



Editorial

Transcatheter closure of patent ductus arteriosus in adults

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Patent ductus arteriosus (PDA) is usually identified in childhood but sometimes is not detected until adult life. The adult patient with PDA is sometimes associated with congestive heart failure, and pulmonary hypertension [1–3]. The mortality is estimated to be 1.8% per annum [1].

Indication of PDA closure

PDA closure is indicated in the following situations: (1) the presence of a PDA (except of the silent PDA and severe irreversible pulmonary vascular diseases); (2) closure of the silent PDA remains controversial and should not be routinely performed. However, silent PDA should be closed in the occurrence of an episode of endocarditis; (3) if pulmonary hypertension (pulmonary artery pressure > 2/3 of systemic artery pressure or pulmonary artery resistance > 2/3 of systemic arterial resistance) is present, there must be a net left to right shunt of 1.5:1 or more, or evidence of pulmonary artery reactivity with reversibility test or lung biopsy evidence that pulmonary arterial changes are potentially reversible [3,4].

Transcatheter closure of PDA

Surgical repair has been an established method. However, it may be technically more difficult in adults with possible calcification of PDA, requiring cardio-pulmonary bypass with an anterior approach through a median sternotomy. In addition, advantages of transcatheter closure of PDA include the shorter hospital stay and no scar, compared with surgical repair. Thus, transcatheter closure has become a first-line treatment of most PDA in children and adults.

Although the implantation of a single or multiple coils of various sizes has been a safe procedure to complete occlusion [5], it has limited success of complete closure when PDA is large. The diameter of the PDA must be related to the size of patients, and the proportion of large PDA is more common in older patients [2]. The

introduction of the Amplatzer duct occluder (ADO) offers the possibility to close moderate to large size PDA by catheter intervention, regardless of morphology. PDA closure using ADO has been proven to be a safe and effective treatment [6,7].

Left ventricular dysfunction after closure of PDA

Several studies have demonstrated that PDA closure leads to immediate deterioration of left ventricular (LV) systolic function, which recovered within 6 months in neonates and children [8–10]. Galal et al. reported on a 12-year-old child in whom LV dysfunction after large PDA closure occurred, and described a possible mechanism of deterioration of LV systolic function [9]. It was Starling's effects resulting from the changes in pre-load and after-load subsequent to closure. LV volume overload due to PDA is required to increase LV volume and ejection fraction (EF) stretching the length of LV muscle fiber according to Starling's law. PDA closure results in the sudden reduction of LV volume overload, thereby reducing the LV fiber length and decreasing LVEF. Also, LV ejected blood into only high resistance systemic circulation after PDA closure, although it ejected into both high-resistance systemic circulation and low-resistance pulmonary circulation before closure. They speculated that changes in circulation increased LV after-load. In addition, previous reports have shown that the systolic and diastolic pressure increase immediately after PDA closure [8,10]. The acute changes in blood pressure may also act to increase LV after-load immediately after PDA closure. Apart from the detailed mechanism of LV dysfunction, Eerola et al. evaluated the effects of catheter closure of PDA on the hemodynamic changes including LV systolic and diastolic function in 37 children and concluded that the LV remodeling and changes in systolic function that occurred immediately after closure disappeared within 6 months [10]. In neonates, Kimball et al. showed that wall stress and LV performance were maintained, although blood pressure and systemic resistance increased after closure of PDA [8]. Thus, the ability of LV adaptation for acute changes in pre- and after-load after PDA closure seems to depend on the age of patients, the duration of left to right (L–R) shunt, or magnitude of the shunt. With long-standing L–R shunt, the exposure of LV volume overload leads to morphological changes in the LV and results in impaired function. Thus, the LV function before closure of PDA in adults could be impaired compared with that in neonates and children. Jeong et al. analyzed the LV performance in 45 adult patients and concluded that the LVEF remained low late after PDA closure compared with the pre-closure state. They also showed that pre-closure LVEF was the independent predictor of late normal post-closure LVEF and the pre-closure LVEF > 62% had a

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sensitivity of 72% and a specificity of 83% for predicting late normal EF after closure [11].

In contrast to the previous reports, the LVEF returned to the normal range of 68%, even when pre-closure LVEF was 50% and fibrosis was observed in the LV wall in the case reported by Fujii et al. [12]. Medical therapy such as with angiotensin receptor blockers may affect the recovery of LV dysfunction caused by PDA closure [12].

LV diastolic function could also be impaired in adults with PDA. However, there are few studies examining the LV diastolic function [10]. A recent report showed that LV diastolic dysfunction occurred in adult patients with repaired tetralogy of Fallot (TOF) in whom chronic pulmonary regurgitation impaired right ventricular function, and it was one of the predictors of late onset of arrhythmia [13].

Arrhythmias in adults with PDA

It is well known that atrial fibrillation and/or atrial flutter occur in congestive heart failure due to chronic volume overload by PDA [1–4,14]. However, ventricular tachycardia (VT) is rare in adults with PDA [15]. There is a great risk of VT developing in patients who have undergone a ventriculotomy and/or patching for ventricular septal defect. Less commonly, VT can develop independently of direct surgical scarring whenever a long-standing hemodynamic overload causes ventricular dysfunction or hypertrophy. Lesions of congenital heart disease that can develop VT include aortic stenosis, corrected transposition of the great arteries, Ebstein anomaly, single ventricle, Eisenmenger's syndrome, and unrepaired TOF [15]. PDA is a L–R shunt lesion that results in left heart volume overload. In the case reported by Fujii et al., VT originated from the right ventricular outflow tract, whereas pulmonary artery hypertension was mild [12]. Therefore, it is unclear about the relation between VT and PDA.

In summary, further studies are needed in adults with PDA to clarify: (1) the LV remodeling including diastolic function and arrhythmia before and after closure of PDA; and (2) effects of medical therapy and prevention of the LV dysfunction after closure of PDA.

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