Pulmonary Embolism in Children: A Case Series

Sonam Agrawal¹, Yash Shrivastava², Rishi Bolia³, Prateek K Panda⁴, Indar K Sharawat⁵, Nowneet K Bhat⁶

Abstract

Background: Pulmonary embolism (PE) is a life-threatening event with a mortality of ~10%. It is relatively uncommon in children and literature regarding the condition is sparse. In adults, the classical clinical presentation is with pleuritic chest pain, hemoptysis, and dyspnea, whereas in children, the presentation is often nonspecific.

Materials and methods: Clinical features, risk factors, and outcome of children with PE presenting to our unit between December, 19 and March, 2020 were recorded.

Results: Four children [mean age: 10 (6–16) years, 3 females], all presenting with tachycardia and dyspnea were diagnosed with PE. Different risk factors such as deep vein thrombosis, nephrotic syndrome, softtissue infection, and infective endocarditis (IE) were identified in all patients. One child died while others responded to anticoagulation.

Conclusion: We aim to highlight the importance of timely recognition of PE in children with known risk factors for the same. Early recognition and timely treatment of PE are critical to save lives.

Keywords: Children, Dyspnea, Pulmonary embolism, Tachycardia.

Indian Journal of Critical Care Medicine (2020): 10.5005/jp-journals-10071-23682

INTRODUCTION

Pulmonary embolism (PE) is a serious disease with mortality of $\sim 10\%^1$). It is an important cause of mortality in adults but is less commonly reported in children. Diagnosis of PE is often overlooked due to its low incidence and nonspecific signs and symptoms, which include dyspnea, chest pain, cough, hemoptysis, syncope, tachycardia, hypoxia, and fever. We report four cases of pediatric PE who presented to our intensive care unit (Table 1).

CASE DESCRIPTIONS

Case 1

A 11-year-old male presented with fever, lower limb swelling, and breathing difficulty for 5 days. On examination, he was febrile, with tachycardia and tachypnea. Chest X-ray revealed bilateral in-hemogeneous nodular opacities with cavitatory consolidation. Venous Doppler showed right popliteal vein thrombosis. Computed tomography pulmonary angiography (CTPA) revealed septic PE. Child was treated with intravenous antibiotics and subcutaneous low-molecular weight heparin (LMWH) and other supportive care. However, after 2 days of intensive care stay, the child died due to massive acute pulmonary hemorrhage.

Case 2

A 6-year-old girl, a known case of nephrotic syndrome (NS) presented with breathlessness, anasarca, and decreased urine output for 7 days. On examination she had tachycardia. Her electrocardiogram (ECG) showed sinus tachycardia with ST segment elevation and T wave changes (Fig. 1A). After an echocardiogram raised suspicion of PE, a CTPA was performed which showed acute PE (Fig. 1B) for which anticoagulation was started with LMWH and later switched over to warfarin.

Case 3

A 7-year-old girl presented with abdominal pain in right iliac fossa, fever, and dyspnea for 5 days. On examination, she had

^{1–6}Department of Paediatrics, All India Institute of Medical Sciences Rishikesh, Uttarakhand, India

Corresponding Author: Yash Shrivastava, Department of Paediatrics, All India Institute of Medical Sciences Rishikesh, Uttarakhand, India, Phone: +91 9717295788, e-mail: dr.yash.24@gmail.com

How to cite this article: Agrawal S, Shrivastava Y, Bolia R, Panda PK, Sharawat IK, Bhat NK. Pulmonary Embolism in Children: A Case Series. Indian J Crit Care Med 2020;24(12):1272–1275.

Source of support: Nil Conflict of interest: None

pallor, tachypnea, tachycardia, and tenderness in right iliac fossa. Laboratory analysis showed leukocytosis, raised erythrocyte sedimentation rate and C-reactive protein. Chest X-ray showed heterogeneous opacities in the left lower segment. CT scan revealed an iliopsoas abscess and right iliosacral osteomyelitis. During the hospital stay, she developed right lower limb tenderness and venous Doppler showed right popliteal vein thrombosis. CTPA showed PE. She was treated with antibiotics, LMWH, and surgical exploration with evacuation of pus.

