Cardiology in the Young

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Spontaneous neonatal pulmonary arterial thrombosis – cases, mechanisms, and literature review

Ali Abdelhafiz[1], Alexis L. Benscoter[1,2] and Sarosh P. Batlivala[1,2]

[1]The Heart Institute, Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA and [2]Division of Pediatric Cardiology, University of Cincinnati College of Medicine, Cincinnati, OH, USA

Brief Report

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Corresponding author: S. P. Batlivala; Email: sarosh.batlivala@cchmc.org

Abstract

Spontaneous pulmonary artery thrombosis in neonates is rare and can be life-threatening. Clinical presentation may mimic pulmonary hypertension or CHD. Further, not all children present with identifiable risk factors. We report the case of two infants with pulmonary artery thromboses who underwent rapid diagnosis and therapy, one with percutaneous intervention and the other with anticoagulation. We also conducted a literature review to highlight the importance of early identification and referral to a centre capable of performing appropriate medical and interventional therapies.

Background

Neonatal thrombosis ranges from incidentally diagnosed "clinically unsuspected" thrombi to life-threatening thromboembolism. Neonatal thrombosis, in any form, affects approximately 0.2% of neonatal ICU patients, of which ~ 35% are in the venous circulation.[1] Only ~ 45% of patients have an identified aetiology, which range from extrinsic issues such as sepsis and perinatal asphyxia to intrinsic problems including thrombophilias and CHD.[1] Neonatal pulmonary artery thrombosis is rare and can be life-threatening. We report two infants with left pulmonary artery thrombi of unclear aetiology. We also conducted a systematic review of all available literature for this rare condition.

Case report

The Institutional Review Board approved this report, and the patients' families provided informed consent for publication.

Patient 1

Patient 1 was a male born at 33 + 4 weeks gestation via induced, vaginal delivery due to maternal pre-eclampsia to a G1P1 mother with type 1 diabetes mellitus treated with insulin. Birth weight was 3.024 kg, and he was initially asymptomatic. On day 10 of life, he developed tachypnoea and hypoxaemia requiring oxygen at 1 L/min via nasal cannula. Echocardiogram demonstrated normal intracardiac anatomy and ventricular function, though no flow could be demonstrated in the left pulmonary artery. Subsequent MRI was concerning for a proximal left pulmonary artery thrombus with differential perfusion of 80% to the right pulmonary artery and 20% left pulmonary artery with no flow to the left upper pulmonary artery. Laboratory analysis demonstrated normal complete blood count, blood chemistry, blood/urine cultures, inflammatory markers (c-reactive protein and procalcitonin), prothrombin time, fibrinogen, and antithrombin 3 with elevated activated partial thromboplastin time of 36.2 and factor VIII of 370.1. His condition deteriorated slightly on day 13, ultimately stabilising on oxygen at 6 L/min via high-flow nasal cannula.

He was then transferred to our institution and underwent cardiac catheterisation on day of life 14. Diagnostics demonstrated mildly elevated right ventricular pressure with no gradient to the right pulmonary artery. A large, nearly occlusive filling defect, consistent with thrombus, was identified in the left pulmonary artery, originating near the ductal insertion and extending into the lower left pulmonary artery. Overall left pulmonary artery flow was severely diminished, with minimal-to-no left upper pulmonary artery visualisation. Injection past the defect demonstrated normal distal left pulmonary artery arborisation and normal pulmonary venous return. Mechanical aspiration with the Indigo Cat 3 system (Penumbra Inc., Alameda, CA, USA) was considered but was not readily available.[2] Further, we felt that other reported techniques, such as using vascular plugs to extract the thrombus, had unpredictable risk of vascular damage given the location of this thrombus near the pulmonary artery bifurcation.[3] Left pulmonary artery angioplasty was performed using a 4-mm balloon with complete

