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Advances in Transcatheter Closure for Patent Foramen Ovale with Migraine

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Abstract

Migraine is a recurrent chronic neurovascular disease and the most common primary headache type in clinical practice. Once it occurs, it severely impacts patients' quality of life and work capacity, and is listed by the World Health Organization as one of the most disabling diseases. In recent years, researchers have found that the prevalence of patent foramen ovale (PFO) in migraine patients is significantly higher than in the general population, and some migraine patients who undergo PFO closure surgery experience significant improvement in migraine symptoms. This article reviews the correlation between interventional closure therapy for PFO and migraine.

Full Text

Advances in Interventional Closure for Patent Foramen Ovale with Migraine: A Review

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Abstract

Migraine is a recurrent chronic neurovascular disorder and the most common type of primary headache encountered clinically. Once manifested, it seriously impairs patients' quality of life and working capacity, and has been designated by the World Health Organization as one of the most disabling diseases. In recent years, researchers have discovered that the prevalence of patent foramen ovale (PFO) is significantly higher in migraine patients than in the general population, and that migraine symptoms in some patients improve markedly following PFO closure. This article reviews the correlation between interventional closure and migraine with PFO.

Keywords: migraine; patent foramen ovale; patent foramen ovale closure

Migraine is a common disease in clinical practice, with symptoms that can manifest as unilateral or bilateral pulsating headaches, often accompanied by nausea, vomiting, photophobia, and phonophobia. The pathogenesis of migraine remains unclear, and may be related to genetic, endocrine-metabolic, environmental, and psychological factors. It is precisely this unclear etiology that limits effective treatment of migraine. According to a 2012 epidemiological survey of primary headaches in mainland China, the prevalence of migraine among Chinese adults was 9.3%, with 38.0%, 23.1%, and 47.9% of patients experiencing moderate, severe impacts on life, and requiring effective medical treatment, respectively [1]. Given China' s large population base and high incidence of migraine, a considerable portion of the Chinese population is suffering from migraine. Recent clinical practice has found that the prevalence of PFO is significantly higher in migraine patients than in the general population, and that migraine symptoms can be significantly improved after PFO closure. This article will review the correlation and complications of interventional closure for PFO with migraine, based on relevant literature reports.

1. Anatomical Characteristics of the Foramen Ovale

During embryonic cardiac development, the atrium exists as a single chamber. The primitive single atrium is divided into left and right atria through the formation and fusion of two septa (the septum primum and septum secundum). Beginning in the fourth week of gestation, a thin membrane composed of epithelial cells, collagen fibers, and myocardial tissue develops in the posterior-superior portion of the atrium, known as the septum primum. It grows from the roof of the primitive atrium toward the endocardial cushion. During the growth of the septum primum, its superior portion is gradually absorbed, forming a new channel between the left and right atria called the foramen secundum. On the other hand, on the right side of the septum primum and closely adjacent to it, a membrane grows posteriorly and inferiorly toward the entrance of the superior vena cava. This membrane is called the septum secundum, which gradually grows and partially overlaps the foramen secundum, forming an incomplete septum with an oval window called the fossa ovalis. In this region, the left and right atria are separated only by the thin membrane of the septum primum. A small gap exists at the superior margin of the fossa ovalis, allowing oxygen-rich blood from the fetal right atrium to enter the left atrium through this gap and the foramen secundum. This channel is called the foramen ovale [2]. This anatomical structure enables oxygenated blood from the placental inferior vena cava to pass through this channel into the arterial circulation, bypassing the non-respiring fetal lungs and delivering oxygenated blood to the brain and other organs, which is crucial for fetal development.

After birth, fetal pulmonary respiration allows oxygen to enter the alveoli, leading to the opening of pulmonary arterioles and decreased resistance in the right atrium and pulmonary artery. Meanwhile, increased blood return from the pul-

monary veins to the left atrium leads to elevated left atrial pressure. Through one or both of these mechanisms, left atrial pressure exceeds right atrial pressure, causing the thin membrane of the septum primum to cover the foramen ovale under atrial pressure and undergo fibrous adhesion with the atrial muscle, resulting in closure [2,3]. Approximately 75% of individuals complete this closure within the first two years of life, while the remaining 25% develop a slit-like defect during the closure process, similar to a tunnel, which is known as PFO.

