Unknown vascular compression of the airway in patients with congenital heart disease and persistent lower respiratory symptoms

Tuğba ŞİŞMANLAR EYÜBOĞLU[1], Ayşe Tana ASLAN[1,]*, Çiğdem ÖZTUNALI[2], Sedef TUNAOĞLU[3],
Ayşe Deniz OĞUZ[3], Serdar KULA[3], Betül DERİNKUYU[2], Öznur BOYUNAĞA[2]
[1]Department of Pediatric Pulmonology, Faculty of Medicine, Gazi University, Ankara, Turkey
[2]Department of Radiology, Faculty of Medicine, Gazi University, Ankara, Turkey
[3]Department of Pediatric Cardiology, Faculty of Medicine, Gazi University, Ankara, Turkey

Background/aim: Airway compression (AC) by vascular structures is an important complication of congenital heart disease (CHD) that often goes unrecognized. It is not easy to identify whether CHD patients require additional invasive examinations or not. Therefore, the present study aims to develop an AC diagnostic algorithm for CHD patients.

Materials and methods: CHD patients with persistent respiratory symptoms that were treated between January 2007 and December 2015 were retrospectively reviewed. The following data were recorded for all CHD patients with AC: age, cardiac anomalies, the compressed structure, the airway diameter ratio (ADR), the compressing structure(s), treatment, and follow-up.

Results: During the 8-year study period, 62 of 253 CHD patients had persistent respiratory symptoms, of which 11 cases were diagnosed as AC via bronchoscopy and/or thoracic computed tomography angiography. The most frequently affected structures were the left main bronchus and trachea, and the most common compressing structure was the right pulmonary artery. The ADR was near total compression in 3 patients and >0.50 in 3 patients. During follow-up, 5 of the 11 patients with AC underwent surgery, 2 died, and 4 were followed clinically. Patients with ADR of >0.50 did not require surgery and were followed clinically.

Conclusion: CHD patients with persistent respiratory symptoms associated with lower respiratory airway obstruction should be evaluated via invasive examination. An AC diagnostic algorithm for pediatric CHD patients was developed.

Key words: Airway compression, child, congenital heart disease, computed tomography angiography

1. Introduction

Children with congenital heart disease (CHD) can have respiratory problems caused by increased pulmonary blood flow or compression of the airway due to enlarged or dilated vascular and cardiac structures (1–3). Patients with CHD have a high prevalence of abnormal ciliary motion, which is associated with an increased risk of respiratory symptoms (4). Approximately 1%–2% of children with CHD experience airway compression (AC) by vascular structures (5). AC commonly occurs in patients with anomalies of the aortic arch system and vascular rings (1), and it has been known to cause extrinsic airway malacia (6). The most common anomalies associated with AC in children are those of the vascular rings; rarely reported causes of AC include left-to-right shunt (causing dilatation of the pulmonary arteries), truncus arteriosus, tetralogy of Fallot (in the absence of a pulmonary valve),

and malpositioned or dilated aorta (1,7,8). The most frequently compressed airway structures are the trachea and the main bronchus. Generally, patients with congenital heart diseases are followed with echocardiography and angiography, both of which cannot be used to diagnose AC.

In children, chronic AC is associated with morbidity and mortality; therefore, its definitive diagnosis and optimal treatment are essential (1). Although CHD patients commonly suffer from lower respiratory system symptoms, there is currently no algorithm for identifying patients that should undergo further examination with procedures such as bronchoscopy and thoracic computed tomography angiography (CTA). Therefore, the present study aims to develop an AC diagnostic algorithm for pediatric CHD patients. This algorithm is based on a retrospective analysis of pediatric CHD patients referred to

the Pediatric Pulmonology Department due to persistent/prolonged respiratory symptoms. CHD patients diagnosed with AC were analyzed in terms of cardiac anomalies, the relationship between vascular structures and the airway, treatment, and follow-up.

2. Materials and methods

We retrospectively reviewed the files of pediatric patients (<18 years of age) with CHD referred to the Pediatric Pulmonology Department of Gazi University, Ankara, Turkey, between January 2007 and December 2015 due to persistent/prolonged respiratory symptoms. Any congenital heart anomalies were recorded. Persistent/prolonged respiratory symptoms were defined as follows: ≥2 episodes of pneumonia during a 1-year period or ≥3 episodes since birth (9), cough with or without sputum of ≥4 weeks in duration (10), failed extubation, and wheezing that did not resolve following bronchodilator treatment.

Patients with the following were excluded from the study: AC due to a known cause (e.g., vascular ring or double aortic arch), chronic lung disease (e.g., cystic fibrosis, immunodeficiency, or primary ciliary dyskinesia), respiratory disease (e.g., asthma, pneumonia, malacia), and diseases known to cause respiratory symptoms (e.g., gastroesophageal reflux and tracheoesophageal fistula via first-line examination, including chest X-ray, pulmonary function test, acute phase reactant measurement, sputum culture, sweat chloride test, barium esophagram, immunoglobulin level measurement, and tuberculin skin test). Patients were evaluated in detail if they required any additional invasive examinations to diagnose AC, including flexible bronchoscopy and thoracic CTA. We noted any airway anomalies observed via bronchoscopy. Thoracic CTA was performed in patients with AC in order to determine the relationship between vascular structures and the airway. Some patients were unable to undergo bronchoscopy due to hemodynamic instability and a high risk of anesthesia-induced complications; these patients underwent thoracic CTA to evaluate the airway as well as cardiac, vascular, and pulmonary structures.

