ORIGINAL ARTICLE

Characteristics and transcatheter closure of patent ductus arteriosus in patients living at moderate to high altitude in Eastern Anatolia

Doğu Anadolu Bölgesi'nde orta veya yüksek rakımda yaşayan hastalarda, patent duktus arteriyozusun karakteristiği ve transkateter kapatılması

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ABSTRACT

Objective: The incidence of patent ductus arteriosus (PDA) is greater among patients living at high altitude. In this population, the ductal diameter is often larger and pulmonary hypertension is more frequent. The aim of this study was to evaluate the hemodynamic and morphological features of PDA and transcatheter closure procedures performed with various devices in a group of patients living at high altitude in Turkey.

Methods: The data of 327 patients who lived at an altitude of at least 1600 m above sea level and who had undergone cardiac catheterization for isolated PDA between May 2010 and July 2018 were retrospectively analyzed.

Results: The mean age was 7.33±7.67 years, and 62.4% of the patients were female. The mean ductal diameter was 3.74±2.14 mm. Pulmonary hypertension was present in 57.8%. Transcatheter closure was performed in 322 patients, with a 97.3% success rate. The Amplatzer duct occluder I (ADO I) was used most often, as well as off-label use of the Amplatzer vascular plug II (AVP) and the Amplatzer muscular ventricular septal defect occluder (AMVSDO). Pulmonary artery pressure decreased immediately in the vast majority after percutaneous closure. Transient left ventricular systolic dysfunction after ductal closure was seen only rarely. Follow-up was uneventful.

Conclusion: Transcatheter PDA closure can be performed with high success rate in highlanders. Off-label devices may be required for these procedures. Pulmonary hypertension is frequent but regresses after ductal closure. Transient left ventricular dysfunction after transcatheter closure is rarely seen in these patients and resolves without any medication.

ÖZET

Amaç: Patent duktus arteriyozus (PDA) insidansı yüksek rakımda yaşayanlarda artmıştır. Bu hasta grubunda, duktus daha geniştir ve pulmoner hipertansiyon daha sıktır. Bu çalışmanın amacı, orta veya yüksek rakımlı yerleşim yerlerinde yaşayan olgularda, PDA'nın morfolojik ve hemodinamik özellikleri ile birlikte, farklı cihazlarla transkateter PDA kapatılmasının incelenmesidir.

Yöntemler: Mayıs 2010 ile Temmuz 2018 tarihleri arasında, 1600 metre rakımın üzerinde yaşayan ve izole PDA nedeni ile kalp kateterizasyonu uygulanmış 327 hasta geriye dönük olarak incelendi.

Bulgular: Ortalama yaş 7.33±7.67 yıl idi ve hastaların %62.4'ü kızdı. Ortalama duktus çapı 3.74±2.14 mm idi. Olguların %57.8'inde pulmoner hipertansiyon mevcuttu. Hastaların 322'sine (%97.3) transkateter PDA kapama işlemi uygulanmıştı. Amplatzer duct occluder I en çok kullanılan cihazdı. Amplatzer vascular plug (AVP) II ve Amplatzer musküler ventriküler septal defekt occluder (AMVSDO) etiket dışı kullanılan cihazlardı. Hastaların çok büyük bir kısmında kapama işlemi sonrası pulmoner arter basıncında hızlı düşüş izlendi. İşlem sonrası nadir sayıda olguda geçici sol ventrikül disfonksiyonu gözlendi. İzlemde tüm olgularda pulmoner hipertansiyon geriledi ve sol ventrikül fonksiyonları ek tedavi gerektirmeksizin normale geldi.

Sonuç: Yüksek rakımlı yerleşim yerlerinde yaşayan olgularda transkateter PDA kapatılması yüksek başarı oranına sahiptir. Bu olgularda etiket dışı cihazların kullanılması gerekebilir. Pulmoner hipertansiyon sıklıkla görülür ancak işlem sonrası geriler. Transkateter PDA kapatılması sonrası geçici sol ventrikül disfonksiyonu nadiren bu hastalarda görülür ve ek tedavi gerektirmeden izlemde düzelir.

