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Different Etiological Factors for Pulmonary Hypertension in The Neonatal Form of Scimitar Syndrome - Case Report and Literature Review

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Abstract

Introduction: Pulmonary hypertension in infant Scimitar syndrome (SS) is multifactorial, and 60% of patients had two or more causes. The most crucial factor is a significant left-to-right shunt due to aortopulmonary collaterals (APC). The transcatheter occlusion of APC could reduce the shunt and be a definitive treatment in patients with PH caused only by a left-to-right shunt.

Case Outline: A 10-day-old early-term infant (body weight 2290g) from a twin pregnancy was transferred to our hospital because of inferior right-sided pulmonary atelectasis and prolonged dependence on mechanical ventilation. Echocardiography and CT scan point out infantile type SS, a huge abnormal systemic supply to the lung from the abdominal aorta, and right lung hypoplasia. We decided to perform percutaneous occlusion of abnormal arterial vessel due to the patient's age, body weight and respiratory problems that may worsen after surgical ligation. The massive tortuous anomalous vessel arising from the abdominal aorta supplying blood to a localized region of the right lower lung was occluded with Amplatzer Vascular Plug 4. The echocardiographic examination 7 days after the procedure revealed elevated pulmonary blood pressure (PG at the patent ductus arteriosus (PDA) was estimated at 17 mmHg) and the presence of residual flow through the AVP and scimitar vein. The PDA and the anomalous vessel were closed percutaneously using detachable Coils in a second procedure. At the end of the procedure, complete vessel occlusion was achieved.

Conclusion: This experience suggests that percutaneous procedures are safe and effective for occluding anomalous vessels in the neonatal period.

Keywords: Neonatal Scimitar Syndrome, Pulmonary Hypertension, Amplatzer Vascular Plug 4, Neonatal Interventional Cardiology

Abbreviations: SS: Scimitar Syndrome; PH: Pulmonary Hypertension; ASD: Atrial Septal Defect; CT: Computed Tomography; IVC: Inferior Vena Cava; APC: Aortopulmonary Collaterals; WSPH: World Symposium on Pulmonary Hypertension

Introduction

Scimitar syndrome (SS) is an infrequent inborn cardiopulmonary anomaly first described in 1836, and it was defined as a partial anomalous pulmonary venous connection with a typically shaped anomalous vein draining into the lower caval vein. Scimitar syndrome includes a constellation of abnormalities such as right pulmonary artery hypoplasia, right lung hypoplasia or pulmonary sequestration, total or partial anomalous pulmonary venous return of the right lung (scimitar vein), and cardiac dextroposition [1,2]. The incidence ranges from 1 to 5 per 100,000, but the true incidence may be higher because of silent clinical presentation [1]. Considering differences in clinical presentation, SS is divided into infantile and adult types. Patients with the infantile form had respiratory distress clinical presentation and right heart failure early in life due to pulmonary hypertension (PH); consequently, it always requires intervention [1,3]. A two-staged strategy has been suggested for the percutaneous management of patients with SS [4].

Herein, we report a 24-day-old newborn with SS with an anomalous arterial supply from the aorta decedent to the hypoplastic lung, which was successfully percutaneously closed. Nevertheless, embolization was performed in the newborn period, pulmonary hypertension has not been resolved due to multifactorial etiology, so the patient underwent pneumonectomy.

Written informed consent was obtained from the patient's parents to publish any potentially identifiable images or data in this article.

Case Report

A 10-day-old early-term infant from a twin pregnancy was transferred from the maternity ward to our hospital due to inferior right-sided pulmonary atelectasis and protracted mechanical ventilation dependency. At birth, it weighed 2290 grams and had an Apgar score of 9. Because of signs of respiratory distress, the newborn was intubated, and surfactant was administered. The first radiograph after birth showed atelectasis of the right lower lobe of the lung with moved cardiovascular shadow in the right hemithorax. Considering the maintenance of radiological findings, the newborn was referred to a tertiary pediatric facility for further diagnostic evaluation.