Following intravenous injection of a nonionic contrast agent, nonelectrocardiography-gated MDCT images were obtained with a 64-slice scanner (LightSpeed VCT, GE Healthcare Technologies, Milwaukee, WI, USA) with the following scan parameters: voltage: 100–120 kV, 80-tube current: 80–200 mAs, collimation: <1 mm, and rotation speed: 0.50 s. All images were processed with a minimum intensity projection algorithm and were evaluated for the presence of tracheal and/or bronchial compression using a lung window (window width: 1500 HU; window level: –500 HU). When present, the severity of AC was assessed

with axial images and airway diameter ratio (ADR) (11). In cases of bronchial compression, the ADR was defined as the ratio of the diameter of the narrowest point of the bronchus to the diameter of the normal bronchus, proximal to the distal bifurcation (Figure 1).

The term 'near-total compression' was used to describe cases in which the diameter of the compressed airway was barely discernible in reformatted images. For tracheal compression, ADR was defined as the ratio of the diameter at the narrowest point of the trachea to the diameter of the normal trachea. The measurement of the normal tracheal diameter was performed just proximal to the area of narrowing. Both the compressed and normal tracheal diameters were measured on the same axis. All measurements were obtained using the lung window setting. Anatomical structures responsible for each segment of AC were also noted using the soft tissue window setting. In patients with AC, the following demographics were recorded: age, cardiac anomalies, respiratory complaints, compressed structure, compressing structure(s), ADR, treatment, and follow-up. Statistical analyses were performed using SPSS 16.0 for Windows (SPSS Inc., Chicago, IL, USA). Data are expressed as mean ± SD. The protocol for this study was approved by the Ethics Committee of the School of Medicine of Gazi University.

3. Results

During the study period, 4095 patients were treated in the pulmonology department; among them, 253 (6%) had CHD and were referred to the pediatric pulmonology department with respiratory symptoms. Among these 253 CHD patients, 62 had persistent and/or prolonged respiratory symptoms, and 51 (82%) of these were diagnosed via first-line examination (pneumonia 26%; asthma 19%; gastroesophageal reflux 13%; laryngomalacia 13%; atelectasis 11%; postinfectious cough 7%; swallowing dysfunction 3%; chronic sinusitis 3%; immunodeficiency 3%; tuberculosis 1%; pseudoaneurysm 1%) and 11 were previously evaluated (at least once before) via echocardiography and/or cardiac catheterization, not being diagnosed via first-line examination. Cardiac catheterization is not routine in every CHD case. Instead, it is mainly performed for patients with planned surgery, as seen here in Patient 4. All 11 patients underwent CTA, and bronchoscopy was performed for 3 of them.

Eleven (17%) of the 62 patients that had persistent and/or prolonged respiratory symptoms were diagnosed with AC by cardiac and vascular structures. The mean age of the CHD patients with AC was 2.8 ± 3.1 years and 4 (36%) were male. The mean AC symptom duration was 8.45 ± 8.74 months. The most common symptoms were wheezing (63%) and recurrent pneumonia (45%). The characteristics
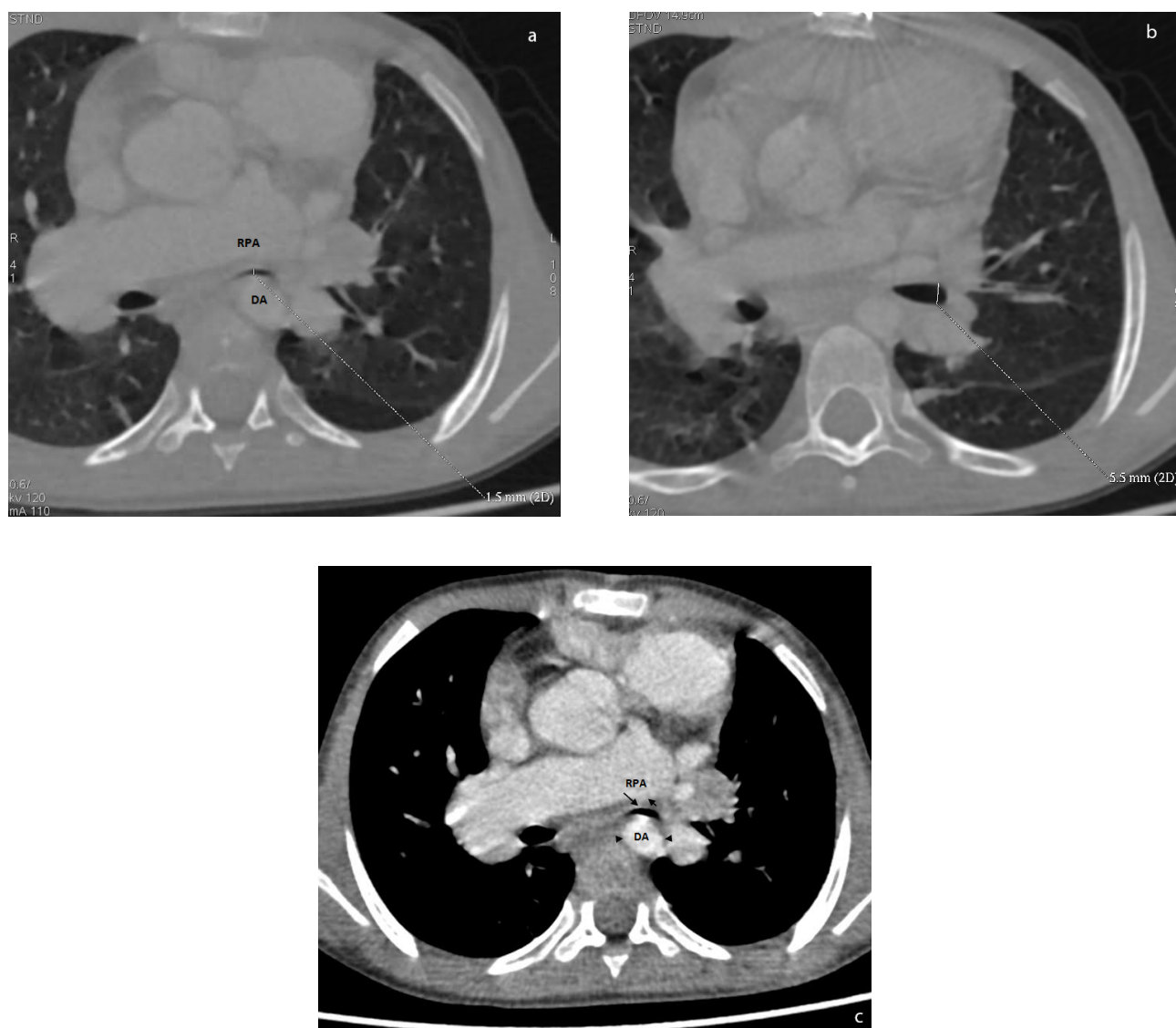
Figure 1. To calculate the ADR, the diameter of the narrowest point of the compressed bronchus (A) and the diameter of the normal bronchus proximal to its distal bifurcation (B) were measured using axial CT and the lung-window setting. Axial CT image in mediastinal window settings (C) shows compression of the left main bronchus (long arrow) between the right pulmonary artery (RPA, short arrows) anteriorly and the descending aorta (DA, arrowheads) posteriorly.