2. Relationship Between Patent Foramen Ovale and Migraine

In clinical practice, numerous scholars have observed that patients with PFO often present with recurrent headache complaints, and the detection rate of PFO in migraine patients is also higher than in the general population. These clinical observations suggest a certain association between PFO and migraine. In 1998, Del et al. [4] first described the association between right-to-left shunt (RLS), stroke, and migraine with aura. In this study, 44 patients with migraine with aura were compared with 73 patients under 50 years of age with focal cerebral ischemia and 50 controls without cerebrovascular disease symptoms or migraine history. All subjects underwent bilateral transcranial Doppler contrast examination. The results showed that 18 of 44 migraine patients (41%) had RLS, compared with 8 of 50 controls (16%) ($P<0.05$). Twenty-six of 73 cerebral ischemia patients (35%) had RLS. The study concluded that the prevalence of RLS in migraine with aura patients was significantly higher than in normal controls and similar to that in young stroke patients. Schwedt et al. [5] conducted a systematic review of case-control studies published up to 2008 and concluded that migraine with aura was more common in PFO patients than in the general population. Anzola et al. [6] compared the frequency of RLS in 113 patients with migraine with aura, 53 patients with migraine without aura, and 25 age-matched controls. PFO was present in 48% of subjects with migraine with aura, which was statistically different from controls, but there was no difference between subjects with migraine without aura (23%) and controls (20%). Wilmshurst et al. [7] first reported that migraine symptoms could be improved after PFO closure. Among 37 patients undergoing PFO closure, 21 (57%) had a history of migraine, including 16 with migraine with aura and 5 with migraine without aura. After PFO closure, 7 of 16 patients with migraine with aura (44%) experienced complete migraine resolution, while 8 of the remaining 9 patients showed improvement in migraine frequency and severity. Among 5 patients with migraine without aura, 3 (60%) experienced complete migraine resolution. Butera et al. [8] applied meta-analysis to systematically search relevant clinical studies from Bio Medcentral, Google Scholar, and PubMed between January 2000 and December 2008, including 522 PFO patients with migraine who underwent PFO closure. Complete migraine cure was achieved in 46% (95% CI 25-67%) of patients, while 83% (95% CI 78-88%) experienced migraine relief or significant improvement. Therefore, numerous studies have demonstrated an association between PFO and migraine, and that PFO closure can improve

migraine symptoms.

3. Pathophysiological Mechanisms of Migraine Induced by Patent Foramen Ovale

To date, no unified conclusion has been reached regarding the pathophysiological mechanisms by which PFO causes migraine. This article summarizes the main hypotheses currently proposed for PFO-induced migraine.

3.1 Paradoxical Embolism Theory

The paradoxical embolism theory is one of the hypotheses supported by many scholars. This theory proposes that under normal conditions with a closed foramen ovale, microemboli in the venous system (such as air, microthrombi, etc.) are filtered out during passage through the pulmonary circulation. When PFO is present, microemboli from the venous system can pass directly from the right atrium to the left atrium through the foramen ovale, bypassing the pulmonary circulation and entering the systemic circulation directly, thereby causing arterial embolism and reduced blood supply to the corresponding arterial territories, triggering migraine attacks. Nozari et al. [9] found that injecting microparticles or air microemboli (similar to microemboli in veins) into the carotid arteries of mice could trigger cortical spreading depression (CSD) without causing cerebral infarction. CSD can activate the trigeminal nerve conduction pathway, leading to stimulation in pain-sensitive innervation areas and thereby triggering migraine [10]. This animal experiment provides good support for this theory.

3.2 Vasoactive Substance Theory

In patients with PFO and migraine, right-to-left shunting within the atria allows certain vasoactive substances in venous blood (such as serotonin, calcitonin gene-related peptide, etc.) to bypass the pulmonary circulation and directly enter the systemic circulation, causing sudden elevation of these vasoactive substances in arterial blood. These substances would normally be inactivated during passage through the pulmonary circulation and would not enter or would minimally enter the systemic circulation. The sudden increase in vasoactive substance levels (such as serotonin) may be an important component in triggering cortical spreading depression, which subsequently activates pain-sensitive fibers of the trigeminovascular system, leading to migraine attacks [11].

