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The Author Response

Suspected Anaphylactic Reaction Associated with Microemulsion Propofol during Anesthesia Induction

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To the Editor:

We prepared a case report entitled 'Suspected Anaphylactic Reaction Associated with a Microemulsion of Propofol during Anesthesia Induction' (1). However, one reviewer commented that we should have considered midazolam (2), remifentanil (3), rocuronium (4), ephedrine (5), dexamethasone (6), epinephrine (7), poloxamer 188 (8), and polyethylene glycol (9) in addition to a microemulsion of propofol (10).

The referenced study (10) concerned propofol not a microemulsion of propofol. In that study, tryptase was normal and the patient had the allergy history to soybean, so they speculated the soybean of the intralipid as the cause of anaphylactic reaction.

The mentioned study was about hypersensitivity to midazolam (2). In the study, IgE immunoassay result was in normal. But, after 3 months the inspection, result of intradermal test was positive in the midazolam. It is difficult to diagnose Kounis syndrome, which is a type I hypersensitivity initiated from mast cells with degranulation. The source for a referenced study was wrong, because subject of that study was transdermal fentanyl, not remifentanil (3).

Li et al.'s report (7) had no relation with hypersensitivity (hypersensitivity reaction). The contents of the study was that bimatoprost lowered ocular hypertension generated by the topical dexamethasone effectively and the topical dexamethasone raised the ocular pressure falling by the bimatoprost.

Of course, all medicines mentioned could directly or indirectly result in a patient's anaphylactic reaction rarely. However, in the present case, since injected drugs prior to the reaction were sequentially remifentanil and microemulsion propofol, the authors illustrated the anaphylactic reaction by microemulsion propofol administration among many factors. The correspondence mentioned that fentanyl could increase IgE mediated anaphylaxis, but because it was not administered to the patient, it seemed not to be an appropriate example.

We thought that the cause of the reaction was remifentanil or microemulsion propofol . The skin test was negative and the negative result showed no presence of the IgE antibody. Therefore the non-IgE-mediated anaphylactic reaction was estimated.

In addition, PP188 added to a microemulsion propofol has been reported as a cause for a non-IgE-mediated anaphylactic reaction known as the C-activation-related pseudoallergy (11). Of course, the authors do not deny the probability that there could be a false negative reaction.

Clinically, we agree that the reaction could be caused by many potential factors not one. The authors did not consider intracoronary mast cell activation (12). In our case, there was no EKG change including ST segment elevation and there was a decrease in blood pressure and tachycardia. We were unable to verify by coronary angiography if there was a vasospasm. The possibility for Kounis syndrome seemed small.

The correspondence stated that sulfite was included in the epinephrine, but epinephrine is still the drug of choice for anaphylaxis to sulfite-sensitized patients even though its use is controversial. In addition, we do not have access to preservativefree epinephrine.

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