of patients with AC as well as their bronchoscopy and thoracic CTA findings are shown in Table 1.

The left main bronchus (63%) and trachea (45%) were the most commonly compressed structures. ADR was near total in 3 patients, >0.50 in 3 patients, 0.5 in 1 patient, and <0.5 in 4 patients. Airway and vascular structure pathologies, as well as ADR values in the CHD patients with AC, are shown in Table 2. CTA images of the compressed structures and compressing structures in Patients 1, 2, 6, 7, and 8 (all with AC) are shown in Figures 2–6. Left atrium compression was present in two patients. In Patient 6, atrium compression was due to diastolic dysfunction and left ventricular wall thickening, stemming from idiopathic

hypertrophic subaortic stenosis and volume load owing to ventricular septal defect (VSD). Left atrium dimensions of Patient 6 were (AP × CC × ML) 5.5 × 4 × 3.3 cm, and the z score was 8.36 (12).

In Patient 7, atrium compression was due to coarctation of the aorta and volume load because of VSD. Left atrium dimensions of patient 7 were (AP × CC × ML) 4 × 5.5 × 4 cm and the z score was 8.95 (12).

Compression findings are not an expected condition in patients with a normal course of descending aorta. However, compression was found in Patients 3, 4, 6, 7, 8, 9, and 11. In these patients, the dilated pulmonary artery had approached the chest wall and caused the bronchial

Table 1. Characteristics of patients with airway compression (AC).

No.	Age (months)	Sex	Diagnosis*	Symptoms	Symptom duration (months)	Bronchoscopy	Thoracic CTA
1	2.5	M	VSD, PAH, AAA	Wheezing	2	ND	AC
2	4	M	PDA, PFO	Extubation failure	4	ND	AC
3	6	M	ASD, VSD, TA, MA	Wheezing recurrent pneumonia	6	ND	AC
4**	6	F	Multiple ASD, large VSD, history of surgery for pulmonary banding	Wheezing, chronic cough	1	ND	AC
5	7	F	AVSD, ASD, PDA	Wheezing	6	ND	AC
6	9	F	IHSS, VSD, MR	Wheezing recurrent pneumonia	6	ND	AC
7	18	F	CoA, operated VSD	Wheezing	1	AC	AC
8	60	F	Dilated pulmonary artery	Chronic cough	12	ND	AC
9	62	F	Corrected TOF, PR, MR, residual mild PS, operated PA	Recurrent pneumonia	5	AC	AC
10	86	F	TGA, VSD, AAA, dilated pulmonary artery	Recurrent pneumonia	24	AC	AC
11**	91	M	VSD, operated CoA, PDA	Wheezing recurrent pneumonia	26	ND	AC

AAA: Anomaly of the aortic arch; ASD: atrial septal defect; AVSD: atrioventricular septal defect; CoA: coarctation of the aorta; CTA: computerized tomography angiography; IHSS: idiopathic hypertrophic subaortic stenosis; MA: mitral atresia; MR: mitral regurgitation; ND: not done; PA: pulmonary atresia; PAH: pulmonary arterial hypertension; PDA: patent ductus arteriosus; PFO: patent foramen ovale; PR: pulmonary regurgitation; PS: pulmonary stenosis; TA: truncus arteriosus; TOF: tetralogy of Fallot; VSD: ventricular septal defect.
*Patients previously evaluated via echocardiography and/or cardiac catheterization at least once.
**Swallowing dysfunction was present in Patients 4 and 11. Despite appropriate treatment, i.e. gastrostomy, respiratory symptoms continued. They were then evaluated for vascular compression.

structures to be compressed between the pulmonary arteries and the descending aorta.

