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CLINICAL CASE

Hemodynamic effects of acute tension pneumothorax in a term newborn. A case report

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Abstract

Background: *Pneumothorax (PTX) occurs more frequently in the neonatal period than in any other period of life. Lung ultrasound (LU) is considered the gold standard for diagnosis and treatment.* **Clinical case:** *We describe the case of a term male newborn who developed a tension pneumothorax (tPTX). A quick echocardiographic assessment for post-processing measurements was performed, demonstrating right ventricular failure and elevation of pulmonary vascular resistances as seen in tension physiology, which resolved quickly after drainage.* **Conclusions:** *The right ventricle easily handles varying amounts of preload, but it rapidly decompensates with an acute rise in afterload. LU cannot differentiate PTX from tPTX; however, echocardiographic assessment can.*

Keywords: *Tension pneumothorax. Lung ultrasound. Targeted neonatal echocardiography. Newborn.*

Efectos hemodinámicos del neumotórax a tensión en un neonato de término. Reporte de caso

Resumen

Introducción: *El neumotórax (NTX) se presenta con mayor frecuencia en el periodo neonatal que en cualquier otro periodo de la vida. El ultrasonido pulmonar (UP) se considera el método de referencia para su diagnóstico y tratamiento.* **Caso clínico:** *Describimos el caso de un recién nacido a término que desarrolló un neumotórax a tensión (NTXt). Se realizó una evaluación ecocardiográfica rápida para realizar mediciones de posprocesamiento que demostró insuficiencia ventricular derecha y elevación de las resistencias vasculares pulmonares, observada en la fisiología de tensión, que se resolvió rápidamente tras el drenaje.* **Conclusiones:** *El ventrículo derecho maneja con facilidad niveles variables de precarga, pero se descompensa rápidamente con un aumento agudo de la poscarga. El UP no puede diferenciar el NTX del NTXt, pero la evaluación ecocardiográfica sí.*

Palabras clave: *Neumotórax a tensión. Ultrasonido pulmonar. Ecocardiografía funcional neonatal. Neonato.*

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Introduction

Pneumothorax (PTX) occurs more frequently in the neonatal period than in any other period of life and is associated with increased mortality and morbidity. Symptomatic PTX occurs in about 0.05-0.1% of all live births, and in very low birthweight infants, this rate can achieve 3.8-9%[1-3]. Among preterm neonates born at < 32 weeks of gestation, PTX is significantly associated with increased hospital length of stay, mortality, bronchopulmonary dysplasia, and intraventricular hemorrhage[3,4]. Birth trauma, neonatal resuscitation, meconium aspiration, underlying lung diseases, and positive pressure ventilation are the main causes of PTX in newborns[3,5]. Prompt recognition that a PTX is causing or exacerbating respiratory distress is critical to improve outcomes and decrease morbidity and mortality related to this condition[6].

Clinical case

Term male neonate, born at 38 weeks of gestation to a 35-year-old mother (Gravida 4, Para 3) with a history of recurrent urinary tract infections, treated empirically without documented cultures. Delivery was through cesarean section. At birth, the neonate was non-vigorous and required aggressive resuscitation, including positive pressure ventilation, chest compressions, and one dose of epinephrine. Apgar scores were 3, 5, and 8 at 1, 5, and 10 minutes, respectively. Birth weight was 3050 g. The newborn developed tachypnea, grunting, and jitteriness, progressing to the need for continuous positive airway pressure (CPAP) at 6 cmH_2O with an FiO_2 of 30%. He was subsequently transferred to a tertiary care center.

At admission, the newborn was febrile and tachycardic. He was intubated after CPAP failure and started on mechanical ventilation. Chest radiography (CXR) revealed patchy bilateral infiltrates, raising suspicion of early-onset sepsis. Empirical antibiotic therapy with ampicillin and amikacin was initiated. Lung ultrasound (LU) performed by the on-call fellow showed pleural sliding, a dense B-line pattern, and a collapsed bronchogram. Cardiac point-of-care ultrasound (POCUS) demonstrated adequate contractility and a left-to-right shunt through a patent foramen ovale (PFO).