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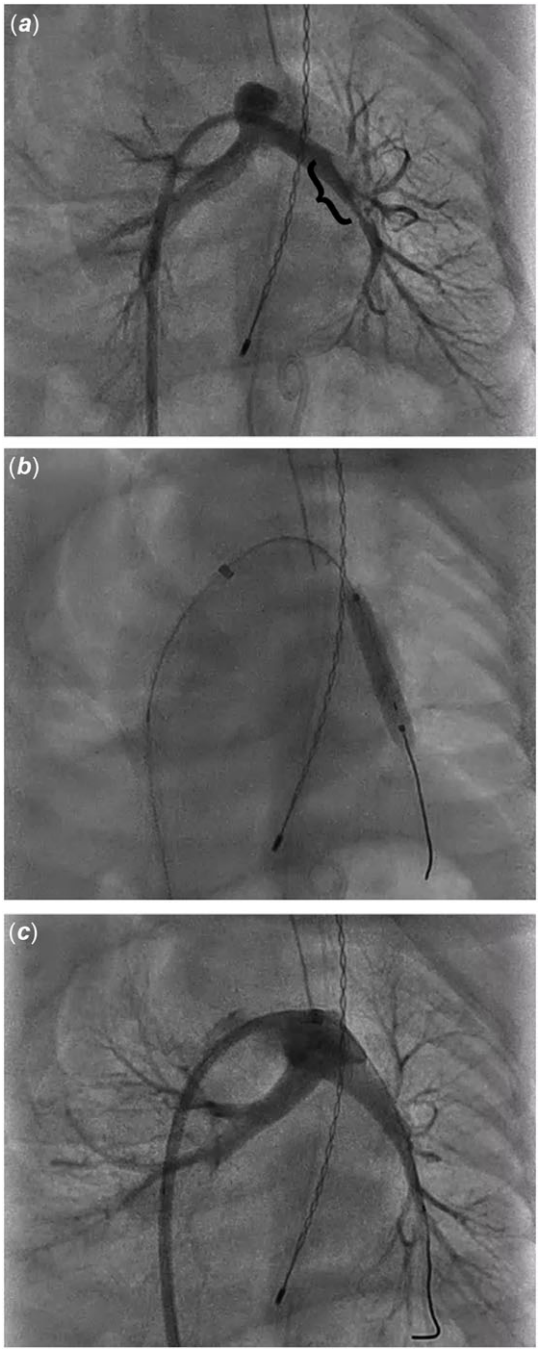

Figure 1. Fluoroscopic scenes of the cardiac catheterisation of patient 1. ***a***) Initial angiogram in the main pulmonary artery demonstrates a filling defect in the left pulmonary artery ({}) consistent with a nearly occlusive thrombus; ***b***) single frame of the balloon thromboplasty; and ***c***) final angiogram in the MPA demonstrating resolution of the filling defect with normal flow in both the right and left pulmonary arteries.

angiographic resolution of the obstruction and mild residual gradient ~ 15 mmHg (Fig. 1 and Videos 1–3).

He was weaned to air the evening of the procedure. In collaboration with haematology, therapeutic enoxaparin was initiated the night of the intervention with a plan for at least 6 months of therapy. Subsequent workup has revealed normal Factor V Leiden, Protein S, and Protein C levels; no prothrombin gene mutations were identified.