On admission, breath sounds were decreased on the right side, with bilateral rare inspiratory crackles and a systolic ejection murmur of II /VI in the third intercostal space. Two-dimensional echocardiography showed the right pulmonary artery maldevelopment and a moderate secundum-type atrial septal defect (ASD). Color Doppler examination revealed that the two anomalous pulmonary veins without stenosis entered the dilated inferior vena cava (IVC) just below the diaphragm with laminar flow. The blood vessel separated from the abdominal aorta and directed toward the right lung. Computed tomography (CT) showed anomalous systemic supply to the right lower lung lobe from the abdominal aorta (pseudo sequester), partial anomalous vein return of two right pulmonary veins, which drained to the suprahepatic part of the inferior vena cava, hypoplasia of the right lung with and right pulmonary artery (Figure 1). We diagnosed the infantile type of scimitar syndrome. Fiberoptic bronchoscopy pointed out bronchomalacia of the right bronchus. A controlled echocardiographic examination revealed severe pulmonary hypertension (moderate tricuspid regurgitation, pressure gradient of 75 mmHg; mild pulmonary regurgitation, pressure gradient of 37 mmHg); sildenafil was added.

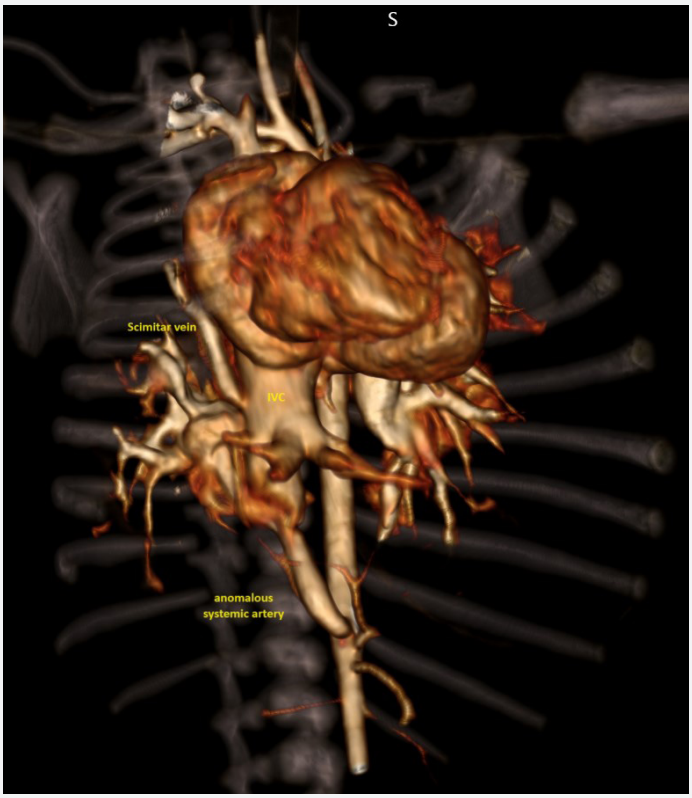

Figure 1: CT finding of our patient pointer out anomalous systemic supply to the right lower lung lobe from the abdominal aorta (pseudo sequester), partial anomalous vein return of two right pulmonary veins, which drained to the suprahepatic part of the inferior vena cava.

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On the 24th day of life, a body weight of 2.8 kg, cardiac catheterization, and angiography via femoral vein and artery with 4-French were performed under general endotracheal an aesthesia. An initial heparin bolus of 100 units/kg was administered. The mean pulmonary artery pressure was 51 mmHg, and the pulmonary to systemic blood flow (Qp/Qs) ratio was 2:1. The right pulmonary artery branch was significantly narrower than the left. Aortography showed the massive anomalous artery arising from the right anterior wall of the abdominal aorta above the celiac trunk, supplying blood to a localized region of the right lower lung (Figure 2). Its narrowest diameter was 4.3 mm; its largest transverse diameter was 6.8 mm, and its length was 19 mm. We selected a device 30% (7 mm) larger than the narrowest artery diameter [Amplatzer Vascular Plug 4]. The device was placed in the second curve via a 4F multipurpose catheter. Control angiography revealed slower flow and low contrast intensity through the deviated blood vessel and the absence of distal translocation of the device. After the procedure, according to the Institutional protocol, a continuous infusion of heparin was prescribed for 24 hours.

