Authors' reply

Dear Editor,

We thank the authors of the letter for their valuable remarks. In our study entitled "Predictors of neurologically favorable survival among patients with out-of-hospital cardiac arrest: A tertiary referral hospital experience" published in the April 2017 issue of Archives of The Turkish Society of Cardiology, in 58.1% of cases, the etiology of the cardiac arrest was not identified.[1] Therefore, in the present study exploring the relationship between cardiac arrest etiology and electrocardiographic (ECG) markers would be challenging. Interestingly, in one study, the authors found that 21% of the patients with an occluded coronary artery had no ST elevation on ECG.[2] Although in the literature, different results have been reported regarding the usefulness of ECG markers in predicting cardiac arrest etiology,[2,3] in our study, definitive conclusions could not be made due to limited prehospital data. As we stated in the limitations section, transfer to a tertiary referral hospital of patients who had worse prognosis and who needed advanced care might have led greater number of asystole ECGs on admission. Finally, we agree with the comment that prolonged transportation time may also contribute to the difference in number of asystole as initial rhythm.

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Amoxicillin/clavulanate allergic reaction, implantable defibrillator shock, and Kounis syndrome: Pathophysiological considerations

Dear Editor,

Implantable cardioverter/defibrillator devices were introduced in humans in 1980 as first-line treatment or prophylaxis for patients at risk of lethal ventricular tachyarrhythmias, such as ventricular tachycardia and ventricular fibrillation.[1]

In a very interesting report published in Turk Kardiyoloji Dernegi Arsivi,[2] a 54-year-old male patient suffering from non-ischemic dilated cardiomyopathy who had cardioverter defibrillator implantation developed acute allergic reaction with itching, nausea, and retrosternal chest pain, and subsequently experienced pre-syncope and defibrillator shock following 1000 mg peroral amoxicillin/clavulanate (Croxilex 1000 mg, I.E. Ulagay İlaç Sanayii Türk A.Ş., Istanbul, Turkey) for upper respiratory tract infection. Although his past history was unremarkable for any allergic reaction, smoking, hypertension, diabetes mellitus, or dyslipidemia, coronary angiography demonstrated normal coronary arteries with vasospasm at the ostium of the right coronary artery and defibrillator interrogation revealed ventricular fibrillation.

This report raises the following issues with regard to the defibrillator components and their antigenicity, the non-ischemic dilated cardiomyopathy, and the causality of the allergic reaction.

1. Both defibrillators and pacemakers are made of 2 implantable components: the generator and the pacing lead.[3] The generator is covered by a titanium capsule. The pacing leads, which are flexible, insulated conductor wires, are attached to the capsule through the pacemaker's header. The headers are made from poly (methyl methacrylate) and polydimethylsiloxane

(silicone rubber). The conductor wires consist of an alloy of nickel, cobalt, chromium, and molybdenum, and the pacing electrodes are made of platinum-iridium alloy. The defibrillator leads, in particular, have at the tip additional 1 or 2 defibrillation electrodes (shock coils) that are usually made of platinum or platinum iridium alloy or of tantalum with platinum coating.[4] The leads are most commonly insulated with one of the following: polyurethane, silicone rubber, some copolymers of silicone and polyurethane, expanded polytetrafluoroethylene or polychloroparaxylene (parylene).[5]

All of the above 8 metals and 5 polymers have been implicated in inducing hypersensitivity reactions.[6]

2. The possibility of an immune response as cause of non-ischemic dilated cardiomyopathy has received increasing attention and evidence of unusual mast cell activity in myocardial tissue from a patient with non-ischemic dilated cardiomyopathy suggested that immediate hypersensitivity may play a role in the initiation or progression of this disease.[7] Subsequently, several possible causes have been named, including autoimmune, viral, or postviral mediated myocardial inflammation induced by the effector cells of the immune system, such as cytotoxic T-lymphocytes, natural killer cells, mast cells, and macrophages;[8] direct effect of inflammatory mediators, such as Inducible nitric oxide synthase activation, cytokines released by the infiltrating lymphocytes, macrophages, or endothelial cells; and antibody action against the beta receptor, myolemma, membrane receptors or proteins, mitochondrial or microsomal membrane, or enzymes.[9]

Therefore, the allergic reaction to amoxicillin/clavulanate seems to have taken place in an immune environment.

3. Amoxicillin/clavulanate allergic reactions are commonly encountered antibiotic-induced allergic reactions. The patient's past history was free of any allergy, but the combination of metals and polymers of the defibrillator, the possible immunological background of the underlined disease, and the antibiotic administration make the manifestation of Kounis syndrome type I coronary spasm possible. It is known that patients simultaneously exposed to several allergens have more symptoms than monosensitized individuals.[10] Furthermore, immunoglobulin E antibodies with different specificities can have additive effects, and small, even subthreshold number of them can join forces and trigger the cells to release their mediators.[11] This can happen when the patient is simultaneously exposed to several antigens, as was the described patient.

Thus, careful patient disease background and consideration of drug side effects should be taken into account. The decision to prescribe a drug where there is a history of previous immune environment requires careful assessment of the risks and potential benefits.

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Predictors of neurologically favorable survival among patients with out-of-hospital cardiac arrest: A tertiary referral hospital experience

To the Editor,

We read the article entitled "Predictors of neurologically favorable survival among patients with out-of-hospital cardiac arrest: A tertiary referral hospital experience" by Kevser Gulcihan Balci et al.[1] with great interest. We want to thank the authors for this valuable contribution to the literature.

Both in-hospital and out-of-hospital cardiac arrest are very challenging entities, though the prognosis is worse for the latter. Cardiac arrest, independent of etiology, can cause very severe disabilities, including neurological complications. Early efforts with medical and mechanical cardiopulmonary resuscitation (CPR) and effective airway management (EAM) are necessary. In case of inadequate circulation and oxygenation of vital organs, death or serious disabilities caused by ischemic encephalopathy are inevitable. Neurological complications, in particular, lead to increased expenses, both for the patient's family and for the country, by increasing hospitalization duration and necessity for physical treatment. So we wonder about some issues in this trial. The first point is about EAM. Even though CPR guidelines have proposed chest compressions rather than breathing in order to minimize the time to initiation of blood circulation,[2] oxygenation, which is best provided with EAM like endotracheal intubation, is very important for both cardiac and neurological well-being. There are some trials with contradictory survival results regarding out-of hospital airway management.[3,4] In our country, unfortunately, many patients are transported to hospital without being intubated or with esophageal intubation, especially if the response crew is untrained. We wonder if there is any difference between successful return of spontaneous circulation and failure with respect to intubation timing. The second point, which is directly related to the result of this trial, is neurological disability. It is very difficult for cardiologists to analyze and record the neurological status of patients in a retrospective trial. Interpretation of Cerebral Performance Category scale by a cardiologist may not be enough for classification, so we wonder if a neurologist evaluated all of the patients in this study. It would be a better and more objective way to obtain more valuable results. Aside from these points, this trial is enlightening work about cardiac arrest and neurological consequences.

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