Hemodynamic consultation: On day 2 of admission, sudden desaturation (70%) with tachycardia and hypotension occurred, so the hemodynamics team was paged. Vital signs: Heart rate 180, blood pressure (right arm) 50/32 mmHg, respiratory rate 68, and saturation 70%. Ultrasound assessment: Given the sudden respiratory deterioration, a modified SAFE protocol (Sonographic Algorithm for Life-Threatening Emergencies) was performed. Ultrasound revealed the absence of pleural sliding with an A-line pattern, no B-lines, and the presence of the stratosphere sign on M-mode. No lung point was identified. Considering the previous findings of a dense B-line pattern, a diagnosis of tension PTX (tPTX) was made.

While preparations for chest drainage were underway, a rapid hemodynamic assessment for post-processing measurements was conducted. Findings included pulmonary hypertension with evolving right ventricular (RV) dysfunction and low cardiac output. In the left ventricle (LV), there was low pulmonary venous return, a hyperdynamic pattern, and reduced cardiac output. Hemodynamic instability resolved following drainage of the tPTX. Table 1 summarizes the hemodynamic parameters before and after chest drainage. Figure 1 depicts imaging findings.

Vitals after drainage: Heart rate 136, blood pressure 72/45 mmHg, respiratory rate 50, and saturation 93%. The chest tube was maintained for 6 days, and the patient was extubated on day 10. Antibiotics were administered for 7 days with a negative blood culture. The newborn was discharged at day 16 and enrolled in a high-risk follow-up clinic.

Discussion

In newborns, the pores of Kohn, Martin's, and Lambert's channels and the intersegmental bronchioles, which are effective collateral channels in normal lungs, are not well developed. This makes it difficult for the high-pressure air in these groups of alveoli to pass easily into adjacent, unexpanded alveoli, causing overdistended alveoli to rupture, and air can escape into the perivascular sheaths and cause a pneumomediastinum or PTX[6].

Conventionally, tPTX has been defined as the presence of intrapleural pressure exceeding atmospheric pressure throughout the entire respiratory cycle[7]. However, confirming this requires pleural manometry, a technique that has not been systematically studied in humans. Consequently, many clinicians rely on a clinical definition, which includes the presence of mediastinal shift on imaging accompanied by "hemodynamic instability."[6,7] Nevertheless, there is no standardized definition of hemodynamic instability, and radiographic signs such as mediastinal shift and diaphragmatic flattening may also appear in non-tPTX[7].

PTX begins with the rupture of an over-distended alveoli. The air escapes along the perivascular connective

Table 1. Hemodynamic variables before and after tension pneumothorax drainage

Parameter	Before	After (1h)
Vital signs		
Heart rate (bpm)	180	136
Blood pressure (mmHg)	50/32	72/45
Respiratory rate (rpm)	70	50
Saturation (%)	70	93
RV		
TAPSE (mm)	7	9
FAC (%)	23	39
S' (cm/s)	4.2	8.2
RVO (mL/kg/min)	124	274
RV longitudinal strain (%)	14	18.3
VRi (PAAT/RVET)	0.17	0.34
IVS	Paradoxical	Round
LV		
S/D (cm/s)	21/22	41/38
E/A (cm/s)	40/68	48/64
E/A ratio	0.59	0.75
LVO (mL/kg/min)	142	180
Simpson's biplane ejection fraction	79%	60%
Shunts		
PFO	Right to left	Left to right
Cerebral hemodynamics		
MCA RI	0.64	0.71
MCA PI	1.04	1.44

bpm: beats per minute, rpm: respirations per minute; FAC: fractional area change; IVS: interventricular septum; PAAT: pulmonary artery acceleration time; MCA: medium cerebral artery; PI: pulsatility index; PFO: patent foramen ovale; RI: resistance index; RV: right ventricle; LV: left ventricle; RVET: right ventricular ejection time; RVO: right ventricular output; TAPSE: tricuspid annular plane systolic excursion; VRi: vascular resistance index.

tissue sheath into the pleural space[8]. The classic description of tPTX involves mechanical shifting of the mediastinum with equalization of cardiac filling pressures and cardiogenic shock, as it was evolving in our case[9,10].

In experimental studies, tPTX has been defined to cause a decrease of > 50% of ventricular output (VO) or cardiac index from baseline value[9], as the point at which ipsilateral pleural pressures became positive throughout the respiratory cycle[11], "cardiovascular collapse," or injection of > 120% of total lung capacity (induced PTX)[12]. In another animal model, tPTX was defined as a positive intrapleural pressure > 1 mmHg and a significant deviation of hemodynamic parameters, including a decline in VO > 20%[13]. In our patient, right ventricular output found at the time of tPTX was 55% lower than what was documented after drainage.

