LETTER TO EDITOR

Reactivation of Plasma Butyrylcholinesterase by Pralidoxime Chloride in Patients Poisoned by WHO Class II Toxicity Organophosphorus Insecticides

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To the Editor,

We commend Eddleston and coworkers for investigating the utility of butyrylcholinesterase (BuChE) activity as a surrogate marker for pralidoxime effectiveness in organophosphate (OP) poisoned patients (Konickx et al., 2013). Although the authors analyzed data from two of their previous studies (Eddleston et al., 2005, 2009), only the randomized controlled trial data demonstrated significant changes in BuChE with pralidoxime treatment. Based on the data presented in Figure 3 (Konickx et al., 2013), pralidoxime administration improves BuChE within the first hour following ingestion of diethoxy OP. Also, patients administered pralidoxime rapidly following ingestion had an increase in BuChE activity compared to those with late administration (Fig. 4). Although the group analysis did not show improved mortality or reduced need for intubation (Eddleston et al., 2009), we question whether analysis of a subgroup of patients who were both administered pralidoxime early and had evidence of reactivation of BuChE would have shown an improved clinical outcome. It would also have been useful to know whether the subsequent decrease in BuChE after initial rise following pralidoxime administration correlated with either worsening clinical status or the need for other treatment interventions.

The appeal of a serum biomarker for assessing pralidoxime efficacy is to provide an objective measure of the need for ongoing treatment. We hope the authors will continue to assess whether time to pralidoxime administration postexposure and initial rise in BuChE concentrations is related to clinical status as well as mortality and/or need for mechanical ventilation.

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REFERENCES

