letters to the editor

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Yew tree poisoning: a near-fatal lesson from history (1)

Editor – We read with interest and concern Jones *et al*'s case report of yew tree poisoning in which they appear to advise induced emesis in the management of overdose (*Clin Med* April 2011 pp 173–5), a therapy not advised by the UK National Poisons Information Service (NPIS) for over 20 years. While syrup of ipecac was used historically in management of poisoned patients, the American Academy of Clinical Toxicology and the European Association of Poisons Centres and Clinical Toxicologists published a position statement in 2004 no longer recommending routine administration of syrup of ipecac in poisoned patients.[1] This was as a result of limited evidence for improved outcome following its use in clinical studies, together with a significant risk of aspiration particularly in those with reduced consciousness. Equally importantly administration of syrup of ipecac may delay administration or reduce effectiveness of activated charcoal, the gastric decontamination agent of choice.

Yews (*Taxus spp., Taxaceae*) are poisonous evergreen shrubs common throughout the UK. Toxicity is primarily related to the cardiotoxicity of taxine alkaloids, present in all plant parts except the scarlet aril (berry).[2] Management of yew poisoning is largely supportive as no antidote exists. We would remind readers that TOXBASE®, the online database of the NPIS, carries current advice on best practice in poisons management.[3] After charcoal, further management includes fluid resuscitation, monitoring of cardiac rhythm and, in severe cases, inotropic support and cardiac pacing. Use of digoxin-specific FAB antibody fragments has been proposed on account of structural similarity between digitalis and the taxine molecule.[4] NPIS advice on TOXBASE reflects this publication but stresses that there is no information available to guide dosing.

This case report describes a rare cause of poisoning in the UK but highlights the wider issue of gastric decontamination in poisoned patients, and that use of syrup of ipecac has now become obsolete. We strongly recommend that your readers obtain up-to-date advice from NPIS when managing rare poisons.

EUAN SANDILANDS
Specialist registrar, clinical toxicology

NICK BATEMAN
Professor in clinical toxicology and director

NPIS Edinburgh

References

1 Anonymous. Position statement: ipecac syrup. *Clin Toxicol (Phila)* 2004;42:133–43.
2 Wilson CR, Sauer JM, Hooser SB. Taxines: a review of the mechanism and toxicity of yew (*Taxus spp.*) alkaloids. *Toxicon* 2001;39:175–85.
3 TOXBASE®. National Poisons Information Service. www.toxbase.org
4 Cummins RO, Haulman J, Quan L *et al.* Near-fatal yew berry intoxication treated with external cardiac pacing digoxin-specific FAB antibody fragments. *Ann Emerg Med* 1990;19:38–43.

Yew tree poisoning: a near-fatal lesson from history (2)

Editor – Jones *et al* should be congratulated on the presentation and successful management of a challenging clinical case (*Clin Med* April 2011 pp 173–5). The lady within the case ingested a significant amount of yew tree leaves and shoots. Ultimately this resulted in two cardiac arrests mediated by ventricular tachycardia (VT). Notably despite DC cardioversion attempts, the patient had two boluses of intravenous amiodarone. The authors comment after the first cardiac arrest that the corrected QT interval (QTc) was greater than 600 ms.

Amiodarone is established as a class III antiarrhythmic agent. Its efficacy at suppressing ventricular arrhythmias is well recognised. A large proportion of this evidence was obtained in ventricular arrhythmias following an acute ischaemic insult. However, its efficacy has also been demonstrated in cardiomyopathies. Hence due to its actions it is now recommended within the resuscitation guidelines for life-threatening ventricular arrhythmias. A well recognised side effect, among others, of amiodarone is the prolongation of the QT interval. It is also well known that QTc prolongation predisposes to torsades de pointes and sudden cardiac death.

However, with overdoses and poisonings, the situation is less clear. Tricyclic antidepressants are commonly encountered as chosen agents for potential suicide. In toxic quantities they may also potentiate the QTc interval. The currently advocated recommendations are not to use amiodarone within this context due to its ability to generate further arrhythmias and prolong QTc. Reports of yew tree derivatives and poisoning are infrequent and though the pharmacological actions are well documented, the treatment of such an episode remains unclear. Others have used magnesium sulphate and sodium bicarbonate within similar clinical contexts to correct cardiac dysrhythmia. Ultimately with a history compatible with poisoning or the deliberate ingestion of cardio active agents, it may be prudent to be cautious with the management of cardiac dysrhythmias and to avoid, if possible, the concomitant usage of antiarrhythmic agents.

Perhaps such cases should be recorded on a national or regional basis, to facilitate