Polymorphic Ventricular Tachycardia Induced by Cetirizine Hydrochloride

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Citation

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Abstract

Torsade de pointes (Tdp) associated with first generation antihistamines is well-known but due to non-sedating antihistamines is rare. Terfenadine and astemizole are the main culprits but ventricular tachycardia (VT) due to cetirizine hydrochloride to our knowledge is not described due to its cardiac action not leading to QTc prolongation or induce Tdp. We describe a patient who presented with ventricular tachycardia (VT) while he was on cetirizine and itraconazole for his skin disease.

CASE REPORT

A fifty-year old Indian patient working in a store was brought to our accident and emergency department with history of burning chest pain and palpitations since morning. He was found to be pulseless, in shock but conscious. Cardiac monitor showed wide QRS tachycardia (Fig1).

{image:1}

He was DC- shocked repeatedly, a total of 7 times but without any response. He received 3 bolus doses of Amiadarone each of 150 mgs followed by further 300/360 J shocks with only a transient response. At this stage patient was given 2gms of magnesium sulphate IV boluses but again with no response. The resident in ER (emergency room) tried two boluses of Lidocaine each of 100 mgs but without any response. At this stage patient was shifted to our ICCU drowsy with VT rate 160/min and BP of 70/50. An attempt to introduce a transvenous pacemaker for pacing to control VT failed because of inability to achieve a venous access since the patient was very irritable. It was decided to continue amiodarone infusion 1.2 g/24hrs and fortunately for us addition of IV Inderal 1mg helped us to convert VT to NSR (normal sinus rhythm) (Fig2).

{image:2}

The monitor was still showing short episodes of VT, later replaced by ventricular bigeminy, which fully resolved in next 12 hours.

CVS (Cardiovascular system) examination revealed normal heart sounds, with no S3/S4. On the second day of admission, patient was in mild left ventricular failure (post-CPR effect) with normal vital signs. Chest X-ray confirmed pulmonary congestion with normal heart size. Serum urea and creatinine were mildly elevated which normalized during the hospital stay; serum electrolytes were normal all through; serum Magnesium was 1.1.CK/LDH(CK,75,3089,CK-MB 65,73) and LDH(340,540,320) were both elevated. Blood counts showed mild leucocytosis(14.5) only ,PLT 270 and Hg of 14.5 g%. Electrocardiogram post-conversion did not confirm acute myocardial infarction(MI); echocardiographic evaluation on the second day did not reveal any evidence of valvular disease or wall motion abnormalities.

He was managed as a case of MI till second day when we could take a full history, which revealed that he was suffering from psoriasis; recently he had cough and sneezing for which he had been prescribed cetirizine 10 mg once a day as also itraconazole100 mg a day since one week. These medications were discontinued once patient was admitted to CCU.

Subsequent hospital course was uneventful; he was discharged home without any medications. He was seen in outpatient department after two weeks having no cardiac complaints and with a normal ECG and echocardiogram.

DISCUSSION

Second - generation antihistamines are lipophobic and offer the advantages of a lack of CNS and cholinergic effects such as sedation and dry mouth, which are commonly seen in

first-generation antihistamines. Their longer duration of action also enables more patient- friendly dosing regimens which increases patient compliance. Terfenadine has been withdrawn from the U.S market since 1997,4 second generation antihistamines are currently widely available: astemizole, loratidine, cetirizine and fexofenadine. Terfenadine and astemizole both produce significant cardiac QT prolongation that may progress to a rare but fatal ventricular tachycardia known as torsade de pointes ([3,4,5]).

While terfenadine has been withdrawn due to its adverse side effects profile, significant warnings were issued for astemizole ([4,6]). These two drugs prolong cardiac repolarization when the elimination is impaired such as by liver disease or drugs that inhibit 3A family of cytochrome P450. Prolongation of cardiac repolarization is due to blockade of one or more of cardiac potassium channels that determine the action potential duration ([4]). Pharmacokinetic profiles of loratidine and cetirizine are reflective of the advantages of these agents as non-cardiotoxic antihistamines, as they do not prolong repolarization or induce torsade de pointes([1,2]).So in our case the occurrence of VT due to cetirizine is surprising. However, since patient was on itraconazole also it might have raised the levels of cetirizine by interfering with metabolism through cytochrome enzymes,as itraconazole has been shown to raise the levels of cisapride ([4,5]) and lead to cardiac fatality. Cisapride has since been withdrawn The patient was not having any organic heart disease and no MI was documented; the VT self-terminated once the culprit drugs were withdrawn or metabolized.

We want to caution against the use of over the counter antihistamines and drug interactions which might be fatal ([5]).

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