LETTER TO EDITOR

Plasma Butyrylcholinesterase as a Marker of Clinical Outcome in Diethyl Organophosphorus Insecticide Poisoned Patients Treated With Pralidoxime

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Dear Sir,

We are grateful for Lucyk and colleagues’ interest in our work on the value of plasma butyrylcholinesterase (BuChE) as a marker of pralidoxime effect. We note their interest in the association between early BuChE reactivation and clinical outcome.

Most importantly, the original randomized controlled trial (RCT; Eddleston et al., 2009) assessed the effectiveness of pralidoxime in patients proven by laboratory analysis to have been poisoned by the organophosphorus (OP) insecticide, chlorpyrifos. Both red cell acetylcholinesterase (AChE; Eddleston et al., 2005) and BuChE (Konickx et al., 2013) inhibited by chlorpyrifos are rapidly reactivated by pralidoxime. Unfortunately, in a preplanned subgroup analysis of these patients we found no evidence of benefit from pralidoxime (case fatality: placebo arm 2/42 [4.8%] vs. pralidoxime arm 6/54 [11.1%]; odds ratio 3.34 [95% CI 0.57–19.37]) despite clear reactivation of AChE in diethyl OP poisoned patients (Eddleston et al., 2009, Fig. 3).

Concerning intubation, this occurred earlier in patients receiving pralidoxime (and reactivating their cholinesterases) than those receiving placebo in the RCT (Eddleston et al., 2009, Fig. 7). Looking at patients with confirmed diethyl OP poisoning (the great majority of whom ingested chlorpyrifos), there was no reduction in need for intubation after pralidoxime administration (proportion intubated: placebo arm 6/50 [12.0%] vs. 10/62 [16.1%]; odds ratio 1.41 [95% CI 0.47–4.19], unpublished). The median (interquartile range, IQR) time to admission and treatment was 4.4 (2.9–7.6) h in the two arms of the study.

Of note, patients in the placebo arm, with no reactivation of their red cell AChE (Eddleston et al., 2009) or BuChE (Konickx et al., 2013), did better than patients in the pralidoxime arm, suggesting that even early reactivation of BuChE is unlikely to be associated with major benefit.

Due to the resources available for the study, data on less severe markers of OP poisoning (such as atropine requirement) were not recorded for the hours after pralidoxime administration. We are therefore unable to check for evidence of less severe clinical deterioration as BuChE became inhibited again.

A larger study, with more patients and more detailed collection of clinical data, may be able to provide the data Lucyk and colleagues would like to see. However, due to the lack of any apparent clinical role for BuChE in OP poisoning (Lotti, 2001), it seems highly unlikely that the BuChE activity would be a more useful marker than red cell AChE in OP insecticide poisoning.

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REFERENCES