Case 4

A 16-year-old female presented with fever, cough, and dyspnea for 20 days. On examination she had tachycardia and a continuous murmur best heard in the left second intercostal space. Chest X-ray showed multiple cavitatory lesions in the right lung with right-sided hydropneumothorax (Fig. 2). Echocardiography showed a large patent ductus arteriosus (PDA). CTPA was suggestive of septic emboli. Child was treated with IV antibiotics, LMWH, and intercostal drain for hydropneumothorax.

DISCUSSION

The PE in the pediatric population is less common when compared with adults though exact incidence is not known, as many cases

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	Patient 1	Patient 2	Patient 3	Patient 4
Age	11 years	6 years	7 years	16 years
Sex	Male	Female	Female	Female
Weight (kg)	28	30	28	44.5
Height (cm)	120	112	113	155
BMI (kg/m²)	15	24	22	19.7
Presentation	Fever	Seizure	Abdomen pain Fever	Fever
	Lower limb swelling	Hypotension		Cough
	Respiratory distress			Difficulty in breathing
Risk factor for pulmonary embolism	Right popliteal vein thrombosis	Nephrotic syndrome	Right hip osteomy- elitis with right psoas abscess with septic thrombophlebitis	Infective endocarditis MRSA
Vitals				
Heart rate	136/minutes	150/minutes	142/minutes	126/minutes
Respiratory rate	36/minutes	28/minutes	28/minutes	22/minutes
SpO ₂	90%	97%	95%	96%
Blood pressure	100/60 mm Hg	106/72 mm Hg	110/66 mm Hg	110/60 mm Hg
Investigation				
D-dimer	5.5 mg/L			
ECG	Sinus tachycardia	Sinus tachycardia ST-segment elevation	Sinus tachycardia	Sinus tachycardia
Echocardiography	Dilated RA and RV RV dysfunction Moderate TR Severe PAH	Dilated RA and RV Mild TR	Dilated RV with mild PAH	ACCHD, 10 mm large PDA, Left to right shunt >2:1. Dilated LA and LV. Mod- erate TR. Severe PAH
Venous Doppler of lower limbs	Right popliteal vein thrombosis	Normal	Right popliteal vein throm- bosis	Normal
CT-pulmonary angiography	Random peripheral predominant nodules and cavitatory consolidation in bilateral lung parenchyma (feeding vessel sign seen) with mild bilateral pleural effusion-septic emboli	Acute thrombus in posterior basal segmental branch (3rd order) of the right descending pulmonary artery	Pulmonary thromboembo- lism involving segmental branches of left lower lobe pulmonary artery with possible segmental pulmonary infarct in left basal segments of left lower lobe	Thick-walled cavities in B/L lung parenchyma with air fluid levels? Septic emboli with large PDA (10 mm) with dilated MPA, RPA, LPA, LA, and Left ventricle
Outcome	Died	Discharged and in follow-up	Discharged and in follow-up	Discharged and in follow-up PDA closure planned

Table 1: Clinical features, salient investigations and outcome of children with pulmonary embolism

RA, right atrium; RV, right ventricle; LA, left atrium; LV, left ventricle; PDA, patent ductus arteriosus; MPA, main pulmonary artery; RPA, right pulmonary artery; LPA, left pulmonary artery; PAH, pulmonary arterial hypertension

remain unrecognized. In an autopsy series, Buck et al.² reported that ~50% of children with PE had signs and symptoms of PE in the ante-mortem period and the diagnosis was considered only in a third of them. Studies have shown an incidence of PE of 0.3 to 1.4 per 10,000 children per year,^{1.3} which is increasing in recent years. Carpenter et al.⁴ reported a 200% increase in PE in hospitalized children in the United States, increasing from 9.8 to 24.7 per 10,000 over a period of 14 years. PE has a bimodal age of presentation and is more common in neonates and adolescents.^{1.3,5}

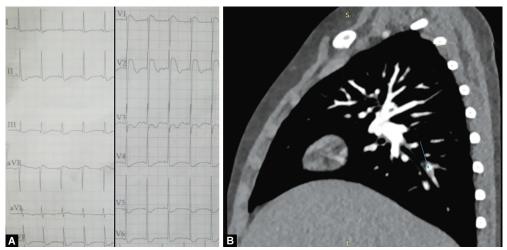
Neonates have high risk of PE due to indwelling central venous catheter,³ while obesity³ and oral contraceptives^{1,3,5} are the most common risk factor in adolescents. Deep vein thrombosis (DVT) and hypercoagulable states are other important risk factors.⁶ We identified risk factors for PE in all children in the current series.