3.3 Transient Hypoxemia Theory

PFO-induced right-to-left shunting causes transient hypoxemia, leading to migraine attacks. This theory explains that when patients with PFO suddenly cough or perform a Valsalva maneuver, transient elevation of right atrial pressure exceeds left atrial pressure, resulting in right-to-left shunting that causes transient hypoxemia and triggers migraine attacks [12].

3.4 Genetic Theory

Wilmshurst et al. [13] analyzed echocardiographic reports of 71 migraine patients from 20 families and found that among patients with migraine with aura and atrial shunting, 15 of 21 first-degree relatives (71.4%) of patients with significant RLS also had migraine with aura, compared with 3 of 14 first-degree relatives (21.4%) of patients without significant shunting. The study demonstrated that atrial shunting follows an autosomal dominant inheritance pattern and has genetic correlation with migraine with aura.

4. Interventional Closure Provides a New Treatment Approach for PFO with Migraine

Migraine is a chronic disease characterized by recurrent attacks, with drug therapy as the mainstay of treatment [14]. Treatment strategies are divided into preventive therapy and acute therapy based on the stable and attack phases of migraine symptoms. The goals are: 1) during stable phases, preventive therapy reduces attack frequency, severity, and disability risk; 2) during acute attacks, therapy rapidly and sustainably relieves headache, reduces headache recurrence, and restores patients'normal lives. However, clinical use of migraine medications has revealed that a considerable proportion of patients are insensitive to drug therapy or have medication overuse, and long-term drug therapy also causes many side effects. These issues compel clinicians to urgently seek new treatment methods. In recent years, studies have found that PFO closure can improve clinical symptoms in patients with PFO and migraine, providing a new approach for migraine treatment, particularly for those with refractory headaches and poor response to medication.

Anzola et al. [15] conducted a case-control study of 50 migraine patients who underwent PFO closure surgery and 27 migraine patients who received drug therapy. There were no differences in basic clinical data between the two groups. After 12 months of follow-up, 18 of 50 migraine patients (36%) who underwent PFO closure experienced complete migraine resolution, and 39 (78%) showed improvement in migraine symptoms. In the 27 patients who received drug therapy, 23 (85%) had no relief or worsening of migraine symptoms. PFO closure showed significantly greater improvement in migraine compared with drug therapy, with no difference in efficacy between migraine with aura and migraine without aura. Wahl [16] conducted a retrospective analysis of 150 migraine patients who underwent PFO closure, showing that 51 patients (34%) experienced complete migraine resolution, and 72 (48%) had varying degrees of improvement in migraine attack frequency, severity, and duration. Among 96 patients with migraine with aura who underwent PFO closure, 68 (70%) experienced complete migraine resolution. Xing et al. [17] evaluated the safety and efficacy of PFO closure for migraine in a Chinese population, following up 125 patients with PFO and migraine after closure and comparing HIT-6 scores with a drug therapy group. The impact of migraine decreased by 73.6%, proving

the effectiveness of PFO closure for migraine treatment ($p<0.001$).

5. Complications of Interventional PFO Closure

In the past, repair of PFO could only be performed through open-chest surgery with cardiopulmonary bypass, which carried high surgical risk, numerous postoperative complications, and complex postoperative management. Most hospitals could not perform such procedures, so few patients underwent surgery solely for PFO. With the development of minimally invasive cardiac surgical techniques, interventional PFO closure technology has gradually matured. China began performing interventional PFO closure in the early 21st century, and with continuous updates of interventional devices and improving operator skills, PFO interventional closure procedures increased significantly after 2012, along with deepening understanding of PFO. Numerous foreign studies have confirmed the safety of interventional PFO closure through long-term follow-up of postoperative patients [18, 19]. China started performing these procedures later, and there are currently few large-data analytical studies on complications of interventional PFO closure.

5.1 Device Thrombosis

In a meta-analysis [20] that searched Medline, Embase, and Scopus databases from 1973 to 2012, 28,142 patients who underwent interventional closure procedures (atrial septal defect closure and PFO closure) were included. The most common complication was device thrombosis, with an incidence of 1.0% (95% CI 0.8%-1.0%). Krumsdorf et al. [21] evaluated the incidence of thrombosis after interventional closure in 1,000 patients, showing a device thrombosis rate of 1.2%, with significant differences between different devices: AMPLATZER (0%), HearoSEAL (7.1%), STARFlex (5.7%), and Helex (0.8%). The mechanism of device thrombosis remains unclear. Some scholars believe it results from fibrin deposition on the device surface or blood turbulence that enhances coagulation function [22]. For prevention of device thrombosis, drugs targeting the thrombosis pathway are used, generally antiplatelet and anticoagulant agents. However, there are currently no unified standards for the regimen, dosage, or duration of antiplatelet and anticoagulant drug use, which is mainly based on some biological evidence [23]. No comparative studies of different antithrombotic drug regimens have been reported.

