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Case Report

Spontaneous pneumothorax associated with *Staphylococcus aureus*

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ABSTRACT

Pneumothorax is a condition in which there is free air between the parietal and visceral pleural sheets. The neonatal stage is the most common period when it develops during childhood. It is a problem that requires urgent intervention. We presented the case of a spontaneous pneumothorax in a newborn with positive culture of *S. aureus*.

Keywords: Pneumothorax, *S. aureus*, Spontaneous

INTRODUCTION

Pneumothorax is a condition in which there is free air between the parietal and visceral pleural sheets. The neonatal stage is the most common period when it develops during childhood. It is a problem that requires urgent intervention. Symptomatic pneumothorax is reported in 0.08% of all live births. It is reported 1-2% in newborns, 5-7% in those with birthweight below 1500 g, but 30% in patients with an underlying pulmonary pathology and requiring mechanical ventilation.[1,2] It is an entity commonly found in neonatal intensive care units, causing high mortality and morbidity, especially in premature infants. Early diagnosis is necessary, as well as adequate treatment to reduce complications and mortality.[3,4-6]

Sepsis is the third most common cause of death among newborns, with about 225,000 newborns dying each year worldwide, with *S. aureus* being one of the most isolated pathogens. Data on the microbial etiology of neonatal sepsis and the antimicrobial resistance profiles of its causative agents are needed to inform targeted and effective treatment and prevention strategies.[7-20]. We presented the case of a neonate neonatal *S. aureus* sepsis who developed a spontaneous pneumothorax.

CASE REPORT

Admission diagnosis

Pneumothorax, bacterial pneumonia, shock, staphylococcemia, staphylococcal postulosa impetiginized dermatoses, suspected congenital pulmonary malformation were the admission diagnosis.

Discharge diagnoses

The discharge diagnosis were congenital toxoplasmosis, systemic arterial hypertension, toxic liver disease, with cholestasis, to rule out immunodeficiency. A 7-day-old boy was admitted to the pediatrics department for febrile symptoms and papuloerythematous lesions with purulent content predominantly on the face, neck and head. Saturation of 88% was detected in the morning, so O_2 was indicated by approximation at 0.5 per minute, achieving saturations of 99%. On physical examination she was active and reactive, good coloration, adequate hydration, hemodynamically stable, no evidence of low output, normocephalic, normotensive anterior fontanel, multiple scab-like lesions scattered on face, scalp, postauricular region and thorax with xerosis on hands, feet and abdomen, pustules on the skin of the scalp, normodynamic precordium with murmur in mitral focus

II/VI, without irradiation, distended abdomen, soft and depressible, with peristalsis present, of adequate intensity and frequency, with apparent discomfort on palpation, hepatomegaly of 4-5 cm below the costal ridge. On 26 April 2022 respiratory deterioration with oxygen in nasal prongs and increased work of breathing, observed in radiography with left pneumothorax, endotracheal intubation, as well as endopleural tube placement. Three days after his admission he was unstable, hypoperfused, with tendency to bradycardia, dobutamine was started and bilateral pleural seals were placed due to bilateral pneumothorax, obtaining 220 ml in total. Chest X-ray with bilateral pneumothorax and images with consolidation at basal and right medial level. 26 April 2022: Hb 12.2, hto 37.5, platelets: 109 mil, PT: 13.5, PTT: 28.3, lactate. 28 April 2022: 0.8, Hb 9.3, hto 27.7, platelets 135,000, WBC 11,900, lymphocytes 1940, neutrophils 7430. On stage III mechanical ventilation, aspiration with abundant bronchial secretions, thick and yellowish. The patient required evaluation by pediatric pulmonology for bilateral spontaneous pneumothorax to rule out any congenital pulmonary malformation and for specialized management. He was referred to the third level of care. Chest CT was performed on 27 April 2022 where radiologically multiple nodular lesions were reported randomly distributed and some of them had their radial center shined with air and in both basal regions pulmonary consolidation was observed with data of air bronchogram, as well as a discrete posterobasal pleural effusion contiguous with left pneumothorax.

Metabolic-renal

During his stay he had anuria previously, which required furosemide, with partial improvement of uresis, with uresis of 1.8 ml/kg/hour, so a dose of furosemide was administered, with improvement. With a history of hyperglycemia, which required insulin infusion for approximately 4 hours, last capillary glycemia: 45 mg/dl with GKM: 2.8, corrected calcium: 8.76, renal function preserved and electrolytes within parameters.

Gastric

Previously fasting, enteral stimulation was started at 24 ml/kg/day, tolerated adequately so far. With stools in the previous shift without mucus or blood.