Case 1 had DVT as a risk factor, which is an important risk factor for PE in adults but is uncommon in children. The incidence of DVT is 5 to 14 per 10,000 pediatric hospital admission per year in various studies.^{6–8} Possibility of a thrombophilic state was considered; however, workup could not be done as it was deferred initially due to the acute presentation and the child died subsequently.

Case 2 had NS which is an important risk factor for PE.^{5,9,10} In NS, abnormality in coagulation cascade predisposes to venous thromboembolism and PE.^{10,11}

Case 3 had osteomyelitis of the right hip and a psoas abscess, which lead to septic PE. Septic PE is less common in children. Softtissue abscess and osteomyelitis are common causes of septic PE.¹²

Case 4 had PDA with infective endocarditis (IE), hydropneumothorax requiring inter-costal drainage and methicillin-



Figs 1A and B: (A) 12-lead ECG showing sinus tachycardia and ST-segment elevation in lead V1-V2; (B) CT pulmonary angiography showing nonenhancing thrombus in posterior basal branch of right posterior descending artery

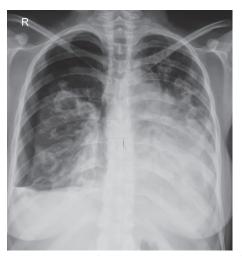


Fig. 2: Chest X-ray showing multiple cavitatory lesions in right lung with right-sided hydropneumothorax

resistant *Staphylococcus aureus* infection. Wong et al.¹² found that *S. aureus* was the causative organism in 9 out of 10 cases, 8 of which were methicillin resistant. In this series, three patients had pneumothorax like our case.

The ECG in PE shows sinus tachycardia, $S_1Q_3T_3$ pattern and ST segment changes.^{1,5} All our cases had sinus tachycardia, and case 2 had ST segment elevation in lead V1-V2. Chest X-ray in case of septic emboli shows multiple peripherally located areas of nodularity and cavitation, as seen in cases 1 and 4. D-dimer is sensitive but not specific for PE, it is not validated for use in children.^{1,5} Echocardiography shows right ventricular dilation and dysfunction, and pulmonary hypertension. Venous Doppler ultrasound is useful to diagnose DVT. CTPA^{1,5} is the diagnostic test of choice and has advantage of being widely available. Pulmonary angiography is the gold standard¹ but is invasive and not routinely performed. Venous Doppler ultrasound, echocardiography, and CTPA were performed in all cases (Table 1).

All four cases had tachycardia and dyspnea at presentation, which was attributed to other causes initially leading to a diagnostic delay of approximately 24 hours. On retrospectively looking into the cause of delay, it was noticed that PE was not kept in the initial differential diagnosis due to nonspecific clinical features as a presenting complaint and the relative rarity of PE in children even though a risk factor was present in all. Pointers which lead us to diagnosis of PE are as follows: case 1 venous Doppler showed DVT and D-dimer was raised, case 2 ECG and echocardiographic findings raised possibility of PE, case 3 CT abdomen showed right iliopsoas abscess and venous Doppler right popliteal vein thrombosis, and in case 4, PDA with IE and worsening of respiratory status. Cases 1 and 2 had submassive PE and cases 3 and 4 had low risk PE according to American Heart Association (AHA) guidelines.¹³

Management involves anticoagulation after hemodynamic stabilization. LMWH is given in acute phase and switched over to warfarin after 5–10 days. Duration of anticoagulation⁵ for first episode of venous thromboembolism is 3–6 months, long-term for patients with chronic risk factors and lifelong for patients with of congenital thrombophilic disorders. Most children recover uneventfully with mortality ~10% in children. All our children were treated with LMWH for 5–7 days and are now on warfarin for 6 months. One child died due to pulmonary hemorrhage and other three improved with anticoagulation.

CONCLUSION

The PE is rare in children and a high index of suspicion is required to diagnose it in patients with risk factors. ECG and echocardiography help to suspect PE and CTPA is important to confirm the diagnosis. Early recognition and timely treatment of PE is critical to save lives.

ACKNOWLEDGMENT

The authors thank Dr Aakriti Kapoor for providing the images of cases.

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