

Transient ECG Changes Associated with Right-Sided Pneumothorax – A Case Report

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Received April 03, 2021; Revised May 11, 2021; Accepted May 20, 2021

Abstract Relatively few cases have been reported of electrocardiographic findings associated with pneumothoraces. This case report seeks to add to the literature regarding the transient ECG findings associated with a right-sided tension pneumothorax. Based on our extensive review, these findings have not been previously documented. Our patient was found to have a right-sided tension pneumothorax after intubation for acute hypoxic respiratory failure secondary to COVID-19 pneumonia. A 12-lead ECG revealed an accelerated junctional rhythm, left axis-deviation, and ST depressions in the anterolateral leads. All of these abnormalities resolved on repeat ECG two hours later after insertion of a chest tube. Based on these findings, we propose a possible mechanism explaining these ECG findings. Although not all pneumothoraces present with ECG findings, this case demonstrates that physicians must be aware of these changes, particularly because they may present as a mimicker of myocardial infarction.

Keywords: ECG findings, pneumothorax, case report

Cite This Article: Ryan Lee, Whitney Liehr, and Candice Lovelace, "Transient ECG Changes Associated with Right-Sided Pneumothorax – A Case Report." *American Journal of Medical Case Reports*, vol. 9, no. 9 (2021): 456-459. doi: 10.12691/ajmcr-9-9-5.

1. Introduction

Few studies have looked at the electrocardiographic changes in patients with pneumothorax. The first studies to look at ECG tracings in pneumothorax were written in the early 1900s when pneumothoraces were artificially induced in the treatment of pulmonary tuberculosis before its replacement with chemotherapeutics in the mid-1900s [1]. Even ECG textbooks such as Dubin's Rapid Interpretation of EKG's only make passing references to ECG changes in pneumothorax. The most common changes that have been described include right axis deviation, reduced QRS amplitude in precordial leads, and T wave inversion. Most recently, there have also been some case reports of ECG changes that mimic other life-threatening conditions including ST elevations and depressions [2,3,6,7] and even a S1Q3T3 electrocardiographic pattern [4]. In this case report, we describe a patient with right-sided pneumothorax with ECG abnormalities that mimic the findings of an acute myocardial infarction. Notably, these ECG abnormalities resolved shortly after resolution of the pneumothorax. This case seeks to add to the literature by informing readers of the transient ECG findings that can arise in a right-sided pneumothorax. This case shows that the ECG tracings can sometimes mimic other pathology, and under the right conditions, the symptoms and ECG findings of a pneumothorax may be misdiagnosed with other emergent pathologies such as acute myocardial infarction and lead to delay in treatment of the underlying problem.

2. Case Narrative

The patient is a 76-year-old man who presented to our medical center for fatigue and shortness of breath. He was found to have COVID-19 pneumonia with associated hypoxia and admitted. His comorbidities included coronary artery disease, diabetes mellitus type 2, and hyperlipidemia. Baseline ECG obtained on the day of admission showed a normal sinus rhythm with no abnormalities. The patient was treated conservatively, but the patient's oxygen requirements steadily increased each day. Sixteen days into hospitalization, a rapid response was called on the patient for worsening hypoxic respiratory failure. Despite receiving 60 liters per minute of oxygen via high flow nasal cannula, the patient had oxygen saturations in the low 70% and was acutely agitated. The patient was emergently intubated and transferred to the ICU.

Upon arrival in the ICU, the patient's vital signs were abnormal. Temperature was 36.5C, heart rate 81, blood pressure 79/57 mmHg, and SpO2 90% on volume control ventilator assistance with an FiO2 of 80%. Physical exam revealed a sedated, ventilated man with bilateral crackles and expiratory wheezes. Laboratory studies collected in the ICU showed an arterial blood gas of pH 7.36, pCO2 34, pO2 53, HCO3- 19 on an FiO2 of 80%, white blood

cell count of 19.46 K/ μ L, and hemoglobin level of 11.8 K/ μ L. Electrolyte panel showed no abnormalities except for an elevated BUN of 42 mg/dL and creatinine of 1.41 mg/dL.