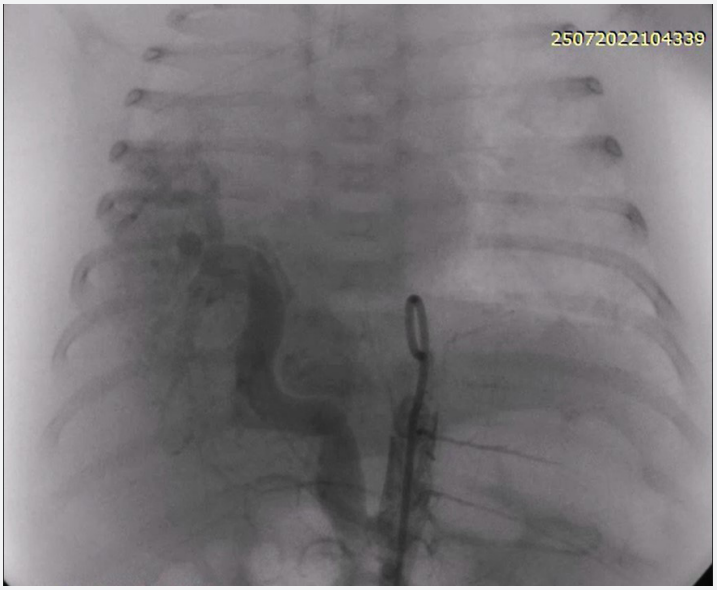

Figure 2: The aortography showed the massive anomalous vessel arising from the right anterior wall of the abdominal aorta above the celiac trunk, supplying blood to a localized region of the right lower lung.

A week after the procedure, the control echocardiography revealed an increased pulmonary blood pressure (peak pressure gradient at the patent ductus arteriosus was 17 mmHg). Residual flow through the AVP and scimitar vein (laminar venous flow) was registered. The PDA type C was 7 mm long, and the pulmonary and aortic ends were 2.8 and 3.5 mm in diameter, respectively (Figure 3 a, b). The PDA and the anomalous artery were closed percutaneously in the second procedure. The procedure was performed with a 4-French sheath and catheters through the right femoral artery. Angiography of the anomalous vessel showed the presence of residual flow through the AVP (Figure 3c), whereas aortography showed that the narrowest diameter of the PDA was 2 mm. The detachable Cook Coil 5x5 was positioned below the AVP and released, resulting in complete vessel occlusion, confirmed on control angiography after 5 minutes. On the other hand, a thinner collateral vessel was observed below the devices (Figure 4a). The second, more delicate supply collateral vessel was occluded with the detachable Flipper Coil 3x3 without residual flow (Figure 4b). The detachable Flipper coil 3x5 was appropriately positioned and released in the PDA. Complete vessel occlusion was executed at the end of the procedure (Figure 4c).

After the intervention, the patient had recurrent right-sided pneumonia as a possible consequence of bronchomalacia and dependence on MV, so we performed a right pulmonary lobectomy after 1.5 months. A median sternotomy revealed an injected, poorly ventilated lung and a pneumonectomy was performed. Due to severe respiratory acidosis, high-frequency oscillatory MV was applied after the surgical procedure. After seven days, conventional MV was continued with a gradual reduction in conditions and satisfactory gas exchange. The patient became septic on the 14th after the surgery and died after two days.

