Effect of systemic inflammation in the cardiac surgery performed on elderly

Yıshlarada yapılan açık kalp cerrahisinde sistemik enflamasyonun etkisi

We would like to congratulate the authors for their study (1). Certainly, human life-span is being prolonged and mean age of the persons undergone cardiac surgery increased in parallel with increase of the quality of life. In this context, cardiac surgery in elderly has become a popular subject. Contrary to general belief, in parallel with the study by Kara et al. (1) many studies report that outcomes of the open heart surgeries performed on the patients with an advanced age were not poor (2, 3). However, we would like to specify that we have some questions regarding this issue. In many studies, inflammatory response is stated to increase in advanced ages (3-5). In relation to that, since one of the factors affecting open-heart surgery is known to be systemic inflammatory response, which is induced by open heart surgery itself, are not these outcomes expected to be poorer if this response is greater in elderly people? We would like to know authors’ opinion on this point.

Another issue we would like to mention is that in a similar study, it was reported that open surgery outcomes were influenced from the blood amount which is preoperatively used in the people younger than 80 years old, but this negative effect was not observed in the patients in mid-eighties (3). We believe that it will add value to study of Kara et al. (1), if they have any data related to this interesting result.

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Author’s Reply

Dear Editor,

We read the letter to the Editor concerning our article titled “The quality of life after cardiac surgery in octogenarians and evaluation of its early and mid-term results”. We are grateful to the authors’ interest in the subject and for their critiques.

Whether or not an octogenarian, all patients who underwent cardio-pulmonary bypass (CPB) suffer from the SIRS induced by contact of blood with non-physiological surfaces during CPB, surgical trauma, ischemia-reperfusion in various organs, changes in body temperature, complement activation, endotoxin, leucocyte activation as a result of release of cytokine and adhesion molecules, free oxygen radicals, arachidonic acid metabolites, platelet activating factor, and formation of substances like nitric oxide and endothelin. This resulting SIRS is a defense mechanism created to protect the organism in situations caused by mentioned pathological stimuli (1). The most important subject here is the severity of systemic inflammatory response during CPB and its’ damage on organs. If, activated as a natural defense mechanism, SIRS, continues by uncontrolled activation of different humoral and cellular paths it is named as (SIRS), a pathological condition. And this condition can induce a rather complex, very difficult-to-control clinical process that can present with 90% mortality. Clinically, inflammatory response as a result of SIRS is observed in the form of myocardial failure, shortness of breath, nephritic and neurological system disorders, bleeding disorders, and multi-organ failure like hepatic disorders during the postoperative period (2). SIRS incidence is reported at 2% in all cases who underwent CPB (3). There are many factors triggering SIRS incidence during CPB and these triggering factors may create different responses in every patient (4). It was reported that the cause of this different response could be the different activation or damage of triggering factors on the endothelium (2).

In summary, as we have mentioned above, SIRS occurs as a result of many factors and the severity of the resulting inflammatory response can be different in persons with the same clinical symptoms. More importantly, there is no consensus on a biochemical parameter considered to show systemic inflammatory response clearly and as correctly as in nephritic dysfunction, diabetes, and atherosclerosis. However, patients with high risk profiles (e.g. multiple comorbidity, diabetes, low functional capacity) and a risk SIRS occurrence can be detected. However, a study conducted by Litmathe et al. (2) report SIRS rate to be 11% even in patients in the high risk group for which they considered a potential risk of perioperative SIRS occurrence. Therefore, uncontrolled humoral and cellular activation secondary to different endothelial effects and damage of increased inflammatory response with old age in situations such as diabetes, hypertension, hypercholesterolemia or nicotine addiction, which are specific risk factors for atherosclerosis, can lead to the occurrence of SIRS.

As a result, we do not agree with the authors’ opinions that the outcomes should have been worse in octogenarian patients based on the factors we tried to explain above. Nonetheless, mortality is reported to be 12.5% in our article (5). As suggested in our article, patients with...
high risks of perioperative SIRS occurrence can in fact be detected and early mortality and morbidity reduced with a detailed analysis of the preoperative physiological and functional conditions of patients, comorbid diseases, and myocardial functions.

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A 52-year-old male patient was admitted to our center with a diagnosis of inferior myocardial infarction following a minor surgical procedure. At the initial evaluation, his clinical appearance was unstable and he was classified as Killip 3. The patient was intubated and transferred to the cardiac catheterization laboratory for primary intervention. Total thrombotic occlusion was observed in the dominant right coronary artery. Angioplasty and stent deployment were performed with a final distal TIMI 2 flow. Aspirin (300 mg), clopidogrel (600 mg), and an intracoronary loading dose of tirofiban and heparin (60 u/kg) were given during the procedure. A maintenance dose of tirofiban and anticoagulation therapy, including heparin, were not continued due to bleeding risk. Even so, minor bleeding was observed at the surgical site and pressurized compression was performed at that site in order to stop the bleeding. Eight hours after the procedure, ventricular fibrillation was observed and repeat angiography showed acute stent thrombosis. At that point, percutaneous thrombectomy with an aspiration catheter was performed and yielded a final distal TIMI 2 flow. In addition, combined therapy of ticagrelor and tirofiban perfusion was begun to decrease further thrombosis risk. However, excessive bleeding occurred at the surgical site, which required massive transfusion. After a week of supportive treatment, the patient was discharged from the hospital. Final echocardiography revealed a 45% ejection fraction with inferior and posterior wall hypokinesia.

Perioperative myocardial infarction is associated with high morbidity and mortality due to accompanying sympathetic activation, enhanced oxidative stress, a prothrombotic and proinflammatory environment, and also marked bleeding risk (2). Similar to our case, administration of antplatelet and anticoagulant agents could trigger massive bleeding even after a minor surgical procedure. On the other hand, optimal antplatelet and anticoagulant therapy are crucial in preventing stent thrombosis in such a specific clinical circumstance. Thrombus aspiration without balloon angioplasty is a logical approach to restricting the necessity of intensive high-dose antplatelet and anticoagulant therapy. Stent deployment should be avoided in such instances; proven dosage and duration of antplatelets and anticoagulants should be used in case of stent deployment because of an enhanced prothrombotic environment, despite the existing high bleeding risk.

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