A central venous catheter was placed in the patient's right internal jugular vein, and the patient was started on norepinephrine and vasopressin for hemodynamic support. After placement, a portable supine chest x-ray was obtained. Imaging showed a new right tension pneumothorax. This is shown in Figure 1. An ECG was also collected at that time due to a new abnormal cardiac tracing seen on the cardiac monitor. The 12-lead ECG strip can be seen in Figure 2. It revealed an accelerated junctional rhythm, wide QRS complexes with a dominant S wave in V1 and broad monophasic R waves in the lateral leads suggestive of a new left bundle branch broad, left axis deviation, and ST depressions in the anterolateral leads. A chest tube was promptly inserted into the right thorax. After chest tube insertion, another ECG was obtained. This one occurred two hours after the initial ECG. The 12-lead ECG strip obtained after chest tube insertion can be seen in Figure 3. It revealed a normal

sinus rhythm with one irregular premature ventricular complex.



Figure 1. Portable supine chest x-ray of the patient showing a right-sided tension pneumothorax

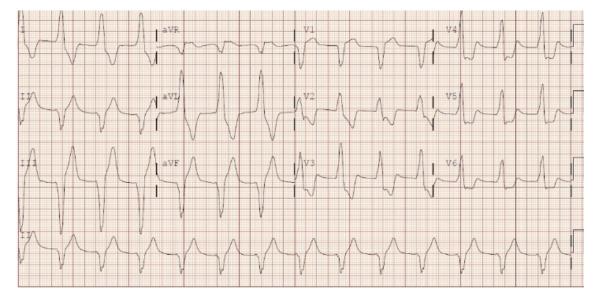


Figure 2. ECG tracing of the patient at the time of the right-sided pneumothorax

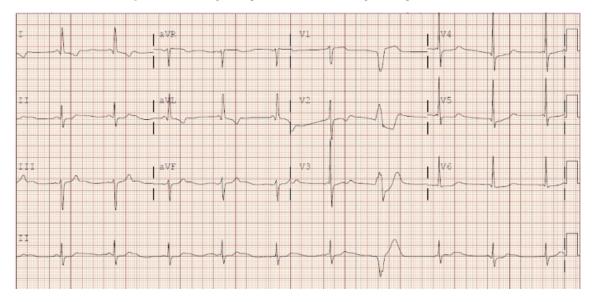


Figure 3. ECG tracing after chest tube insertion

Three days after these events, the patient had a transthoracic echocardiogram which showed no wall motion abnormalities. Follow-up portable chest x-ray after chest tube placement can be seen in Figure 4.

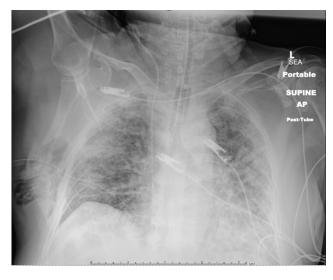


Figure 4. Portable supine chest x-ray of the patient showing insertion of the chest tube in the right hemithorax and resolution of the pneumothorax

3. Discussion

In the literature, most patients with a right pneumothorax had some sort of abnormality noted on ECG, but no specific pattern has ever been reliably replicated. Furthermore, ECG findings associated with right-sided pneumothorax have been reported less and have also been noted to be less pronounced when compared to a left-sided pneumothorax [5]. Krenke et al looked at forty patients with pneumothoraces, which was the largest cohort we could find in the literature [1]. In their study, there were 18 cases of right-sided pneumothorax. They found twelve cases with right-sided axis change, 5 cases with left axis change, and 1 case with no axis change. Other abnormalities noted include a case with negative T waves in III and aVF and two cases with incomplete right bundle branch block. Finally, they also noted three cases with significant decreases with the precordial QRS amplitude. One pediatric case was described by Paige and Spalding where ECG changes were the first indicator of a right-sided pneumothorax in an anesthetized child [9]. In their case, there was an increase in the amplitude of the QRS complex in leads II and V5 and reversal of the polarity of the QRS complex in lead V5 from positive to negative.