Hemato-infective

Initial data of systemic inflammatory response, positive peripheral blood culture for *S. aureus* taken on 23 April 2022 and secretion of dermal lesions with *S. aureus* in both sensitive to vancomycin, in antibiotic regimen with cefotaxime at 200 mg/kg/day and vancomycin 30 mg/kg/day. Bronchial secretions were cultured, positive for *Klebisella Ozoenae*. TORCH was also requested, only with results of Epstein Barr virus already reported as negative, herpes virus negative, the rest pending. Labs 26 April 2022 Hb 12.2 g/dl Hto 37.5% MCV 103.90 MCH 33.8 pg/cell platelets 109 thousand total neutrophils: 52.6% leukocytes 6.8 lymphocytes 24.8% 1.69%, INR 1.23 PT 13.5 TPT 28.3 PCR 213.7. (4/28/22): hb 9.3, hto 27.7, platelets 135 000, leukocytes 11 900, lymphocytes 1940, neutrophils 7430. He required a third level evaluation by immunology to rule out any immunodeficiency.

Neurology

He was kept under sedoanalgesia with fentanyl at 5 mcg/kg/hour, maintaining RASS-4. Reactive to stimuli, no abnormal postures, no presence of seizures, no anticommittants, symmetrical eyes, pinpoint pupils due to sedoanalgesia were observed.

Laboratory and cabinet studies

04/22/2022: BH: Hb 17.2, Ht 50.6, platelets: 100 000, leukocytes 4 100, lymphocytes 1310, neutrophils 2180. QS: glucose 103.0 mg/dl, urea 12.00 mg/dl, creatinine 0.40 mg/dl. ES: phosphorus 3.8 mg/dl, calcium 9.7 mg/dl, chlorine 103.0 mmol/l, potassium 4.80 mmol/l, sodium 138.0 mmol/l, magnesium 1.60 mg/dl. CRP 53.4. EGO: PH 7, density 1.015, proteins 25 mg/dl, glucose normal, ketone negative, bilirubin negative, blood 10 apr eri/ul, leukocytes negative, nitrites negative.

Sediment

Leukocytes 1-2 cells/u, erythrocytes 5-7× field were seen. Cells were negative. Epithelial C. scarce yeast negative, bacteria negative, crystals negative. 04/23/2022: Blood culture positive for *S. aureus*, susceptible to: ciprofloxacin ≤0.5 s daptomycin 0.5 s doxycycline ≤0.5 s gentamycin ≤0.5 s levofloxacin 0.25 s linezolid 2 s moxifloxacin ≤0.25 s rifampicin ≤0.5 s tetracycline ≤1 s tigecycline ≤0.12 s trimethoprim/sulfametho ≤10 s vancomycin 1 s nitrofurantoin ≤16 S. 04/24/2022: BH: Hb 12.7, Ht 40.4, platelets: 156 000, leukocytes 9 100, lymphocytes 2840, neutrophils 4840 PCR 433. BH: Hb 13.8, Ht 41.9, platelets: 166 000, leukocytes: 9 800, lymphocytes 4030, neutrophils 4220. Direct Coombs negative. INR 1.19, TP 13.1, TTP 27.6, Lactate 2.4. glucose 49, urea 16, direct Bilirubin 0.22, indirect Bilirubin 2.53. B. total 2.75. TGP 8, TGO 19, FA 75. phosphorus 4.4, calcium 9.1, chlorine 100, potassium 4.6, sodium 142, magnesium 1.9. DHL 254. Venous blood gas: pH: 7.28, pCO2: 49, pO2: 29, HCO3: 23, CO2 24.5, Sat 46%. 04/25/2022: PCR 400.2, ACS. Anti-epstein B virus not reactive, ACS. Anti-herpes 1 and 2 (HSV-1/2) IgM 0.50. Positive culture of secretion for *S. aureus* 04/26/22: hb 12.2, hto 37.5, platelets: 109 mil, PT: 13.5, PTT: 28.3, blood glucose 139.0 mg/dl urea 16.00 mg/dl serum creatinine 0.20, P 4.4 mg/dl, Ca 8.5 mg/dl, Cl 98.0 mmol/l K, 3.80 mmol/l Na, 137.0 mmol/l, Mg 1.60 mg/dl, DHL 300, neonatal bilirubins: BT 1.1 Total protein 5.40 a/g ratio, 0.7 globulin, 3.2 g/dl albumin, 2.30 g/dl tgp/alt 7.00 u/l tgo/ast 28 u/l alkaline phosphatase 60. 04/26/22: blood glucose 67mg/dl urea 16.00 mg/dl serum

