

# Reversible Congestive Heart Failure After Percutaneous Closure of a Large PDA in a 34-Year-Old Woman

Ramazan Kargin<sup>1</sup> MD, Soe Moe Aung<sup>1</sup> MD, Ozkan Candan<sup>1</sup> MD, Nihal Ozdemir<sup>1</sup> MD, M. Yunus Emirođlu<sup>1</sup> MD

<sup>1</sup> Kartal Koşuyolu Yüksek İhtisas Eğitim ve Araştırma Hastanesi, Department of Cardiology

## ABSTRACT

Patent ductus arteriosus is a congenital heart disease which can cause chronic volume overload leading to congestive heart failure. A 34-year-old woman with rest dyspnea was found to have patent ductus arteriosus and echocardiogram revealed a markedly dilated left ventricle (8.9 cm) and severely-compromised left ventricle systolic functions (ejection fraction~24%). The patent ductus arteriosus was successfully closed percutaneously with Amplatzer occluder device. The patient was discharged on optimal dosages of ramipril, metoprolol, furosemide, spiranolactone and aspirin. On the follow-up after 18 months, the symptoms were found to have regressed and echocardiographic parameters improved (ejection fraction~55%).

**Key Words:** Patent ductus arteriosus; Congestive heart failure; percutaneous closure.

## ÖZET

### *34 Yaşındaki Kadın Hastada Geniş PDA'nın Perkütan Yolla Kapatılmasıyla Konjestif Kalp Yetersizliğinin Gerilemesi*

Konjenital bir kalp hastalığı olan patent duktus arteriyozus (PDA) kronik volüm yüküne bağlı olarak konjestif kalp yetersizliğine neden olabilmektedir. 34 yaşında istirahatde nefes darlığı olan bir kadın hastada PDA ile ekokardiyografik olarak ileri derece dilate sol ventrikül (8.9 cm) ve bozulmuş sol ventrikül sistolik fonksiyonu saptadık (ejeksiyon fraksiyonu~24%). Patent duktus arteriyozus Amplatzer tıkaçıcı alet ile başarılı bir şekilde kapatıldı. Hasta optimum doz ramipril, metoprolol, furosemide, spiranolactone ve aspirin tedavisi ile taburcu edildi. 18 aylık takip sonucunda semptomlarda gerileme ve ekokardiyografik parametrelerde düzelme saptandı.

**Anahtar Kelimeler:** Patent duktus arteriyozus; Konjestif kalp yetmezliği; perkütan kapama.

## INTRODUCTION

Transcatheter methods for closure of patent ductus arteriosus (PDA) have been known as an effective technique for many years (1-3). Moreover, in patients with congestive heart failure, regular follow-up and prescription of suitable medications for heart failure are essential after PDA has been successfully closed.

We present a case of an adult with a large PDA and severely reduced left ventricular ejection fraction, which benefited from percutaneous PDA closure with an Amplatzer occluder device (AOD) and medical treatment of congestive heart failure (CHF).

## CASE REPORT

A 34-year-old woman presented to our clinic with increasing symptoms of CHF. Examination revealed a tachycardia (120 bpm, rhythm was atrial fibrillation), blood pressure 130/70 mmHg, a raised venous pressure, fine bilateral basal crepitations, a loud second heart sound, a loud systolic murmur and a mild diastolic murmur at the left first intercostal space. Her chest X-ray showed cardiomegaly and increased pulmonary vascular markings. She was a non-smoker and was abstinent of alcohol. Her past history was otherwise unremarkable.

Transthoracic echocardiogram revealed a markedly dilated left ventricle, with end-diastolic dimension (LVEDD) 8.9 cm (Normal Range 3.7–5.6 cm), left ventricle end-systolic dimension (LVSD) 7.8 cm, global hypokinesis, severely impaired systolic function (ejection fraction~24%) (Figure 1) and pulmonary artery systolic pressure (PASP) 70 mmHg. Moreover, a PDA with left to right shunt was detected.

## Address for reprints:

Ramazan Kargin, MD

Kartal Koşuyolu Yüksek İhtisas Eğitim ve Araştırma Hastanesi, Department of Cardiology, 34865 Cevizli-Kartal Istanbul, Turkey

Telephone: +90 216 325 46 97 - 0505 565 33 25 Fax: +90 216 459 63 21 e-mail: ramazankargin@yahoo.com

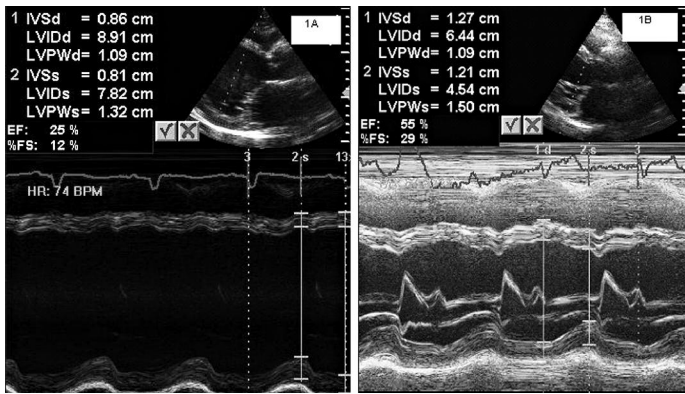


Figure 1: Parasternal long axis view demonstrating the left ventricular enlargement (A) and the improved left ventricular parameters (B).