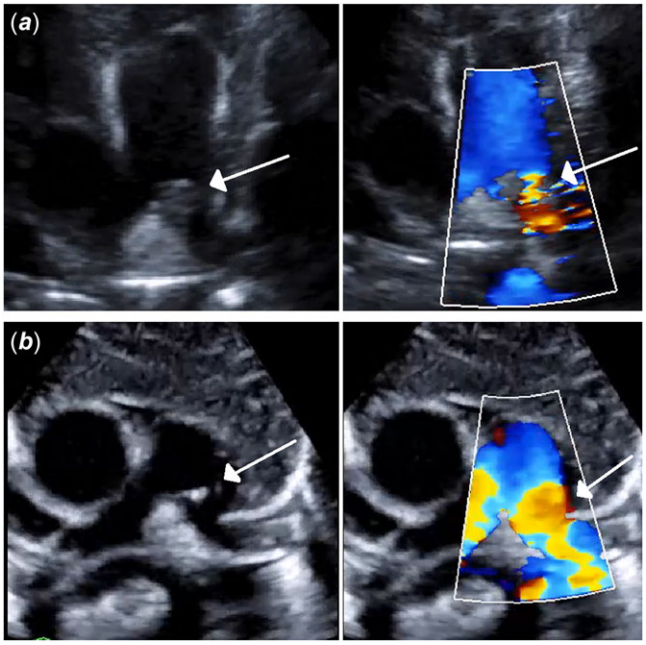

Figure 2. Echocardiographic images of the left pulmonary artery thrombus in patient 2. ***a***) Initial 2D echocardiography (left) demonstrates a large echogenic mass (arrow) at the base of the LPA, in the region of the patent ductus arteriosus insertion. Colour Doppler (right) demonstrates significant flow aliasing (arrow), consistent with a significant obstruction. ***b***) Subsequent images after 5 days of anticoagulation demonstrating a smaller thrombus with improved flow (arrows).

Patient 2

Patient 2 was a male newborn born at 38 weeks gestation after an uncomplicated pregnancy to a G1P1 mother. There were no identifiable maternal risk factors for neonatal thrombosis. His mother was GBS + and did not receive appropriate intrapartum antibiotics, so he underwent screen for sepsis – blood and urine cultures remained negative after 72 hours – and received prophylactic ampicillin and gentamicin for 48 hours. He did well immediately after birth but developed tachypnoea and hypoxaemia 8 hours after birth, being stabilised on oxygen at 2 L/min via nasal cannula. Laboratory analyses were all normal, including complete blood count, urinalysis, cultures, and inflammatory markers. Chest X-ray did not demonstrate acute pulmonary pathology, and a systolic murmur was auscultated at the left upper sternal border radiating to the left axillae, prompting an echocardiogram. The echocardiogram demonstrated normal cardiac anatomy with a large echogenic mass, consistent with thrombus, at the base of the left pulmonary artery; a patent ductus arteriosus was not identified (Fig. 2 and Videos 4–5).

He was transferred to our hospital and started on a therapeutic heparin infusion, which was transitioned to enoxaparin after 4 days. His symptoms slowly improved, and he was weaned to air on day-of-life 3. Repeat echocardiogram on day 5 demonstrated a decrease in thrombus size, so he was discharged with close follow-up. As with patient 1, screening haematologic workup was unrevealing with normal Protein C, Protein S, activated partial thromboplastin time, prothrombin time, and INR levels with no identified prothrombin mutations.

Systematic review

We utilised the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) methodology to conduct a

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Table 1. Review of clinical characteristics, therapies, and outcomes for all identified infants with neonatal pulmonary artery thrombosis.

Year	Author	Demographics	Age*	Maternal risks	Perinatal risks	Symptoms	Imaging findings	Therapy details	Aetiology	F/U duration	Outcome
1986	Clapp	M FT 3.9 kg	At birth	None	Meconium stained fluid	No symptoms with hypoxaemia	- CXR: decreased pulmonary vascular markings - Echo: thrombus in PA bifurcation with flow obstruction to PAs (poorly defined), no PDA - Cath angiogram: thrombus in proximal LPA, extending into MPA, with no LPA flow	Invasive: opted against tPA given medical knowledge of the era; intubated but hypoxaemia persisted so surgical thrombectomy on DOL 3	Possible peripartum stress	9 months	Clinically well, no ongoing issues
2004	Goble	F FT AGA	At birth	DM, smoking, drugs of abuse (cocaine, alcohol), and ibuprofen	None	Cyanosis at birth	Echo: Base of LPA thrombus with near complete flow obstruction, no PDA	Medical: NCO2, enoxaparin x 2 months	Intrauterine PDA closure and intrapartum stress; normal prothrombotic evaluation	3 months	No symptoms, thrombus resolved at 3 months
2004	Goble	M FT AGA	At birth	HTN	None	Hypoxemia at birth	Echo: Base of LPA thrombus with minimal-to-no flow obstruction, no PDA	Medical: NCO2, aspirin	Intrauterine PDA closure; normal prothrombotic evaluation	8 months	No symptoms; tiny residual, organised thrombus at 8 months with no flow obstruction
2004	Goble	M 33 weeks AGA	19 days	None	None	Murmur at discharge physical, no respiratory symptoms	Echo: small, LPA thrombus with no flow obstruction; no PDA	None (observation)	Unrevealing; normal cardiac and pulmonary anatomy, prothrombotic evaluation not performed	No additional	No follow-up given lack of clinical findings
2007	Kenny	M FT 2.2 kg	12 days	Pre-eclampsia	None	Respiratory distress with cyanosis	- Echo: RV hypertension, LPA not well visualised with concern for isolated LPA - Catheterisation: retrospectively interpreted as large thrombus at base of LPA extending into MPA, near completely occlusive of LPA flow	Invasive: cath given concern for isolated LPA. Angiography at time interpreted as severe LPA stenosis, so stent implant. Enoxaparin for 3 months post-stent	Unrevealing; normal cardiac and pulmonary anatomy, normal prothrombotic evaluation	6 months	Overall well, with residual LPA stenosis requiring repeat intervention