With regard to tension physiology, early models of tPTX supported a theory of progressive mechanical compression that resulted in "kinking" of mediastinal structures and right heart compression, culminating in cardiogenic shock[9,14]. Later studies, however, have provided evidence to suggest that central hypoxia actually represents the primary physiologic insult, with preservation of VO by compensatory mechanisms, including tachycardia and increased negative intrathoracic pressure on the contralateral hemithorax until late in the physiologic process when sudden cardiovascular collapse occurs [9,10,14,15]. It appears that both models of tension physiology (mechanical compression and central hypoxia) are accurate and that tPTX represents a collection of related but significantly varying pathophysiologic processes[9]. Therefore, alveolar collapse with normal pulmonary perfusion establishes that the main mechanism of hypoxemia is shunt (in our patients, shown as a right-to-left PFO). In case of hypoxic vasoconstriction, the dead space mechanism could also be associated[8,9,16]. Our patient showed tachycardia, hyperdynamic LV pattern, and cerebral compensatory vasodilation that improved after drainage.

Hypoxic pulmonary vasoconstriction leads to a redirection of the blood to the alveoli with higher oxygen tension, increasing pulmonary vascular resistance (PVR) by 50-300%, and pulmonary blood flow in a lung affected by a PTX can decrease to 25-35% of the total blood flow[16]. The redirection of blood to the unaffected lung by increasing PVR in the affected lung may prevent a decrease in blood oxygenation[17]. Our patient showed increased PVR with an index (inversely proportional to PVR) of 0.17 at the time of tPTX that immediately improved to 0.34 (almost normal) after drainage.

Although the RV easily handles varying amounts of preload, it rapidly decompensates with an acute rise in afterload[5]. Coronary perfusion to the RV occurs in systole and diastole, compared with primarily diastolic flow in the LV, making the RV dependent on systolic blood pressure[18].

At the first assessment, our patient presented evolving RV failure and elevation of PVR as seen in tension physiology[14,16,18]. Hypotension was also present, which causes further deterioration of acute RV failure due to reduced transseptal gradient and coronary perfusion (the double hit phenomenon); the RV diameter can be significantly reduced preceding arrest[17,18].

Rapid diagnosis of tPTX in the NICU is essential to prompt life-saving management in the newborn[19,20]. The SAFE protocol for the suddenly decompensating infant is a tool for rapid screening for the most common life-threatening complications needing immediate attention[20,21]. The average time to perform diagnostic tests in these studies was 5.3 ± 5.6 min for LUS versus 19 ± 11.7 min for CXR[5].

