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Case Report

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Pulmonary embolism in a developing country. a case report

Abstract

Introduction: Pulmonary embolus (PE) refers to obstruction of the pulmonary artery or one of its branches by material that originated elsewhere in the body.

Case presentation: This was a 5-year-old, Caucasian girl (weight, 20 kg) without preceding symptoms, she was admitted to our Hospital on 07 July 2015; previous history relevant only for valproic acid during the last year for seizures. She was at school, and had a sudden loss of consciousness followed by tonic seizure activity. She was brought by paramedics to the emergency room, where she was asymptomatic.

Discussion: There are few cases in the literature of PE in a previously healthy child. In pediatric patients PE has a mortality of 30%, mostly because the delay in diagnosis and treatment. In children it has been proven that 96% of PE patients have at least one risk factor and almost 50% of them have 2 or more risk factors.

Conclusion: The diagnosis of pulmonary embolism is difficult and complex in the pediatric population, however in the presence of syncope and refractory hypoxemia is important to think in PE and start the diagnostic approach.

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Introduction

Pulmonary embolus (PE) refers to obstruction of the pulmonary artery or one of its branches by material that originated elsewhere in the body. Different from adults, in children the PE is rare, with the estimated incidence at 0.07/10 000 children, which is lower than the 2–5% incidence reported in adults.[1,2] Virchow described a pathologic triad for thrombosis formation: endothelial injury, stasis of blood flow, and blood hypercoagulability. In adults most thrombi arise in the deep venous system of the lower extremities, pediatric thromboembolic disease is not so easily characterized, however it has been described a bigger incidence in the upper venous system. Once embolization into the pulmonary artery occurs, depending on the size of the clot, symptoms may range from hemodynamic collapse to pleuritic chest pain.[3]

The most common risk factors in pediatric patients are: central venous catheter, malignancy, cardiac surgery, other major surgery, and infection/sepsis. It has been identified a directly proportional risk based on the severity of the disease and the presence of two or more risk factors, mainly those multiple trauma and undergoing major surgery. The Prospective Investigation of Pulmonary Embolism Diagnosis reported the most common signs and symptoms in patients with PE and no preexisting cardiopulmonary disease, no single finding or combination of these signs or symptoms was felt to be sensitive or specific enough to confirm or exclude the diagnosis of PE.[4] Fortunately in pediatrics, the patients with PE usually have one or two risk factors and some of the following symptoms: tachycardia, crackles, dyspnea, pleuritic chest pain, cough and/or hemoptysis.

The gold standard for diagnosing PE is angiography, however, the lung ultrasound is taking more relevance due to the ease of the study to the patient's bed and by combining echocardiography pulmonary ultrasound diagnosis becomes more sensitive and specific. Another useful study is angiotomography, an increasingly accessible in hospital units study. Chest radiography and electrocardiogram show nonspecific changes so cannot be considered reliable for establishing the diagnosis. D-dimer, produced by the breakdown of fibrin is considered reliable to exclude the diagnosis of pulmonary embolism in patients at low risk score, however in children the presence or absence of a value of D-dimer positive does not exclude the need for perform an imaging study.

When a patient presents with suspected PE, initial resuscitative therapy should focus upon oxygenating and stabilizing the patient. Resuscitative therapy may range from supplemental oxygen to ventilatory support, hemodynamic support, and empiric anticoagulation. There are different types of anticoagulation available for the treatment of PE in children; unfractionated heparin, thrombolytic agents, thrombectomy, vitamin K antagonists and low-molecular-weight heparin, with the latter two the most commonly used.

Case Report

This was a 5-year-old, Caucasian girl (weight, 20 kg) without preceding symptoms, she was admitted to our Hospital on 07 July 2015; previous history relevant only for valproic acid intake 25 mg/kg/day during the last year for seizures. She was at school, and had a sudden loss of consciousness followed by tonic seizure activity. She was brought by paramedics to the emergency room, where she was asymptomatic. Respiratory rate was 32 breaths/min; heart rate 88 beats/min; blood pressure 95/60 mm Hg; and oxyhemoglobin saturation of 99% room air; she was with Glasgow coma scale of 15 and bilateral fine rales at pulmonary and mild tachypnea at physical examination. Laboratory workup showed hemoglobin 12.9 g/dL, leukocytes, 20,400/ml, platelets 445/ml; creatinine0.6 mg/dL; serum albumin, 3.4 g/dL; cholesterol 131; AST, 156; ALT, 37; venous blood gas analysis pH 7.28; PCO_2 36 mmHg; PO_2 40 mmHg; HCO_3 16.9; lactate 4.4 mmol/L. No abnormalities were seen in her chest radiograph (Figure 1).