During follow-up, 5 of the 11 CHD patients with AC underwent surgery; 2 of these were for primary cardiac correction (Patients 1 and 6) and 3 were for AC (Patients 2, 9, and 10). In addition, 2 other patients died during follow-up. Patient 3 was unable to undergo surgery for cardiac correction and died due to cardiac failure while on the heart transplantation waiting list. Patient 4 was scheduled for surgery and died during cardiac catheterization. Another patient was lost to follow-up, and 3 were followed clinically. The mean duration of follow-up was 18.09 ± 14.58 months. All patients with an ADR of <0.50 required surgery, and those with an ADR of >0.50 were followed clinically. Types of treatment, duration of follow-up, and patient status at the last follow-up visit are shown in Table 2.

4. Discussion
Because cardiac and pulmonary pathophysiologies are strongly correlated, the management of patients with CHD is quite complex. Pulmonary complications of CHD can be structural in nature and can be caused by anatomical compression of the airway; this is a commonly unrecognized complication of CHD. For pediatric CHD patients with wheezing, stridor, respiratory distress, apneic or cyanotic spells, and atelectasis, AC must be included in the differential diagnosis (2). Currently, it is not known how to identify CHD patients that require additional examinations such as bronchoscopy and/or thoracic CTA. Therefore, a diagnostic algorithm might be useful in such patients. In the present study, wheezing and recurrent pneumonia were the most common respiratory symptoms in the pediatric CHD patients, and the mean duration of their respiratory symptoms was 8.4 months. Interestingly, Patient 8 had respiratory symptoms for 24 months and was referred to the pediatric pulmonology department with suspected tuberculosis. We suspect that the diagnosis of AC was delayed in all patients in this study based on the duration of their symptoms. A delayed diagnosis of AC is significant, because long-term AC is known to cause tracheobronchomalacia (2). Moreover, Patient 8 had severe bronchiectasis in her left lung due to recurrent infections.

The morbidity and mortality rates associated with central airway obstruction are higher in patients with

Table 2. Airway and vascular structures, ADR, treatment, and follow-up of 11 patients.

No.	Affected structures	ADR	Compressing structures	Treatment	Duration of follow-up (months)	Patient status
1	TR RMULB IB	0.29 0.40 0.49	RPA/thoracic vertebra body RPA/azygous vein RPA/azygous vein	Surgically repaired VSD tracheostomy	8	Alive
2	TR RMB IB	NT NT NT	Right brachiocephalic trunk/esophagus RPA RPA	Tracheostomy	8	Alive
3	LLLB	0.50	RPA/descending aorta	Clinical follow-up	12	Died**
4	LMB	0.46	LSPV/descending aorta	Referred for surgery	1	Died***
5	TR	0.32	LPA	Lost to follow-up	42	Unknown
6	LMB RMLB	0.20 NT	LSPV-left atrium/descending aorta Left atrium	Surgically repaired VSD, IHSS	42	Alive
7	LMB	0.23	Left atrium/descending aorta	Clinical follow-up****	24	Alive
8	LMB	0.53	LPA/descending aorta	Clinical follow-up	12	Alive
9	LMB TR	0.27 0.56	RPA/descending aorta RPA	Operated pulmonary artery plication	23	Alive
10	LMB TR*	NT 0.15*	Pulmonary trunk Pulmonary trunk	Lobectomy	26	Alive
11	LMB	0.66	LSPV/descending aorta	Clinical follow-up	1	Alive

ADR: Airway diameter ratio; IB: intermediate bronchus; IHSS: idiopathic hypertrophic subaortic stenosis; LLLB: left lower lobe bronchus; LMB: left main bronchus; LPA: left pulmonary artery; LSPV: left superior pulmonary vein; NT: near-total airway compression; RMLB: right middle lobe bronchus; RMULB: right main and upper lobe bronchus; RPA: right pulmonary artery; TR: trachea.

*Asymmetric compression of the left side of the distal trachea in an anterior–posterior direction; ADR calculated based on anterior–posterior axis measurements of the left side of the trachea.

**Patient 3 was inoperable for cardiac correction and died due to cardiac failure while on the heart transplantation waiting list.

***Patient 4 was scheduled for surgery, but died during cardiac catheterization.

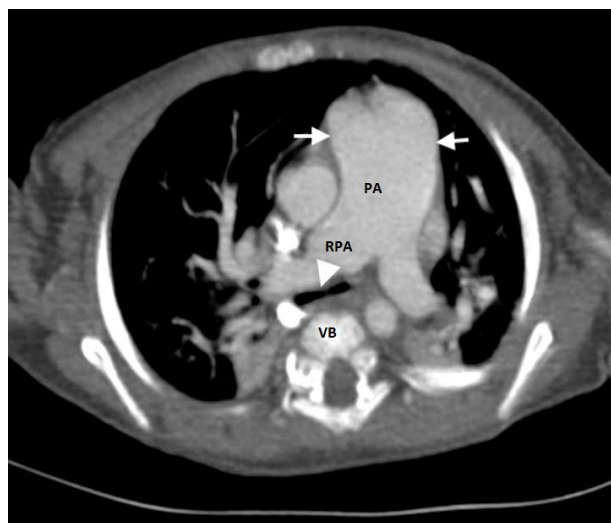****Patient 7 was followed clinically because of a high risk of repeated surgery.