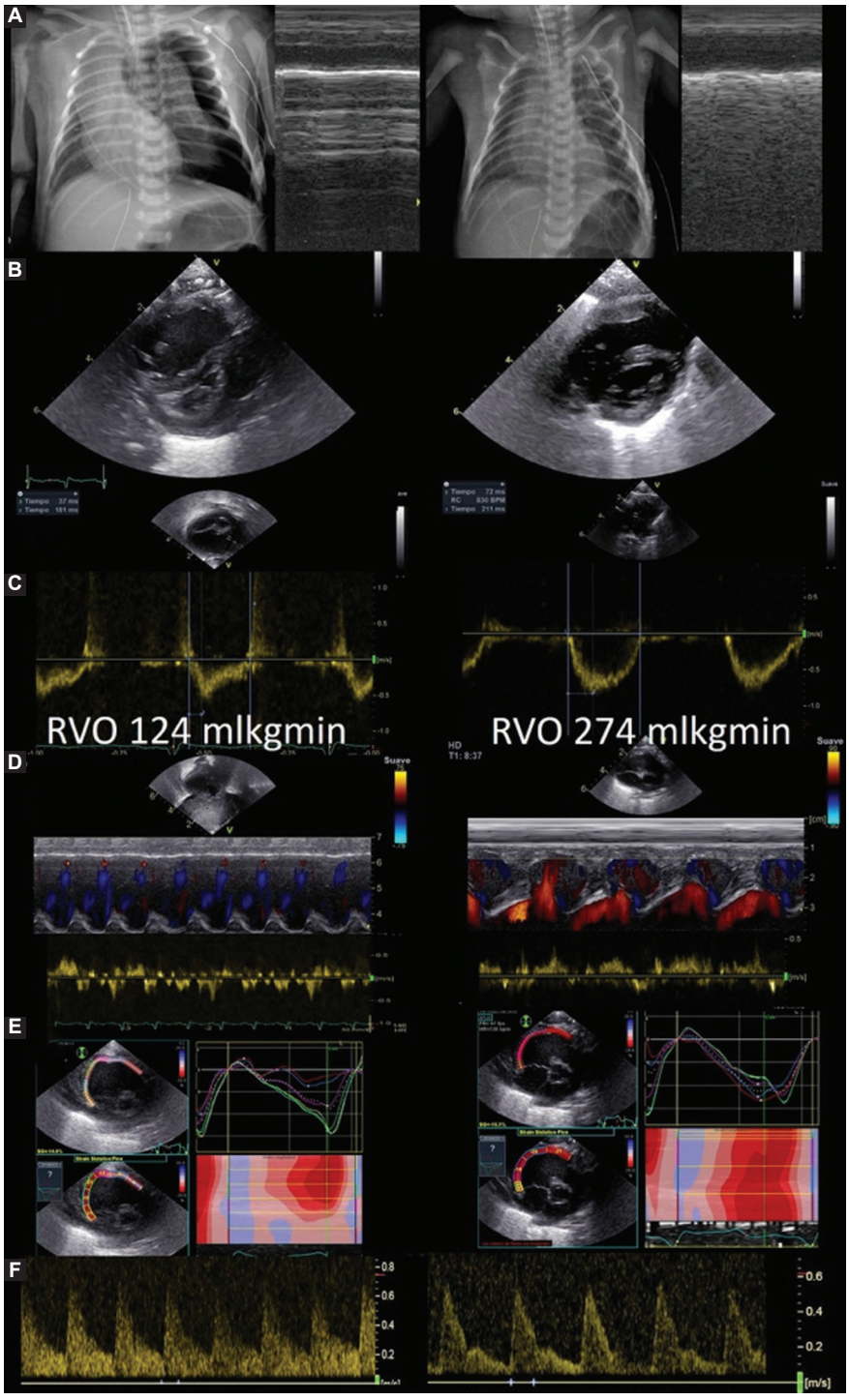

Figure 1. Hemodynamics before and after tension pneumothorax drainage. **A:** CXR with corresponding LU M-mode comparing tPTX and normal pleural sliding (Stratosphere sign vs Seashore sign). **B:** paradoxical vs round shaped interventricular septum. **C:** pulsed doppler of the pulmonary artery showing short PAAT and PVRi (inversely proportional to PVR) that improved after drainage. Low RVO that augmented 55% after drainage. **D:** color and pulsed Doppler of the PFO showing a right to left shunt at the time of tPTX that turned left to right after one hour. **E:** RV longitudinal strain improving from –14% (impaired) to –18.3% (almost normal). **F:** MCA showing compensatory vasodilation and then normal pattern. CXR: chest radiography; RV: right ventricular; LU: lung ultrasound; tPTX: tension pneumothorax; PAAT: pulmonary artery acceleration time; PVR: pulmonary vascular resistance; RVO: right ventricular output; PFO: patent foramen ovale.

In Mexico, the algorithm by Yousef et al.[21] was adapted by our group[22] including consolidated core steps to support a high-risk newborn, aiding clinicians managing cardiac arrest[23], and adding views to verify correct intubation. It also integrated the approach suggested by Kharrat and Jain, where patients are categorized as cardiac arrest, hemodynamic decompensation, and respiratory decompensation[24].

Conclusions

Bedside ultrasound evaluation shows that basic training is sufficient to allow operators, regardless of prior ultrasound experience, to quickly screen for cardiac tamponade, PTX, and pleural effusion. In our experience, basically trained fellows have been able to recognize and treat tPTX and tamponade[22]. Importantly, LU cannot differentiate PTX from tPTX[16]. Cardiac ultrasound (targeted neonatal echocardiography), however, is capable of discerning between the two (small hyperkinetic cardiac chambers or hypokinetic right ventricle, dilated IVC, mediastinal shift, low VO, increased PVR)[14,15]. In our patient, as soon as PTX was identified, a quick cardiac ultrasound was performed for post-processing measurements, and tension physiology was described.

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Conflicts of interest

The authors declare no conflicts of interest.

Ethical considerations

Protection of humans and animals. The authors declare that no experiments involving humans or animals were conducted for this research.

Confidentiality, informed consent, and ethical approval. The authors have obtained approval from the Ethics Committee for the analysis of routinely obtained and anonymized clinical data, so informed consent was not necessary. Relevant guidelines were followed.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing of this manuscript.

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