5.2 Device Erosion

Device erosion is a rare complication. During device erosion, some complications may be sudden and life-threatening, drawing attention to this potentially fatal complication. In 2004, Amin et al. [24] first reported device erosion, describing 28 patients with hemodynamic changes after atrial septal defect closure, with an incidence of 0.1%. Nineteen patients (68%) developed symptoms within 72 hours after closure, 8 (29%) were diagnosed between 5 days and 8 months post-

operatively, and 1 (3.5%) developed pericardial effusion 3 years after surgery. Twenty-six patients (92%) experienced cardiac perforation, with locations including the left atrial roof, right atrial roof, and aortic root. Subsequently, Amin et al. [25], in a study evaluating all complications occurring during or after Amplatzer PFO device closure and recommending technical measures to reduce complications, found that two patients experienced device erosion, with an incidence of 0.018%.

5.3 Arrhythmias

Atrial fibrillation is the most common type of arrhythmia after PFO closure, with an incidence of 2%-5% [26]. Staubach et al. [27] followed up 1,349 patients who underwent PFO closure and found that 53 patients (3.9%) developed new-onset atrial fibrillation, which is higher than in the elderly population. Taaffe et al. [28] followed up 660 patients for 30 days after closure and found that the incidence of atrial fibrillation with Amplatzer, Helex, and STARFlex devices was 4% (3/220), 0.9% (2/220), and 5% (8/220), respectively. The incidence of atrial fibrillation may vary with different devices, but there is no correlation between device size and atrial fibrillation. Currently, there are few studies on how to manage post-closure atrial fibrillation. Some literature [29] recommends treatment with classic anticoagulant and antiarrhythmic drugs, while catheter ablation may be used for refractory atrial fibrillation. Additionally, there have been reports of atrioventricular block after PFO closure [30], possibly because the atrioventricular node is located near Koch' s triangle at the edge of the interatrial septum, which may be damaged during PFO device placement, leading to cardiac electrophysiological disturbances.

5.4 Residual Shunt

Small residual shunts are common after PFO closure, while large residual shunts are rare. Transthoracic echocardiography commonly used in clinical practice often cannot detect residual shunts, requiring transesophageal echocardiographic contrast examination, which is a semi-invasive procedure that patients are often reluctant to undergo during follow-up. Clinically, transesophageal echocardiographic contrast examination is typically used to evaluate residual shunts only when patients show no relief of original symptoms or experience symptom recurrence [26]. This situation may lead to missed diagnosis of some patients with residual shunts after closure. Hornung et al. [31] followed up 660 patients who underwent PFO closure for 5 years and found that 24 patients (3.6%) developed severe residual shunts requiring re-implantation of closure devices. The incidence of severe residual shunts differed significantly among devices (P=0.0038), with rates of 2 (0.9%), 15 (6.8%), and 7 (3.2%) for Amplatzer, Helex, and CSS devices, respectively. Additionally, studies [22] have shown that residual shunts after closure can cause significantly increased coagulation activity, leading to thrombotic events. This demonstrates that residual shunt complications cannot be ignored.

5.5 Other Complications

Other complications reported in the literature include infective endocarditis, device allergy, and valve injury [32-34]. In pediatric closure procedures, because children are in a growth and development phase, there is limited research data on whether implanted devices can be tolerated long-term as the child grows. However, some literature has mentioned that echocardiographic and magnetic resonance imaging studies after implantation of large devices in children [35, 36] have shown that the distance between the device and surrounding structures increases with age, which may reduce the risk of long-term complications.

