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Case Study

A Case Report on Ranitidine Induced Anaphylaxis

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Abstract

Ranitidine since its discovery in the 1980s is a widely used prescription and OTC medication. Its potency and well – tolerability have made it the first - line drug for treating heartburn, acid reflux and peptic ulcers. This case report describes ranitidine associated anaphylaxis in a 54-year-old man who was admitted to the emergency department following a motor vehicle accident. The main objective of this article is to bring awareness among healthcare professionals on the occurrence of rare and unanticipated reactions like anaphylaxis with a drug used routinely in clinical practice. The case report also emphasizes the importance of proper collection and documentation of the patient's medication and allergy history in preventing the occurrence of severe reactions to drugs.

Keywords: Drug allergy, anaphylaxis, hypersensitivity, ranitidine

INTRODUCTION

Hypersensitivity as an immunological dysfunction is defined as the exaggerated or inappropriate response of the immune system which is mostly targeted at innocuous antigens with consequent tissue damage.[1] Hypersensitivity reactions are triggered by drugs, food substances and insect stings.

Drug induced hypersensitivity reactions have variable clinical presentations ranging from mild urticaria to severe anaphylaxis. The World Allergy Organization classifies drug allergies to immediate and delayed reactions. Immediate reactions occur rapidly after drug administration usually within one hour and are IgE - mediated. IgE - mediated reactions clinically present as urticarial angioedema or as anaphylaxis. Anaphylaxis is an acute hypersensitivity reaction due to exposure to a previously encountered antigen. On the first exposure the susceptible individual gets sensitized to the offending agent and the immune system makes antibodies (IgE) against the offending agent which are attached to the membrane of mast cells. On re-exposure, the offending agent binds with the specific antibodies targeted against it. This binding activates the mast cells and basophils to release

the pre-formed chemicals such as histamine, leukotrienes, prostaglandins and many more responsible for the signs and symptoms associated with the reaction. The reaction may include generalized hives, itching, flushing, erythema, respiratory distress, vascular collapse, systemic shock and death. Ranitidine, is a competitive H2 receptor blocker. The mechanism of action involves reversible inhibition of H2 receptors in gastric parietal cells causing a reduction in both gastric acid volume and concentration. Its acid - lowering effect is more pronounced for basal and nocturnal acid secretion than it is for food - stimulated secretion. Additional effects of ranitidine include a decrease in pepsin formation. The drug is FDA - approved for treating gastric ulcers, duodenal ulcers, stress ulcers, gastritis, Zollinger – Ellison syndrome, GERD, prophylaxis of aspiration pneumonia and as an adjuvant in certain cases of urticaria who do not adequately respond to H1 antagonist.[2] The overall incidence of side effects is lower with the use of ranitidine. Common side effects are headache, diarrhea or constipation, dizziness. Rashes are infrequent.

However, ranitidine induced anaphylaxis has been reported in diverse clinical settings including preoperative aspiration prophylaxis, [3] obstetric settings, and pediatric population in relieving epigastric discomfort.[4] Fatal anaphylaxis due to ranitidine has also been reported. The findings of a recent study based on the 10 years of data retrieved from the Eudravigilance database of suspected adverse drug reactions, reveal that out of 6917 reported hypersensitivity reactions, 12.43% were immune allergic and seventeen cases were fatal anaphylactic reactions to ranitidine.[5] The analysis of 8 years of the pharmacovigilance data from Korea showed that of all ranitidine induced reactions, 17% were anaphylactic reactions.[6] Evidence from the literature suggests that anaphylaxis to ranitidine occurs irrespective of the route of administration. It is diagnosed in patients receiving the drug through either route both oral and parenteral. This is a case report of a patient who developed anaphylaxis after receiving intravenous ranitidine.

Case History

A 54 - year - old male was admitted to the hospital following a motor vehicle accident, with the chief complaints of back pain, right thigh pain, headache, nausea and giddiness. The patient had an abrasion measuring 0.5 cm × 0.5 cm above the left eye and 0.2 cm × 0.2 cm in the left wrist. The patient had no comorbidities. He was then treated with IVF NS 1 unit @ 75 ml/hr, inj. Paracetamol – 1g IV BD, Inj. Ranitidine – 50 mg/2 ml ampoule and C. Cephalexin – 250 mg.

The patient complained of difficulty in breathing, giddiness, chest tightness, and generalized pruritus within minutes after receiving the injection ranitidine. On examination, the patient was conscious, oriented, afebrile and was found to be tachypneic. The BP was 90/60 mmHg, PR was 104 beats/min, and SpO2 was 93% obtained by pulse oximetry. Based on the signs and symptoms, it was confirmed that the patient was suffering from ranitidine - induced anaphylaxis and management of anaphylaxis was commenced.