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The incidence of patent ductus arteriosus (PDA) is about 1/2000 live, full-term births, and constitutes 5% to 10% of all congenital heart defects.[1] The incidence is reported to be greater among individuals living at high altitude.[2–4] Additionally, the diameter of the arterial duct tends to be larger and the pulmonary artery pressure (PAP) tends to be higher in highland patients.[5] The barometric pressure, the pressure of inspired oxygen, and the fraction of inspired oxygen is 760 mmHg, 149 mmHg, and 20.9%, respectively, at sea level. All decrease with increasing altitude.[6] Hypoxemia and persistent pulmonary hypertension can lead to delayed closure of the ductus arteriosus, which is thought to explain the increased prevalence of PDA at high altitude.[3] Percutaneous closure of PDA with various devices is a well-defined procedure with excellent results.[7–10] However, there are few studies in the literature focusing on catheter closure of PDA in patients living at high altitude,[5,11,12] and it is considered a challenging issue.[13,14] The aim of this study was to evaluate the hemodynamic and morphological features of PDA and investigate transcatheter closure with various devices in patients living at moderate or high altitude in Eastern Anatolia.

Abbreviations:

ADO	*Amplatzer duct occluder*
AVP	*Amplatzer vascular plug*
AMVSDO	*Amplatzer muscular ventricular septal defect occluder*
CDO	*Cera/Ceraflex duct occluder*
LVEF	*Left ventricular ejection fraction*
mPAP	*Mean pulmonary arterial pressure*
mSAP	*Mean systemic arterial pressure*
PAP	*Pulmonary artery pressure*
PDA	*Patent ductus arteriosus*
PVR	*Pulmonary vascular resistance*
SVR	*Systemic vascular resistance*

METHODS

The records of 327 patients who were born and living at an altitude of at least 1600 m above sea level and underwent cardiac catheterization for isolated PDA between May 2010 and July 2018 were retrospectively analyzed. All of the procedures were performed at a tertiary hospital located at an altitude of 1700 m above sea level. Demographic, clinical, echocardiographic, angiographic, and hemodynamic data of the patients were analyzed as well as follow-up data.

Patient selection

The study population was from various sites in Eastern Anatolia that have an altitude of between 1600 m and 2700 m. Patients who had additional structural cardiac anomalies were not included in the study in order to obtain more accurate hemodynamic data about isolated PDA. Informed consent was obtained from the patients or the parents if the patient was <18 years old.

Catheterization procedure

A 50 mg/kg dose of cephazolin was administered 30 minutes prior to catheterization. The procedure was performed under local anesthesia with or without conscious sedation in adults, and deep sedation or general anesthesia in pediatric patients. Both the femoral artery and vein were cannulated in all of the patients. Intravenous heparin was administered at a dose of 50 units per kg, with additional doses if necessary. A hemodynamic study, including pulmonary and systemic arterial pressure, as well as pulmonary (Qp) and systemic (Qs) flow, was performed for all of the patients, and pulmonary vascular resistance (PVR) and systemic vascular resistance (SVR) were calculated for patients with pulmonary hypertension (mean PAP obtained with cardiac catheterization >25 mmHg) whose mean pulmonary arterial pressure to mean systemic arterial pressure ratio (mPAP/mSAP) was >0.5. Descending aorta angiograms were performed with lateral and right anterior oblique projections. On the basis of aortography performed in the left lateral projection, the PDA was categorized according to the Krichenko classification system.[15] A residual duct remaining after either a transcatheter or surgical closure was described as a type R duct.[5] Balloon sizing of the duct was performed in a case of pulmonary hypertension and a large PDA. In patients with normal pulmonary arterial pressure or a mPAP/mSAP of <0.5, the size of the device selected was such that the pulmonic end of the occluder shank would be at least 1.5 mm to 2 mm larger than the narrowest diameter of the duct. If the patient had significant pulmonary hypertension, a device 1 or 2 sizes bigger was used. The size of a vascular plug was at least 1.5 times the ductal diameter for patients with normal pulmonary arterial pressure, and 2 times the ductal diameter for patients with pulmonary hypertension. The duct was closed without any additional study in patients with normal pulmonary arterial pressure and in patients with an mPAP/mSAP of <0.5. In patients with an mPAP/mSAP of between 0.5 and 0.66, a test occlusion was performed by occluding the duct with a low profile balloon catheter or by opening the implanted device in the duct before release. The device was released if at least a 20% decrease in the mPAP was observed.

