Secondary contamination in organophosphate poisoning

Sir,
We read with interest the article by Stacey et al. discussing their experience with treatment of organophosphate/organophosphorus (OP) pesticide poisoning.1 This is an important topic, given the large number of exposures and deaths from poisoning with this class of pesticide worldwide.2 A number of important points have been raised by these authors, on which we would like to comment.

In this case, advanced protective garments were used by treating staff which interfered with patient care, the ED was closed down temporarily, staff were taken off duty (placing a strain on other hospital areas) and the Chemical Incident Response Service were involved. Despite the delay in institution of such procedures, no person was poisoned other than the patient with intentional poisoning. The question remains: was the response taken for this incident necessary?

For secondary contamination to occur, there must be direct contact between staff and the pesticide. This may occur when the pesticide has been spilled on skin or clothes, or if contained in body fluids such as vomit and diarrhoea. OP pesticides are formulated in hydrocarbon solvents, which are volatile and have a characteristic offensive smell. The OP itself is not volatile, in contrast with OP gases used in chemical warfare, so unless the pesticide is aerosolized, the risk of OP toxicity from inhalation is low.

As noted by Stacey et al., secondary OP contamination and poisoning is most likely to occur when people (including attending relatives, emergency services or health workers) unfamiliar with these pesticides do not exercise appropriate precautions. Although the risk of secondary poisoning and steps to minimize it are discussed frequently by doctors managing these exposures, the issue is infrequently raised in the literature and not without controversy.

For example, the most publicized case on this topic was that of Geller et al.3 In the absence of measurements of cholinesterase activity, the relative contribution of pesticide (if any) and solvent has been debated.4,5 In the paper by Stacey et al., cholinesterase activity was not measured and no staff member was judged to be poisoned. Indeed, many of the symptoms they describe could be consistent with solvent effects or anxiety.

Delays in diagnosis and treatment (including decontamination) have the potential to put staff at risk of secondary contamination. In developed countries, where the incidence of OP poisoning is low, delays in diagnosis have been noted in patients who are unable to provide a history (see references 6,7 and D. Roberts, unpublished data), but adverse outcomes to staff were not reported in these cases.

In Asian and South American countries, where thousands of OP poisoned patients are treated every year and resources are limited (e.g. full protective suits with self-contained ventilation as discussed by Stacey et al. are not available in Sri Lankan hospitals), the likelihood of secondary poisoning would appear to be higher. But adverse events to staff have not been reported.

From our own experience, the risk of secondary OP poisoning is low, perhaps negligible, when universal precautions are used. We have treated >700 patients with OP poisoning in the last 2 years at Anuradhapura General Hospital, Sri Lanka. During this time, despite frequent medical review of these patients, only brief episodes of nausea and dyspnoea have been reported by doctors. These reactions are infrequent, following close contact with severely poisoned patients, and symptoms settle quickly with fresh air. Inhalation of solvent from the breath or clothes of a patient is the most likely cause for these effects. Hospital wards in Sri Lanka are largely open, which may improve ventilation and contribute to the apparent lack
of secondary poisoning of hospital staff. However, we are not aware of reports of more severe toxicity in relatives or ambulance officers who transfer patients. We have not measured cholinesterase activity in staff with close contact because there has never been a clinical or other indication to do so.

The development of guidelines for management of patients poisoned with OP pesticides must protect not only the staff, but also the patient. It is not unknown for patients who are critically ill with OP poisoning to be managed in ambulance receiving bays with minimal staff, or for patient transfers to be refused by aeromedical services. These concerns are based mostly on theoretical risks, rather than known complications, in the interest of staff safety. On the basis of current evidence, it is difficult to support such management. Similar concerns have been raised by others in response to over-cautious management of rare poisonings.8

Our recommendations for treatment of OP pesticide-poisoned patients differ from those of Stacey et al. Universal precautions will prevent most cases of secondary contamination. These should be used by all staff when treating any critically ill patient, including OP pesticide poisoning. Resuscitation and atropinization must be performed in the first instance: it is these measures that will initially save the patient's life. Dermal and gastrointestinal decontamination of the patient may then be performed. It is unlikely that staff who come into direct contact with the pesticide will suffer toxicity if they wash the affected area immediately with plain soap and water; unnecessary delays should not occur while specialized soaps are located. Chemical Incident Response Services do not appear to be necessary for routine decontamination of the environment. Treatment of patients in clinical areas with adequate ventilation may be important to prevent mild systemic symptoms from inhalation of the solvent.

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Cancer and hereditary haemochromatosis

Sir,
In their recent review,1 Limdi et al. appear to have understated the frequency of cancer in patients with hereditary haemochromatosis (HH). While everybody is aware of the risks of hepatocellular cancer in male patients with haemochromatosis and cirrhosis, the risks of non-hepatic malignancies are not widely appreciated. In one study,2 the relative risk for non-liver malignancies was 1.8 compared to non-HH liver diseases, even after exclusion of confounding factors such as alcohol, hepatitis B and C and family history, and 20 compared to the general population. A direct causal role for iron in triggering malignancy has been postulated. Even patients who are simple carriers of C282Y or H63D mutation or those who have raised ferritin but negative for the HH mutations also have higher risks of malignancy compared to the healthy population.3,4

With the wide availability of genetic screening for HH, the coming years will see many new cases of HH coming to light. This may become a major service issue in the face of non-existent liver services in many DGHs.

At our hospital, all patients with HH are entered in a database and are followed up by a specialist