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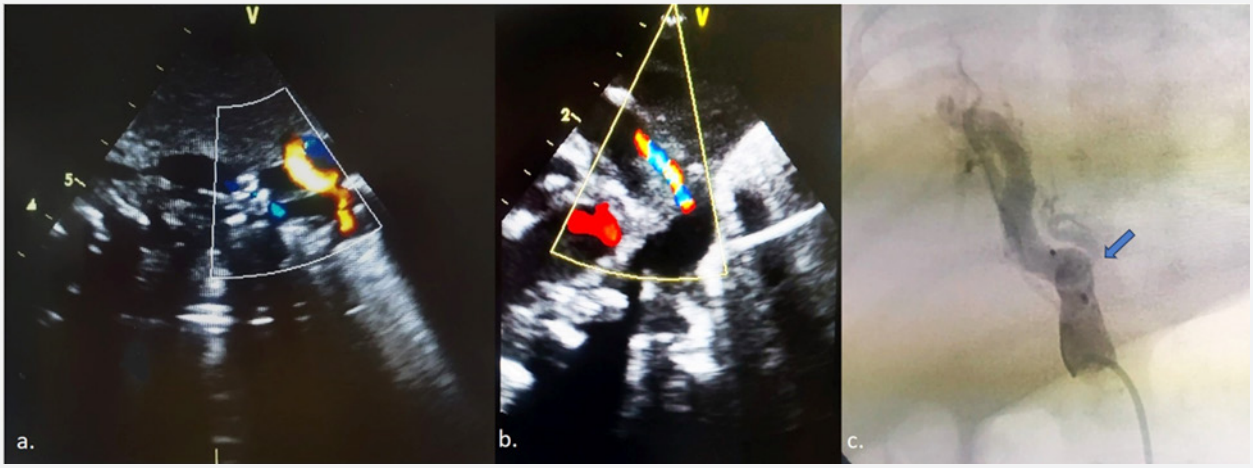

Figure 3: The control echocardiographic examination revealed the presence of residual flow through the AVP and scimitar vein and the patent ductus arteriosus type C (Figure 2a and 2b). Angiography of the anomalous vessel showed the presence of residual flow through the AVP4 (Figure 2c); AVP4 marks with an arrow.

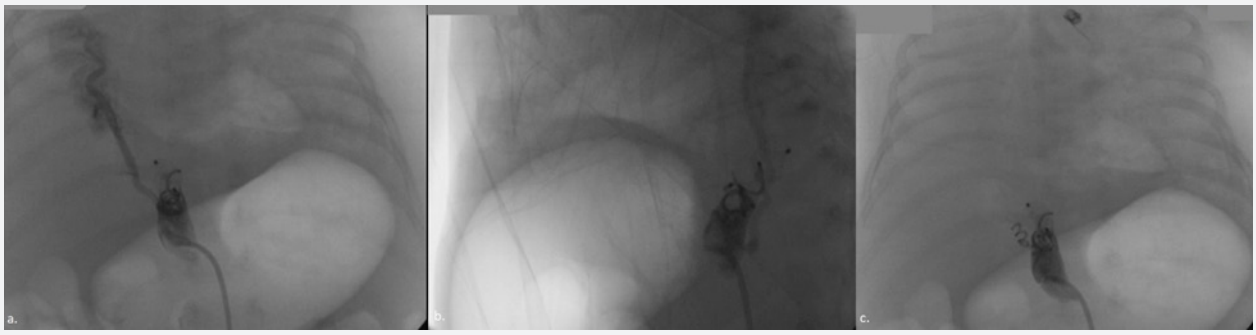

Figure 4:

Angiography of the anomalous vessel showed the presence of residual flow through the AVP. In contrast, the detachable Coil 5x5 (Cook Medical Inc., Bloomington, IN, USA) was positioned below the AVP and released, resulting in complete vessel occlusion, confirmed on control angiography after 5 minutes.

A thinner collateral vessel was observed to detach below the site where the AVP and coil were positioned. The second thinner supply collateral vessel was occluded with the detachable Flipper Coil 3x3 without residual flow.

at the PDA, the detachable flipper coil 3x5 was appropriately positioned and released. At the end of the procedure, complete vessel occlusion was achieved.

Discussion

Pulmonary hypertension in infant Scimitar syndrome could result from one or more factors: 1. the extensive left-to-right shunt through the abnormal pulmonary vein, 2. the left-to-right shunt from the anomalous systemic arterial supply to the right lung, 3. the right lung hypoplasia with underdevelopment of pulmonary vascular bed, 4. pulmonary vein stenosis and obstruction, and 5. other inborn cardiac malformations [5]. Our 3-week-old infant became symptomatic due to PH, probably caused by a combination of left to right shunt due to the abnormal feeding arterial vessel and right lung hypoplasia with a small right pulmonary artery.

