Allergic Angina Syndrome in Anesthesia and Diabetes

Sir,

We have read with great interest the report published in *Indian Journal of Critical Care Medicine*[1] concerning a 55-year-old male diabetic patient who developed tachycardia, bronchospasm, high airway pressure, increased blood pressure, reduced oxygen saturation, and reduced left ventricular ejection fraction with electrocardiographic ST elevation in the anterior leads following induction anesthesia with rocuronium, propofol, and fentanyl intravenous administration. The patient was diagnosed as rocuronium-induced Kounis syndrome and recovered with intravenous nitroglycerin, hydrocortisone, budesonide, and salbutamol nebulization. Subsequent coronary arteriography did not reveal obstructive coronary artery disease. The authors correctly diagnosed and treated the

patient. However, this report raises the following issues as far as perioperative anesthesia-induced allergy in diabetic patients.

Intravenous administration of rocuronium, propofol, and fentanyl is a routine practice for induction anesthesia, and the authors correctly used this combination. However, these three agents have been incriminated as inducing mild or severe hypersensitivity reactions.[2] Clinical studies have shown that patients, prone to allergy, have more symptoms than monosensitized individuals when simultaneously exposed to several anesthetics, as in the described case. This is based on findings that subthreshold numbers of immunoglobulin E antibodies with different specificities may have additive effects and can join forces to trigger mast cells to release their mediators. This can happen when the patient is simultaneously exposed to the corresponding antigens.[3]

The described patient's blood pressure was increased during the allergic reaction (190/72 mmHg), and his clinical picture and clinical course were mild that is a bit unusual in rocuronium-induced allergic reactions. Indeed, the patient was diabetic and such patients are refractory to allergic inflammatory diseases. In alloxan-induced diabetes, there is a clear downregulation of local allergic inflammatory responses.

Experiments in diabetic rats have shown that skin mast cells' and histamine release is reduced. Administration of insulin in diabetic rats restores basal mast cell numbers.[4] Furthermore, patients with diabetes present markedly reduced lethality when they suffer severe allergic reactions. This refractoriness is associated with mast cell depletion and overexpression of endogenous corticosteroids.[5]

In this patient, the development of allergic reaction was not associated with signs of shock, the left ventricular ejection fraction was dropped only to 40%, and he recovered with intravenous nitroglycerin, hydrocortisone, budesonide, and salbutamol nebulization. Neither epinephrine not intravenous fluid replacement had been necessary. This is another example denoting that the human heart and especially the coronary arteries can be the primary site and the target of an allergic reaction resulting in the development of Kounis syndrome. The view, therefore, that the registered cardiac damage during severe anaphylactic reactions might be due to peripheral vasodilation cannot be supported in this case.[6]

Therefore, detailed medical history, regarding the coronary arteries as the primary target of allergic reactions and considering, in medicine and especially in anesthesia, "the fewer the better" could be of paramount clinical importance for the patient's safety.

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Conflicts of interest

There are no conflicts of interest.

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