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Table 1. (*Continued*)

2007	Lytrivi	M	11 days	h/o DVT (mat GM also)	None	Fever and tachypnoea	- CXR: hyperlucency in left chest - CTA: large, lobulated cystic cavity left lung - Echo: large thrombus at base of LPA, near completely occlusive	Invasive: stabilised with O2, ultimately performed left pneumonectomy	Large, cystic lesion of left lung with in utero LPA thrombus (postnatal large, organising LPA thrombus)		
2008	Sawyer	M FT	4 hours	DM, Factor V Leiden heterozygosity, PE with prior pregnancy	None	Tachypnea, hypoxaemia	- Echo: RV HTN, minimal left pulmonary vein flow with concern for pulmonary vein stenosis - Catheterisation: diagnosed thrombus, no intervention	Medical then invasive: heparin infusion x 3 days after initial cath with no improvement; repeat catheterisation with thrombectomy; post-cath enoxaparin x 6 months	Inherited Factor V Leiden heterozygosity for FVR506Q	6 months	No symptoms; normal lung perfusion scan
2010	El Hassan	M 35 weeks 2.4 kg	20 min	DM (controlled), 2 SAB	Pre-term labour (vaginal, no major stressor)	Acute respiratory failure requiring intubation, then ECMO	- CXR: right tension pneumothorax - Echo: RV hypertension, thrombus at base of LPA with complete flow obstruction - CTA: thrombus in LPA with no distal flow, normal RPA, hypoplastic L lung with arterial collaterals	Medical (with ECMO): Intubated, ECMO for worsening OI; heparin while on ECMO, no other antithrombotics	Maternal DM and possible intrauterine LPA injury given mild left lung hypoplasia and collaterals; normal prothrombotic evaluation	18 months	F/U imaging at 10 with improved aeration of left lung; DC from NICU at 128 DOL on NCO2; doing well at 18 months
2012	Jadhav	M FT 2.5 kg	15 days	None	Oligohydramnios with meconium stained fluid	Acute respiratory distress and cyanosis	- Echo: RV dilation and hypertension, thrombus at base of RPA with complete flow obstruction - CTA: thrombus occluding RPA with small thrombi in multiple LPA lobar branches	Medical: tPA gtt x 24 hours, heparin gtt transitioned to enoxaparin	Pre- and peripartum stress; normal cardiac and pulmonary anatomy, negative prothrombotic evaluation	3 months	Clinically improved after 12 hours tPA, no symptoms at follow-up with no residual thrombus; enoxaparin d/c at 3 months

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Table 1. (*Continued*)

