

Repair of the left coronary artery originating from right coronary sinus with intramural course

Sağ koroner sinüsten çıkan intramural seyirli sol koroner arter tamiri

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Isolated coronary artery anomalies occur with up to 1% of the general population^[1] and 5.6% of patients undergoing cardiac catheterization.^[2] Anomalous aortic origin of the right coronary artery (AAORCA) is 6 to 10 times more common than the anomalous aortic origin of the left coronary artery (AAOLCA).^[1] However, AAOLCA is seen four to six times than the AAORCA, according to the autopsy series.^[3,4] The American Heart Association (AHA) reported that coronary anomalies are the second most common cause of death (19%) in young athletes.^[5] Due to the risk for sudden cardiac death (SCD) as a first presenting symptom, AAOLCA is considered a surgical indication when diagnosed; even patients have no symptoms.^[6] In general, AAORCA is a benign lesion, and intervention may be needed in case of evidentiary myocardial ischemia. Compression between great arteries, small or slit-like stenotic ostium, acute take-off angle with potential for kinking at the exit of the aortic wall, compression of the intramural part within the aortic wall, or the commissure of the aortic valve are the possible mechanisms for myocardial ischemia and SCD.^[7]

A six-year-old boy was diagnosed with AAOLCA and referred to our hospital. The child was asymptomatic. Echocardiography revealed the suspicion of a coronary anomaly and computed tomography (CT) angiography revealed AAOLCA.

In this video, the technical details of the surgical repair of AAOLCA are presented.

TECHNIQUE

After a standard midline sternotomy, aortic purse-string sutures were placed distally in the ascending aorta, proximal to the origin of the innominate artery. Venous bicaval cannulation was performed. Cardiopulmonary bypass (CPB) was established with moderate hypothermia (32°C to 34°C). Dissection between the aorta and main pulmonary artery was done before cross-clamping. The aortic clamp was placed as close to the aortic cannula as possible. Tepid blood cardioplegia was administered and repeated every 20 min until releasing the cross-clamp. The left side of the heart was vented via patent foramen ovale. Transverse aortotomy was performed above the sinotubular junction. A special care was taken to avoid injury to coronary arteries. There were no coronary ostia in the left coronary sinus. Both coronaries were arising from the right coronary sinus as a single coronary ostium and immediately divided into the left and right coronaries. The ostium of the left coronary artery (LCA) seemed narrower than the right one. A coronary probe (1.5 mm) confirmed the intramural course of LCA. The main pulmonary artery was transected below the bifurcation and suspended to gain exposure. The aortic end of the intramural part of the LCA on the left coronary sinus was determined. A vertical incision was performed from aortotomy to the ending point of the intramural part. The incision was extended to the LAD-Cx bifurcation. The posterior part of the newly created coronary ostia was fixed to the aortic wall by using interrupted 7/0 prolene

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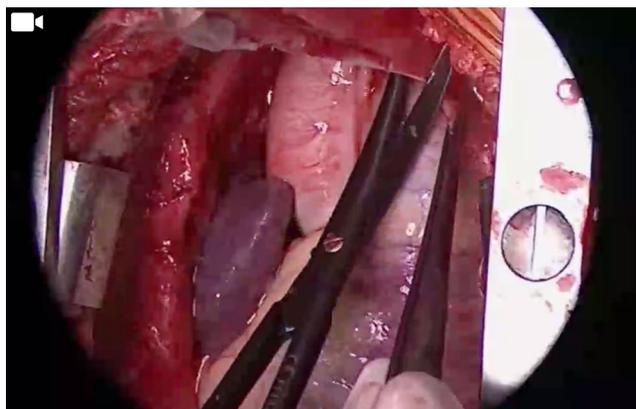
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Video 1. The anatomical repair of a left coronary artery originating from the right coronary sinus with intramural course.

