Correspondence

Fatal anaphylactic reaction to oral diclofenac sodium

To the Editor:
Diclofenac sodium is commonly used in pain clinic and peri-operatively. We report a fatal anaphylactic reaction to oral diclofenac in a nine-year-old girl hospitalised for "transient synovitis". She had no history of atopy or cardio-respiratory illness and her clinical investigations were unremarkable. After symptomatic improvement on oral nimsulide and limb traction, oral diclofenac 25 mg twice daily was prescribed at discharge. At home, after a first dose of diclofenac, she complained of body aches, itching, showed cutaneous flushing and became febrile (38.9°C). Attributing her symptoms to fever, a second dose was given within two hours. Soon, the girl became restless, developed a generalised body rash and choking sensation. Within 35 min, she was brought to the ICU gasping, cyanosed, pulseless, with mid-dilated weakly reactive pupils and was intubated. Aggressive hemodynamic and respiratory support was provided but failed to resuscitate the child.

The second dose of diclofenac probably lead to a fulminant allergic reaction while delayed hospitalisation likely proved fatal. Should routine sensitivity tests be performed with diclofenac sodium? This drug is considered safe with a worldwide experience of 7.6 million patients per year.[1] Van der Klauw *et al.* found 30 cases[2] of probable anaphylaxis to diclofenac in 992 reports on drug reaction. A fatal drug reaction following *im* diclofenac has been reported,[3] but sodium metabisulphite preservative as a cause was not ruled out. In a singular report of reaction to oral diclofenac, the patient survived due to prompt resuscitation.[4]. It is therefore essential to remain aware of the toxic profile of all drugs. Prompt recognition and treatment of serious complications can help avert such untoward outcomes.

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Effects of pirenzepine, omeprazole, lansoprazole, and rabeprazole on human neutrophil functions

To the Editor:
Anesthesia and surgery inhibit several neutrophil functions[1] involved in antibacterial host defence mechanisms. Pirenzepine (a muscarinic receptor antagonist) and omeprazole, lansoprazole, and rabeprazole (proton pump inhibitors) are administered peri-operatively for prophylaxis against aspiration pneumonitis or stress ulceration.[2–4] Critically ill patients, potentially immunocompromised, often receive these gastric protective agents for the same purpose. This is why, after institutional approvement and informed consent, we assessed chemotaxis, phagocytosis, and superoxide (O_2-) and hydrogen peroxide (H_2O_2) production of neutrophils isolated from 12 healthy adult volunteers in the presence of pirenzepine, omeprazole, lansoprazole, or rabeprazole at clinically relevant concentrations, four times, and ten times these concentrations, according to methods described previously.[5] We also measured intracellular calcium ion ($[Ca^{++}]i$) concentrations and O_2- and H_2O_2 production by the xanthine- xanthine oxidase system. Although pirenzepine, omeprazole, and lansoprazole had no effect on neutrophil chemotaxis, phagocytosis, O_2- and H_2O_2 production, and the stimulant-induced elevation of $[Ca^{++}]i$, rabeprazole inhibited these functions / responses in a dose-dependent manner (Figure). Rabeprazole impaired chemotaxis at clinically relevant concentrations. All drugs failed to scavenge O_2- and H_2O_2 generated by an acellular system. Inhibition of calcium mobilization may have contributed, in part, to