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ARDS in a Paediatric Patient: An Unusual Case of Systemic Edema and Respiratory Failure

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Abstract : Acute Respiratory Distress Syndrome (ARDS) is a major cause of respiratory failure in critically ill patients, often requiring mechanical ventilation. It is commonly caused by conditions like pneumonia, sepsis, gastric aspiration, or severe trauma. The condition is characterized by non-cardiogenic pulmonary edema, hypoxemia, and inflammation, leading to impaired gas exchange. This case describes an 8-year-old previously healthy child who presented with a week-long history of swelling, starting with facial puffiness and progressing to lower limb edema, followed by tachypnea, shortness of breath, and reduced urine output. On examination, the child exhibited tachycardia, hypotension, and prolonged capillary refill, suggesting circulatory instability. Chest X-ray and CT scans revealed bilateral ground-glass opacities and consolidation, confirming ARDS. Despite negative microbiological results, the child received comprehensive supportive care, including fluid resuscitation, high-flow nasal cannula therapy, vasopressors, antibiotics, and diuretics. After three days of treatment, the child showed improvement, with stable vital signs, decreased edema, and normalized urine output. Upon discharge, the child was stable and prescribed pantoprazole, amoxicillin-clavulanate, and ursodeoxycholic acid. This case highlights the importance of early diagnosis and aggressive management in paediatric ARDS cases to ensure positive outcomes.

Index Terms **- Tachycardia, Edema, Hypoxemia, Fluid resuscitation, Shortness of breath, vasopressors.**

INTRODUCTION

Acute Respiratory Distress Syndrome (ARDS) is a prevalent cause of respiratory failure in critically ill individuals, marked by the rapid onset of non-cardiogenic pulmonary edema, hypoxemia, and the requirement for mechanical ventilation. It is most frequently triggered by conditions like pneumonia, sepsis, gastric content aspiration or severe trauma. ARDS is seen in roughly 10% of all intensive care unit patients gollaby[(1)]. PARDS can be induced by both direct and indirect lung injuries. Direct causes include pneumonia, aspiration, inhalation injury, lung contusion, chest trauma, and submersion accidents. Sepsis, shock, pancreatitis, trauma, cardiopulmonary bypass, transfusion-related acute lung injury (ALI), burns, and increased intracranial pressure are examples of indirect causes[(2)]. Pathology in the lung arises in the environment of dysregulated inflammation, improper activity of leukocytes and platelets and uncontrolled activation of coagulation pathways[(3)]. According to the researchers, the mortality rate for PARDS was as high as 17%, while the prevalence was 3.2%[(4)]. Actually, one of the required criteria for diagnosing ARDS was the presence of bilateral lung infiltrates as shown by chest radiography together with "refractory" hypoxemia and decreased respiratory system mechanics (stiff lung). The dependent lung regions showed the largest distribution of CT densities, suggesting that the ARDS lung was non homogeneously affected by the illness process[(5)]. Patients taking NIV or HFNC should have their PARDS and potential PARDS diagnosed using Pao_2/Fio_2 or Spo_2/Fio_2 [(6)]. Supportive care for ARDS consists of addressing the underlying injury, providing nutritional support, preventing venous thromboembolism and stress ulcers and providing mechanical ventilation[(7)].

CASE PRESENTATION

An 8-year-old previously healthy child was admitted with a one-week history of generalized swelling, initially involving facial puffiness and later progressing to the lower limbs. Two days before admission, the child developed shortness of breath, tachypnea, and decreased urine output. On examination, the child was tachycardia (136 bpm), hypotensive (85/50 mmHg), and had prolonged capillary refill time (4 seconds). Chest X-ray showed bilateral haziness with ground-glass opacities in Fig:1, and a chest CT scan confirmed ground-glass opacification in both lungs in Fig 2.

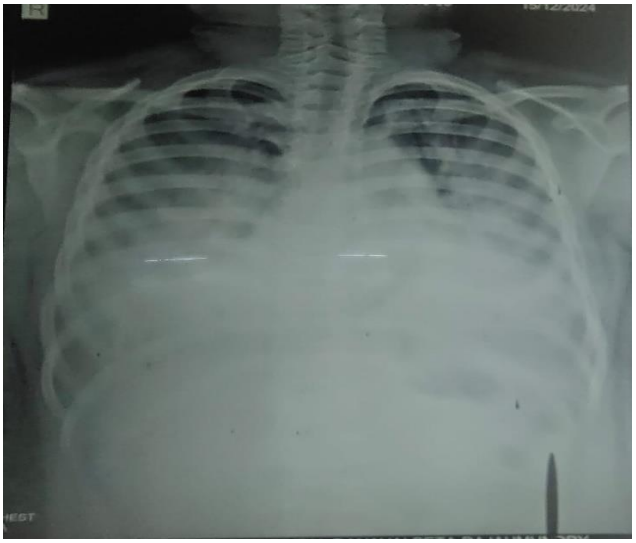

Figure 1 chest x ray

The chest X-ray shows widespread diffuse opacities in both lungs, which is a key indicator of Acute Respiratory Distress Syndrome (ARDS). The lungs appear cloudy with extensive alveolar infiltrates, pointing to fluid buildup caused by increased capillary permeability. The usual lung clarity is lost, suggesting pulmonary edema or consolidation. Air bronchograms, where the air-filled bronchi stand out against the fluid-filled alveoli, are also present, further supporting the diagnosis of ARDS. Importantly, there is no significant enlargement of the heart, which helps distinguish ARDS from cardiogenic pulmonary edema. These radiographic signs, along with clinical symptoms like severe hypoxia and respiratory distress, strongly suggest ARDS and should prompt further evaluation with a CT scan and clinical correlation.