If the patient had an mPAP/mSAP of >0.66, an acute pulmonary vasoreactivity test with 100% oxygen and inhaled nitric oxide or inhaled iloprost was performed and the PDA was closed if a vasoreactive response was observed. In these patients, hemodynamic measurements were repeated after the device was opened in the duct as a test occlusion before release. If at least a 20% reduction in the mean mPAP and PVR/SVR ratio was seen without a change in cardiac output or a significant residual shunt, the device was released. These steps were repeated after release of the device. A final angiogram was performed to confirm the device position and evaluate any residual shunt. Additional angiogram and pressure measurements were performed if there was a suspicion of pulmonary arterial or descending aorta stenosis associated with the device.

PDA was defined as non-correctable in patients with a PVR of >8 WUxm^2, a negative test occlusion, or negative acute pulmonary vascular reactivity test. Surgical closure was performed for patients with large but correctable ducts that were unsuitable for transcatheter closure or a failed transcatheter closure.

Follow-up

A clinical examination and an echocardiographic evaluation were performed for all of the patients the day after the procedure; 1, 3, and 6 months after the procedure; and once or twice per year thereafter during follow-up.

Statistical analysis

The statistical analysis was performed using IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA). Demographic and clinical variables were summarized with descriptive statistics. Categorical variables were reported as absolute frequency and percentage, whereas median, mean, and SD were used for continuous variables. Paired t-tests were used to compare baseline and postprocedural numerical variables. A chi-square test was used to compare ordinal and categorical variables. The Pearson test was performed for bivariate correlations.

Ethical standards

All of the procedures performed were in accordance with ethical standards and the study protocol conformed to the 1964 Helsinki Declaration and its later amendments or comparable standards. Informed consent was obtained from all of the study patients.

RESULTS

The mean age and weight at catheterization was 7.33±7.67 years (median: 5 years; range: 5 months-40 years) and 21.29±15.62 kg (median: 16 kg; range: 5–76 kg), respectively, and 204 (62.4%) patients were female. Of the patients studied, 34.6% (n=113) were under 2 years of age, 20.5% (n=67) were between 2 and 5 years, 36.3% (n=119) were between 5 and 18 years, and 8.5% (n=28) were older than 18.

Most of the ducts were type A (68.5%, n=224) according to the Krichenko classification. The distribution of ductal types is shown in Figure 1. The mean narrowest diameter of the duct at the pulmonary end

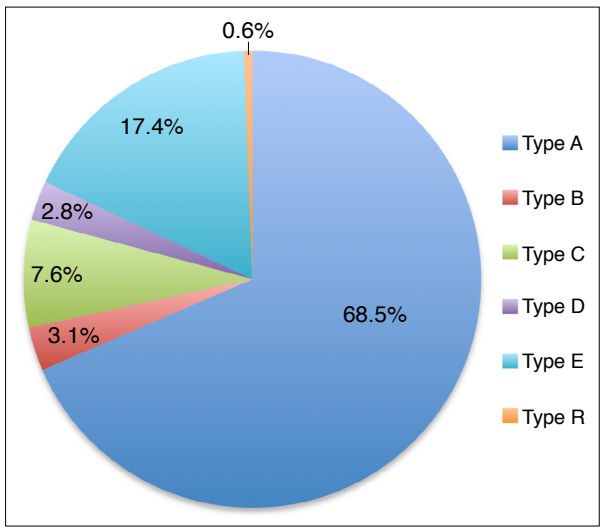

Figure 1. Distribution of the ductal type of all of the study cases (n=327).

