Letter to the Editor

Cardiac Arrest Associated with Intravenous Ranitidine

Gholamali Mollasadeghi, MD; Evaz Haydarpour, MD;
Touraj Babaie, MD; Kamaleddin Tabatabai, MD

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Among the H_2 receptor blockers, ranitidine is the most effective antagonist and appears to lack some of the side effects that are seen with other H_2 receptor blockers such as cimetidine. We describe a case who received intravenous ranitidine rapidly, which led to a potentially fatal reaction.

A 25-year-old nurse asked her colleague to inject 50 mg of ranitidine intravenously to relieve her gastric pain. She had a one-week history of gastric pain, which decreased with food and antacids. She was a fit woman and had a history of ulcerative colitis that was treated with corticosteroids two years ago. In particular, she had no history of cardiac or electrolyte abnormality. She had taken ranitidine orally previously many times, but she had not taken it recently.

After the administration of ranitidine intravenously, the patient developed cardiac arrest. Fortunately, this event occurred in the intensive care unit of the hospital, and resuscitation equipment was available. Cardiopulmonary resuscitation was successful, but cardiovascular support was needed for 28 hours. With mechanical ventilation after tracheal intubation, secondary complications due to hypoxia were not seen. Ten minutes after beginning cardiopulmonary resuscitation, electrolyte and blood glucose concentration and electrocardiogram were all normal. Transthoracic echocardiography was done and left ventricular ejection fraction was reported to be about 35%. An echocardiogram and electroencephalogram were done five days after arrest and were normal, and she was discharged after 7 days. This case of cardiac arrest associated with ranitidine occurred in a patient who was relatively fit and apparently without any predisposing factors. Unfortunately ranitidine was not diluted, and was injected rapidly.

The H_2-receptor antagonists are effective in increasing gastric pH and in decreasing gastric volume by reducing gastric acid secretion. Ranitidine offers the advantages of prolonged period of protection and fewer side effects than cimetedine.[1] If given intravenously, ranitidine has a faster onset of action and better protection than cimetidine and other H_2 receptor antagonists.[2] Cimetidine is more cost-effective, but it may cause sedation and confusion and; therefore, we can not use it as a premedication. Compared with fasted patients who did not receive ranitidine, patients who received fluids plus oral ranitidine 2 to 3 hours before the induction of anesthesia had lower residual gastric volume, higher pH values, and a decreased incidence of thirst.[3]

Cardiac arrest has been reported with older H_2 receptor blockers (cimetidine), as have other life-threatening complications.[4] Minor side-effects of ranitidine such as headache, nausea, and constipation (which are usually resolved with continued and chronic therapy) are well documented.[5] Many cases have been reported as ranitidine-induced bradycardia.[6]

Reversible chorea is another side effect of ranitidine.[7] Acute confusion can be seen after intravenous injection and oral use.[8] Chest pain is seen with ranitidine without

any angiographic findings.[9] The adverse cardiac effects of ranitidine may be due to H_2 receptor antagonism in coronary smooth muscle, via vasoconstriction and decreasing myocardial blood supply. An increase in the plasma histamine level is the other reason for adverse cardiac effects of ranitidine.[10] Other mechanisms to be considered include a cholinergic action mediated by cholinesterase inhibition.

References

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