Year	Author	Demographics	Age*	Maternal risks	Perinatal risks	Symptoms	Imaging findings	Therapy details	Aetiology	F/U duration	Outcome
2012	Jadhav	F FT AGA	3 days	None	Brief birth asphyxia with minimal treatment	Cyanosis and murmur	- Echo: RV hypertension, small thrombus at base of LPA with mild flow obstruction - CTA: confirmed echo findings, no anatomic abnormalities	Medical: heparin gtt x 48 hours then enoxaparin x 3 months	Possible peripartum stress; normal cardiac and pulmonary anatomy, negative prothrombotic evaluation	D/C x 3 days, lost to follow-up	Clinically improved after 24 hours heparin; thrombus decreased in size on echo after 3 days
2012	van Schendel	F, 37 weeks 2.1 kg	Hours after birth	None	Third-trimester oligohydramnios with impaired fetal growth	Respiratory distress, diminished left lung auscultation, and systolic ejection murmur	- CXR: diminished left vascular markings and lung volume - Echo: large thrombus at base of LPA with complete flow occlusion of LPA and moderate RPA obstruction - CTA: confirmed vascular findings, noted hypoplastic left lung with small cysts and collaterals to left lung	Medical then invasive: initial heparin gtt, but thrombus extended into RPA. Surgical thrombectomy several days later (thrombus was old and organised)	Infant with Protein C deficiency; FHx of heterozygous type 1 Protein C deficiency	8 months	Doing well, no recurrent symptoms
2013	Miyoshi	M, 31 weeks, 1.7 kg	At birth	None	Fetal hydrops with chylothorax; three thoracoamniotic shunts; pre-term labour (29 weeks) treated with ibuprofen	Immediate respiratory distress, death in hours	- No imaging - Autopsy = well-developed PAs with sub-acute thrombus adherent to ductal insertion in PAs	None	Pre- and peripartum stress; normal cardiac and pulmonary anatomy, no thrombotic disorder evaluation	D**e**ath	
2014	DeMeo	M, 26 weeks, 660 gm	20 days	DM, hypertension	Pre-term labour, breech; UVC (DOL 0-6), LLE PICC DOL6 onwards	Acute respiratory distress, (NCO2 to CPAP), new systolic murmur at LUSB	- CXR = decreased left vascular markings - Echo = base of LPA thrombus with near complete LPA flow obstruction, no PDA - CTA = confirmed vascular findings, no other anatomic abnormalities	Medical: systemic tPA (bolus, 40hr gtt); enoxaparin x 6 weeks	Maternal DM and suspected culture negative sepsis (acquired prothrombotic state); intracardiac lines	6 weeks	Symptoms resolved, no recurrence

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Table 1. (*Continued*)

2016	Isik	M 32 weeks 1.6 kg	6 days	DM-2, 4 prior SAB	Prolonged rupture of membranes (3 days); suspected chorioamnionitis	Acute respiratory distress, tachycardia, and cyanosis	- CXR = decrease vascular markings bilaterally - Echo & CTA = large thrombus from base of LPA, extending into RPA with significant obstruction to both pulmonary arteries, no PDA	Medical: tPA (daily bolus x 2) then enoxaparin (started 5 days after last tPA bolus)	Maternal DM and suspected culture negative sepsis; normal prothrombotic evaluation	3 months	No symptoms; thrombus resolved on imaging
2018	Kimhi	F FT AGA	14 days	No known thrombophilia but 5 SABs	None	Severe dehydration (hyperNa and pre-renal azotemia), hypotension, and cyanosis.	- Echo: normal cardiac anatomy, RV hypertension, large thrombus at bifurcation of branch PAs - CTA: confirmed large thrombus causing severe RPA and moderate LPA obstruction	ECMO stabilisation then surgery: Stabilised on ECMO at outside institution; surgical embolectomy performed when diagnosis confirmed; post-operative enoxaparin for 1 year	Infant with homozygous MTHFR gene mutation, coupled with severe dehydration	1 year	Doing well, no symptoms; thrombus resolved so enoxaparin discontinued at 1 year
2019	Odackal	M 39 weeks 3.3 kg	Hours after birth	None	None	Hypoxemia shortly after delivery; clinical decline over 24 hours despite HFOV, 100% O2	- CXR = normal without evidence of pulmonary process - Echo=RV hypertension, large thrombus in distal MPA with significant occlusion of both RPA and LPA - CTA = confirmed thrombus findings with complete RPA and significant LPA obstruction; thrombus appeared chronic; cystic right lung and hypoplastic left main bronchus	Medical (with ECMO): placed on ECMO with improvement. Enoxaparin started DOL 14. CTA at 6 weeks showed resolution of LPA thrombus and decreased size in RPA; significant decompensation at 9 weeks, echo with severe LPA stenosis and unchanged RPA thrombus, so performed cardiac catheterisation with LPA stent implant	Cystic right lung, left main bronchus stenosis	9 months	Underwent stent dilation at 9 months of age, but clinically well. Remains on enoxaparin.