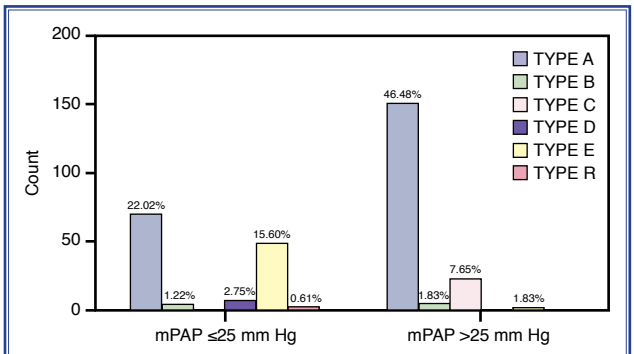

Figure 2. Type of duct observed in patients with and without pulmonary hypertension.

Table 1. Demographic and angiographic features of the patients according to age groups

	<2 years (n=113)	2–≤5 years (n=67)	5–≤18 years (n=119)	>18 years (n=28)	All patients (n=327)
Mean age, mean±SD	1.42±1.13	3.9±0.93	10.22±3.41	27.12±5.84	7.33±7.67
Gender (female), (%)	60.2	62.7	59.7	82.1	62.4
Weight (kg), mean±SD	8.78±1.92	14.18±2.90	28.91±10.27	56.89±10.27	21.29±15.62
Duct size (mm), mean±SD	3.23±1.52	3.34±1.76	4.01±2.35	5.62±2.94	3.74±2.14
Duct morphology,[5, 15] (%)					
Type A	69.9	68.7	63.0	85.7	68.5
Type B	1.8	6	2.5	3.6	3.1
Type C	6.2	9	9.2	3.6	7.6
Type D	2.7	3	3.4	3.6	2.8
Type E	19.5	11.9	21.8	3.6	17.04
Type R	–	1.5	–	3.6	0.6
Pulmonary hypertension (%)	51.3	53.7	62.2	75.0	57.8
mPAP before closure (mmHg), mean±SD	25.58±12.76	26.13±11.76	33.02±17.22	46.67±19.72	30.21±16.14
mPAP/mSAP before closure, mean±SD	0.39±0.20	0.37±0.18	0.42±0.20	0.54±0.20	0.41±0.20

mPAP: Mean pulmonary arterial pressure; mSAP: Mean systemic arterial pressure; SD: Standard deviation.

was 3.74±2.14 mm (median: 3.5 mm; range: 1–14 mm). The average mean PAP was 30.21±16.14 mmHg (range: 9–86 mmHg). In all, 57.8% (n=189) of the patients had pulmonary hypertension, using a definition of a mean PAP of >25 mmHg obtained with catheterization. The distribution of ductal types according to mean PAP is shown in Figure 2. The average mPAP/mAoP ratio was 0.41±0.20. Patients with pulmonary hypertension were classified into 3 groups according to simultaneously measured mPAP and mSAP. Of the group, 78 (41.3%) had an mPAP/mSAP of <0.5, in 69 cases (36.5%) the result was 0.5–0.66, and 42 cases (22.2%) had a ratio >0.66. Demographic and angiographic features of the patients according to age group are shown in Table 1.