Figure 2. Axial CT image shows an enlarged pulmonary trunk (PT, arrows). The distal trachea (arrowhead) is compressed between the enlarged right pulmonary artery (RPA) and the thoracic vertebral body (VB) (Patient 1).
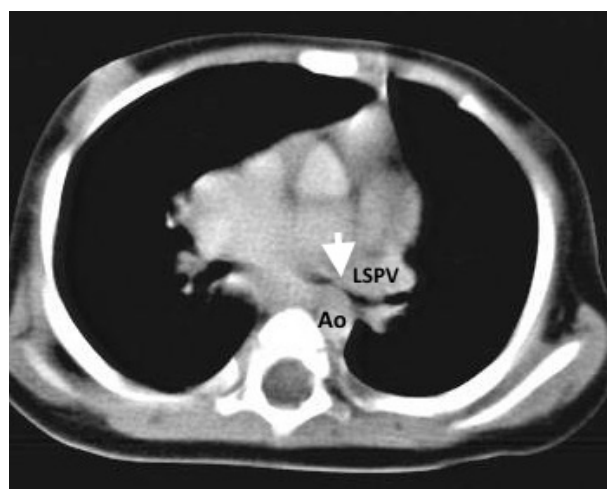
Figure 3. Axial CT image shows compression of the left main bronchus (arrow) between the left superior pulmonary vein (LSPV) and the descending aorta (Ao) (Patient 6).
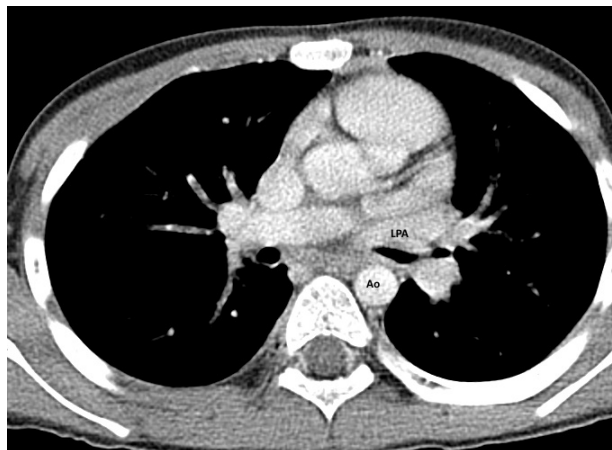
Figure 4. Axial CT image in mediastinal window settings shows compression of the left main bronchus between the left pulmonary artery (LPA) anteriorly and the descending aorta (Ao) posteriorly (Patient 8).

Figure 5. Axial CT image in mediastinal window settings shows the compression of the right main bronchus between the azygous vein posteriorly (Azy) and the right pulmonary artery (RPA) anteriorly (Patient 2).

CHD than in those without (13,14). The central airway in children is easily susceptible to compression, which can result in atelectasis, pneumonia, and respiratory failure, all of which are difficult to tolerate for patients with CHD (15). In the present study, 2 CHD patients with AC died during follow-up; Patient 3 was unable to undergo surgery for cardiac correction and died while on the heart transplantation waiting list, whereas Patient 4 died during cardiac catheterization 1 month after the diagnosis of AC. Cardiac catheterization is a more invasive procedure than thoracic CTA, and children undergoing this procedure require anesthesia. Moreover, patients undergoing cardiac catheterization are exposed to a higher dose of radiation than those undergoing thoracic CTA. Avoiding radiation is especially important for pediatric patients, as there is a known association between radiation exposure and the development of cancer. Although cardiac catheterization is necessary for hemodynamic assessment, it cannot reveal airway compression. It has been reported that cardiac catheterization exposes patients to 15-fold more radiation than thoracic CTA (16). Exposure to ionizing radiation increases the lifetime risk of cancer (17), and children are generally more sensitive to radiation than adults for 25% of cancer types, including leukemia and cancers of the thyroid, skin, breast, and brain (18). Any decision to perform a procedure exposing a child to high levels of radiation should be made carefully.

Whereas bronchoscopy is an effective method for observing airway narrowing and other pathologies, including

malacia, plastic bronchitis, infection, endobronchial lesion, and foreign bodies, children undergoing the procedure require general anesthesia. Nevertheless, bronchoscopy remains the first-line examination for evaluating the airway in children and is reported to be safe for use in children with CHD (19,20). In the present study, 3 of the 11 patients with AC were evaluated via bronchoscopy. However, these children also had to undergo thoracic CTA to observe the anatomical relationship between the compressed airway and the vascular structures. Bronchoscopy could not be performed in 8 patients due to hemodynamic instability and a high risk of anesthesia-related complications; in these patients, AC was diagnosed via thoracic CTA. When planning additional examinations in pediatric patients with CHD, patient status (hemodynamic stability, bleeding risk) and the risk of anesthesia-related complications should be considered. Magnetic resonance (MR) angiography is another technique that can be used in children to avoid exposure to ionizing radiation, but children undergoing MR require general anesthesia. Gadolinium-based contrast agents used in MR angiography can be toxic to infants (21), yet they are commonly used to obtain thoracic vessel images of optimal quality. Moreover, MR angiography is a lengthy procedure that is not as effective as CT for observing the associated abnormalities of the foregut, airway, and lungs (22,23).