creatinine 0.40, P 5 mg/dl Ca 8.76 mg/dl Cl 104 mmol/l K 4.60 mmol/l Na 137.0 mmol/l Mg 1.50 mg/dl, neonatal bilirubins: BT 1.1, Direct 0 Indirect 1.1. VDRL negative. Arterial blood gas: pH: 7.42, pCO2: 48, pO2: 146, HCO3: 31.1, Sat 99%. Lactate 0.5. Chest CT on 04/27/22 where radiologically multiple nodular lesions were reported randomly distributed and some of them had their radial center lucid with air and in both basal regions pulmonary consolidation was observed with data of air bronchogram, as well as a discrete post-basal pleural effusion contiguous with left pneumothorax.

Current evolution

The current evolution remained stable with better control of blood pressure. No supplemental oxygen, with good respiratory pattern, oxygen saturation greater than 90%. Pneumology attributed the imaging changes to an infectious process. Tolerating feeding on demand, no vomiting or abdominal distension, with good suction, bowel movements present. Euthermic in management with tmp/smz and clindamycin. No neurological alarm data was present.

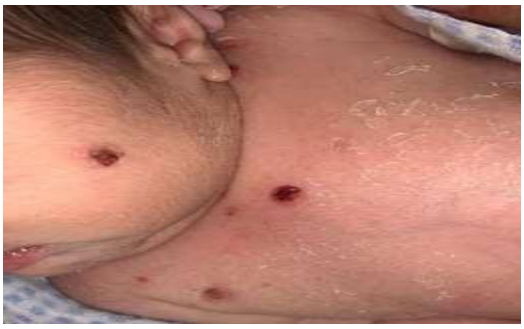

Figure 1: Papulomacular lesions and melisseria crusts on trunk and face.

Figure 2: Pustular lesion on scalp.

Diagnosis

Congenital toxoplasmosis

Within his diagnostic approach with suspected immunodeficiency, toxoplasma IgG >300 IU/ml and IgM 4.28, pcr for toxoplasmosis positive, cranial tomography with multiple and uncountable parenchymal calcifications. Increased titers and immunoglobulins for toxoplasma, as well as hepatomegaly and splenomegaly. Blood biometry with persistent plateletopenia and tac with multiple calcifications, management with trimetropin sulfamethoxazole with pyrimethadine and folinic acid is started and should be maintained for 6 months.

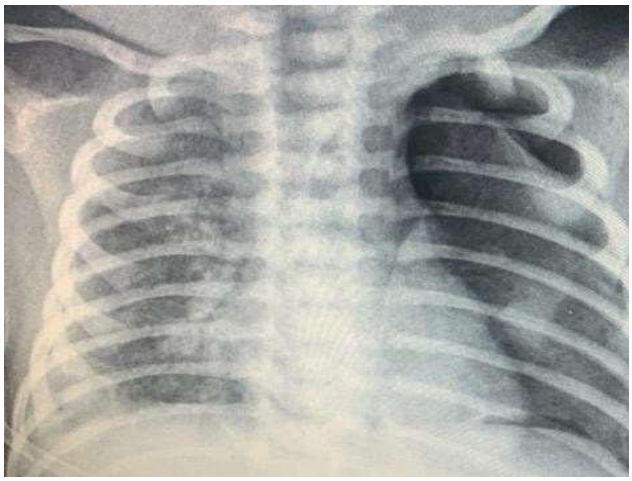

Figure 3: Left apical spontaneous pneumothorax, preintubation.

Probable multifactorial systemic arterial hypertension

Elevation of blood pressure above the 95th percentile in more than 3 continuous measurements, management with hydralazine is initiated with good response. Renal Doppler ultrasound was performed reporting discreetly elevated vascular resistance max. 0.9, nephrology refers probable multifactorial cause, suggests continuing management with hydralazine until reaching the maximum dose and adding or changing to amlodipine.

Rule out immunodeficiency

Given the perinatal history and initial clinical presentation, an alteration in immunity is suspected, immunology referring IgG 1821.28, IgA 62.96, IgM 103, C3 90, C4 10.3, within the approach of primary immunodeficiency only IgG, IgA and IgM elevation was found, which speaks of a polyclonal response secondary to the antigenic stimulus resulting from the infectious process, with normal complements. Therefore, primary immunodeficiency is ruled out, with continuous follow-up by the outpatient immunology department.