6. Summary and Outlook

In summary, the correlation between PFO and migraine is undeniable. However, the exact role that PFO plays in the pathogenesis of migraine remains uncertain, as none of the existing hypotheses alone can fully convince. Numerous studies have demonstrated that interventional PFO closure is characterized by few postoperative complications and high safety. For PFO patients with refractory migraine and poor response to drug therapy, PFO closure can be considered an alternative treatment option. For patients with PFO and migraine undergoing closure, there is currently no unified standard for postoperative antithrombotic drug regimens to prevent thrombosis, which may require further clinical practice. We believe that with deepening clinical practice, continuous development of molecular biotechnology, ongoing updates in basic research, and improving surgical techniques, research into the physiological and pathological mechanisms of PFO with migraine will become more profound, and closure treatment for patients with PFO and migraine will become safer and more reliable.

[1] Yu S, Liu R, Zhao G, et al. The prevalence and burden of primary headaches in China: a population-based door-to-door survey[J]. Headache, 2012,52(4):582-591.
[2] Hara H, Virmani R, Ladich E, et al. Patent foramen ovale: current pathology, pathophysiology, and clinical status[J]. J Am Coll Cardiol, 2005,46(9):1768-1776.
[3] 郑庆厚, 朱鲜阳. 卵圆孔未闭的诊断与治疗 [J]. 介入放射学杂志, 2008(07):527-531.
[4] Del S M, Angeli S, Leandri M, et al. Migraine with aura and right-to-left shunt on transcranial Doppler: a case-control study[J]. Cerebrovasc Dis, 1998,8 (6): 327-
[5] Schwedt T J, Demaerschalk B M, Dodick D W. Patent foramen ovale and migraine: a quantitative systematic review[J]. Cephalalgia, 2008,28(5):531-540.
[6] Anzola G P, Magoni M, Guindani M, et al. Potential source of cerebral embolism in migraine with aura: transcranial Doppler study[J]. Neurology, 1999,52(8):1622-1625.
[7] Wilmshurst P T, Nightingale S, Walsh K P, et al. Effect on migraine of closure of cardiac right-to-left shunts to prevent recurrence of decompression illness or stroke or for haemodynamic reasons[J]. Lancet, 2000,356(9242):1648-1651.

[8] Butera G, Biondi-Zoccai G G, Carminati M, et al. Systematic review and meta-analysis of currently available clinical evidence on migraine and patent foramen ovale percutaneous closure: much ado about nothing?[J]. Catheter Cardiovasc Interv, 2010,75(4):494-504.
[9] Nozari A, Dilekoz E, Sukhotinsky I, et al. Microemboli may link spreading depression, migraine aura, and patent foramen ovale[J]. Ann Neurol, 2010,67 (2):221-229.
[10] Teive H A, Kowacs P A, Maranhao F P, et al. Leao' s cortical spreading depression: from experimental "artifact" to physiological principle[J]. Neurology, 2005,65(9):1455-1459.
[11] Saengjaroentham C, Supornsilpchai W, Ji-Au W, et al. Serotonin depletion can enhance the cerebrovascular responses induced by cortical spreading depression via the nitric oxide pathway[J]. Int J Neurosci, 2015,125(2):130-139.
[12] Naqvi T Z, Rafie R, Daneshvar S. Original Investigations. Potential faces of patent foramen ovale (PFO PFO)[J]. Echocardiography, 2010,27(8):897-907.
[13] Wilmshurst P T, Pearson M J, Nightingale S, et al. Inheritance of persistent foramen ovale and atrial septal defects and the relation to familial migraine with aura[J]. Heart, 2004,90(11):1315-1320.
[14] 李舜伟, 李焰生, 刘若卓, 等. 中国偏头痛诊断治疗指南 [J]. 中国疼痛医学杂志, 2011,17(02):65-86.
[15] Anzola G P, Frisoni G B, Morandi E, et al. Shunt-associated migraine responds favorably to atrial septal repair: a case-control study[J]. Stroke, 2006,37(2):430-
[16] Wahl A, Praz F, Tai T, et al. Improvement of migraine headaches after percutaneous closure of patent foramen ovale for secondary prevention of paradoxical embolism[J]. Heart, 2010,96(12):967-973.
[17] Xing Y Q, Guo Y Z, Gao Y S, et al. Effectiveness and Safety of Transcatheter Patent Foramen Ovale Closure for Migraine (EASTFORM) Trial[J]. Sci Rep, 2016,6:39081.
[18] Sondergaard L, Kasner S E, Rhodes J F, et al. Patent Foramen Ovale Closure or Antiplatelet Therapy for Cryptogenic Stroke[J]. N Engl J Med, 2017, 377 (11):1033-1042.
[19] Saver J L, Carroll J D, Thaler D E, et al. Long-Term Outcomes of Patent Foramen Ovale Closure or Medical Therapy after Stroke[J]. N Engl J Med, 2017, 377(11):1022-1032.
[20] Abaci A, Unlu S, Alsancak Y, et al. Short and long term complications of device closure of atrial septal defect and patent foramen ovale: meta-analysis of 28,142 patients from 203 studies[J]. Catheter Cardiovasc Interv, 2013,82(7):1123-1138.
[21] Krumsdorf U, Ostermayer S, Billinger K, et al. Incidence and clinical course of thrombus formation on atrial septal defect and patient foramen ovale closure devices in 1,000 consecutive patients[J]. J Am Coll Cardiol, 2004,43(2):302-309.
[22] Rodes-Cabau J, Palacios A, Palacio C, et al. Assessment of the markers of platelet and coagulation activation following transcatheter closure of atrial septal defects[J]. Int J Cardiol, 2005,98(1):107-112.
[23] Lock J E, Rome J J, Davis R, et al. Transcatheter closure of atrial septal