Transcatheter closure was performed in 322 patients (98.5%). Three patients (0.9%) were referred for surgical ligation due to unsuitable anatomy for a transcatheter closure or the lack of suitable device at the time. Two patients (0.6%) with severe pulmonary

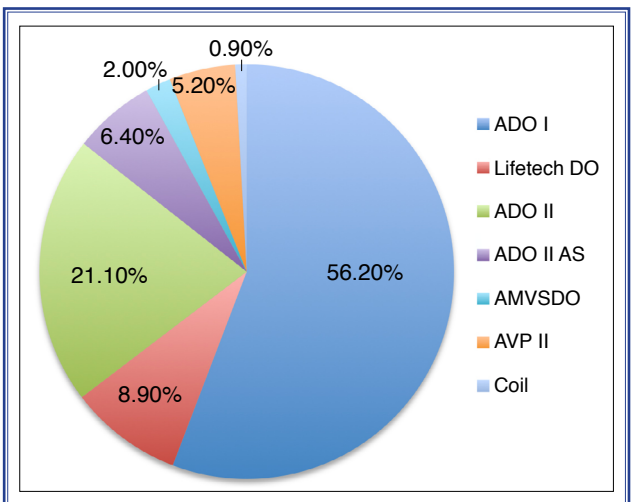

Figure 3. Use of various devices for transcatheter patent ductus arteriosus closure (n=322).

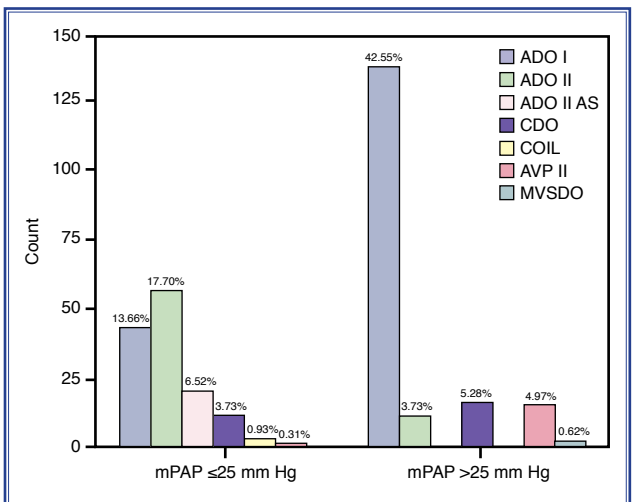

Figure 4. Distribution of the devices used according to mean pulmonary artery pressure

arteria hypertension, a negative acute pulmonary vascular reactivity test, and a negative occlusion test result were considered non-correctable PDA and targeted therapy for pulmonary hypertension with bosentan was initiated. The Amplatzer duct occlude I (ADO I) (St. Jude Medical, Inc., St. Paul, MN, USA) was the most commonly used device for percutaneous device closure of PDA. Other devices and coils used can be seen in Figure 3 and the devices used according to mPAP are provided in Figure 4.

PDA closure was also achieved with off-label use of the Amplatzer vascular plug II (AVP II) (St. Jude Medical, Inc., St. Paul, MN, USA) and the Amplatzer muscular ventricular septal occluder (AMVSDO) (St. Jude Medical, Inc., St. Paul, MN, USA). Device implantation was successful in 313 patients (97.3%). Complete occlusion was achieved immediately in the catheter room in 269 cases (83.5%), while mild residual shunting was present in 46 (14.3%) and moderate residual shunting in the remaining 7 patients (2.2%). Echocardiographic examination of these patients performed on the day after the procedure demonstrated no residual shunting in 78.2% of the patients (n=36) with mild residual shunting and 57.1% (n=4) of those with moderate residual shunting. The average mPAP decreased to 17.67±8.57 mmHg immediately and only 16.8% (n=54) had pulmonary hypertension after transcatheter closure. Of those, 49 patients (90.7%) had an mPAP/mSAP of <0.5, and 5 (9.3%) were 0.5–0.66. No patient had an mPAP/mSAP >0.66 after closure. The mean fluoroscopy time was 8.7±6.1 minutes.

