True left ventricular aneurysms and rupture/Midterm survival following repair of a giant left ventricular true aneurysm ruptured during operation and associated with papillary muscle rupture

Gerçek sol ventrikül anevrizmaları ve rüptür/ Papiller adele rüptürü ile birlikte görülen ve operasyon sırasında rüptüre olan dev sol ventrikül gerçek anevrizmasının onarımı sonrası orta dönem yaşam süresi

Left ventricular aneurysm develops in 2-8 weeks in 15% of patients with myocardial infarction. The circumstance has been qualified to formation of aneurysm if necrosis is transmural and less than 40% of left ventricular wall area. Ahead of triggering factors, which activate those is steroid use and hypertension (1).

True left ventricular aneurysms generally occur as a result of transmyocardial infarction which ameliorates following acute occlusion of left anterior descending (LAD) or dominant right coronary artery (RCA). Angiographically, insufficient collateral flow is pointed out. Acute occlusion of LAD and insufficient collateral flow are probably essential terms for dyskinetic left ventricular aneurysms. About 88% of left ventricular aneurysms develop following anterior infarctions. While the second common cause of aneurysms is inferior wall infarctions, the ones occupying posterior wall rarely lead to formation of aneurysm (2, 3).

It has been shown in the experimental studies that myocyte death starts after 20 minutes of coronary occlusion. In the area which will evolve to aneurysm in a few days, loss of trabeculations and accumulation of fibrin deposits with thrombus development in half of patients commence. Many myocytes are necrotic and extravascular bleedings may be seen in those necrotic milieus. Impairment of systolic and diastolic functions can be observed in this period. Inflammatory cell infiltration and necrotic cell lyses set in 2-3 days. While cell lysis ends in 5-10 days, destruction of collagen takes longer. Those events lead to increased wall strain and decreased contractile force. Myocardial rupture may arise on post-infarct days 5-10. When the fibrosis advances in this area the possibility of rupture weakens. As indicated by Laplace's law, contraction loss in tapered infarcted region makes outward bulging from surrounding environment with preserved contraction (3).

Aneurysmatic field is clinched to pericardium with avascular adhesions in 90% of cases. This aneurysmatic media is seen as prune like or dimpling under cardiopulmonary bypass through inspection. It is also defined as noncontractile protrusion during both systole and diastole angiographically. In case of angina, congestive heart failure, thromboemboli, ventricular tachycardia, surgical treatment is required (2).

We want to cite some points concerning the recently published case report by Çakıcı et al (4) because of the features that we mentioned above. First of all, the aneurysm defined in the article is not a giant one but a medium sized one. There is no clear description about how far the aneurysm developed following the infarction. When the findings like early operation, acute pulmonary insufficiency were taken into account, one can assume the aneurysm as newly developed. However, the definition given for that area matches with the definition for akinetic-dyskinetic rather than aneurysm. There is no proof in the literature that new developing ischemic mitral insufficiency gives rise to left ventricular aneurysm rupture.

True left ventricular aneurysms cannot be ruptured without hard manipulations. Over and above, such hard manipulations should not be put into practice. In literature, there are reports related to their rupture (5). Yet in this case, in a ventricle with pre-formed aneurysm reinfarction should be kept in mind. By then, the defined case should be free wall rupture following the myocardial infarction rather than a true aneurysm. As pointed out in this report, especially in a case with unperformed ventriculography, one should not handle the ventricle without cross-clamping the aorta to avoid from thrombus mobilization (even though there are echocardiographic views, it can not rule out the thrombus) (4).

The intraaortic ballon pump (IABP) has been implemented as explained in the report. When the IABP operates properly, it decreases the left ventricular workload and strain (4). Therefore, the device could be expected to prevent rupture in the aneurysmatic region.

We thank the authors for presenting this interesting case report.

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Author's reply

Dear Editor,

In our case, left ventricular aneurysm was detected by follow-up echocardiographic examination 8 weeks after the myocardial infarction. However, during the operation there were no tough manipulations and aneurysm rupture occurred during routine bicaval cannulation when the inferior vena cava's cannula was placed. Since there was no echocardiographic investigation before myocardial infarction, it is hard to consider whether it is a preformed aneurysm. But, after the interval time when echocardiography was applied, the patient was followed up at cardiology and cardiovascular surgery clinics and it is known that there was no new myocardial infarction. Also, there were no signs of ventricular wall rupture as pericardial adhesions and pericardial hematoma following the pericardiotomy.

