Archives of Emergency Medicine, 1989, 6, 63–65

CASE REPORT

Prolonged Q-T interval following astemizole overdose

R. O. BISHOP & P. L. GAUDRY
Accident and Emergency Department, Westmead Hospital, Sydney, Australia

SUMMARY

Astemizole overdose has been reported to cause torsades-de-pointes and in the present case it caused prolongation of the Q-T interval. Astemizole overdoses should be managed in a similar way to overdose with other cardiotoxic drugs.

INTRODUCTION

Antihistamines have not typically been associated with cardiac arrhythmias, but a newer H₁ receptor antagonist, astemizole was reported to cause torsades-de-pointes following overdose (Craft, 1986). The present case report is an astemizole overdose which resulted in prolongation of the Q-T interval. Astemizole is an H₁ receptor antagonist which has limited sedatory effect due to poor blood-brain barrier penetration. It also has little anticholinergic or betaadrenergic blocking effects. Its long half life (up to 100 h) and active metabolites make it suitable for once daily administration. The combination of these properties give it significant advantages over older antihistamines in the treatment of atopic conditions such as allergic rhinitis. It is therefore expected that its use will become more widespread and as a consequence there will be an increased availability for intentional and accidental overdosage.

CASE REPORT

A 16-year-old girl took 25 tablets of astemizole (total dose 250 mg) following an argument with her mother, 2 h prior to presentation to the accident and emergency department. She complained of slight drowsiness but had no other symptoms and examination was normal. Thirty ml of Ipecac was given on admission. The patient's electrocardiograph (ECG) was monitored and a 12-lead electrocardiograph was performed (Fig. 1). This showed sinus tachycardia (100/min) with a Q-T interval of 0·45 sec and Q-Tc of 0·59 sec.

The patient was subsequently given oral charcoal and admitted for continuous ECG

Correspondence: Dr P. L. Gaudry, Director, Accident and Emergency Department, Westmead Hospital, Westmead, New South Wales 2145, Australia.
monitoring. Her serum biochemistry was normal (serum potassium was 3.8 mmol/l and calcium 2.31 mmol/l). She was monitored for 48 h, during which time no arrhythmias were noted. During this time, the Q-T interval returned to normal, she had developed no other side effects of the overdose and she was discharged.
DISCUSSION

Electrocardiographic changes have long been used as a guide to the severity of certain drug overdoses, in particular tricyclic anti-depressants (Gosselin et al., 1976). Of recent interest has been the production of a prolonged Q-T interval and the precipitation of the ventricular arrhythmia known as torsades-de-pointes by certain drugs such as antiarrhythmic agents, antidepressants and phenothiazines (Stratmann & Kennedy, 1987). In a clinical trial of astemizole in standard dosages (30 mg per day), there were no electrocardiographic effects (Craft et al., 1987). In one reported astemizole overdose, a 14-year-old girl took 200 mg and again there were no electrocardiographic effects (Kingswood et al., 1986). However, the case reported by Craft et al. (1986), and the present case report, suggest that astemizole overdoses are not benign, and they should be managed in a similar way to overdose with other cardiotoxic drugs. All patients should receive therapy aimed at decreasing absorption (oral charcoal) and all should have a 12-lead electrocardiograph and continuous ECG monitoring until any ECG abnormalities present are reversed.

Unfortunately, the limited clinical experience of astemizole overdoses makes it impossible to be more specific about therapy. Questions such as precisely how long these patients should be monitored and whether the Q-T interval can be used as a guide to severity or the likelihood of torsades-de-pointes remain to be answered. Similarly the management of astemizole-induced torsades-de-pointes remains empirical. Experience from other drug overdoses suggests that overdrive pacing or isoprenaline infusion would be favoured modalities (Stratmann & Kennedy, 1987).

ACKNOWLEDGEMENTS

The authors thank the Poisons Information Centre at The Children’s Hospital at Camperdown and the Pharmacy Department at Westmead Hospital for advice on the potential toxic effects of astemizole.

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