

## Case Report

# A rare cause of pediatric respiratory distress: pulmonary embolism

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### ABSTRACT

Pediatric Pulmonary Embolism (PE) is a very rare condition as compared to adults. However, the incidence of pulmonary embolism in children is increasing as a result of accurate and timely diagnosis. Authors hereby reporting a 7-year-old female child who presented to us with acute onset respiratory distress and hypotensive shock, with no other significant illness in the past. The diagnosis of pulmonary embolism was made based on chest x ray, electrocardiogram and echocardiography. After ascertaining the diagnosis of PE, child was started on unfractionated heparin infusion along with other supportive measures. But the child went into refractory shock followed by asystole, could not be revived. Pulmonary embolism is a rare and potentially fatal, condition that often goes unrecognized among the pediatric population. There should be high index of suspicion on PE in a child who presented with sudden onset respiratory distress with other supportive radiological, 2d echo and lab findings.

**Keywords:** Echocardiography, Heparin, Pediatric pulmonary embolism, Respiratory distress

### INTRODUCTION

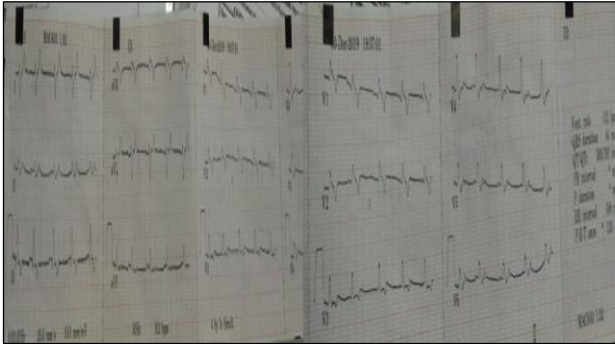
Paediatric PE was described almost two centuries ago by von Löschner, still knowledge of pediatric PE remains fragmented.<sup>1</sup> Historically, infection was thought as a common cause of pulmonary embolism in children, but it is becoming clear that PE is increasingly related to cancer, congenital heart disease, acquired and inherited thrombophilia, and central line placements.<sup>2</sup> Studies suggests that the incidence of PE in children is 8.6-57 per 100,000 in hospitalized children, and 0.14-0.9 per 100,000 in general population of non-hospitalized children.<sup>3</sup> Authors hereby reporting a 7-year-old female child presenting to us with history of respiratory distress and shock with no other significant past history or contributing factors with acute massive pulmonary embolism.

### CASE REPORT

A 7-year-old female child referred to hospital with history of fever of 4 days duration and rapid breathing and chest pain since that day morning. At admission child

was found to have cyanosed with feeble pulse and cold peripheries. Child was immediately shifted to pediatric intensive care unit with oxygen on flow. The initial physical examination revealed the following findings: heart rate of 180/min, respiratory rate of 66/min, blood pressure of 60/30 mmhg, presence of central cyanosis, saturation at room air was 70 %, with decreased air entry on left upper lung fields. The child was started on 100% oxygen through non rebreathing mask and slow intravenous fluid boluses given along with monitoring liver size and started on inotropes in view of fluid refractory shock. According to the history and physical examination findings initial diagnosis made was severe pneumonia with septic shock or myocarditis with cardiogenic shock. Arterial blood gas done showed severe hypoxaemia (pao<sub>2</sub> of 40) with severe metabolic acidosis with high lactate. Electrocardiography (ECG) done showed sinus tachycardia with right bundle branch block with S1Q3T3 (Figure 1). Chest x ray done showed left upper lobe haziness (Figure 2). These clinical symptoms and signs raised a suspicion of PE. Echocardiography done showed dilated right atrium and right ventricle with large right ventricular thrombi or

mass (Figure 3) with occluded left pulmonary artery (Figure 4) suggesting pulmonary embolism. Investigations done revealed leucositosis (36500 cells/cumm) with elevated C reactive protein (>100). Troponin I level was normal.