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Table 1. (*Continued*)

Year	Author	Demographics	Age*	Maternal risks	Perinatal risks	Symptoms	Imaging findings	Therapy details	Aetiology	F/U duration	Outcome
2021	Laviolette	M 40 weeks 3.1 kg	Hours after birth	None	PROM x 7d	Hypoxemia immediately after birth; normal work of breathing	- Echo: RV hypertension, large thrombus at base of LPA with no LPA flow; no PDA - CTA: confirmed large, completely occlusive LPA thrombus	Medical: initially enoxaparin with unchanged CTA after 24 hours, so tPA gtt (41 hours total) with complete resolution on CTA DOL 4; then enoxaparin x 3 months for thromboprophylaxis	Unrevealing: normal cardiac and pulmonary anatomy, normal prothrombotic evaluation	6 months	Doing well, no symptoms
2021	Shrimanth	F 40 weeks	3 days	None	Meconium-stained fluid, no asphyxia	Tachypnea, hypoxaemia requiring NCO2	- Echo = thrombus at base of LPA with moderate flow obstruction - CTA = confirmed echo findings	Medical: heparin infusion x 2 days, then enoxaparin x 6 weeks	Unrevealing: normal cardiac and pulmonary anatomy, normal prothrombotic evaluation	10 weeks	Clinically well; thrombus resolved after 6 weeks enoxaparin
2021	Villeda	M 34 weeks 2.2 kg	13 days	None	C/S for PROM	Cyanosis and respiratory distress	- CXR = diminished vascular markings - Echo=RV hypertension, thrombus in mid-RPA with complete flow obstruction	Medical: tPA gtt x 22 hours, then transitioned to enoxaparin (with a heparin bridge) for 6 months	Unrevealing: normal cardiac and pulmonary anatomy, family refused prothrombotic evaluation	6 months	Clinically well, enoxaparin d/c after 6 months, lost to follow-up thereafter
2022	Inagi	M FT 2.4 kg	At birth	None	Suspected asphyxia	Hypoxemia at birth	- Echo = thrombus in LPA with minimal LPA flow obstruction initially - CTA = ductal aneurysm with thrombus	Medical then invasive: heparin gtt initially; worse clinical status on DOL6 with enlarged thrombus and complete LPA flow obstruction on echo; emergency surgical thrombectomy with ductal aneurysm resection	Ductal aneurysm; normal prothrombotic evaluation	Unclear	Clinically well at follow-up (duration unspecified)

AGA = appropriate for gestational age; C/S = caesarean section; CTA = CT angiography; CXR = chest X-ray; DM = diabetes mellitus; DOL = day of life; ECMO = extra-corporeal membrane oxygenation; F = female; FHx = family history; FT = full term; gtt = infusion; HFOV = high-frequency oscillatory ventilation; HTN = hypertension; LPA = left pulmonary artery; M = male; NCO2 = nasal cannula oxygen; PDA = patent ductus arteriosus; PE = pulmonary embolism; PICC = peripherally inserted central catheter; PROM = premature rupture of membranes; RPA = right pulmonary artery; RV = right ventricle; SAB = spontaneous abortion; tPA = tissue plasminogen activator; wk = week.
*Perinatal risks include central venous lines if noted.

matic review.[4] Data from published reports, which are limited and disparate, were inadequate to perform a meta-analysis. Table 1 provides a concise review of published cases.