Conservative management of pulmonary hypertension in the newborn requires intubation and ventilator support, sedation, sildenafil, and diuretic therapy [4-6]. The final management of scimitar patients implies a two-staged procedure. The systemic collaterals occlusion at the first stage showed clinical improvement by reducing left-to-right shunt and pulmonary artery pressure. Although surgery is considered for a hemodynamically significant left-to-right shunt, the transcatheter occlusion of the systemic collaterals could reduce the shunt and be a definitive treatment in patients with PH caused only by a left-to-right shunt [4,7]. We decided to perform percutaneous occlusion due to the patient's age, body weight, and respiratory problems that may worsen after surgical ligation. Transcatheter embolization with coils was an option, but we decided to occlude the vessel with AVP due to the lower recanalization rate and great vessel diameter.

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Table 1: The table shows a literature review of cases and case series with infant form Scimitar syndrome in whom percutaneous occlusion of aberrant feeding vessel was achieved percutaneously.

Author	Year	No of Patients	Months of Age	Body Weight (g)	Gender	Clinical Presentation	Device	Outcome
Al Rukban H et al. [1]	2014	16 (in 7 pts occlusions)	55 days (1-730)		female 56%	Tachypnea 56.25% Heart failure 25% Failure to thrive 12.5% Cyanosis 6.25%	Coils	One died; Pulmonary pressure normalization after an average of 19 mounts
Bentham JR et al. [11]	2010	1	7			tachypnoea	AVP4	No follow-up
Örün UA [12]	2011	1	9	5200	female	cough, tachypnea, and feeding difficulty	AVP4	one week after the procedure, normal right heart size with significant clinical improvement was noted
Gursu H et al. [13]	2023	1	30				Amplatzer Piccolo occluder	
Wang Z et al. [14]	2016	1	12	9500	male	recurrent pneumonia, sweating, growth retardation	Mreye embolisation coils	clinically well at the 12-month follow-up assessment
Mazandarani M et al. [7]	2022	1	10	2800 (on delivery)	female	Respiratory distress	3 MReye Flipper PDA Closure Detachable Coil	Second occlusion 3 months later
Saltik L et al. [15]	2017	1	21		male	fever and cough	AVP4	Successful transcatheter embolisation
Recker F et al. [16]	2022	6	neonatal period			Prenatal diagnosis	Coil and Plugs occlusion	3/6 died during the first six months
Weber H et al. [17]	2015	1	3		female	cough, congestion and fussiness, desaturation	3 Gianturco coils	Successful occlusion; after 3 days - surgical repair (ASD and TV)
Pamukcu O et al. [18]	2023	1	13 years		female	dyspnea	ADO II	No follow-up
Awasthy N et al. [3]	2014	1	6	2500 (on delivery)	female	respiratory distress	Gianturco coils	Death (respiratory infection)
Aslan E et al. [19]	2015	1	2	3800	female	heart murmur and respiratory distress	AVP2	10 days SpO2 98%, respiratory rate 32/min
Parappil H et al. [20]	2015	1	9 days	3200 (on delivery)	male	respiratory failure	AVP and tornado coils	Successful occlusion
Wang K et al. [21]	2022	22 infants 12 adults (in 15 pts occlusions)	4 months (1 day to 11 months) diagnosis time		male 75.0%	Tachypnea 63.4% Heart failure 27.3% Pneumonia 22.7% Cyanosis 13.6%	Coils	without requiring further interventional or
Bo I et al. [22]	2016	10 patients (in 5 pts occlusions)	42 (3–312)		females 40%	Tachypnoea 90% respiratory distress 30%, recurrent chest infections or wheezing 50%, exercise intolerance with haemoptysis 10%		2 symptom-free, 2 mild wheezing, 1 exercise intolerance