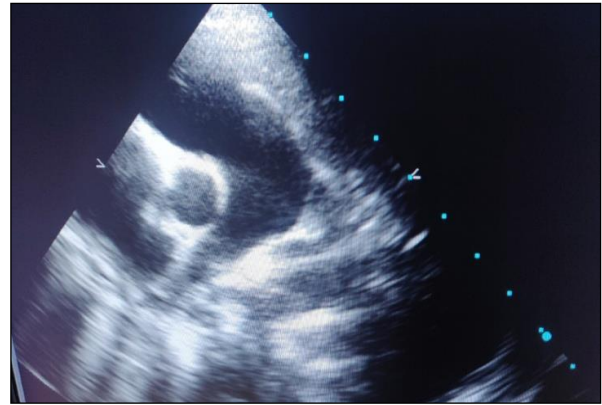
**Figure 1: Right ventricular strain with right bundle branch block and S1Q3T3 pattern.**



**Figure 2: Left upper zone haziness suggestive of left upper lobe infarction.**



**Figure 3: Dilated right atrium and right ventricle with large right ventricular thrombi.**



**Figure 4: Absent left pulmonary artery flow suggestive of left main pulmonary artery embolism.**

In PICU after fluid boluses and inotropic support, child was intubated and mechanically ventilated in view of fluid refractory catecholamine resistant shock. After ascertaining the diagnosis of PE, child was started on unfractionated heparin infusion (75 units/kg loading) and planned for thrombolysis by alteplase and CT angiography. But the child went into bradycardia followed by asystole. Expired within 2 hours of admission.

#### DISCUSSION

PE is a relatively rare entity in pediatric population and often difficult to diagnose because of nonspecific clinical symptoms and signs.<sup>4</sup> Studies suggests that the incidence of PE in children is 8.6-57 per 100,000 in hospitalized children, and 0.14-0.9 per 100,000 in general population of non-hospitalized children.<sup>3</sup> Despite knowing predisposing risk factors and the associated symptoms and signs, the diagnosis of PE often delayed because of the presenting disorder may often overlap with other clinical condition.<sup>5</sup> Unlike adults pediatric PE may occur without any classic symptomatology.<sup>6</sup> Classic symptoms of PE when present include increased shortness of breath, pleuritic chest pain, hemoptysis, cough and syncope. Signs of PE include tachycardia, tachypnoea and signs of right heart failure.<sup>2,7</sup> In this case, child presented with rapid breathing, chest pain and cyanosis, on examination child was found to have tachycardia, tachypnoea and hypotensive shock with severe hypoxaemia. The incidence of PE in pediatric population depicts a bimodal distribution, with the highest incidence in infants and teenagers.<sup>8</sup> The most common predisposing factor for pediatric PE was found to have the presence of central venous catheter.<sup>9</sup> Other risk factors for which can be listed for pediatric PE are infection, dehydration, malignancy, congenital heart disease, systemic lupus erythematosus, thrombophilic disorders, prolonged total parenteral nutrition. In this case authors could not find any significant risk factor, authors concluded the cause of PE could be either due to thrombophilia or septic thrombi.

As for the diagnosis concerned there are some laboratory and imaging modalities which can guide the diagnosis of PE in children. Electrocardiogram, echocardiography, arterial blood gas and chest x-ray, are few easily available modalities to diagnose PE in suspected children. CT pulmonary angiography is a primary imaging technique for the diagnosis of PE because of its speed reliability and ability to specifically detect other pathologies.<sup>10</sup> Arterial blood gas analysis in PE typically shows hypoxemia, hypocapnia and respiratory alkalosis.<sup>11</sup> In this case arterial blood gas analysis showed hypoxemia with metabolic acidosis (because of shock). Electrocardiogram in PE can show sinus tachycardia, right axis deviation, right bundle branch block and S1Q3T3 pattern.<sup>12</sup> In this case electrocardiogram showed all the classical features. Echocardiogram in PE can depict right ventricular dilatation, hypokinesis, abnormal motion of interventricular septum and tricuspid regurgitation.<sup>13</sup> In this case echocardiography showed dilated right atrium and right ventricle with large right ventricular thrombi with absent flow in the left main pulmonary artery.

Due to the lack of proper guidelines and many controversies, the management of pediatric PE have been extrapolated from that of adults.<sup>14,15</sup> Initial therapy for pediatric PE remains either Unfractionated heparin (UFH: 75 U/kg over 10 min intra-venous followed by 20 U/kg/h for patients >1 year, 28 U/kg/h for <1 year of age) and low molecular weight heparin (LMWH: 1 mg/kg twice daily subcutaneously for patients >2 months of age, 1.5 mg/kg twice daily subcutaneously for patients <2 months of age). Pharmacological or mechanical thrombolysis can also be beneficial for the management of pediatric PE.<sup>16</sup> In this case, authors started the child on unfractionated heparin infusion and planned for pharmacological thrombolysis.

## CONCLUSION

PE is a very rare in pediatric population, but highly fatal condition that often goes unrecognized. There should be high index of suspicion who presents with classical signs and symptoms along with the presence of other risk factors. The mainstay of treatment remains heparin (unfractionated or low molecular weight heparin) along with pharmacological or mechanical thrombolysis.

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