Discussion

Though thrombosis in children is fairly common, isolated pulmonary artery thrombus is rare with no reliable incidence estimate. Therapies depend on the clinical condition as well as the child's age. With our cases, we opted for an interventional procedure for patient 1 given that he was nearly 2 weeks old at the time of diagnosis. Conversely, we opted for medical management in patient 2, who was diagnosed just hours after birth and only requiring nasal cannula oxygen.

Virchow's triad provides insight into the development of pulmonary artery thrombi. A plausible mechanism in common with all pulmonary artery thrombi is endothelial disruption related to closure of the patent ductus arteriosus at its insertion in the PA roof, serving as the nidus for the thrombus. In most reported cases, another process was identified that contributed to stasis of flow (e.g., intracardiac lines) or created a prothrombotic state (e.g., serious peripartum bacterial infection and inherited thrombotic disorder).[5]

Our systematic review identified 20 reported cases of pulmonary artery thrombi in neonates, with a general review provided in Table 1.[6–22] Aetiologies varied but can be broadly categorised into those with intrinsic and extrinsic predisposing factors. Intrinsic issues include anatomic anomalies and thrombotic disorders, while extrinsic issues largely comprised either a serious infection, maternal conditions that predispose to neonatal thrombosis (e.g., diabetes mellitus), or perinatal asphyxia with severe stress.

As outlined in the table, anatomic anomalies associated with pulmonary artery thrombus include premature closure of the patent ductus arteriosus, pulmonary artery/ductal vascular anomalies, as well as cystic pulmonary disease.[7,9,22] Patients with any form of thrombophilia are potentially at risk, with reports of Protein C and Factor V Leiden deficiencies.[10,13] Per our review of the literature, a notable number of cases did not have a clear identifiable risk factor.[7,8,19–21] And others were noted to be associated with risks that are unclear, such as meconium-stained amniotic fluid or very brief birth asphyxia requiring "minimal resuscitation."[6,12,20] The therapies were also significantly disparate, ranging from no intervention, to systemic anticoagulation and tissue plasminogen activator, transcatheter angioplasty, and even surgical thrombectomy. Some patients were also initially stabilised on extra-corporeal membrane oxygenation. No significant therapy-related complications were described in the reports, including no bleeding complications with tissue plasminogen activator and no procedure-related complications after catheterisation or surgery.

Diagnosing a pulmonary artery thrombus begins with recognising it as part of the differential. Asymmetric chest X-ray vascular markings are often the first clue. An echocardiogram will typically identify the thrombus, though some obstructions may occur beyond the pulmonary artery hilum so may be missed by echocardiography. Though diagnostic angiography was reported in many older cases, we recommend proceeding to CT angiography if echocardiographic findings are unusual (e.g., decreased flow to one branch pulmonary artery or pulmonary veins).[10,13]

Finally, management should be directed by the clinical condition as well as identified aetiologies. Medical management with anticoagulants may be reasonable for patients with mild symptoms requiring minimal treatments (e.g., nasal cannula oxygen), while systemic tissue plasminogen activator, transcatheter angioplasty, surgical thrombectomy, and even extra-corporeal membrane oxygenation may be indicated for more ill patients with larger occlusive/near-occlusive thrombi or evidence of right ventricle strain.[10,15,17]

Conclusion

Pulmonary artery thrombus is a rare and serious condition in neonates with variable aetiologies. Clinicians should be vigilant for this issue, especially since the condition may masquerade as primary pulmonary hypertension. Earlier diagnosis will allow for appropriate therapy which can range from medical management to surgical thrombectomy and even extra-corporeal membrane oxygenation support. Ongoing larger sample size studies need to be conducted to determine the significance of such associations.

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Competing interests. None.

Ethical standard. This content of this manuscript consists of review of medical care and outcomes only and did not involve human subjects experimentation. As such, the manuscript complies with the ethical standards of the Helsinki Declaration of 1975, as revised in 2008, and has been approved per policy of the Cincinnati Children's Hospital Institutional Review Board.

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