High neurological risk

Risk factors, normal transfontanelar ultrasound, tac with brain parenchyma with presence of countless supratentorial parenchymal calcifications. There is adequate differentiation of gray-white matter, which preserves the usual location, thickness and volume. Assessed by neurology without alarm data, but

considered high risk. Otacoustic emissions pass bilaterally. Cardiology evaluation without evidence of myocarditis, endocarditis or congenital heart disease, ophthalmology both eyes with transparent media, retina and optic nerve without alterations.

Cholestasis probable multifactorial etiology

Elevation of direct bilirubin, infectious process with toxoplasmosis may be contributing, management with ursodeoxycholic acid.

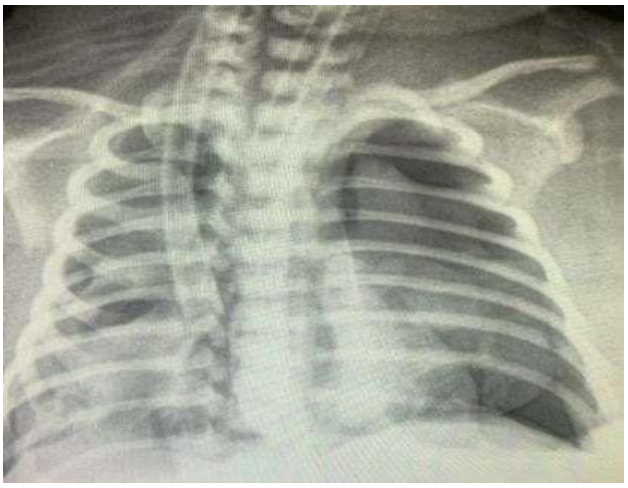

Figure 4: Bilateral postintubation pneumothorax.

Figure 5: Bilateral thoracostomy.

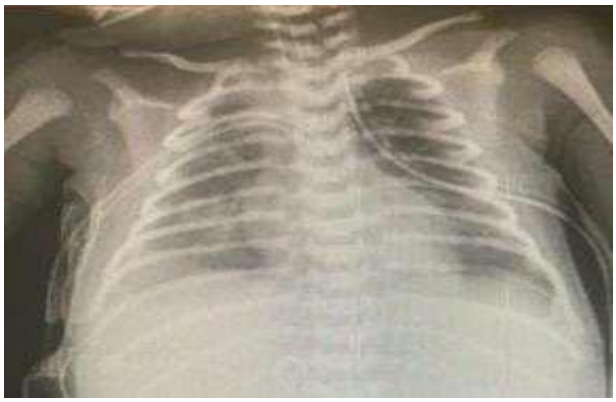

Figure 6: Post thoracostomy chest X-ray.

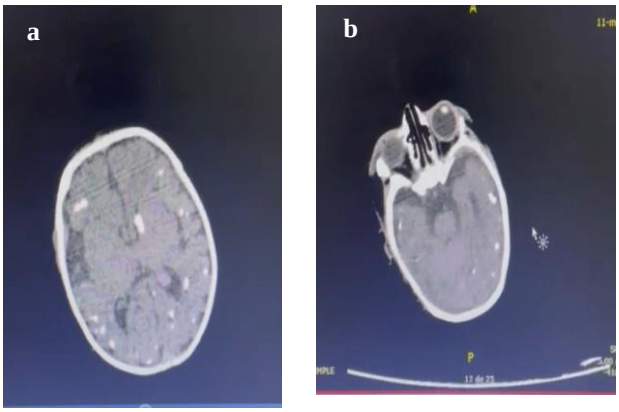

Figure 7 (a and b): Supratentorial parenchymal calcifications.

DISCUSSION

The incidence of spontaneous pneumothorax varies from 1-2%.[20] It can occur spontaneously (idiopathic) or secondary to various underlying lung diseases. Most *Pneumothoraces* occur as a result of birth injury, shoulder dystonia, prematurity, pneumonia, meconium aspiration syndrome or assisted ventilation. Spontaneous pneumothorax in a termneonate is a very rare entity. Very few case reports described bilateral spontaneous pneumothorax in a newborn. A similar case was published by Arora et al in a newborn with spontaneous pneumothorax and moderate respiratory distress.[20] In general, reports in the literature point to the same risk factors and also agree on the rarity of the picture, with pneumonia being a clear risk factor.[1-6]

CONCLUSION

Bilateral *Pneumothorax* due to *S. aureus* is a rare clinical entity, especially in neonates. In the present case it was possible to identify a causative agent by blood culture and culture of the lesions. It is important to identify signs and symptoms early, especially in such a nonspecific period as the neonatal stage, in order to establish appropriate management. Naturally, this must be individualized for each patient.

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