Rogers et al. (6) developed a diagnostic algorithm for tracheal compression in children with vascular anomalies. However, there is still a lack of data concerning whether CHD patients with symptoms indicative of distal AC
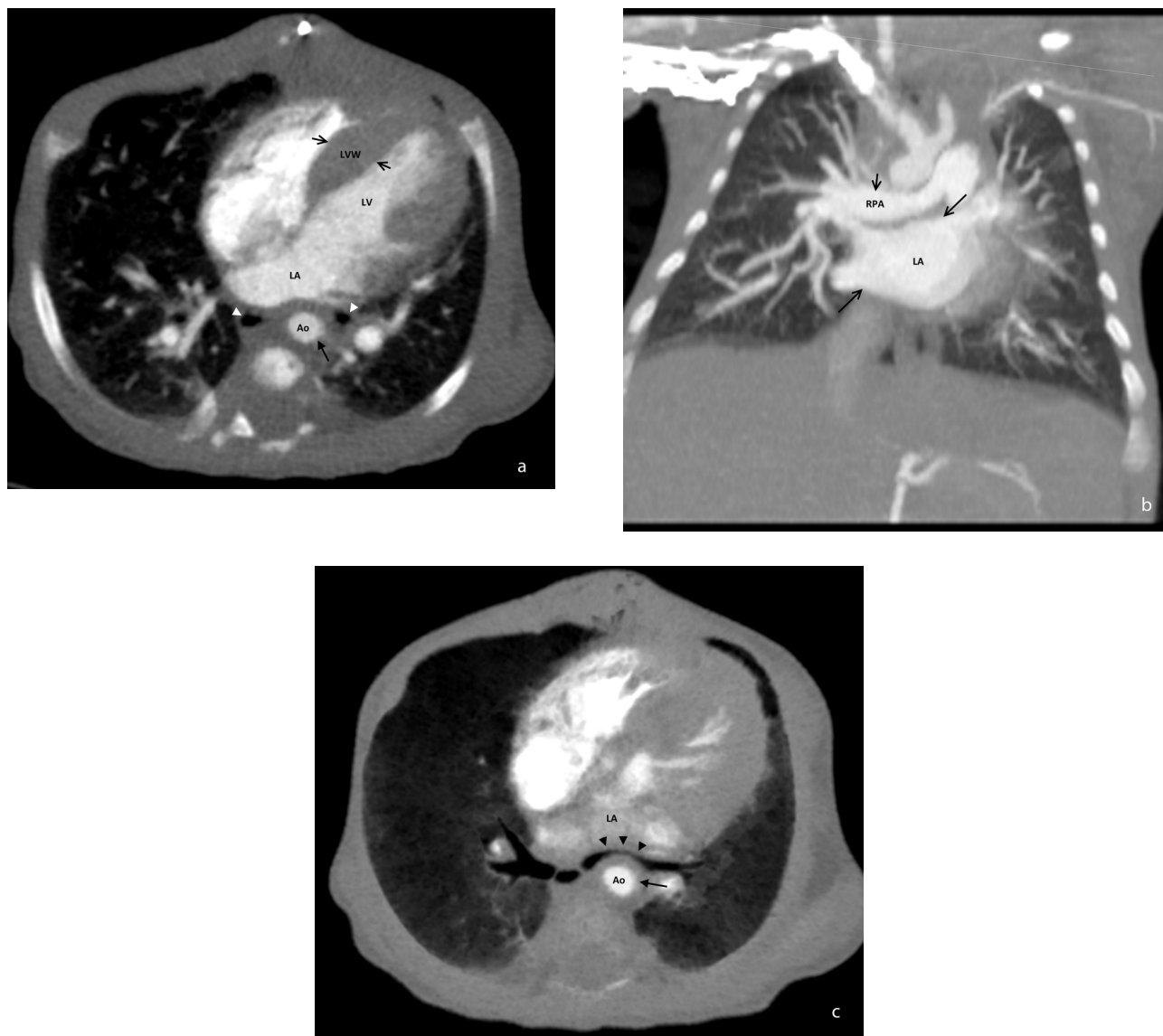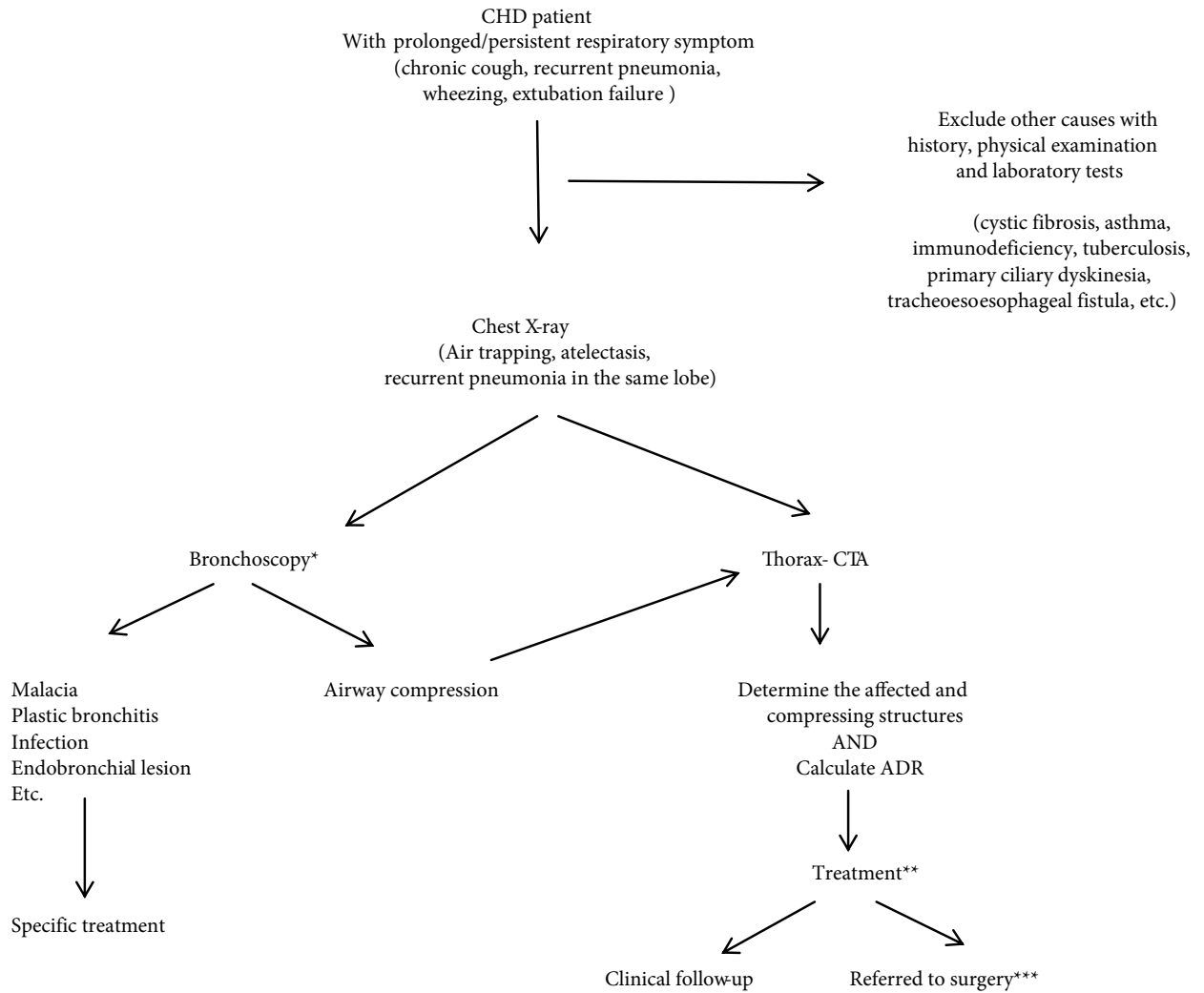
Figure 6. (A) Axial CT image in mediastinal window settings shows the thickening of the left ventricular wall (LVW, short black arrows), right and left main bronchi (white arrowheads), and the descending aorta (Ao, long black arrow). (B) Coronal MIP (maximum intensity projection) image shows the enlarged left atrium (LA, long arrows) and the right pulmonary artery (RPA, short arrow). (C) Axial minimum intensity projection image shows compression of the left main bronchus (arrowheads) between the left atrium (LA) anteriorly and the descending aorta (Ao) posteriorly (Patient 7).

