ions can react with high- and low-affinity IgE antibody receptors, the known FCγRI, FCγTII, FCεRI, and FCεRII receptors, situated on platelet surface and trigger the thrombotic cascade (2). Implanted devices, therefore, constitute an ideal surrounding for endothelial damage and dysfunction together with hemorheologic changes and turbulence as well as platelet dysfunction, coagulation, and fibrinolytic disturbances. Metals and polymers are great sensitizers that are able to produce corresponding IgE antibodies. Nickel, chromium, and cobalt induced hypersensitivity reactions in 14%, 4%, and 9% patients in the United stated and in approximately 20%, 4%, and 7% patients, respectively, in Europe (3).

In a report of patients who were allergic to nickel, as evidenced by cutaneous patch skin tests and suffering from interatrial shunts and having full nitinol Amplatzer occluder device and the low nitinol Premere closure implanted, a Kounis syndrome-like disease was developed in eight of nine patients (4). The symptoms these patients experienced were chest discomfort, exertional dyspnea, asthenia, palpitations, worsening of migraine headaches, and mild leukocytosis between postoperative days 2 and 3. All symptoms were resolved within one week with prednisone and clopidogrel administration. Interestingly, in the same report, two patients with negative skin patch testing who had occluder system implantation had postoperative atrial fibrillation that was resolved with antiarrhythmic treatment.

Thrombus formation can occur up to 5 years after Amplatzer device implantation, but this is rare. This can be explained by the fact that hypersensitivity inflammation goes through three phases: the early phase that lasts minutes; the late phase that lasts from 2 h to 2 days; and the chronic phase that follows a continuous, persistent, and repetitive allergen exposure and lasts as long as the allergen is present.

Thrombus formation on the Amplatzer device has emphasized the need for critical attitude in decision making in percutaneous closure of patent ovale (5). We therefore believe that careful history of contraindications and hypersensitivity with monitoring of inflammatory mediators and lymphocyte transformation studies would be helpful before such device implantation.

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Author`s Reply

To the Editor,

We appreciate the authors of the letter that emphasizes the hypersensitivity inflammation after the occlusion of defects with devices that contain various metals. In our report entitled "Huge thrombus formation 1 year after percutaneous closure of an atrial septal defect with an Amplatzer septal occluder" published in the Anatolian Journal of Cardiology 2016; 16: 63-4. The significance of long-term follow-up of these patients was demonstrated, and the accurate duration and type of antiplatelet therapy, and preference of imaging technique after device implantation was considered (1).

As you mentioned, nickel allergy can be the cause of systemic reactions such as chest discomfort, palpitation, and migraine headache with or without aura in patients undergoing percutaneous atrial septal defect and patent foramen ovale closure (2). As our patient had no symptoms like chest pain, palpitation, or headache, the patient's condition was not suggestive of Kounis syndrome. Furthermore the electrocardiogram was normal. However, we do agree that hypersensitivity reactions to nickel may be more common than expected in the patients that underwent defect occlusion, particularly with devices having high nickel content.

In one study, it was reported that all patients developed Kounis syndrome within 2 and 3 days after device occlusion, and all these patients presented clinical features of this syndrome (3). Although late hypersensitivity can develop and last as long as the allergen is present, it was not clear whether this reaction could occur without any symptoms that were associated with Kounis syndrome.

On the basis of the above clinical observations, we believe that nickel allergy was not the cause of thrombus formation in our patient.

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Admission serum potassium level is associated with in-hospital and long-term mortality in ST-elevation myocardial infarction

To the Editor,

I have read the article entitled "Admission serum potassium level is associated with in-hospital and long-term mortality in ST-elevation myocardial infarction" by Uluganyan et al. (1) with great interest, recently published in the Anatolian Journal of Cardiology 2015; 16: 10-15. The investigators reported that admission serum potassium (sK) level of >4.5 mmol/L was associated with increased long-term mortality, and significant relation was detected between sK levels of <3 mmol/L and ≥5 mmol/L and ventricular arrhythmias. A previous study demonstrated that mean sK level above 4.5 mmol/L is associated with increased mortality, and sK levels between 3.5 and 4.5 mmol/L is the optimal range suggested for acute MI patients (2). Rate of ventricular fibrillation or cardiac arrest was relatively stable across a wide range of mean post-admission potassium levels, except for extreme values (<3.0 and ≥5.0 mEq/L) (2). Another study revealed that long-term mortality was lowest in patients with potassium levels of 3.5 to <4.0 mEq/L, whereas mortality was higher in patients with potassium levels of ≥4.5 or <3.5 mEq/L (3).

However, because of some confounding factors, I would like to emphasize on some important points to clarify the findings of Uluganyan et al. (1). First, sK level is a very changeable parameter, and many factors affect the sK levels such as drugs, kidney function, and insulin therapy (4,5). Because insulin therapy affects sK level, lack of in-hospital sK follow-up period is a big gap, particularly for patients on insulin therapy. In addition, it is not mentioned whether patients were on standard insulin therapy or patients on insulin infusion were excluded. Second, there was no data regarding the severity and extensiveness of coronary artery disease and PCI procedure and the success rate of total revascularization. Third, they have mentioned ventricular arrhythmias but did not mention the type such as postperfusion ventricular arrhythmias ; postperfusion ventricular arrhythmias are known to be benign, and there is no need for treatment. Fourth, the kind of diuretic treatment that was administrated is not clear. They should have classified diuretic treatments such as the use of loop diuretics, thiazides, and potassium-sparing diuretics.

In conclusion, although the relation between cardiovascular events and sK levels was shown in several studies, further randomized clinical trials are needed with close follow-up of sK levels because many factors may easily affect sK levels.

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Author`s Reply

To the Editor,

We thank the author(s) for their special comments on our study entitled "Admission serum potassium level is associated with in-hospital and long-term mortality in ST-elevation myocardial infarction" published in the Anatolian Journal of Cardiology 2015; 16: 10-15. In the study, we determined the association between cardiovascular outcomes and admission serum