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1 In the last decades , number of drugs were withdrawn due to cases of TdP , leading to intensify assessment of drug QT interval .

2 In our case , hydroxyzine was identified as the only drug able to induce QTc interval prolongation in the patients active medication .

3 Moreover , a hydroxyzine overdose was detected probably due to high posology , bile ducts obstruction , and liver impairment -LRB- rise in GPT -RRB- .

4 Cetirizine is the hydroxyzines major active metabolite ; unfortunately , its blood level dosage was not performed .

5 We suppose that combination of hydroxyzine overdose and transient renal failure could have lead to cetirizine accumulation .

6 However , cetirizine seems to be safe regarding ventricular repolarization , even at supratherapeutic dose .

7 Theres only one published clinical case of hydroxyzine-induced QT prolongation .

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8 This case describes recurrent syncope during hydroxyzine treatment at usual posology in a 34-year-old woman presenting a hERG mutation .

9 hERG gene codes for the subunit of a potassium channel -LRB- I Kr -RRB- implicated in the ventricular repolarization .

10 Experimentally , it was shown that hydroxyzine could be proarrhythmic by blocking hERG channels and therefore prolonging action potential duration , thus inducing TdP .

11 In the French pharmacovigilance database , we found 24 cases of QT prolongation mentioning hydroxyzine with a suspected accountability assumed in 7 cases .

12 We publish here the first clinical case of hydroxyzineassociated QT prolongation with drug dosage data .

13 The association of hydroxyzine , especially at high posology , and others already well-known TdP risks factors -LRB- including hypokalemia and bradycardia -RRB- could induce QT prolongation and lifethreatening TdP .

The present work underlines the recent addition of

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