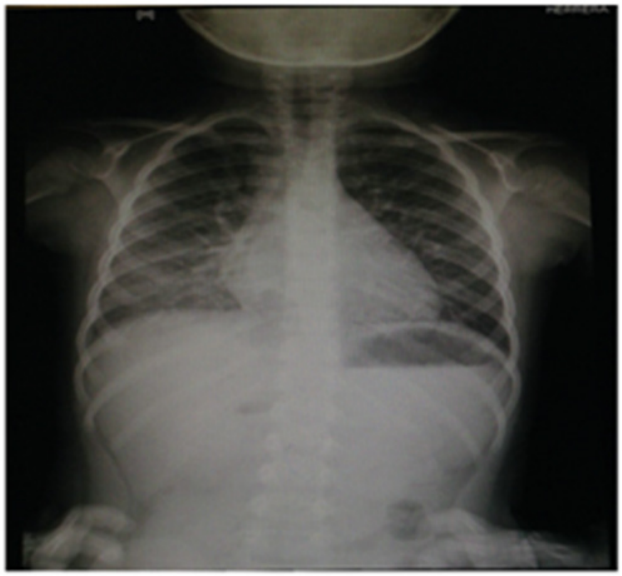

Figure 1 Chest X-ray, 5 years old female with no pathological findings

Considering the previous signs and laboratory parameters; treatment was started with antibiotic ceftriaxone 50 mg/kg and requirement fluid therapy; she evolved with cardiopulmonary deterioration; with persistent desaturation with 100% inspired oxygen, increased respiratory effort and sinusal tachycardia; we performed endotracheal intubation followed by clinical deterioration with hypotension, bradycardia and cardiac arrest; we started advance CPR, during the maneuvers she developed ventricular arrhythmias, fibrillation with no response to treatment and no return to spontaneous circulation, declaring time of death 10:00 in the morning. An autopsy was performed and reported the presence of a pulmonary thrombosis and an atrioventricular intracavitary thrombi (Figure 2 & 3).

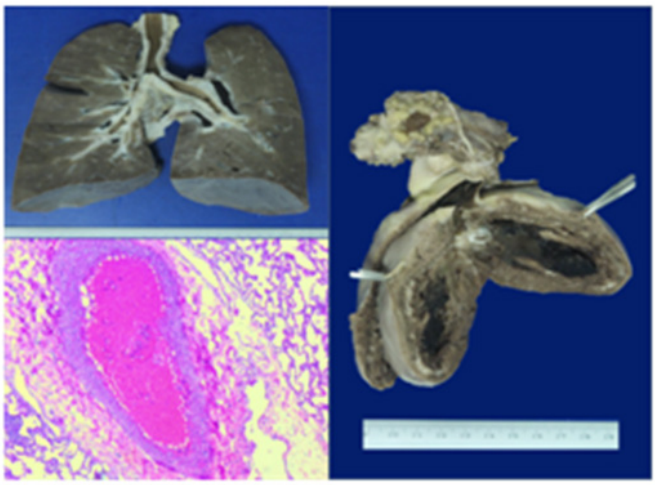

Figure 2 & 3 Pulmonary thrombosis and atrioventricular intracavitary thrombi.

Discussion

There are few cases in the literature of PE in a previously healthy child. In pediatric patients PE has a mortality of 30%, mostly because the delay in diagnosis and treatment. In children it has been proven that 96% of PE patients have at least one risk factor and almost 50% of them have 2 or more risk factors. The presentation of symptoms in our patient was unspecific and without any associated risk factor, which delay the diagnosis leading to hemodynamic instability that conditioned an event of cardiac arrest and ultimately death.[5]

Diagnosis of PE is difficult, the laboratory test are unspecific, including D-dimer who have a predictive negative value of 96%, D-dimer with a negative value of 96 % to discard the diagnosis with ≤400ng / dl values in adults has not been studied in the pediatric population. Chest radiography and electrocardiogram may be normal in up to 50 % of patients with pulmonary embolism and angiotomography and ultrasound are operator - dependent and not available in most hospitals in Latin American countries. In conclusion, the diagnosis of pulmonary embolism is difficult and complex in the pediatric population, however in the presence of syncope and refractory hypoxemia is the responsibility of the treating physician to think in PE and start the diagnostic approach to not delay treatment and reduce the risk of lethal complications. Better guidelines indicating diagnostic investigations and appropriate management are required to improve the care and outcomes in children.

Competing Interests

The author(s) declare(s) that there is no conflict of interest regarding the publication of this paper.

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None.

Conflict of Interest

There were no financial interest or conflict of interest.

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