sutures. A patch of glutaraldehyde-treated autologous pericardium was used to close the aortocoronary incision. Therefore, neo-ostium for LCA was created in the left coronary sinus as described by Pascal Vouhé.^[6] This patch ostioplasty technique created a new large left coronary ostium and allowed the original coronary ostium and intramural pathway to open. Distal pulmonary incision was extended to the left pulmonary artery. In this way, pulmonary artery anastomosis was moved to the left side to create a more expansive space for the interarterial region. After re-anastomosing the transected pulmonary artery, aortotomy was closed with deairing. Fibrin glue was applied to improve hemostasis. The patient was weaned from CPB with sinus rhythm. The CPB and cross-clamp times were 116 and 98 min, respectively. The postoperative period was uneventful. The patient stayed one day in the intensive care unit and seven days in the ward. There was no sign of ischemia in the postoperative electrocardiography (ECG) and echocardiography examination. A written informed consent was obtained from the parents and/or legal guardians of the patient.

COMMENTS

According to one of the pathological series, 59% of patients with AAOLCA died before the age of 20 years.^[8] Patients with anomalous aortic origin of the coronary arteries (AAOCA) are mostly asymptomatic. Non-invasive tests, stress ECG, and echocardiography are mostly insufficient to demonstrate myocardial ischemia, even in symptomatic patients. Documented anomalous origins of the coronary arteries with a history of chest pain or syncope is the absolute indication for surgery. The surgical indication in asymptomatic patients is still controversial. Mosca and Phoon^[7] suggested that, in asymptomatic patients

with the AAORCA, surgical treatment might be considered for those patients who would continue to pursue strenuous competitive events or had worrisome anatomic features such as very narrow coronary ostia, documented long intramural coronary segments, and dominant right coronary artery. As previously mentioned, although AAOLCA is seen less, but carries a much higher risk of SCD comparing AAORCA. Therefore, diagnosing the anomalous aortic origin of the LCA may consider surgical intervention due to the increased risk of SCD.^[6] Expert consensus guidelines from the American Association for Thoracic Surgery (AATS) recommend that AAOCA with symptoms and asymptomatic patients with AAOLCA and an interarterial course should be offered surgery (Class I).^[9] Asymptomatic patients with AAORCA should be evaluated for inducible ischemia, and if asymptomatic and without ischemia, they may be observed and allowed to resume competitive athletics (Class IIa). However, documented myocardial ischemia via a moderate grade stress test (radioisotope myocardial perfusion imaging or stress echocardiogram) may require surgical treatment.^[10] Briefly, most authors have concluded that documented myocardial ischemia is the absolute surgical indication, and the presence of intramural course and length are the critical elements for myocardial ischemia.^[11]

Coronary reimplantation, coronary artery bypass grafting, unroofing, and patch ostioplasty are some different surgical techniques for the management of AAOCA.^[12] Aortic insufficiency and recurrent myocardial ischemia are the most common causes of morbidity after surgical correction of AAOCA. Reimplantation technique is limited in case of the single coronary ostium and intramural course, as in our case. Unroofing of the intramural part may be hazardous to the aortic valve commissures and may cause aortic insufficiency. Jegatheeswaran et al.^[13] reported that freedom from mild aortic insufficiency was 77% in patients with commissural manipulation and 88% in those without commissural manipulation at three years of follow-up. Also, the unroofed part of the coronary artery may be constricted with the surrounding aortic wall. Patch ostioplasty technique creates a new, secondary coronary ostium in the left coronary sinus. Coronary flow is provided by two coronary ostia without manipulation commissure in patch ostioplasty. Gaillard et al.^[14] reported a study including 61 patients who underwent surgical repair of AAOCA. They concluded that anatomical repair might provide treating the entire intramural segment, relocating the ostium at the appropriate sinus level, and correcting any take-off angle, unlike unroofing.

One consideration that remains does aneurysmal dilatation can occur in patch ostioplasty? Gaillard et al.^[14] reported that aneurysmal dilatation occurred in patients treating with long saphenous veins, but not in the patients those treated with autologous pericardium. Although longer and further follow-up are needed, we believe that this technique is safe, effective, and reproducible.

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