should undergo additional examinations or not. Therefore, we developed a diagnostic algorithm for AC in order to provide optimal clinical care while weighing the risks of additional examinations associated with radiation versus those associated with additional sedation and exposure to general anesthesia (Figure 7). When a child presents with persistent/prolonged symptoms of lower respiratory airway obstruction severe enough to warrant further workup, we believe that the child must first undergo anamnesis and physical examination, followed by chest X-ray and exclusion of pneumonia, cystic fibrosis, asthma, immunodeficiency,

gastroesophageal reflux, primary ciliary dyskinesia, and tuberculosis (based on history and, when necessary, diagnostic tests such as the sweat chloride test, pulmonary function test, and immunological workup). If chest X-ray reveals air trapping, atelectasis, and recurrent pneumonia in the same lobe, AC should be suspected and bronchoscopy and/or thoracic CTA should be performed in accordance with the patient's status. If bronchoscopy reveals airway malacia, plastic bronchitis, infection, endobronchial lesion, etc., specific treatment should be given; however, thoracic CTA is required in cases of suspected vascular compression to

CHD patient
With prolonged/persistent respiratory symptom
(chronic cough, recurrent pneumonia,
wheezing, extubation failure)

Exclude other causes with
history, physical examination
and laboratory tests

(cystic fibrosis, asthma,
immunodeficiency, tuberculosis,
primary ciliary dyskinesia,
tracheoesoesophageal fistula, etc.)

Chest X-ray
(Air trapping, atelectasis,
recurrent pneumonia in the same lobe)

Bronchoscopy*

Thorax- CTA

Malacia
Plastic bronchitis
Infection
Endobronchial lesion
Etc.

Airway compression

Determine the affected and
compressing structures
AND
Calculate ADR

Specific treatment

Treatment**

Clinical follow-up

Referred to surgery***

(ADR: airway diameter ratio; CHD: congenital heart disease; CTA: computorized tomography angipgraphy)

* Patients should be evaluated for the risks of anesthesia.

** Treatment should be decided based on every patient's clinical results, symptoms duration and severity, and ADR.

*** Treatment should be decided with the pediatric pulmonologist, pediatric cardiologist, and cardiovascular surgeon. It could be considered in patients in whom ADR< 0.50.

Figure 7. A diagnostic algorithm for airway compression in CHD.

determine the relationship between the airway and vascular structures. If a patient's status contraindicates general anesthesia, thoracic CTA may be the best option. Thoracic CTA will reveal the affected and compressing structures, allowing one to calculate the ADR. Treatment should be based on the patient's clinical findings, the duration and severity of symptoms, and the ADR. Although the present study included a small patient population, we determined that patients with ADR of <0.50 required surgery, and those with ADR of >0.50 could be followed clinically.