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We chose AVP 4, which requires only a 4-Fr diagnostic catheter for delivery [8-10], but the complete embolization was achieved using Cook coils. Bentham et al. presented the first case with an infantile form of Scimitar syndrome using the new AVP 4 for arterial occlusion. At the same time, Örün et al. occluded arterial feeding vessels a year later with the same device [11,12-20]. They performed successful cardiac catheterization in 7- and 9-month-old children. On the other hand, despite successfully feeding vessel occlusion, some literature data showed a high mortality rate of infants with symptomatic Scimitar syndrome, around 40% (Table 1). Wang K et al. presented 22 patients with infant-type Scimitar syndrome and 12 with adult type. In 15 patients, the aortopulmonary collaterals (APC) were occluded percutaneously, and all the patients did not require further interventional or surgical treatment. Still, in their patient's group, 7 died, 6 had APC, and all had pulmonary hypertension [21-24]. Awasthy N et al. presented a patient who was successfully percutaneously treated but died due to respiratory infection [3].

Although we successfully performed feeding vessel occlusion, our patient's pulmonary hypertension persisted. In most Scimitar syndrome cases, pulmonary hypertension is multifactorial, and 60% of patients had two or more causes. The most crucial factor is a significant left-to-right shunt due to aortopulmonary collaterals. The reversibility of PH demonstrates this in most patients after the suppression of a cardiac left-to-right shunt or of a significant systemic supply to the right lung. Nevertheless, perinatal pulmonary vascular maldevelopment and underdevelopment due to right lung hypoplasia led to the high prevalence of pulmonary hypertension in neonates in this series [25]. At birth, an X-ray of our patient pointed out a complete opacification of a hemithorax, while on the CT examination, right lung hypoplasia with a small right pulmonary artery was described, which could be the second reason for PH. Pretricuspid shunt (atrial septal defect) was the reason for PH in almost 15% of patients [23]. Due to pathogenesis complicity, scimitar syndrome has been added to the fifth group of the World Symposium on Pulmonary Hypertension (WSPH) classification, which includes clinical conditions with unclear or multifactorial mechanisms for PH [23].

Chowdhury U et al. suggested lobectomy in patients with persistent right-sided pneumonia on a grossly hypoplastic lung [24]. Hence, we decided to perform a partial lung resection months after percutaneous occlusion due to bronchomalacia of the right bronchus, persistent right-sided pneumonia and mechanical ventilation dependency.

Pulmonary hypertension in Scimitar syndrome is multifactorial. In some patients, transcatheter occlusion of the feeding vessel could resolve the PH after several months, but more of them underwent more procedures. Nevertheless, the mortality rate is still high due to several reasons of PH. According to the available Scholar database, this is the patient with the lowest body weight with the infantile form of Scimitar syndrome in whom embolization of the systemic feeding with detachable coils was performed. Our experience suggests that percutaneous abnormal vessel occlusion in the neonatal period is safe and effective. Still, although catheterization was done early in life, we did not resolve PH with the procedure. Consequently, the PH in our patient probably was the combination of left-to-right shunt and right lung hypoplasia.

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Ethical approval: approved by local ethical committee.

Authors contribution: All authors have been active participants in the research (including participation in the conception, execution, and writing of the manuscript).