In some previous investigations, the similar sized aneurysms were defined as "giant" or "large" (1-5). So, we think there are subjective opinions about the definition of aneurysm size in the literature.

Thanks to author for their interest to our case.

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Incomplete Kawasaki disease: a pediatric diagnostic conflict

İnkomplet Kawasaki hastalığı: Pediatrik tanısal zorluk

Dear Editor,

We read with interest the article "Incomplete Kawasaki disease: a pediatric diagnostic conflict" by Çelik et al (1). They reported two children with incomplete Kawasaki disease (KD) who responded to 2 g/kg single dose intravenous immunoglobulins (IVIG) and 3 mg/kg aspirin (1).

Sonobe et al. (2) recently reported that the coronary artery abnormality (CAA) prevalence of incomplete KD (18.4%) was higher than that of complete KD (14.2%) among 15,857 cases with KD (83.9% complete KD and 16.1% incomplete KD) using the data from the 17th Japanese nationwide survey of KD. Because late diagnosis of KD increases the risk for coronary artery abnormalities, Minich et al. (3) also recently suggested that clinicians should maintain a high index of suspicion of KD in the infant who is younger than 6 months and has prolonged fever even with incomplete criteria. Nevertheless, it is not easy to suspect KD at an early stage of the disease.

Therefore, Sinha et al. reported that erythema at the site of BCG inoculation might be a useful diagnostic tool even for incomplete KD (4). Using this phenomenon, we previously diagnosed and reported a 9-week-old male infant with incomplete KD who was initially treated with high-dose IVIG (2 g/kg) and oral aspirin (100 mg/kg) (5). Initial echocardiography was normal in this patient, but a giant aneurysm of right coronary artery (RCA) was newly developed one week later. Intravenous dexamethasone and oral methotrexate were given due to rapidly progressive coronary artery aneurysm, but those treatments were not effective. On 38th hospital day, we performed coronary angiography, which demonstrated multiple giant aneurysms with sluggish blood flow on the entire RCA and a stenosis on the proximal anterior descending branch of the left coronary artery. Because he had had a prolonged course of severe coronary involvement refractory to intensive medical therapies, surgical intervention, such as plication of dilated coronary artery, was tried. However, the patient died from acute cardiorespiratory failure shortly after weaning from cardiopulmonary bypass (5).

Although there has been no effective therapy in patients with incomplete KD resistant to IVIG and aspirin, one of our authors previously reported the beneficial effect of low-dose oral methotrexate on 4 patients with Kawasaki disease (age 8 months - 8 years) resistant to IVIG (6). However, methotrexate could not cease the rapid progression of coronary artery aneurysm associated with incomplete KD in our young patient (5).

The diagnosis of Kawasaki disease in very young infants is often difficult because of its rarity and atypical presentation. Although BCG reactivation may help us to suspect incomplete KD at an early stage of the disease, CAA can develop within a relatively short time in contrast to the patients of Çelik et al. (1). Therefore, not only early diagnosis but also more aggressive therapy will be important to prevent sudden cardiac death in incomplete KD and further studies should be performed to elucidate the epidemiology and natural course of incomplete KD in different ethnic populations.

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Author's reply

Dear Editor,

In Kawasaki disease (KD) early diagnosis and specific treatment is essential to avoid mortality. As the authors emphasize, Kawasaki disease is a still diagnostic dilemma for pediatricians, especially with it's atypical or incomplete presentations. Recently, the patients who does not fit the fulfill criteria were considered as incomplete or atypical Kawasaki by the specific signs and exclusion of other causes as we discussed before (1).

Independent predictors were well-defined in KD (2). They have included protracted fever, presumably reflecting worse vasculitis, anemia, elevated white blood count, low albumin, elevated C-reactive protein, male gender and age younger than 1 year (2). As we understood from the author's case, the patient had at least two risk factors (early age and male gender).