Complications

The most common complication was a transient loss of femoral artery pulse, which was seen in 10 (3.1%) and considered a minor complication. Device embolization in the catheter room occurred in 4 patients (1.2%) as a major complication. Late device embolization occurred in only 1 patient (0.31%), who also had Down syndrome. The occluder device embolized to the descending aorta 24 hours after the percutaneous closure. In 1 patient, there was a slight protrusion of the AVP II device into the pulmonary artery without evidence of significant stenosis. Immediate protrusion of the ADO I device into the aorta, which caused iatrogenic coarctation and required surgical device removal, occurred in 1 patient.

Follow-up

The mean follow-up duration was 28.29±17.15 months (median: 25 months; range: 1 month-8 years). M-mode echocardiography revealed mild left ventricular systolic dysfunction with a left ventricular ejection fraction (LVEF) of 45% to 55% following the closure of the duct in 10 patients (3.1%). All of these patients had pre-procedural left ventricular enlargement and pulmonary hypertension; 8 (80%) were classified as type A, while 2 (20%) were type C, with a narrowest diameter of the ductus of at least 4.7 mm. No medication was initiated. The left ventricular function normalized within 2 to 4 weeks after the procedure in all cases.

Complete occlusion was observed in all patients after 6 months. None of the patients displayed evidence of pulmonary hypertension on echocardiography 6 months after the percutaneous closure.

DISCUSSION

Morphology and hemodynamics

At high altitude, the barometric pressure, the pressure of inspired oxygen, and the fraction of inspired oxygen are reduced, and as a consequence, pulmonary vascular smooth muscle becomes thickened and the lumen narrows, leading to pulmonary hypertension.[3] The incidence of PDA is greater among those living at high altitude due to a sequence of physiological and pathological changes.[2,4,11] A female predominance has been reported in patients with PDA. Zheng et al.[4] reported a clear female predominance in highlanders, and our study results confirm this finding.

The PDA diameter also tends to be larger in these patients and it is associated with elevated pulmonary hypertension. The mean ductal diameter of our patients was 3.61±2.15 mm, which was less than that reported in some previous studies.[5,11,12] This may be due to a lower altitude or the number of cases in our study. There was a high correlation between the narrowest diameter of the duct and the persistence of pulmonary hypertension, as expected (r=0.82; p<0.001).

Our findings were consistent with the literature, confirming that a type A (conical) duct is the most frequent duct type associated with high altitude.[5,11–13] In our study, 68.5% of the ducts were classified as type A. The frequency of type C has been reported to vary

from 2.6% to 26.2%.[5,11,12] A type C duct was seen in 7.6% of the cases in the present study. Pulmonary hypertension was present in 67.9% in patients with a type A duct and in 100% of patients with type C duct. We did not find any data regarding a relationship between duct type and pulmonary hypertension in the literature, and therefore cannot compare our findings, but we suggest that there may be a close correlation based on our experience. Pulmonary hypertension was present in all of the patients with a type C duct and more than two-thirds of the patients with a type A duct. The frequency of type E was greater in our study compared with similar studies, whereas the number of those classified as type D and type B was similar to previous reports. Type R was observed in only 2 patients, 1 of whom had a residual shunt after surgical ligation, and the other had a large residual shunt related to malposition of an ADO II device implanted about 1 year previously at another institute. The former case was successfully closed with an ADO II device and the latter with the ADO I device.