defects. Experimental studies[J]. Circulation, 1989,79(5):1091-1099.
[24] Amin Z, Hijazi Z M, Bass J L, et al. Erosion of Amplatzer septal occluder device after closure of secundum atrial septal defects: review of registry of complications and recommendations to minimize future risk[J]. Catheter Cardiovasc Interv, 2004,63(4):496-502.
[25] Amin Z, Hijazi Z M, Bass J L, et al. PFO closure complications from the AGA registry[J]. Catheter Cardiovasc Interv, 2008,72(1):74-79.
[26] 何璐, 张玉顺. 单中心 1336 例经导管封堵卵圆孔未闭患者主要并发症回顾性分析 [J]. 中国介入心脏病学杂志, 2019,27(06):309-314.
[27] Staubach S, Steinberg D H, Zimmermann W, et al. New onset atrial fibrillation after patent foramen ovale closure[J]. Catheter Cardiovasc Interv, 2009,74 (6):889-895.
[28] Taaffe M, Fischer E, Baranowski A, et al. Comparison of three patent foramen ovale closure devices randomized trial (Amplatzer versus CardioSEAL-STARflex versus Helex occluder)[J]. Am J Cardiol, 2008,101 (9):1353-1358.
[29] Li X, Wissner E, Kamioka M, et al. Safety and feasibility of transseptal puncture for atrial fibrillation ablation in patients with atrial septal defect closure devices[J]. Heart Rhythm, 2014,11(2):330-335.
[30] Johnson J N, Marquardt M L, Ackerman M J, et al. Electrocardiographic changes and arrhythmias following percutaneous atrial septal defect and patent foramen ovale device closure[J]. Catheter Cardiovasc Interv, 2011,78(2):254-261.
[31] Hornung M, Bertog S C, Franke J, et al. Long-term results of a randomized trial comparing three different devices for percutaneous closure of a patent foramen ovale[J]. Eur Heart J, 2013,34(43):3362-3369.
[32] Wertman B, Azarbal B, Riedl M, et al. Adverse events associated with nickel allergy in patients undergoing percutaneous atrial septal defect or patent foramen ovale closure[J]. J Am Coll Cardiol, 2006,47(6):1226-1227.
[33] Habib G, Lancellotti P, Antunes M J, et al. 2015 ESC Guidelines for the management of infective endocarditis: The Task Force for the Management of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM)[J]. Eur Heart 2015, 36(44):3075-3128.
[34] Hiraishi M, Tanaka H, Motoji Y, et al. Impact of Right Ventricular Geometry on Mitral Regurgitation After Transcatheter Closure of Atrial Septal Defect[J]. Int Heart J, 2015,56(5):516-521.
[35] Raboisson M J, Hugues N, Dahdah N, et al. Large Amplatzer atrial septal occluder in growing children: an echographic study[J]. Cardiol Young, 2015,25 (3):468-475.
[36] Lapierre C, Hugues N, Dahdah N, et al. Long-term follow-up of large atrial septal occluder (Amplatzer device) with cardiac MRI in a pediatric population[J]. AJR Am J Roentgenol, 2012,199(5):1136-1141.

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