References

1. Al Rukban H, Al Ghaihab M, Tamimi O, Al-Saleh S (2014) Clinical spectrum of infantile scimitar syndrome: A tertiary center experience. Ann Pediatr Cardiol 7(1): 29-33.
2. Abdul Aziz A, Thomas S, Lautner D, Al Awad EH (2018) An Unusual Neonatal Presentation of Scimitar Syndrome. AJP Rep 8(2): e138-e141.
3. Awasthy N, Tomar M, Radhakrishnan S, Iyer KS (2014) Scimitar syndrome: A novel management approach for palliation in a sick infant. J Cardiol Cases 10(2): 48-50.
4. Ghandi Y, Farsi A (2019) Scimitar Syndrome: Pathology, Clinical Presentation, Radiographic Features, and Treatment. Int J Pediatr 7(12): 10611-10620.
5. Kahrom M, Kahrom H (2009) Scimitar syndrome and evolution of managements. Pan Afr Med J 3: 20.
6. Mordue BC (2003) A case series of five infants with scimitar syndrome. Adv Neonatal Care 3(3): 121-132.
7. Mazandarani M, Ahani Azari A, Lashkarbolouk N, Shahkar L (2022) Scimitar Syndrome in a Newborn. J Compr Ped 13(2): e124204.
8. Barwad P, Ramakrishnan S, Kothari SS, Saxena A, Gupta SK, (2013) Amplatzer vascular plugs in congenital cardiovascular malformations. Ann Pediatr Cardiol 6(2): 132-140.
9. Wang W, Li H, Tam MD, Zhou D, Wang DX, et al. (2012) The amplatzer vascular plug: a review of the device and its clinical applications. Cardiovasc Intervent Radiol 35(4): 725-740.
10. Ramakrishnan S (2015) Vascular plugs - A key companion to Interventionists - 'Just Plug it'. Indian Heart J 67(4): 399-405.
11. Bentham JR, Wilson N (2010) Occlusion of anomalous systemic arterial supply in Scimitar syndrome using the new Amplatz vascular plug IV. Cardiol Young 20(3): 349-350.
12. Örün UA, Öcal B, Doğan V, Şenocak F, Karademir S (2011) Transcatheter occlusion of tortuous feeding vessel with new vascular plug in an infant with pulmonary sequestration with Scimitar syndrome. J Cardiol Cases 4(3): e160-e162.

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13. Gursu H, Ozelce B, Cetin I (2022) Successful occlusion of a feeding artery with Amplatzer Piccolo Occluder in a patient diagnosed with Scimitar syndrome. Cardiol Young 27: 1-2.

14. Wang Z, Cai X (2016) Transcatheter intervention in a child with scimitar syndrome. Cardiovasc J Afr 27(3): e9-e11.

15. Saltik L, Ugan Atik S, Bornaun H (2017) Transcatheter correction of Scimitar syndrome: occlusion of abnormal pulmonary venous drainage and vascular supply in an infant. Cardiol Young 27(8): 1627-1629.

16. Recker F, Weber EC, Strizek B, Herberg U, Brockmaier K, et al. (2022) Prenatal Diagnosis and Outcome of Scimitar Syndrome: A Case Series of Six Patients. J Clin Med 11(6): 1696.

17. Weber H, Parikh K (2015) Scimitar Syndrome in a 3-Month-Old Infant. Neonatol Today 10: 1-5.

18. Pamukcu O, Sunkak S, Tuncay A, Erkan GN, Baykan A, et al. (2022) Off-label Use of ADO II® in the Closure of Various Congenital Heart Defects. JPA 3(3): 114-118.

19. Aslan E, Tanıdır İC, Saygı M, Onan SH, Güzeltaş A (2015) Simultaneous transcatheter closure of intralobar pulmonary sequestration and patent ductus arteriosus in a patient with infantile Scimitar syndrome. Turk Kardiyol Dern Ars 43(2): 192-195.

20. Parappil H, Masud F, Salama H, Sajjad ur Rahman (2015) Scimitar syndrome with absent right pulmonary artery and severe pulmonary hypertension treated with coil occlusion of aortopulmonary collaterals in a term neonate. BMJ Case Rep 2015: bcr2014208743.

21. Wang K, Xu X, Liu T, Gao W, Guo Y (2022) Treatment and prognosis of Scimitar syndrome: A retrospective analysis in a single center of East China. Front Cardiovasc Med 9: 973796.

22. Bo I, Carvalho JS, Cheasty E, Rubens M, Rigby ML (2016) Variants of the scimitar syndrome. Cardiol Young 26(5): 941-947.

23. Bonnet D, Szezepanski I, Delacourt C, Malkezadeh-Milani S, Lévy M (2022) Multifactorial pulmonary hypertension in infantile scimitar syndrome. Arch Cardiovasc Dis 115(3): 142-150.

24. Chowdhury UK, Anderson RH, Sankhyan LK, George N, Pandey NN, et al. (2021) Surgical management of the scimitar syndrome. J Card Surg 36(10): 3770-3795.

25. Mathew B, Lakshminrusimha S (2017) Persistent Pulmonary Hypertension in the Newborn. Children (Basel) 4(8): 63.

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