In conclusion, pediatric CHD patients with prolonged/ persistent respiratory symptoms (including lower respiratory airway obstruction) should be evaluated via bronchoscopy and/or thoracic CTA to determine the relationship between vascular structures and the airway. CTA could be considered as a first-line option in patients with CHD and persistent respiratory symptoms, especially in cases with a high risk of complications related to anesthesia or invasive examinations.

References

1. An HS, Choi EY, Kwon BS, Kim GB, Bae EJ, Noh CI, Choi JY, Kim WH, Lee JR, Kim YJ et al. Airway compression in children with congenital heart disease evaluated using computed tomography. Ann Thorac Surg 2013; 96: 2192-2197.

2. Healy F, Hanna B, Zinman R. Pulmonary complications of congenital heart disease. Paediatr Respir Rev 2012; 13: 10-15.

3. Sadoh W, Osarogiagbon W. Underlying congenital heart disease in Nigerian children with pneumonia. Afr Health Sci 2013; 13: 607-612.

4. Harrison MJ, Shapiro AJ, Kennedy MP. Congenital heart disease and primary ciliary dyskinesia. Paediatr Respir Rev 2016; 18: 25-32.

5. Singhal M, Gupta P, Singh RS, Rohit MK, Sodhi KS, Khandelwal N. Cardiovascular causes of pediatric airway compression: a pictorial review. Curr Probl Diagn Radiol 2015; 44: 505-510.

6. Rogers DJ, Cunnane MB, Hartnick CJ. Vascular compression of the airway: establishing a functional diagnostic algorithm. JAMA Otolaryngol Head Neck Surg 2013; 139: 586-591.

7. Leonardi B, Secinaro A, Cutrera R, Albanese S, Trozzi M, Franceschini A, Silvestri V, Tomà P, Carotti A, Pongiglione G. Imaging modalities in children with vascular ring and pulmonary artery sling. Pediatr Pulmonol 2015; 50: 781-788.

8. Habbema L, Losekoot TG, Becker AE. Respiratory distress due to bronchial compression in persistent truncus arteriosus. Chest 1980; 77: 230-232.

9. Hoving MF, Brand PL. Causes of recurrent pneumonia in children in a general hospital. J Paediatr Child H 2013; 49: 208-212.

10. McCallum GB, Bailey EJ, Morris PS, Chang AB. Clinical pathways for chronic cough in children. Cochrane Db Syst Rev 2014; 22: CD006595.

11. Jang WS, Kim WH, Choi K, Nam J, Kim JT, Lee JR, Kim YJ, Kim GB. Aortopexy with preoperative computed tomography and intraoperative bronchoscopy for patients with central airway obstruction after surgery for congenital heart disease: postoperative computed tomography results and clinical outcomes. Pediatr Cardiol 2014; 35: 914-921.

12. Pettersen MD, Du W, Skeens ME, Humes RA. Regression equations for calculation of z scores of cardiac structures in a large cohort of healthy infants, children, and adolescents: an echocardiographic study. J Am Soc Echocardiog 2008; 21: 922-934.

13. Corno A, Giamberti A, Giannico S, Marino B, Rossi E, Marcelletti C, Kirklin JK. Airway obstructions associated with congenital heart disease in infancy. J Thorac Cardiovasc Surg 1990; 99: 1091-1098.

14. Robotin M, Bruniaux J, Serraf A, Uva M, Roussin R, Lacour-Gayet F, Planché C. Unusual forms of tracheobronchial compression in infants with CHD. J Thorac Cardiovasc Surg 1996; 112: 415-423.

15. Griscom N, Wohl M, Kirkpatrick J. Lower respiratory infections: how infants differ from adults. Radiol Clin North Am 1978; 16: 367-387.

16. Watson TG, Mah E, Joseph Schoepf U, King L, Huda W, Hlavacek AM. Effective radiation dose in computed tomographic angiography of the chest and diagnostic cardiac catheterization in pediatric patients. Pediatr Cardiol 2013; 34: 518-524.

17. Kamiya K, Ozasa K, Akiba S, Niwa O, Kodama K, Takamura N, Zaharieva EK, Kimura Y, Wakeford R. Long-term effects of radiation exposure on health. Lancet 2015; 386: 469-478.

18. UNSCEAR. Scientific Annex B: Effects of Ionizing Radiation on Children. Volume II: Sources, Effects and Risks of Ionizing Radiation. Vienna, Austria: United Nations Scientific Committee on the Effects of Atomic Radiation; 2013.

19. Cerda J, Chacón J, Reichhard C, Bertrand P, Holmgren NL, Clavería C, Sánchez I. Flexible fiberoptic bronchoscopy in children with heart diseases: a twelve years experience. Pediatr Pulmonol 2007; 42: 319-324.

20. Lee SL, Cheung YF, Leung MP, Ng YK, Tsoi NS. Airway obstruction in children with congenital heart disease: assessment by flexible bronchoscopy. Pediatr Pulmonol 2002; 34: 304-311.

21. Thomsen HS; European Society of Urogenital Radiology (ESUR). ESUR guideline: gadolinium-based contrast media and nephrogenic systemic fibrosis. Eur Radiol 2007; 17: 2692-2696.

22. McLaren CA, Elliott MJ, Roebuck DJ. Vascular compression of the airway in children. Paediatr Respir Rev 2008; 9: 85-94.

23. Phelan E, Ryan S, Rowley H. Vascular rings and slings: interesting vascular anomalies. J Laryngol Otol 2011; 125: 1158-1163.