NEONATAL HEMODYNAMICS Research Centre

Case Report

Hemodynamic effects of acute tension pneumothorax.

Carolina Michel-Macías[1], Eunice Valeria Serpa Maldonado[2], José Guadalupe Mantilla Uresti[3] Daniel Ibarra-Ríos[2]

1.Neonatology Department, Instituto Nacional de Perinatología. 2. Neonatology Department, Hospital Infantil de México Federico Gómez. 3. Neonatology Department, Hospital Central "Dr. Ignacio Morones Prieto".

Key physiological insight/learning points:

The RV easily handles varying amounts of preload, but it rapidly decompensates with an acute rise in afterload.

At first assessment, our patient presents with evolving right ventricular failure and elevation of PVR as seen in tension physiology, that resolved quickly after tension pneumothorax drainage.

Ultrasound driven protocols for crashing infant in the NICU allows timely and efficient response and invasive procedures can be guided.

Background: Pneumothorax occurs more frequently in the neonatal period than in any other period of life and is associated with increased mortality and morbidity. Symptomatic pneumothorax occurs in about 0.05% to 0.1% of all live births, and in very low birthweight infants this rate can achieve 3.8% to 9%[1, 2, 3]. Among preterm neonates born at less than 32 weeks GA, pneumothorax is significantly associated with mortality, BPD and IVH[3,4]. Birth trauma, neonatal resuscitation, meconium aspiration, underlying lung diseases or positive pressure ventilation are the main causes of pneumothorax in newborns[3,5].

Birth History: Term male neonate, born at 38 weeks of gestation to a 35-year-old mother (Gravida 4, para 3) mother with history of repetitive urinary tract infections, treatment not specified without cultures. Born by caesarean section was found non vigorous at birth requiring aggressive resuscitation with positive pressure ventilation, chest compressions and one dose of adrenaline. Apgar score was reported as 3, 5, 8 at 1, 5 and 10 minutes respectively. Birth weight was 3050 g. Tachypnea, grunting and jittery progressed requiring CPAP of 6 cmH_2O with an FiO_2 of 30% and was transferred to third level care.

Medical History: At admission the newborn was found febrile and tachycardic. On chest radiography patchy bilateral infiltrates were noted so evidence of early onset sepsis was considered for which antibiotic coverage with ampicillin and amikacin was given. The fellow on call found lung ultrasound at admission with pleural sliding, dense B-Line pattern, and collapse bronchogram. Cardiac POCUS with adequate contractility and a left to right patent foramen ovale.

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, Saturation 70%.

Ultrasound assessment: With evidence of sudden respiratory deterioration, a modified SAFE protocol (Sonographic Algorithm for liFe threatening Emergencies) was preformed encountering absence of pleural sliding with A-line pattern, no B lines and under M-mode the stratosphere sign was presented. No lung point was present and with evidence of previous existence of dense B line pattern tension pneumothorax (tPTX) was diagnosed. While

material for chest drainage was prepared a quick assessment of hemodynamics was undertaken finding pulmonary hypertension with right ventricle (RV) evolving dysfunction and low cardiac output. On the left ventricle (LV) low pulmonary venous return, an hyperdynamic pattern with low cardiac output was found. Hemodynamic effects resolved after tPTX was drained. **Table 1** shows hemodynamic variables before and after chest drainage. See **Figure 1** and **Video 1**.

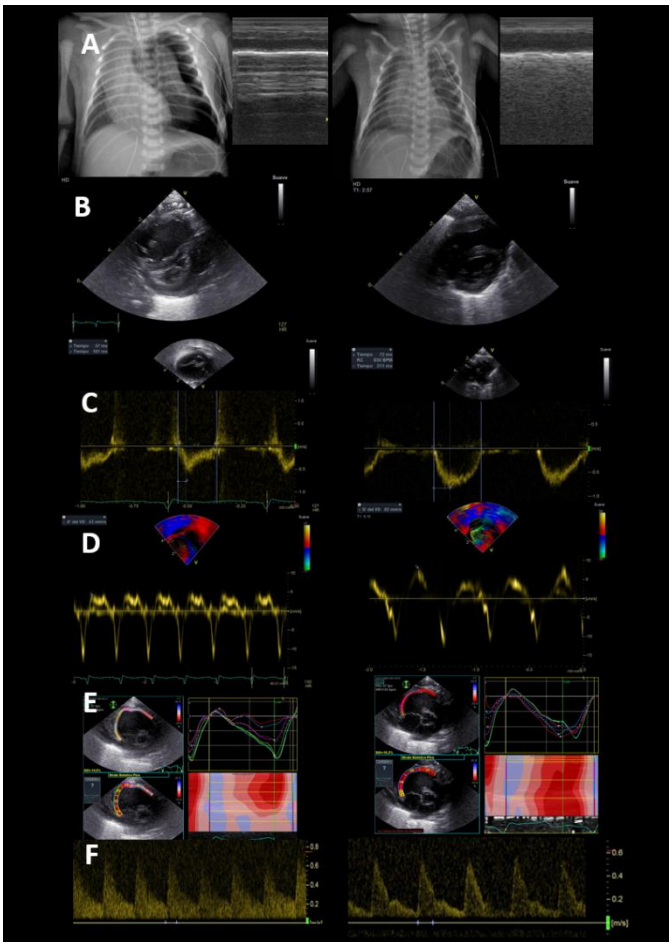
Vitals after drainage: heart rate 136, blood pressure 72/45 mmHg, respiratory rate 50, saturation 93%.

