CASE REPORT

Severe Anaphylactic Reaction to Preoperative Aspiration Prophylaxis

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ABSTRACT

It is a case of severe anaphylactic reaction in a young female who had received aspiration prophylaxis preoperatively. Patient required intensive care and developed severe lactic acidosis. She responded very well to fluid and phenylephrine boluses and later on norepinephrine infusion.

Key Words: *Anaphylaxis. Preoperative period. Aspiration prophylaxis. Ranitidine. H-2 receptor antagonist.*

INTRODUCTION

The incidence of anaphylactic reaction to H-2 receptor antagonists has been reported as 0.3 - 0.7%.[1] These reactions are more common in obstetric population.[2] Though its routine usage as a part of aspiration prophylaxis is not recommended for elective surgery,[3] but patients having risk factor of gastro-esophageal reflux disease, full stomach, obesity; and pregnancy may get benefited.

The present report describes severe anaphylactic reaction to routine prophylaxis.

CASE REPORT

A 27-year female was admitted with diagnosis of right ovarian endometrial cyst and was scheduled for laparoscopic ovarian cystectomy. She had no prior co-morbid condition. Her preoperative assessment including laboratory workup was unremarkable. For the questionable history of gastro-esophageal reflux disease, she was prescribed to have aspiration prophylaxis two hours before coming to operative suite. The regimen constituted syrup sodium citrate 30 ml, and injections metoclopramide 10 mg and ranitidine 150 mg intravenously. Soon after starting the treatment, the patient reported difficulty in breathing and suddenly became unconscious. On monitoring, she had un-recordable blood pressure, while heart rate was 150 beats/minute. Peripheral cyanosis was also there. Auscultation of chest revealed bilateral harsh vesicular breathing with SpO_2 84% on 10 liters face mask. Patient was intubated and responded well to fluid boluses and increments of phenylephrine intravenously.

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After initial management and stabilisation of hemo-dynamic variables, she was shifted to intensive care unit. Immediately after arrival in intensive care, patient became hypotensive, developed tachycardia and peripheral cyanosis. Patient was managed with fluid and 100 micrograms phenylephrine boluses. Meanwhile, the arterial and CVP line were also passed and norepinephrine was started immediately. Patient started to improve clinically after the fluid resuscitation and norepinephrine infusion.

Arterial blood gases at the time of arrival in ICU showed severe metabolic/lactic acidosis with pH of 7.17 and base deficit of -18.2 and lactate of 11.9 mmol/L. After 6 hours of ICU stay, lactate levels return to 3 and arterial blood gases analysis showed pH 7.34, $PaCO_2$ 32.60 mmHg, PaO_2 207.4 mmHg, HCO_3 17.10 mEq/L, base excess at -7.7, and SPO_2 99.60% (on 0.5 FiO_2). All laboratory workups were normal except fibrin degradation product and D-Dimers, which were raised and deranged respectively with INR of 2.1. Further cardiopulmonary workup was done which included echocardiography and CT pulmonary angiography that was absolutely normal.

The working diagnosis was severe anaphylactic reaction, either due to the metoclopramide or ranitidine, as both were administered intravenous, simultaneously. After exploring the past medical history, it was found that patient had history of minor allergic reaction to seafood. Therefore, she was prescribed hydrocortisone 50 mg 6-hourly and chlorphenramine (8 mg) for 5 days. Antihistamine (H2) receptor blocker was not given, considering that it may be the cause of anaphylaxis.

Patient was extubated within 12 hours of ICU admission. She was shifted to special care unit and then to ward from where she was discharged to home after 3 days. The rest of her course in the hospital was unremarkable. Critical care team also advised her to follow allergy clinic for further management plan, however, she did not attend allergy clinic.

DISCUSSION

The symptoms of allergic reactions may vary from mild bronchospasm, flushing or hypotension, requiring symptomatic therapy. However, in severe form, patient may present with cardiovascular collapse, needs aggressive treatment and organ support.[4] Although it was known that the patient developed severe reaction associated with intravenous component of aspiration therapy; but it was not certain whether it was due to metoclopramide or ranitidine. Acute dystonic reactions are the commonly associated adverse event after the administration of metoclopramide.[5] It was considered unlikely to be the cause of anaphylaxis in this patient. However, severe IgE mediated allergic reactions to metoclopramide have also been reported. A young woman with history of asthma and allergy developed severe bronchospasm, urticarial and angioneurotic edema after receiving metoclopramide.[6]

There is a strong association between the route of administration versus the severity of symptoms. In cases, where patients received oral ranitidine, the severity was judged as mild;[7] while anaphylaxis, due to intravenous administration of ranitidine, was associated with cardiovascular collapse.[8] Sripriya *et al*. described the symptoms of cardiovascular collapse after receiving ranitidine in obstetric patient with difficulty in breathing, feeble pulse, tachycardia and hypotension.[9] A similar anaphylactic reaction is reported in a parturient who had received ranitidine as aspiration prophylaxis.[10] Intravenous administration of ranitidine in patient led to severe reaction as indicated by the need of mechanical ventilation and significant rise in lactate level. The pathophysiological effects of anaphylaxis result from immune-mediated release of histamine, leukotrienes, bradykinin, and platelet activating factor. This mediation can cause profound vasodilation, increased vascular permeability with transudation of fluid causing severe hypotension. This results in decreased ventricular filling, reduced cardiac output, and shock-like state.

Epinephrine is considered the drug of choice of treating the vasodilatation associated with anaphylaxis. However, in refractory conditions norepinephrine, metaraminol and Isoproterenol can be used. This patient was given a crystalloid bolus of 500 ml along with the increments of phenylephrine 100 micrograms on showing peripheral cyanosis. She responded very well to it. Meanwhile, she also had arterial and central lines insertion. Norepinephrine was started which was gradually tapered off in 4 hours. After the initial management, it is also very important to find out the cause of allergic reaction. Patient was referred to allergy clinic, but she did not come up and left against medical advice.

Ranitidine is routinely prescribed as pre-medication, but anaphylactic reactions can be devastating with it. Extreme caution and vigilance is required during intravenous administration.

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