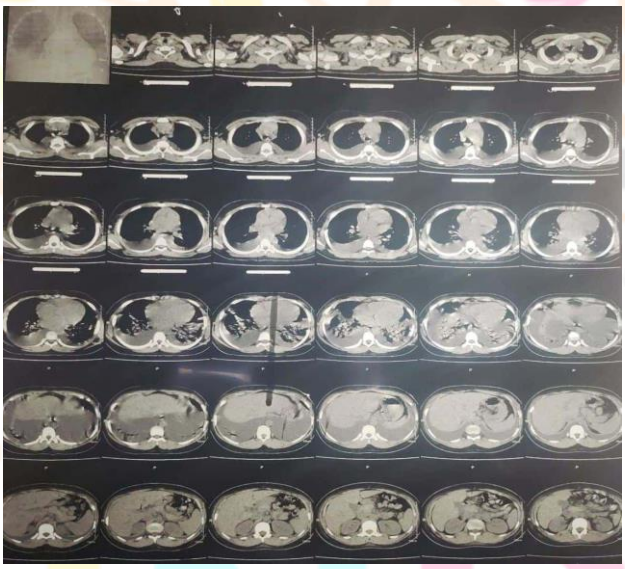

Figure 2 CT scan

The axial CT scans of the chest indicate Acute Respiratory Distress Syndrome (ARDS), characterized by bilateral ground-glass opacities (GGOs), consolidation, and air bronchograms. The GGOs appear as hazy areas of increased lung density, reflecting alveolar fluid accumulation. In some regions, the lungs show dense consolidation, suggesting severe inflammation and fluid infiltration. Additionally, air bronchograms, seen as dark linear structures within consolidated areas, indicate air-filled bronchi surrounded by fluid-filled alveoli. These imaging findings align with the pathophysiology of ARDS, highlighting diffuse alveolar damage and impaired gas exchange.

The child was initially resuscitated with a fluid bolus, but hypotension persisted, and noradrenaline infusion (0.1 mcg/kg/min) was started. The child was placed on high-flow nasal cannula (HFNC) with 60% FiO_2 and 25 liters/min oxygen flow. Treatment included antibiotics (meropenem,doxycycline,ceftriaxone,vancomycin), vasopressors (noradrenaline,dobutamine,),diuretics(furosemide), nebulization (budecort, salbutamol), and analgesics (paracetamol). Despite initial concerns for an infectious or inflammatory etiology, microbiology cultures were negative. After three days of supportive care, the patient's condition improved with stabilization of vital signs, reduced edema, and normalized urine output. At discharge, the child was prescribed pantoprazole (40 mg OD), amoxicillin-clavulanate (625 mg BD) and udiliv (150 mg BD) for five days. The child was discharged in stable condition with no signs of acute respiratory distress or renal failure.

DISCUSSION

ARDS is a common clinical condition of abrupt respiratory failure caused by widespread lung inflammation and oedema[8]. When ARDS develops, patients often have variable degrees of pulmonary artery vasoconstriction and may eventually develop pulmonary hypertension. ARDS has a significant fatality rate and there are few effective therapy options available to combat the illness[9]. ARDS presents with lung inflammation,alveolar edema and hypoxemic respiratory failure. This clinical syndrome's pathophysiology includes inflammatory, proliferative and fibrotic phases as it progresses[10]. An 8-year-old previously healthy child presented with generalized swelling over a week, progressing from facial puffiness to lower limb edema,followed by respiratory distress and reduced urine output. Examination revealed tachycardia, hypotension and prolonged capillary refill time, suggesting circulatory instability. Imaging showed bilateral ground-glass

opacities in the lungs. Despite initial fluid resuscitation, hypotension persisted, requiring noradrenaline infusion. The child was managed with HFNC (60% FiO_2, 25 L/min), broad-spectrum antibiotics, vasopressors, diuretics, nebulization, and analgesics. Microbiological cultures were negative, but with three days of supportive care, the child's condition improved with stabilized vitals, reduced edema, and normal urine output. At discharge, the patient was stable and prescribed a short course of pantoprazole, amoxicillin-clavulanate, and ursodeoxycholic acid. This case highlights the importance of early recognition and comprehensive management of pediatric patients with acute respiratory and circulatory compromise.

CONCLUSION

An 8-year-old child with a one-week history of swelling, tachypnea, and decreased urine output was diagnosed with Acute Respiratory Distress Syndrome (ARDS) after chest X-ray and CT scans revealed bilateral ground-glass opacities and consolidation. Despite negative microbiological cultures, suggesting a non-infectious cause, the child required intensive supportive care, including high-flow oxygen therapy, vasopressor support, fluid resuscitation, and diuretics. With this comprehensive treatment, the child's condition improved, with normalized urine production, reduced edema and stable vital signs. Upon discharge, the child was prescribed supportive medications for gastrointestinal and liver protection with careful monitoring recommended. The child's full recovery demonstrates the importance of early identification and multimodal management in ARDS.

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CONFLICT OF INTEREST

The authors state that they have no financial ties or conflicting interests that might have looked to have influenced the work described in this case report.

STATEMENT OF INFORMED CONSENT :

In order to publish this case report and any related clinical data, the patient gave written informed consent. To protect patient anonymity, all identifying information has been eliminated in compliance with worldwide patient privacy standards and HIPAA regulations.

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