Table 1. Hemodynamic variables before and after tension pneumothorax drainage.

Parameter	Before	After(1h)
RV		
TAPSE mm	7	9
FAC %	23	39
S' cm/s	4.2	8.2
RVO	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	142	180
Simpson's biplane	79%	60%
PFO	Right to left	Left to right
MCA RI	0.64	0.71
MCA PI	1.04	1.44

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

Figure 1. Hemodynamics before and after tension pneumothorax drainage.



A. Radiography with corresponding 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 low output with short PAAT that improves after drainage. D. RV free wall tissue doppler showing improvement of the systolic (S) wave. E. RV longitudinal strain showing improvement. F. MCA showing compensatory vasodilation and then normal pattern.

Follow up: Chest tube was maintained for 6 days, and the patient was extubated at day 10. Antibiotics were administered for 7 days with negative blood culture. Newborn was discharged at day 16 enrolled in high risk follow up clinic.

Discussion

Pneumothorax begins with the rupture of an over-distended alveoli. The air escapes along the perivascular connective tissue sheath into the pleural space[6]. 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[7,8].

In experimental studies, tPTX has been defined to cause a decrease of > 50% of ventricular output (VO) or cardiac index from baseline value[7], as the point at which ipsilateral pleural pressures became positive throughout the respiratory cycle[9], "cardiovascular collapse" or injection of > 120% of total lung capacity to induce pneumothorax[10]. 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 $\geq$ 20%[11].

With regards 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[12,7]. 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 physiologic process when sudden cardiovascular collapse occurs[12,7,8,13]. Our patient showed tachycardia, hyperdynamic LV pattern and cerebral compensatory vasodilation that improved after drainage.

It appears that both of these models of tension physiology are actually correct and that tPTX represents a collection of related but significantly varying pathophysiologic processes[7]. Therefore, alveolar collapse with normal pulmonary perfusion establishes that the main mechanism of hypoxemia is shunt. In case of hypoxic vasoconstriction, dead space mechanism could be also associated[6,14,7].

Hypoxic pulmonary vasoconstriction leads to a redirection of the blood to the alveoli with higher oxygen tension, increasing pulmonary vascular resistance by 50-300%, and pulmonary blood flow in a lung affected by a pneumothorax can decrease to 25-35% of the total blood flow[14]. The redirection of blood to the unaffected lung by increasing pulmonary vascular resistance in the affected lung may prevent a decrease in blood oxygenation[14].

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[15].

At first assessment, our patient presents with evolving RV failure and elevation of PVR as seen in tension physiology[12,14,15]. Hypotension is 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 [15,16].

Rapid diagnosis of tPTX in the NICU is essential to prompt life-saving management in the newborn[17,18]. The SAFE protocol for the suddenly decompensating infant is a tool for rapid screening for the most common life-threatening complications needing immediate attention[2,18]. The average time to perform diagnostic tests in these studies was 5.3±5.6 min for LUS versus 19±11.7 min for a chest X-ray[5].

In our institution we use a modified SAFE protocol that integrates the approach suggested by Kharrat et al, and patients are categorized as cardiac arrest, hemodynamic decompensation, respiratory decompensation [18,19].

Bedside ultrasound evaluation shows that basic training is sufficient to allow operators, regardless of prior ultrasound experience, to quickly screen for cardiac tamponade, pneumothorax, and pleural effusion, in our experience basically trained fellows have been able to recognize and treat tension pneumothorax and tamponade[20].

Importantly, lung ultrasound cannot differentiate PTX from tPTX[16]. Cardiac ultrasound, however, is capable of discerning between the two (small hyperkinetic cardiac chambers or hypokinetic right ventricle, dilated IVC, mediastinal shift)[12,13].

In our patient, as soon as PTX was identified, a cardiac ultrasound was performed, and tension physiology was described.

