Letters to the Editor

Sir,

I read with interest the case report by James et al. on the cerebral venous thrombosis after intravenous immunoglobulin (IVIG) therapy in a patient with immune thrombocytopenic purpura.[1] The case report generally supports the trend of increasing reports of IVIG‑associated thrombotic events in all age groups, including pediatric age group. It is obvious that the tolerance of IG is usually good, but adverse events, including some serious ones, have been reported and may differ among different IG preparations. Thrombotic complications occur in 0.6%–13% of cases and can involve arterial or venous circulation, rarely both.[3] The incidence of thrombotic sequelae appears to vary with the IVIG product composition, rate of infusion, the study population’s disease process, and underlying comorbidities.[4] Treatment with IG has been thought to increase the plasma viscosity, increase and activate platelets, trigger the coagulation cascade through the presence of activated factor XI in some IG preparations, and release vasoactive molecules responsible for vasospasm.[3] I presume that James et al. send an important message to the treating physicians that weighing the risk–benefit ratio must be exercised in planning the use of IG therapy. To limit further evolution of IVIG‑associated thrombotic events in the clinical fields, ensuring sufficient hydration before infusion of IVIG, using the minimal effective dose possible during infusion, considering the use of preparations with lower concentrations of sucrose, and monitoring renal function have been suggested.[4]

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

Mahmood Dhahir Al‑Mendalawi
Department of Paediatrics, Al‑Kindy College of Medicine, University of Baghdad, Baghdad, Iraq

Address for correspondence:
Prof. Mahmood Dhahir Al‑Mendalawi, P . O. Box 55302, Baghdad Post Office, Baghdad, Iraq.
E‑mail: mdalmendalawi@yahoo.com

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
Letters to the Editor

Kounis NG, Koniari I, Tzanis G, Soufras GD,

The more allergens an atopic patient is exposed to, the easier and quicker anaphylactic shock and Kounis syndrome appear: a clinical and therapeutic paradox. J Nat Sci Biol Med 2014;5:240–4.

1. Raut MS, Kar S, Maheshwari A. Awareness of allergic angina syndrome. Indian J Crit Care Med 2017;21:412-3.
2. Kounis N, Kounis G. Anaphylactic cardiovascular collapse during anesthesia: The kounis acute hypersensitivity syndrome seems to be the most likely cause. J Korean Med Sci 2013;28:638-9.
3. Kounis NG, Mazarakis A, Almpanis G, Gkouias K, Kounis GN, Tsigkas G, et al. The more allergens an atopic patient is exposed to, the easier and quicker anaphylactic shock and Kounis syndrome appear: Clinical and therapeutic paradoxes. J Nat Sci Biol Med 2014;5:240-4.
4. de F Carvalho V, Campos LV, Farias-Filho FA, Florim LT, Barreto EO, Pirmez C, et al. Suppression of allergic inflammatory response in the skin of alloxan-diabetic rats: Relationship with reduced local mast cell numbers. Int Arch Allergy Immunol 2008;147:246-54.
5. Carvalho VF, Barreto EO, Díaz BL, Serra MF, Azevedo V, Cordeiro RS, et al. Systemic anaphylaxis is prevented in alloxan-diabetic rats by a mechanism dependent on glucocorticoids. Eur J Pharmacol 2003;472:221-7.
6. Felix SB, Baumann G, Berdel WE. Systemic anaphylaxis – Separation of cardiac reactions from respiratory and peripheral vascular events. Res Exp Med (Berl) 1990;190:239-52.

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 nor 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.[5]

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.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

Nicholas G. Kounis, Ioanna Koniari1, George Tzanis2, George D. Soufras3, George Hahalis

Department of Cardiology, University of Patras Medical School, 1Department of Cardiology, “Saint Andrews” State General Hospital, Patras, 2Department of Cardiology, “Laiko” General Hospital, Athens, Greece, 3Department of Electrophysiology, Royal Brompton Hospital, London, United Kingdom

Address for correspondence: Prof. Nicholas G. Kounis, Department of Cardiology, University of Patras Medical School, Queen Olgas Square, 7 Aratou Street, Patras 26221, Greece. E-mail: ngkounis@otenet.gr

References
1. Raut MS, Kar S, Maheshwari A. Awareness of allergic angina syndrome. Indian J Crit Care Med 2017;21:412-3.
2. Kounis N, Kounis G. Anaphylactic cardiovascular collapse during anesthesia: The kounis acute hypersensitivity syndrome seems to be the most likely cause. J Korean Med Sci 2013;28:638-9.
3. Kounis NG, Mazarakis A, Almpanis G, Gkouias K, Kounis GN, Tsigkas G, et al. The more allergens an atopic patient is exposed to, the easier and quicker anaphylactic shock and Kounis syndrome appear: Clinical and therapeutic paradoxes. J Nat Sci Biol Med 2014;5:240-4.
4. de F Carvalho V, Campos LV, Farias-Filho FA, Florim LT, Barreto EO, Pirmez C, et al. Suppression of allergic inflammatory response in the skin of alloxan-diabetic rats: Relationship with reduced local mast cell numbers. Int Arch Allergy Immunol 2008;147:246-54.
5. Carvalho VF, Barreto EO, Díaz BL, Serra MF, Azevedo V, Cordeiro RS, et al. Systemic anaphylaxis is prevented in alloxan-diabetic rats by a mechanism dependent on glucocorticoids. Eur J Pharmacol 2003;472:221-7.
6. Felix SB, Baumann G, Berdel WE. Systemic anaphylaxis – Separation of cardiac reactions from respiratory and peripheral vascular events. Res Exp Med (Berl) 1990;190:239-52.

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.