Treatment

The immediate intervention included the administration of inj. Adrenaline (1:1000) –0.5 mg – IM. Supportive care measures included O2 inhalation at a rate of 4-6 litres/minute along with salbutamol and ipratropium nebulization every 6 hrs. Inj. Hydrocortisone – 100 mg IV stat was also given to the patient. Fluid management was given with NS 1 litre IV bolus and was maintained at a rate of 100 ml/hr. The patient responded to the treatment. His vitals improved gradually. He recovered within 24 hrs. He was discharged after 2 days. On subsequent days, along with painkillers and antibiotics, T. Gelusil was given to the patient with no complications. Upon inquiry with the patient's relative about past medication and allergy history, we came to know that he had experienced giddiness and generalized itching which resolved without any intervention after taking a 150 mg tablet of ranitidine when he was 52 years old. The patient also reported an allergy to diclofenac. A reasonable timeline between the administration of the drug and the onset of symptoms along with previous reaction to ranitidine revealed by the patient's relative supports the diagnosis of ranitidine induced anaphylaxis. Using the Naranjo algorithm the event was categorized as "probable".

Table 1: Causality assessment using Naranjo adverse drug reaction algorithm

Questions	Yes	No	Don't know	Score
Are there previous conclusive reports on this reaction?	+1	0	0	+1
Did the adverse event appear after the suspected drug was administered?	+2	-1	0	+2
Did the adverse reaction improve when the drug was discontinued/a specific antagonist was administered?	+1	0	0	+1
Did the adverse event reappear when the drug was re – administered?	+2	-1	0	0

Are there alternative causes (other than the drug) that could on their own have caused the reaction?	-1	+2	0	+2
Did the reaction reappear when a placebo was given?	-1	+1	0	0
Was the drug detected in blood (or other fluids) in concentration known to be toxic?	+1	0	0	0
Was the reaction more severe when the dose was increased/less severe when the dose was decreased?	+1	0	0	0
Did the patient have a similar reaction to the same/similar drug in any previous exposure?	+1	0	0	+1
Was the adverse event confirmed by objective evidence?	+1	0	0	0
Total Score				**7**

DISCUSSIONS

It is evident from existing literature that ranitidine was the most common cause of anaphylaxis among H2 receptor antagonists. According to some authors, the presence of a furan ring in the structure of ranitidine is considered to be a probable cause of hypersensitivity reaction.[7] Another probable mechanism is the presence of serum - specific IgE antibodies responsible for true anaphylaxis.[8] Thus, when exposed to a drug capable of causing anaphylactic reactions, pro - inflammatory mediators are released from both mast cells and basophils inducing a severe reaction. Anaphylactic reactions are life-threatening upon re-exposure to the offending agent. However, fatal reactions have been reported on first exposure.

Chuah et al.,[9] in their study described the case of 74 - year -old - male patient with no previous experience of drug allergy who developed a severe anaphylactic reaction on receiving an injection ranitidine, for stress ulcer prophylaxis. The patient was managed with CPCR but did not respond and expired.

The diagnosis of immediate reaction to drugs largely relies on the clinical assessment of signs and symptoms reported by the patient or noticed at the time of reaction. Skin testing is used for conformational diagnosis. A specially prepared allergen is inoculated into the skin which binds to allergen-specific IgE antibody if present. This produces a local reaction commonly described as a “wheal and flare” reaction.

Graded dose challenge testing is used for patients with negative skin testing to confirm the diagnosis. Usually at the beginning, a low dose typically < 1/1000 of a therapeutic dose is given orally and the dose is increased gradually every 30-60 minutes. Serum tryptase levels are used to support the diagnosis of anaphylaxis. Measurement of specific IgE through RAST is also used to detect type one hypersensitivity reactions. It is important to highlight that in the majority of the case studies confirmatory diagnostic tests were performed.

Measurement of serum-specific IgE and intradermal testing was carried out in the case report described by *Chopra et al.,*[10]. A recent study described allergic reactions to oral ranitidine on re – exposure. Skin prick tests and intradermal test was performed and the patient was tested positive. Oral provocation test is considered the gold standard in diagnosing drug induced hypersensitivity reactions. However, due to the risk of severe reaction, it is preferred for patients who test negative for skin tests and for patients in whom skin test is contraindicated. In one of the case studies described by *Demirkan et al.,*[11] OPT was carried out in a woman with 75 mg ranitidine, following which the patient experienced difficulty in swallowing and breathing and throat edema.

We believe that the major limitation of our case report is the lack of confirmatory diagnosis. Several studies have reported cross-reaction with other H2 receptor antagonists in patients allergic to ranitidine.[12] Therefore, it is safe to avoid the use of all the agents under this drug class. Proton pump inhibitors are considered safe alternatives in patients with hypersensitivity reactions to H2 receptor blockers.[13]

CONCLUSION

The patient developed anaphylaxis within a few minutes after receiving injection ranitidine. The timeline between the administration of the drug and the onset of symptoms helped to conclude that the patient was suffering from ranitidine induced anaphylaxis. Past medication and allergy history were not collected and documented from the patient in our case study. The previous reaction to ranitidine recalled by the patient and his relative upon questioning further supports the diagnosis. It is worth noting that proper collection and documentation of the patient’s medication and allergy history could have prevented this undesirable event. Healthcare professionals should be aware of this adverse reaction to ranitidine which helps in early recognition of the event if faced suddenly.

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