Hydrofluoric acid burns from a household rust remover

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TO THE EDITOR: The report by Mangion et al[1] draws attention to a serious risk in the environment. The general public has been increasingly protected against the risk of harm from domestic products by a combination of legal liability actions and government regulation. Thus, the continuing availability to the general public of hydrofluoric acid (HF) in concentrations that are hazardous is something of an anachronism.

While we applaud Mangion and colleagues for raising the issue of HF burns, we feel that their article is deficient in failing to mention a number of important points.

- Topical calcium gluconate has been shown to be more effective in treating HF burns if the preparation contains dimethyl sulfoxide (DMSO).[2]
- There is a great risk of blindness with ocular exposure to HF.
- Slow local injection with 10% calcium gluconate using fine needles, titrating its effect against the patient's pain, is a well described technique. This is another treatment option that could have been tried.
- Nail removal, described by Mangion et al as an "extreme measure", is, unfortunately, often required. It is less likely to be required with the application of DMSO/calcium gluconate solution and retrograde ischaemic intravenous injection of calcium.
- Local excision of contaminated tissue may be required after exposure to concentrated solutions.
- Management should be a team effort from the first moment, involving an intensivist/toxicologist and surgeon, as burns surgeons are trained in the care of HF exposure, and surgery is often needed.

The availability, packaging, and labelling of preparations containing HF have recently been changed. Since 1 December 2001 it has no longer been possible for the general public to purchase any HF preparation stronger than 1%. All preparations now carry prominent labelling drawing attention to the risk of blindness if even dilute solutions of HF get into the eyes. Containers are now less easy to open by children.

These changes have been introduced by the National Drugs and Poisoning Committee of the Therapeutic Goods Administration as a result of an independent review and lobbying by the Australian and New Zealand Burn Association (ANZBA).

The ANZBA guidelines for referral to a specialised burns unit include chemical burns. The peculiar challenge posed by HF burns emphasises the need for the guidelines to be more widely disseminated. Currently, the New South Wales Department of Health has adopted the guidelines, so this policy is official throughout New South Wales.

1. Mangion SM, Beulke SH, Braitberg G. Hydrofluoric acid burn from a household remover. *Med J Aust* 2001; 175: 270-271.
2. Seyb ST, Noordhoek L, Botens S, Mani MM. A study to determine the efficacy of treatments for hydrofluoric acid burns. *J Burn Care Rehabil* 1995; 16(3 Pt 1): 253-257. ❑

Renal protection by angiotensin II receptor antagonists in patients with type 2 diabetes

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TO THE EDITOR: A recent editorial in the Journal attempted to define a role for angiotensin II receptor (AIIR) antagonists in patients with type 2 diabetes.[1] This was in the light of recent trial evidence that these agents reduce progression to renal failure in patients with type 2 diabetes and diabetic renal disease. Unfortunately, the guidelines provided were somewhat confusing and fragmented. I believe that a simpler treatment guide can be constructed, particularly when it is emphasised that the aim in diabetes is to use agents that prevent not only renal failure but also cardiovascular events.

Substantial evidence already supports a role for the angiotensin-converting enzyme (ACE) inhibitor ramipril in treatment of diabetes. The HOPE study included 3577 people with diabetes and another risk factor for cardiovascular disease who were randomised to either placebo or ramipril (10 mg) for 4.5 years.[2] This group had a mean baseline blood pressure of 142/80 mmHg and no clinical proteinuria. Ramipril lowered the risk of the combined primary outcome of myocardial infarction, stroke and cardiovascular death by 25% ($P \leqslant 0.001$), and this effect was independent of blood-pressure lowering. Furthermore, ramipril reduced progression to overt nephropathy by 22%, with a reduction evident in patients with or without proteinuria.[2]

This protective effect of ramipril on renal function is consistent with previous evidence that ACE inhibitors slow progression of chronic renal failure in diabetes,[3,4] and that ramipril slows progression of chronic renal failure in non-diabetic nephropathy.[5]

Whether other ACE inhibitors have the same effect as ramipril on cardiovascular events, and what doses obtain such an effect, remains speculative. Similarly, while there is now evidence that AIIR antagonists also have renal-protective effects, there is no definitive evidence that they protect against cardiovascular events.

In view of this, and in the absence of comparative trials, ramipril (and not other ACE inhibitors or AIIR antagonists) is currently the first choice for treatment for diabetic patients with hypertension, with normotension and microalbuminuria, or with hypertension and micro- or macroalbuminuria. As most patients with diabetes and hypertension require multiple agents to achieve a target blood pressure less than 130/80 mmHg, additional therapy with a β-blocker, diuretic or calcium-channel blocker is often necessary.

The significance of the newly available trial data is that AIIR antagonists provide an alternative to ramipril for reducing progression to chronic renal failure in patients with diabetic nephropathy who cannot tolerate ACE inhibitors because of the side effect of cough.

1. Jerums G, Cooper ME, Gilbert RE, Atkins RC. Renal protection by angiotensin II receptor antagonists in patients with type 2 diabetes [editorial]. *Med J Aust* 2001; 175: 397-399.

Correspondents

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