There is a known association between elevated pulmonary hypertension and high altitude.[3,13] In our study, pulmonary hypertension was present in more than half of the patients. Tefera et al.[11] reported that 47% of the patients in their study had pulmonary hypertension and about one-third had an mPAP/mSAP of >2/3, which was similar to our findings. Although their study population had larger narrowest ductal diameters than we observed, the average mPAP, frequency of patients with pulmonary hypertension, and the ratio of patients with an mPAP/mSAP>2/3 were not very different from our results. Interestingly, in their analysis of a larger population living at higher altitudes than the participants in our study, Bialkowski et al.[5] reported a larger mean narrowest ductal diameter and a lower mean PAP. This may be a result of differences in duct type in the 2 studies. The percentage of ducts classified as type C, which we think is closely associated with high pulmonary hypertension, was 3 times greater in the study conducted by Tefera et al.,[11] while the percentage in our group was 3 times that of Bialkowksi et al.[5] Type E, which is rare and only minimally associated with pulmonary hypertension in our experience, was seen about 3 times more in our study population when compared with that of Tefera at al., and about 1.5 times more than was reported by Bialkowski et al. Though there is a high incidence of pulmonary hypertension in patients with PDA and living at high altitude, a rapid decrease in PAP occurs after ductal closure.[5,11,13] The average mean PAP decreased to 17.67±8.57 mmHg with a mPAP/mSAP <0.5 in almost all cases, and there was no instance of mPAP/mSAP >0.66 after PDA closure. This indicates that the vast majority of cases of pulmonary hypertension are reversible. In our study, only 2 patients, both of whom had large ducts, had irreversible pulmonary vascular disease, as confirmed by balloon test occlusion and acute pulmonary vascular reactivity testing with 100% oxygen and nitric oxide (30 ppm): a 17-year-old female with Down syndrome and a 24-year-old female. Targeted therapy with bosentan was implemented in both cases. Despite the high incidence of pulmonary hypertension, some authors have suggested that high altitude may be protective against irreversible pulmonary vascular disease.[16] Although the mechanism is not clear, pulmonary vasoconstriction likely plays a role in preventing the development of irreversible pulmonary vascular disease.[3,11,16] Transcatheter ductal closure must be performed with caution in patients with a PVR >4.6 WU or a PVR/SVR ratio >0.33, and ductal closure must be avoided in patients with a PVR/SVR ratio >0.66, according to current guidelines.[17]

Transcatheter closure

Transcatheter closure of PDA, which is accepted as first-line treatment for all ages except low-weight infants, is a well-established procedure. However, it may still be considered a challenging procedure in patients living at high altitude and in cases of pulmonary hypertension.[14] The high prevalence of pulmonary hypertension together with large ductal diameters, as well as the frequency of ductal types A and C, which have been associated with difficulties in device closure, may be part of the reason. In the literature, the ADO is the most-used device in these study populations.[5,11,13] In our study too, the ADO was the device used most frequently; coils were used in just a few patients. The results were similar to those of previously reported cases.[5,11,13] The ADO II and the ADO II Additional Size devices were also used. No atrial septal occluder was used in this study population. Off-label use of the AMVSDO and AVP II was also observed. All of the AMVSDO devices and the majority of the AVP II devices were used for closure of type C ducts in patients with pulmonary hypertension. The largest series that included the use of septal occluders

that we found in our review of the literature was that of Zabal et al.,[14] which focused on transcatheter closure of pulmonary hypertensive ducts. In that study, the ADO I was the most commonly used device, and septal occluders were used in 12.5%. They concluded that the ADO I works well for most cases, but sometimes a septal occluder may be required. We successfully used the AMVSDO in 2 patients with a type C duct and pulmonary hypertension. Interestingly, we did not find any data about use of the AVP II in patients at high altitude or with hypertensive ducts. In fact, we believe that the AVP II is the safest device in tubular ducts with pulmonary hypertension and large, long, type E ducts. We used the AVP II with excellent results in 17 patients (5.3%), 13 of whom were classified as type C and all had pulmonary hypertension, while the remaining 4 had a type E duct. No residual shunt or major complication was seen. There are some studies reporting a transient decrease in LVEF after transcatheter or surgical treatment of large ducts.[11,13,18] Tefera et al.[11] reported a high occurrence of LVEF decrease of 26%, and most required additional medication with captopril. Left ventricular dysfunction was normalized within 3 to 6 weeks in all of the patients in that study. We had a small group that demonstrated left ventricular dysfunction, only 0.31% (n=10) of all procedures, and the course was similar to that seen in the literature. The important point is that all of them had left ventricular enlargement before the transcatheter closure and had large ducts (minimum: 4.7 mm). Although the exact mechanism of this dysfunction is not clear, some authors have speculated that it might be related to the rapid decrease in the preload after ductal closure.[11]