Few cases have been published describing ECG changes that mimic other pathologies in patients with right-sided pneumothorax. Alzghoul et al described a case of a 63 year-old with iatrogenic right-sided pneumothorax who had ST-segment elevation in the anterior leads with reciprocal T-wave changes in the inferior leads on 12-lead ECG; similar to our case, there was resolution of the ECG changes after chest tube placement [6]. Chada et al presented a case of a 64 year-old man with right-sided pneumothorax with transient ST segment elevations in V1-V3 and a right bundle branch block [7]. Similarly,

Maheshwari and Mittal showed ST segment elevation and T wave inversions in leads V1-V3 in a right-sided pneumothorax [8]. Additionally, Janssens et al reported a right-sided tension pneumothorax leading to pronounced ST segment elevation in leads II, III, aVF, and V4-V6 [10]. Interestingly, Goddard and Scofield presented a case of an 18 year-old man with a spontaneous right pneumothorax with ECG findings of an S wave in lead I, a Q wave in lead III, and an inverted T wave in lead III -- findings often associated with pulmonary embolism [4]. However, these ECG abnormalities were resolved with treatment of the pneumothorax.

There are many hypothesized reasons for the ECG changes seen in pneumothorax. Yamamoto et al suggested that the changes are primarily due to changes in the heart's anatomic position within the thoracic cavity, including the shifting of the mediastinum and rotation of the heart [11]. Furthermore, the air in the pneumothorax is electrically insulating and will also cause ECG changes. In a right pneumothorax, the heart will be shifted to the left side so more of the right heart is under the V1-V3 leads. Maheshwari et al hypothesized that a right-sided pneumothorax causes right ventricular overload due to a transient increase in pulmonary arterial hypertension [8]. This may explain the ST elevations seen in other cases reported previously.

Unlike other cases, the ECG changes in our patient included conduction abnormalities as well as ST segment depressions in leads V2-V6. Our patient's ECG did not have any discernible P waves and there was a left bundle branch block. These conduction abnormalities may have been induced by ventricular irritability secondary to ischemia. The cause of the ischemia is likely multifactorial, including transient pulmonary arterial hypertension from the pneumothorax as well as relative hypoxia from the patient's underlying hypoxic respiratory failure from COVID-19 pneumonia. The ST depressions seen on ECG may be reflective of ischemia in the right side of the heart rather than the left as more of the heart is in the left hemithorax due to the tension pneumothorax. Impaired coronary blood flow and increased cardiac demand would further exacerbate cardiac ischemia. Related pathologies have also been shown to cause ST-segment changes. Brearley et al reported pneumomediastinum as a mimicker of myocardial infarction [12]. Additionally, Brandão et al showed transient ST-segment elevation in a patient with right pneumothorax and pneumopericardium [13]. These cases highlight the dynamic nature of ECG, particularly in reversible causes of cardiac ischemia.

The rapid resolution of the ECG changes after treatment of the pneumothorax in our case highlights the fact that the underlying cause must be transient and correctable in nature and not due to chronic cardiopulmonary changes.

4. Conclusion

This case report highlights an important lesson. The symptoms of pneumothorax often overlap with the symptoms of other life-threatening pathology such as unstable angina, acute myocardial infarction, or pulmonary embolism. As we have discussed, the ECG findings can also mimic these conditions. Inappropriately treating a pneumothorax with thrombolytics could be devastating. We hope that our readers are better informed about the ECG changes that can arise in right-sided pneumothorax. It is important to avoid misdiagnosis when presented with these findings, especially because of similar symptomatology. Regarding the specific pathophysiologic causes of the ECG tracings in pneumothorax, further research will have to be done. Unfortunately, the dynamic nature of ECG makes this particularly difficult. As our review of the literature shows, the discussions regarding the underlying causes for these ECG changes in pneumothorax are still only hypothetical in nature.

Disclosures

The authors have no conflicts of interest to declare. No funding was provided for this study. Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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