphyrias (AHPs) can present as episodes of acute porphyria with abdominal pain, autonomic dysfunction (hypertension, tachycardia, and gastrointestinal disorders), and deep motor neuropathy. Variegate porphyria (VP) is a type of autosomal dominant hepatic porphyria secondary to protoporphyrinogen oxidase activity deficiency that can present acute neurological manifestations and/or cutaneous photosensitivity. Drugs are the factors most commonly implicated as triggers of acute attacks. We describe a patient with VP who received class Ic antiarrhythmic agents for paroxysmal atrial fibrillation (AF) and presented with an acute episode of porphyria, which consisted of acute abdomen and syndrome of inappropriate antidiuretic hormone secretion (SIADH).

Our patient was a 40-year-old male with a history of VP diagnosed by skin biopsy and elevation of blood aminolevulinic acid and porphobilinogen and fecal protoporphyrins who had a relative with the same condition but no activity to date. The patient experienced various episodes of paroxysmal AF. He was initially treated with flecainide but later switched to propafenone due to digestive intolerance. One week after initiating propafenone therapy, the patient began to have abdominal pain and bloody urine. He came to the emergency room and was referred to the internal medicine department for further study. During the examination, only the diffuse abdominal pain without accompanying signs of peritonism was relevant. The laboratory workup showed normal kidney function, GOT, 41 U/L; GPT, 43 U/L; alkaline phosphatase, 50 IU/L; GGT, 50 IU/L; LDH, 539 IU/L; sodium, 111 mEq/L; and plasma osmolarity, 231 mOsm/L. The urinary tests showed urinary sodium of 109 mEq/L and elevated osmolarity. The heart, thyroid, and adrenal panels were normal. An abdominal ultrasound showed no relevant findings; the Hoesch test was positive. Porphyrin and porphyrin precursor determination in urine showed an increase in porphobilinogen, as well as delta-aminolevulinic acid, coproporphyrin, and protoporphyrins in stools. Based on these findings and the normalization of biochemical and clinical parameters once propafenone was discontinued, an attack of propafenone-induced VP with SIADH as a form of expression was diagnosed. Hematin (5 mg/kg/day) was given for 4 consecutive days, and

**Table 1**
Antiarrhythmic Agents and Safety in Acute Porphyrias

| Antiarrhythmic drugs | Safety/porphyrinogenicity | Alternative |
|---|---|---|
| *Class Ia* | Unsafe | Beta blockers |
| Quinidine, procainamide | Porphyrinogenic | |
| *Class Ib* | Unsafe | Beta blockers |
| Lidocaine | Porphyrinogenic | |
| Mexiletine | Possibly porphyrinogenic | |
| *Class Ic* | Unsafe | Beta blockers |
| Flecainide, propafenone | Possibly porphyrinogenic | |
| *Class II* | Safe | |
| Beta blockers | Nonporphyrinogenic | |
| *Class III* | Unsafe | Sotalol |
| Amiodarone, ibutilide | Probably porphyrinogenic | |
| *Class IV* | Unsafe | Beta blockers |
| Verapamil, diltiazem | Probably porphyrinogenic | Digoxin |
| *Others* | Safe | |
| Digoxin, adenosine | Nonporphyrinogenic | |

beta blocker therapy was started. The patient progressed favorably and the case was presented to the arrhythmia unit to consider AF ablation.

AHPs are characterized by episodes of acute porphyria (EAP) that, if not properly treated, can lead to death. Due to the low incidence of EAP in Spain, health care professionals caring for patients who present their first EAP are unlikely to recognize the condition and, therefore, patients are often not adequately treated and may even be given drugs capable of exacerbating the attack.

In the case of VP, drug exposure plays a significant role.[1] According to the European Porphyria Initiative recommendations, class Ic antiarrhythmic agents are classified as possibly porphyrinogenic and amiodarone as probably porphyrinogenic.[2,3] Although the prevalence of AF in AHPs is low,[4] there is a significant association in its appearance. The antiarrhythmic armamentarium in these patients is limited because most drugs are unsafe and can cause EAP (Table 1). All registers include beta blockers as antiarrhythmic agents to be used in AF, above all to control heart rate, whereas only sotalol appears to be safe for the prevention of recurrences. Both digoxin and adenosine have shown no toxicity and can be used in supraventricular arrhythmias in patients with AHPs. Conversely, Méndez et al.[5] studied 17 patients with acute intermittent porphyria who used amiodarone between 2 and 20 years and concluded that amiodarone was safe. We found no literature reports of AHP associated with class Ic drugs. In patients with porphyrias and AF in whom cardiac rhythm needs to be controlled, percutaneous ablation could be proposed.[6]

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## An Unusual Etiology for Long QT

### *Una extraña etiología para el QT largo*

**To the Editor,**

Long QT syndrome is characterised by a prolongation of the QT interval on the electrocardiogram (ECG), which predisposes to the development of *torsade de pointes* ventricular arrhythmias. QT prolongation is defined as a corrected QT of >450 ms in men and of 470 ms in women.[1] There are two main groups: congenital long QT syndromes, associated with mutations in certain genes, and the acquired variant, associated with environmental factors.[2]

The main cause of acquired long QT is pharmacological, and there is a great variety of drugs associated with prolonging the QT interval.[3] Other causes include electrolyte disorders (mainly hypokalaemia, hypomagnesaemia and hypocalcaemia, toxic substances like organophosphates, liquid protein intake, endocrine disorders like hypothyroidism[4] or phaeochromocytoma, starvation, anorexia nervosa or bradyarrhythmias.