Evaluation of the oxidative state in children with congenital heart disease

Konjenital kalp hastalığı olan çocuklarda oksidatif durum değerlendirilmesi

Cardiovascular disease is a significant cause of death and chronic illness in childhood. However, over the last decades major advances have been made in the diagnosis and treatment of congenital heart disease. Consequently, many children with simple and complex congenital heart defects may now survive to adulthood.

Understanding the pathophysiology of the anatomic malformation is essential to manage these patients. Children with cyanotic congenital heart disease have poorer clinical outcomes than acyanotic patients. It is thought that hypoxia in these children reduces the antioxidant reserve capacity, leading to a greater susceptibility to the oxidative stress of ischaemia. Rokicki et al. (1) have evaluated the antioxidant status of 41 newborns and infants under one year of age and concluded that the imbalance between prooxidant and antioxidant reactions cause enhanced oxidative stress which might be associated with congenital heart defect pathology in infants. Besides, findings of Cheung et al. (2) suggested increased oxidative stress in Kawasaki disease patients with coronary aneurysms was associated with carotid intima-media thickening and stiffening.

There is a delicate balance between free radicals and antioxidants under normal circumstances. Free radicals are usually considered to be deleterious and it is often forgotten that these reactive oxygen species perform many essential functions. However, during critical illness patients are exposed to many factors that may lead to depletion of antioxidant defense mechanisms and increase in free radical production. Reactive oxygen species induced by several diseases can trigger oxidative stress and some evidence suggests that oxidative stress may be the underlying mechanism in the development of atherosclerosis and cardiovascular disease (3, 4).

There is scarce data in literature regarding the association between congenital heart disease and oxidative stress in children with cyanotic and acyanotic congenital heart disease. So, evaluation of the oxidative state might explain why these patients with congenital heart disease cannot adequately maintain their antioxidant defense and lead to deterioration of the patient's condition. In the study by Ercan and colleagues (5), the oxidant and antioxidant values of the cyanotic patients were significantly higher than the acyanotic and control groups. The authors have speculated that due to the underlying anatomical defect, hypoxia develops and increases both the free oxygen radicals and the antioxidant substances for compensation afterwards. The clinical importance of this situation was hypothesized that increased free oxygen radicals might be responsible for the underlying mechanism of several diseases. They also indicated the importance of applying palliative or corrective techniques at the earliest age possible for patients with cyanotic congenital heart disease.

In conclusion, further investigation elucidating the clinical importance of this oxidative state in children with congenital heart disease before and after surgical and medical treatment should be confirmed by larger randomized prospective studies.

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