# 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.

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### 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

improved recognition, understanding and management. Only if comprehensive data are collected, can worrying clinical prognosticators be identified.

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### *In response*

Editor – Sandilands and Bateman raise an important point in regard to the safety of induced emesis and rightly remind us that it is no longer routinely recommended in cases of poisoning. It was not the intention of the previous report to advocate its use as a primary management step in cases of poisoning. Indeed, it can be seen from the case report that this was not part of the management strategy which was guided both by reference to the online TOXBASE and by discussion with the National Poisons Information Service. We concede that there is only anecdotal, dated and rather tenuous basis for the induction of emesis and perhaps the mention of induced emesis in the original report belongs in the same history books as those used by our patient to glean her knowledge of yew toxicity!

In cases of rare poisoning such as this there is little evidence on which to guide management. A review of 10 years' data from the American Association of Poison Control Centers Toxic Exposure Surveillance System revealed only four cases of life-threatening complications of yew ingestion.[1] TOXBASE lists only eight references on which it bases its guidance and information to emergency departments throughout the UK. All of the measures recommended on TOXBASE and highlighted by Sandilands and Bateman were attempted but none appeared to improve the clinical situation at the time.

Given this, it is worth reporting that the young lady described in the case report has presented once again with deliberate self-poisoning with yew foliage. She absconded from the supposedly secure psychiatric unit where she was an inpatient following her previous presentation. She made her way directly to where she knew the yew trees were growing and once again consumed a quantity of shoots and leaves which she washed down with a fizzy drink which she brought specifically for this purpose. Although she was apprehended quickly she was observed continuing to consume yew leaves even after apprehension having hidden some in the pockets of her trousers. She was brought immediately to the emergency department where, because she presented within 60 minutes of ingestion, she was given 50 g of oral-activated charcoal. Although she developed a marked sinus tachycardia, she remained clinically well with a blood pressure of 125/90 mmHg and peripheral oxygen saturations of 99% while breathing room air. She was discharged back to the psychiatric unit the following day. While induced emesis may not be recommended, she has, unintentionally, provided a single patient case-control 'study' into the effectiveness of the early use of oral-activated charcoal. The lack of effect seen from late administration of charcoal is in keeping with current guidelines and published studies showing a steady reduction in toxin absorption with time.[2]

Guha is right to mention the QT prolonging and proarrhythmic potential of amiodarone. While caution with the use of antiarrhythmic medications is certainly prudent, in this case amiodarone was given in the setting of a cardiac arrest with shock refractory ventricular tachycardia (VT) (and later repeated because of the apparent success of the initial administration) and is in keeping with current UK resuscitation guidelines to provide standard advanced life support if cardiac arrest occurs. TOXBASE already acts as a comprehensive database and we hope that publication of rare case reports such as this will help to inform future practice.

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## References

1 Krenzelok EP, Jacobsen TD, Aronis J. Is the yew really poisonous to you? *J Toxicol Clin Toxicol* 1998;36:219–23.
2 American Academy of Clinical Toxicology European Association of Poisons Centres and Toxicologists. Position statement: single-dose activated charcoal. *Clin Toxicol* 1997;35:721–41.
3 Resuscitation Council (UK). *Advanced life support*, 6th edn. London: Resuscitation Council (UK), 2010.

## Angina without 'strangling and anxiety of the breast'

Editor – Cooper and colleagues remind us that cardiac pain may be present only in the neck and arm, without there being any chest pain (*Clin Med* April 2011 pp 201–2). Very rarely, cardiac pain is felt in more unusual positions. Lanza and colleagues reported a case in which cardiac pain consisted purely of headache.[1] The rarity of this presentation may be judged by the fact that the article has never been cited.

I saw a man in his mid-60s who had cardiac pain confined to the vertex of his head. (His exact age was unknown because he was born in a remote village in a developing country and there was no official record of his birth.) He had woken from sleep with sudden onset of severe pain at the top of his head. It was the worse pain he had ever experienced. It lasted about eight hours. There was no meningism or abnormal neurological findings. Subarachnoid haemorrhage was suspected but a computed tompgraphy (CT) brain scan and lumbar puncture were normal. An electrocardiogram (ECG) showed anterior ST elevation consistent with an anterior myocardial infarction. The ECG appearance was initially attributed to a subarachnoid haemorrhage.[2] That view of the ECG was not revised even after subarachnoid haemorrhage was discounted. After discharge from hospital, he reported similar but less severe pain confined to the vertex of his head when walking uphill. It disappeared almost immediately once he rested. He had identical pain associated with anterior ST segment depression during a treadmill exercise test. Coronary angiography showed a single severe stenosis in the left anterior descending