Alcoholic Torsade de Pointes

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INTRODUCTION

Torsade de Pointes (TdP) is a paroxysmal life-threatening ventricular arrhythmia in a very strong relation with increases of the QT interval at ≥ 0.60 sec values. The clinical correlation established an increased risk of TdP in conditions where the QT prolongation is frequent and some of these conditions such as congenital long-QT syndrome, poisoning with organophosphorus compounds, hypokalemia, hypomagnesemia, severe bradycardia and AV blocks are well known in TdP occurrence. Female is also a relative risk factor because of frequent associated hypomagnesemia to this gender. Use of some drugs is associated with TdP and significant QT prolongation has been reported after class IA category of the antiarrhythmic drugs, thioridazine, amantadine, vincamine and psychotropic drugs administration. Undoubtedly, more drugs will be added to the list in the future but now alcohol intoxication in apparently healthy or cardiac subjects is still controversial and ignored in TdP genesis.

OBJECTIVES

To identify a particular etiological form of TdP as a possible and severe life-threatening arrhythmia among alcoholic persons with or without any other previous pathological cardiovascular or pharmacological conditions.

MATERIAL AND METHODS

The alcoholism (more than 80 g alcohol ingestion per day) was proved by careful history (including skilful interrogation in denied alcoholism) and the presence of the typical stigmas and abnormal biological markers (GGT>40 U/l, MCV>93 fl) in 3 women patients with frequent loss of consciousness and iterative episodes of TdP. The prior offending agent (antiarrhythmics and other drugs known to increase the QT interval) as well as the congenital QT prolongation syndrome were excluded. Diagnosis of TdP was established on Dessertenne criteria [cit. 7,10,11] including: QT interval prolongation, premature ventricular beats with Q/T phenomenon, followed by iterative runs of ventricular spindle-shaped waves (200-250/min) with phasic variation in amplitude and polarity resulting in a veritable "torsion" of the QRS complexes around the baseline. All patients were investigated by biological samples for metabolic abnormalities or electrolyte (K, Ca, Mg) and acid-base disorders. Repeated ECG recording and QT- interval determinations were performed. The cure of the episodes of TdP was by infusion of the magnesium sulphate or temporary transvenous pacing (1 case).

RESULTS

Two alcoholic women were admitted for the iterative loss of consciousness in 24 hours before admission and very frequent episodes of TdP on continuous ECG monitoring (Figure 1 and Figure 2). The third alcoholic patient was a woman with uncomplicated myocardial infarction but with prolonged QT interval and iterative episodes of TdP unexplained by dynamic ischemia or prior administration of any drug (Figure 3). The association in the last case of the paroxysmal atrial fibrillation before and after the occurrence of myocardial infarction as well as the progression to the dilated cardiomyopathy were additional arguments for arrhythmogenic and cardiodepressive effects of the alcohol. The QT interval was ≥ 0.60 sec and resulted in normal range after 3-5 days in all cases. The values of GGT were above 40 U/l in all cases with a peak value of 226 U/l but returned in normal range after maximum 14 days of alcoholic abstinence. MCV was above 93 fl in all cases. Alcoholic abstinence and usual therapy mentioned above succeeded in the control of the arrhythmia in all cases but after at least 5 episodes of TdP in 2 cases. The cessation for a long term of the alcohol ingestion resulted in the absence of any arrhythmia for the 2 to 10 years follow-up (case 1 and case 3, respectively).
Figure 1. Paroxysm of TdP recorded at an alcoholic female patient with iterative loss of consciousness. Premature ventricular beats with R/T phenomenon (A). Typical aspect of TdP with “torsion” of variable in amplitude and polarity QRS complexes around the baseline (B). Note the prolongation of the QT interval at 0.60 sec (C).

Figure 2. Paroxysm of TdP recorded at a female patient who denied alcoholism but in the presence of alcoholic stigmas and suggestive biological markers for alcoholism: GGT= 226 U/l, MCV= 97 fl. Note the dominant bigeminal rhythm with R/T phenomenon and QT interval =0.60 sec (bottom record). Asymptomatic after alcohol ingestion cessation and normal ECG recording (QT interval = 0.40 sec) in two years follow-up.
DISCUSSION

Since 1986 an obvious relation was established by Haissaguerre [8] between the occurrence of severe ventricular arrhythmias (including TdP) and the alcoholic intoxication in apparently healthy subjects. In 1993 the QT prolongation and sudden cardiac death have been reported in patients with alcoholic liver disease [4].

In 1996 the interrogation at the European Torsades de Pointes Study Group (EURTOP) [18] concerning a relation between TdP and heavy alcohol consumption didn’t confirm our likewise supposition.

Almost in all cases the alcoholic intoxication occurs over a long time use of alcohol consumption. Some pathological and metabolic abnormalities are induced by alcohol in the structure of the myocardial cells resulting in the increase of the action potential duration.

The most arrhythmogenic causes are the prolongation of the QT interval and hypomagnesemia both induced by alcohol at heavy consumers [13,14,17]

The three cases presented in our brief communication come to support the alcoholic etiology of TdP. The fact that the patients are females was in concordance with clinical expectations: the increased risk of TdP, hypomagnesemia and susceptibility to alcohol consumption are more frequent to the female gender.

An obvious correlation among alcohol consumption, hypomagnesiemia, long-QT interval on ECG and possibility of TdP occurrence is illustrated in Figure 4.
The occurrence of TdP was in absence of other pathological conditions in two cases but alcohol could be a cumulative contributor to the release of a such severe arrhythmia (case 3).

Complete abstinence is the best and sure cure in prevention of TdP recurrence proved by a ten years follow-up.

CONCLUSIONS
TdP is a uncommon but insufficient recognized arrhythmia among alcoholic patients. TdP as a particular and specific arrhythmia caused by alcohol must be evoked when unexplained syncope and apparent death occur to a chronic and excessive consumer. Severe arrhythmia did not repeat if the alcoholic abstinence was complete.

It is proposed the term of alcoholic torsade de pointes as a new etiological form of TdP.

BIBLIOGRAPHY

18. *** European Torsade de Pointes Study Group EURTOP, 100131.2672@compuserve.com or G.Butrous@sghms.ac.uk

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