REFERENCES:

1. Santos-Silva I, Flor-de-Lima F, Rocha G, Alves I, Guimaraes H. Pneumothorax in neonates: a level III Neonatal Intensive Care Unit experience. J Pediatr Neonat Individual Med. 2016;5(2):e050220.doi:10.7363/050220.
2. Sharma D, Farahbakhsh N. Role of chest ultrasound in neonatal lung disease: a review of current evidences. The Journal of Maternal -Fetal & Neonatal Medicine 2019;32(2):doi:10.1080/14767058.2017.137 63
3. Duong HH, Mirea L, Shah PS, Yang J, Lee SK, Sankaran K. Pneumothorax in neonates: Trends, predictors and outcomes. J Neonatal Perinatal Med 2014; 7,1:29-38. Doi: 10.3233/NPM-1473813
4. Migliaro F, Sodano A, Capasso L, Raimondi F. Lung ultrasound-guided emergency pneumothorax needle aspiration in a very preterm infant. BMJ Case Rep 2014; doi: 10.1136/bcr-2014-206803
5. Liu J, Kurepa D, Feletti F, Alonso-Ojembarrena A, Lovrenski J, Copetti R et al. International Expert Consensus and Recommendations for Neonatal Pneumothorax Ultrasound Diagnosis and Ultrasound-guided Thoracocentesis procedure. J. Vis. Exp. 2020. Doi. 10.3791/603791/60836.
6. Connolly JP, Hemodynamic measurements during a tension pneumothorax. Critical Care Medicine 1993; 21;2.
7. Nelson D, Porta C, Satterly S, Blair K, Johnson E, Inaba K et al., Physiology and cardiovascular effect of severe tension pneumothorax in a porcine model. Journal of Surgical Research 2013; March, doi:10.1016/j.jss.2013.05.057.
8. Pavéz P. N. Interacción cardiopulmonar en el neumotórax durante la ventilación mecánica. Caso clínico. Rev Chil Med Intensiv 2021; 36(1):38-45.
9. Rutherford RB, Hurt HH Jr, Brickman RD, Tubb JM. The pathophysiology of progressive tension pneumothorax. J Trauma 1968;8:212.
10. Barton ED, Rhee P, Hutton KC Rosen P. The pathophysiology of tension pneumothorax in ventilated swine. J Emerg Med 1997;15:147.
11. Hurewitz AN, Sidhu U, Bergofsky EH. Cardiovascular and respiratory consequences of tension pneumothorax. Bull Eur Physiopathol Respir 1986;22: 545.
12. Lanks Cw, Correa V. Advantages of Cardiopulmonary Ultrasound in Post-Cardiopulmonary Resuscitation Tension Pneumothorax. J Ultrasound Med 2017; 00:00. Doi: 10.1002/jum.14437.
13. Inocencio M, ChildsJ, Chilstrom M, Berona K. Ultrasound findings in tension pneumothorax: a case report. The Journal of Emergency Medicine, 2017;52,e217-e220.
14. Christophe JJ, Ishikawa T, Imai Y, Takase K, Thiriet M, Yamaguchi T. Hemodynamics in the

pulmonary artery of a patient with pneumothorax. Medical Engineering & Physics 2012;725-732. Doi: 10.1016/j.medengphy.2011.09.016.

15. Wanner PM, Filipovic M. The Right Ventricle-You May Forget It, But It Will Not Forget You. J Clin Med 2020;9, 432, doi. 10.3390/jcm9020432.
16. Caap P, Aagaard R, Sloth E, Lofgren B, Granfeldt A. Reduced right ventricular diameter during cardiac arrest caused by tension pneumothorax- a porcine ultrasound study. Acta Anaesthesiologica Scandinavica 2017;6,813–823doi:2443/10.1111/aas.12911
17. Corsini I, Parri N, Ficial B, Dani C. Lung Ultrasound in the neonatal intensive care unit: Review of the literature and future perspectives. Pediatric Pulmonology 2020; 55:1550-1562. Doi: 10.1001/ppul.24792.
18. Yousef N, Singh Y, De Luca D. "Playing it SAFE in the NICU" SAFE: R: a targeted diagnostic ultrasound protocol for the suddenly decompensating infant in the NICU. Eur J Ped 2021; Jul, doi: 10.1007/s00431-021-04186-w.
19. Kharrat A, Jain A. Guidelines for the management of acute unexpected cardiorespiratory deterioration in neonates with central venous lines in situ. Acta Paediatr 2018; 107(11):2024-2025.
20. Ibarra-Ríos D, Ávila-Ceniceros M, Martínez-Contreras CB, Márquez-González H. Letter to the editor on the original article: the evolution of cardiac point of care ultrasound for the neonatologist. Eur J Pediatr. 2021 Dec;180(12):3577. doi: 10.1007/s00431-021-04183-z.