Complications

Although transcatheter closure of PDA is accepted as a safe procedure, embolization of the device is still a frightening potential complication. The incidence of device embolization is reported to be 2% to 3.9% in patients at high altitude.[11,12] In our study, device embolization occurred in 5 patients (1.5%). Four were instances of early embolization in the catheter room. All of these patients had a type A duct, and all but 1 had pulmonary hypertension. All of the embolized devices were either an ADO I or an ADO II.

We have not had a case of device embolization in 2 years. As our experience grew, we began oversizing the device if there was a concern about embolization. That practice, in addition to off-label use of the AVP II and AMVSDO in some instances, may have prevented device embolization; however, other complications, such as protrusion of the aortic disk into the aorta with ADO devices and protrusion of the proximal disk into the pulmonary artery with the AVP II have been seen. Although a small degree of protrusion into the aorta usually does not cause iatrogenic coarctation and typically regresses with somatic growth, it may still be a problem, particularly in young infants with a large duct and a small descending aorta.

Outcome

The majority of residual shunts after transcatheter closure disappear.[11,14] Echocardiographic evaluation revealed no residual shunting in any member of our study group at the 6-month follow-up. Hemolysis did not occur in any of the patients with residual shunting. The residual pulmonary hypertension seen in some patients after the ductal closure usually regresses with time.[5,11,14] None of our patients had echocardiographic evidence of pulmonary hypertension by the 6-month follow-up visit.

Migration of the occluder device can occasionally be a late complication of the procedure.[19,20] Tefera et al.[11] reported late device migration in an infant with pulmonary hypertension and a large, type C duct that was closed with the ADO device. They detected slow migration of the device to the descending aorta at the third month follow-up visit and operated due to significant coarctation of the aorta. We detected migration of the device to the descending aorta in the second year in a patient with a type C duct that was closed with a 6/6 ADO II. Echocardiography revealed turbulent color flow across the device in the descending aorta, causing a systolic gradient of 35 mmHg without diastolic enhancement. The femoral artery pulses and systemic blood pressure in all extremities were normal. Close follow-up indicated no progression of the migration, so continued careful monitoring was planned and implantation of a covered stent in the descending aorta when necessary.

Limitations of the study

Our study is limited by the lack of a control group and by its retrospective nature. Although echocardiographic examination is very useful for detecting pulmonary hypertension by measuring tricuspid or pulmonary valve regurgitation or the presence of right

chamber dilatation or right ventricular hypertrophy, it is still not a direct measurement. Therefore, the lack of direct measurements of pulmonary artery pressure may be a limitation. On the other hand, we think that a control catheter used as an invasive diagnostic tool is unnecessary and would lead to ethical problems in patients with a good clinical and echocardiographic outcome. Despite moderate to long-term follow-up findings, the majority of the procedures were performed in the last 3 years, so this may be considered a limitation as to comments on long-term outcomes.

Conclusion

PDA in patients living at moderate-to-high altitude is typically larger in size and is highly associated with pulmonary hypertension. Type A is the most frequent type of duct seen in these patients. Type C is closely associated with pulmonary hypertension. Transcatheter PDA closure with various devices can safely be performed despite elevated PAP because the pulmonary hypertension is reversible in the vast majority of cases. The ADO device fits the majority of ducts and has been used safely, though device embolization is most often seen in patients with a type A duct. Oversizing the device may reduce the incidence of device embolization. The off-label use of devices such as the AMVSO and the AVP II may also be required. The AVP II has been used safely and with great success, particularly in patients with type C and large type E ducts. Transient left ventricular dysfunction has been seen rarely in these patients, but usually improves without any medication.

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