



## Flecainide Acetate Poisoning (a case report)

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**Abstract :** Drug poisoning by antiarrhythmics is considered serious with an unfavorable prognosis despite progress in intensive care. Flécaine is a class Ic antiarrhythmic drug according to the Vaughan Williams classification, its effects are responsible for cardiogenic shock by myocardial depression and rhythm and conduction disturbances that it can generate. We report the case of a woman, having ingested a dose of 6 grams of Flécaine® which was at the origin of an acute circulatory failure who did not respond to symptomatic treatment with a fatal evolution. In the absence of a specific treatment, therapeutic management boils down to the administration of catecholamines and molar sodium, which were ineffective in our case.

### 1. Introduction

Flécaine is a class Ic antiarrhythmic drug according to the Vaughan Williams classification of antiarrhythmics. It has a membrane stabilizing effect by blocking sodium channels. The immediate consequence is the inhibiting of the action potential, this inhibition will have negative chronotropic, dromotrope and inotropic effects on the heart. Pro-arrhythmogenic action secondary to the prolongation of the QT space. In addition to these effects, there is a vasodilator action by blocking the calcium flow of the smooth muscle cell.

Flecaine poisoning remains rare: 0.1-0.6% of all drug poisoning but potentially serious with an estimated mortality rate of 22.5% <sup>[1]</sup>

We report in this article a case of a determined suicide attempt in Flécaine in a young woman of 34 years old. The ingestion of this drug was approved and

based on the arguments of the history, clinical signs and data of the electrocardiogram, this in the absence of confirmation by toxicological test. Evolution was quickly fatal despite the resuscitation measures.

## **2. Case observation**

This is a 34-year-old young patient without any pathological history, accompanied by her parents to the medical emergency department of the Oran University Hospital (Algeria) on the grounds of a sudden deterioration in her state of consciousness.

The questioning rediscovered the notion of taking medication two hours before admission, two boxes of flecaine (six grams) following a family conflict. This medication is prescribed for the patient's father on the grounds of arrhythmogenic heart disease.

### **H<sub>1</sub> (in the medical emergency reception unit)**

The clinical examination revealed a comatose patient with a score of 08/15 on the Glasgow scale (verbal response: 2, eye opening: 2, motor response: 4)

The pupils were isocorous reactive with a weak cough reflex. No sensitivomotor deficit was noted, the patient was dyspneic with polypnea at 22 cycles per minute without rattles on auscultation.

However, the hemodynamic state was precarious with blood pressure at 50/30 mm Hg, heart rate at 160 beats / minute with an irregular rhythm, poorly perceived in the extremities which were cold and cyanotic. SPO<sub>2</sub>: 85% in ambient air when capillary blood glucose levels were normal.

The action to be taken was immediately:

- Hospitalization in the vital emergency room
- Two large-caliber venous lines and standard biological assessment.
- Vascular filling: 500cc of 0.9% saline serum.

- Management the upper airways: tracheal intubation and assisted ventilation with pure oxygen (fio<sub>2</sub>: 100%).
  - Transfer to intensive care unit.

### **In intensive care unit**

The shock persisted despite the first initiated vascular filling (total volume 2000 ml) with a blood pressure of 65/35 mm Hg associated with signs of peripheral hypoperfusion. The electrocardiogram (ECG) showed a rapid heart rate with wide QRS (22 seconds) at 160 / min: aspect of Ventricular Tachycardia (VT).

The action to be taken called for an emergency external electric shock (CEE) at 150 joules and 84% sodium bicarbonate infusion: 250cc in 30mn. Persistence of VT elicited a second EEC with an increase in intensity to 250 joules followed by the injection of adrenaline at 1 mg i.v then 2 mg / h as a continuous infusion in view of circulatory inefficiency.

### **In intensive care unit (H<sub>2</sub>)**

We note the onset of Bradycardia at 45 / min followed quickly by asystole which required specialized cardiac resuscitation with external cardiac massage (30 massages for 2 breaths) and administration of adrenaline 1 mg IV / 5 min.

A reperfusion of sodium bicarbonate was decided but without results. Resuscitation was suspended after 30 minutes of circulatory inefficiency.

### **3. Discussion**

We are witnessing a severe form of drug intoxication by:

1 - The nature of the toxic substance: this is a drug with a cardiac tropism with a membrane stabilizing effect (quinidine-like) responsible for a state of shock linked to a negative inotropic effect associated with a vasoplegic component.

2 - The very large ingested dose: a supposedly absorbed dose of 6 grams represents severe intoxication (recommended therapeutic dose: 200 mg / day)

which can rapidly become life-threatening. The power of toxicity of this molecule depends on the ingested dose and its plasma concentration <sup>[1]</sup>.

3 -the clinical presentation is a severe double neurological and cardiovascular syndrome:

Our observation confirms the myocardial depressant effect of Flécaine and the resulting cardiogenic shock. This serious intoxication was the cause of rhythm and conduction disturbances, further aggravating the clinical status.

We have indeed noted in our patient, automaticity disorders (bradycardia), disorders of pathognomonic conduction at the ventricular level (wide QRS).

Intraventricular conduction disorders with wide QRS and prolongation of the QT space are frequent in these intoxications, excitability disorders such as atrial fibrillation, ventricular extrasystoles, and even ventricular tachycardia or fibrillation, with passages in Torsade de pointes by prolongation of the QT have been noted in several observations <sup>[2, 3]</sup>.

4 -resistance to symptomatic treatment

We instituted exclusively symptomatic therapy in our patient by default of specific antidotes. The literature also suggests a purely symptomatic treatment starting with gastric evacuation by lavage which may be effective. In our observation, the gastric lavage was not performed due to the admission delay (greater than 2 hours) and the initial disturbance of consciousness.

The treatment of cardiogenic shock recommended the administration at a continuous infusion of catecholamines (inotropes +, chronotropes +) consisting mainly of adrenaline, isoprenaline and if necessary by dobutamine <sup>[2]</sup>. The patient received a continuous infusion through the central femoral vein of adrenaline from the outset at a rate of 3 mg / hour, the treatment of the membrane stabilizing effect uses molar sodium, whose action would be related to the addition of sodium. Sodium bicarbonate 84% was given twice but without results in this case. Electro systolic training is also generally ineffective given the increased stimulation thresholds.

Resistance to the symptomatic treatment introduced in our patient could be related to the very large ingested dose and probably to metabolic acidosis (gas analysis not performed by technical fault) linked to the perpetuation of the shock.

#### **4. Conclusion**

Flecaine acetate poisoning remains rare but serious due to its complications which are resistant to symptomatic treatment with the rapidity of the onset of the clinical status. It is a difficult purification toxicant with a margin between the therapeutic dose and the toxic dose very narrow. There is currently no specific effective treatment.

Our observation clearly illustrated the therapeutic impasse faced with this serious intoxication characterized by a shock refractory to the various therapies offered.

#### **5. Bibliographical references**

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