Letters to the Editor

Disseminated intravascular coagulation caused by the insecticide Propoxur

Sir,
Haematological side effects due to exposure to insecticides are well known.^{1,2} Laboratory studies have shown that these insecticides and their derivatives can be mutagenic and haematotoxic.³ Exposure to Lindane and DDT have been associated with aplastic anaemia,⁴ Chlorane with refractory megaloblastic anaemia,⁵ and Propoxur combined with DDVP with acute leukaemia.⁶ Hyper- and hypocoagulability after exposure to Sarin, Parathion and Mevinphos have been observed.^{7,8} There is no published record of coagulation disorders caused by Propoxur. Recently we saw a 24 year old female after suicidal ingestion of approximately 120 ml of the household insecticide Propoxur (1% 2-isopropoxyphenyl-methyl carbamate). Immediately after taking it she repeatedly vomited, became confused and lapsed into coma. On examination, she had bradycardia, pin point pupils, frothing from the mouth and twitching of her facial muscles. No improvement was observed after repeated intravenous injections of atropine. Within 4 hours of admission, she developed gross haematuria, bleeding from nose and mouth, large confluent ecchymotic lesions over whole body, bleeding from intravenous sites and hypotension. Lumbar puncture revealed slightly raised proteins and many red blood cells. Haemoglobin was 7.9 g/dl. Total leucocyte count was $12.5 \times 10^9/l$ with neutrophils 78%, lymphocytes 20% and eosinophils 2%. Platelet count was $40 \times 10^9/l$. Prothrombin time (one stage) was more than 120 seconds (against a control of 12 seconds). Partial thromboplastin time with kaolin and thrombin time were more than 120 seconds each (against normal of 36 seconds and 13 seconds respectively). Serum fibrinogen level was 80 mg/dl and levels of fibrin degradation products were $40 \mu\text{g/ml}$ (normal less than $10 \mu\text{g/ml}$).

Blood transfusion and dopamine infusion were started, but the patient expired shortly afterwards. Post-mortem samples from liver, lungs, kidneys and heart showed alveolar oedema in the lungs, but histology of the other organs was unremarkable.

In a study of 1300 cases of anticholinesterase exposure coagulation abnormalities were not clinically significant, mild haematuria (2 cases), thrombophlebitis (1 case) and possibly coronary thrombosis (2 cases) being the only manifestations.³ This may not be entirely true and fatal bleeding may result due to consumptive coagulopathy.

Anoop Misra,
Sanjeev Kapoor,
O.P. Malhotra,
R.R. Singh

Department of Internal Medicine
All India Institute of Medical Sciences
New Delhi-110029, India

References

1. Mastromatteo, E. Haematological disorder following exposure to insecticides. *Can Med Assoc J* 1964, **90**: 1166–1168.
2. Best, W.R. Drug associated blood dyscrasias. *JAMA* 1963, **185**, 286–290.
3. Gibel, W. & Lohs, H. Carcinogenic, dermatotoxic and hepatotoxic effect of organic phosphate pesticides. *Environ Qual Safety* 1975, **4**: 232–234.
4. Sanchez-Medal, L., Castanedo, J.P. & Garcias-Rojas, F. Insecticides and aplastic anaemia. *N Engl J Med* 1963, **269**: 1365–1367.
5. Furic, B. & Trubowitz, S. Insecticides and blood dyscrasias. *JAMA* 1976, **235**: 1720–1722.
6. Reeves, J.D., Driggers, D.A. & Killy, V.A. Household insecticides associated with aplastic anaemia and acute leukaemia in children. *Lancet* 1981, **ii**, 300–301.
7. Holmes, J.H., Starr, H.G., Hanisch, R.C. & Von Kaulla, K.N. Short term toxicity of Mevinphos in man. *Arch Environ Health* 1974, **29**: 84–89.
8. Von Kaulla, K.N. & Holmes, J.H. Changes following anticholinesterase exposure: blood coagulation studies. *Arch Environ Health* 1961, **2**: 168–177.

Clonazepam-induced Tourette syndrome in a subject with hyperplexia

Sir,
Hyperplexia (HPX), or abnormal startle response, refers to a condition in which stimulus (somatosensory, auditory, or visual) results in an exaggerated jumping response. The pathophysiological mechanisms of this disorder are not understood. Fariello *et al.*¹ suggested that interruption of descending thalamic pathways with resultant disinhibition of more caudal brainstem nuclei is important in the production of abnormal startle behaviour. Andermann *et al.*² implied the existence of serotonergic abnormalities since startle-induced epileptic seizures have been successfully treated with clonazepam, a serotonin agonist. We report a patient in whom clonazepam produced Tourette-like symptoms in a patient with HPX.

This 37 year old man developed an exaggerated startle reaction at age 21 years. A shout or any other unexpected stimulus caused him to jump, to flex his upper limbs abruptly, to emit an involuntarily shout, and occasionally to fall to the ground. These startle reactions were absent during sleep. The patient's father had suffered similar symptoms since childhood. Two years before presentation, the patient accepted treatment and was given sodium valproate, which proved therapeutic, but had to be discontinued owing to marked and excessive sedative effects. Four months before presentation, clonazepam was prescribed (average dosage 8 mg/day). This produced marked amelioration of the patients' HPX. Two months later, however, the patient was seen again, for routine evaluation and disclosed frequent eye-blinking, grimacing, episodic neck flexion, arm arching and shoulder shrugging