Right and left heart catheterization with complete analysis of hemodynamic data on each cardiac chamber and great artery confirmed a large PDA. The subject underwent coronary arteriography for evaluation of coronary artery status and revealed normal coronary artery. The descending aortogram in lateral or right anterior oblique view was performed to determine the size and anatomy of PDA. Under fluoroscopic guidance, AOD was successfully deployed. A mild residual left-to-right shunt was present through the device by aortic angiography (Figure 2).

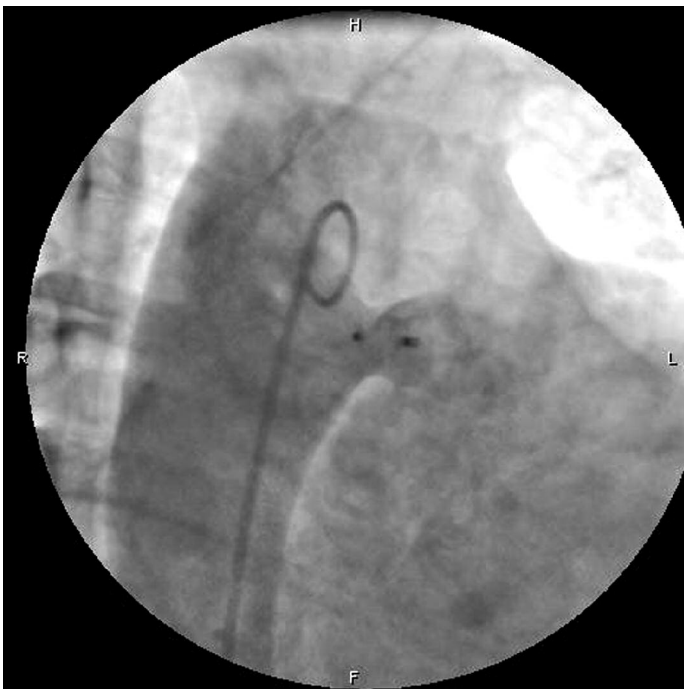


Figure 2: A mild residual left-to-right shunt was present through the amplatzer occluder device by aortic angiography at lateral view.

The symptoms regressed and rhythm returned to sinus rhythm in 24 hours. She was discharged on 300 mg aspirin, 2.5 mg ramipril, 50 mg metoprolol, 80 mg furosemide and 25 mg spironolactone.

The patient was closely followed for 18 months and the medications were administered at maximal tolerable dosages. At the end of 18 months, B-type peptide

(BNP) value decreased from 520 to 40 pg/ml (4) and echocardiographic parameters improved as well. Left ventricle end-diastolic diameter was found to be 6.4 cm, LVSD: 4.5cm, PASP: 30 mmHg and left ventricular ejection fraction (LVEF) ~ 55 % (Fig 1B). Her exercise tolerance was also normal.

## DISCUSSION

This report describes successful transcatheter closure of PDA in an adult with CHF and significant clinical improvement at eighteen months under administration of proper medical therapy. Patients with pulmonary hypertension and PDA who were successfully treated with transcatheter closure and medical therapy for pulmonary hypertension have been reported in literature (1, 2).

In these patients, the reversibility of severe pulmonary arterial hypertension determines management and prognosis (3). Increasing experience with the AOD has made non-surgical closure of even large PDA's simple and safe (3). The Amplatzer Ductal Occluder was successfully applied in our case because the pulmonary hypertension was reversible and PDA was shorter than 10 mm.

The congestive heart failure is often seen in PDA patients due to volume overload. Proper medical treatment is required for heart failure, arrhythmia and pulmonary hypertension. In our patient, normal sinus rhythm was restored after a 24 hour the closure of PDA and a reduction of up to 20 mmHg was seen in PASP. In an early period, significant change in the dimensions of heart chambers was not observed. As known; diuretics, angiotensin converting enzyme inhibitors, beta-blockers and aldosterone antagonist are recommended in heart failure as they have been proven in a number of former studies to have beneficial effects on remodeling, symptoms and survival (5). The release of volume overload with the closure of PDA by AOD and remodelling effects of ramipril, metoprolol, furosemide, and spironolactone were thought to be responsible for improvements in clinical and echocardiographic parameters of our patients.

**In conclusion:** Closure of PDA with AOD and standard treatment of heart failure are beneficial in CHF patients related to PDA.

## REFERENCES

1. Ussia GP, Massimiliano M, Caruso E, Tamburino AR. Combined endothelin receptor antagonist and transcatheter interventional therapy of patent ductus arteriosus with severe pulmonary hypertension. *Int J Cardiol* 2007;116:427-29.
2. Hokanson JS, Gimelli G, Bass JL. Percutaneous Closure of a Large PDA in a 35-Year-Old Man with Elevated Pulmonary Vascular Resistance. *Cong Heart Dis* 2008;3:149-54
3. Yan C, Zhao S, Jiang S, Xu Z, Huang L, Zheng H, et al. Transcatheter closure of patent ductus arteriosus with severe pulmonary arterial hypertension in adults. *Heart* 2007;93:514-18
4. Cheng V, Kazanagra R, Garcia A, Lenert L, Krishnaswamy P, Gardetto N, et al. A rapid bedside test for B-type peptide predicts treatment outcomes in patients admitted for decompensated heart failure: a pilot study. *J Am Coll Cardiol* 2001;37:386-91.
5. Dickstein K, Solal AC, Filippatos G, McMurray JJV, Ponikowski P, Poole-Wilson PA, et al. ESC Guidelines for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2008. *Eur J Heart Fail.* 2008;10:933-89