Electrocardiographic changes in spontaneous pneumothorax

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There are many causes of acute chest pain. In adults with coronary artery disease (CAD) risk factors who present with chest pain, dyspnea, and electrocardiographic (ECG) changes of ST-segment deviation, acute coronary syndrome is considered by physicians. However, other causes for this type of presentation must be considered. Spontaneous pneumothorax is another condition which may mimic acute myocardial infarction (MI). Pneumothorax is often a medical emergency. ECG changes resulting from pneumothorax have been reported previously [1], but remain poorly appreciated. In most cases, the ECG changes revert to normal after resolution of pneumothorax and are not related to any underlying cardiac disease [2,3]. In the largest case series to date of ST-segment abnormalities in spontaneous pneumothorax, we present the ECG changes observed in 15 spontaneous pneumothorax cases that were free from any demonstrable cardiac illness. The potential mechanisms for these changes are discussed.

A total of 66 patients with spontaneous pneumothorax (38 left- and 28 right-sided) were admitted in the emergency department (ED) in a tertiary care hospital, Salem, India over a 3-year period (April 2007 to April 2010). All patients had a 12-lead ECG, chest radiograph, cardiac biomarkers, and echocardiography. A significant ST-segment abnormality was defined as ≥1 mm of ST-segment deviation at the J-point in at least 2 contiguous leads. The percentage of pneumothorax was calculated by two different radiologists, and confirmed by senior radiologist [4]. Statistical analysis was carried out using simple descriptive statistics and the chi-square test to compare those with left- versus right-sided pneumothorax.

Of the 66 patient cohorts, 15 (23%) patients had significant ECG changes. All patients presented to the ED with chest pain and difficulty in breathing. Among 15 patients, there were 9 males and 6 females. Ten patients had left-sided pneumothorax and 5 had right-sided pneumothorax. None had bilateral pneumothorax.

ST elevation was observed among six with left-sided pneumothorax, and five with right-sided pneumothorax (Fig. 1). ST depression was observed in V2 and V3 in four patients with left-sided pneumothorax (Fig. 2). Complete blood count, basic metabolic profile, lipid panel, liver function panel, and cardiac enzymes (at baseline and repeated in 4–6 h) were within normal limits on admission. On aspiration of the pneumothorax, the ECG returned to normal within 60 to 90 min in all patients. Echocardiography and coronary artery angiography revealed no significant abnormalities in these 15 patients. Pulmonary function testing showed a mild restrictive pattern in two patients. The clinical characteristics of patients are given in Table 1. These patients remained well at follow-up of 6 months.

There was no significant difference in the ECG changes between right- and left-sided pneumothorax (p = 0.56, odds ratio (OR) = 1.64, 95% confidence interval (CI) = 0.51–5.26). ST depression was noted only among left sided pneumothorax.
The association of ECG changes and pneumothorax has been well-established [1,6]. In the largest series of ST-segment abnormalities in cases of spontaneous pneumothorax, we did not observe any differences in the ECG abnormalities based on the side of the pneumothorax.

Potentially, pneumothorax can produce many other changes in the ECG [1,7]. Even though more than 70% of air in thoracic cavity is needed to produce ECG changes, we observed such changes in our patients with 35 to 60% air in the chest. It may be due to a lean body mass and smaller thoracic cavity, which makes them more prone to get ECG changes even with smaller percentage of pneumothorax.

In our series, the patients showed significant ST elevation and depression in two or other leads, which abruptly disappeared after the expulsion of air from thoracic cavity [3,8]. Although still controversial, several hypotheses have been explained the 12-lead ECG changes in pneumothorax:

**Air in the retrosternal space**

In pneumothorax, air acts as an insulator between the electrode and the heart. When recorded in the upright position, the ECG has been reported to normalize [7]. The normalization is thought to be due to the majority of air moving into apical aspect of the lung, reducing the amount of air adjacent to the heart.

**Alteration in the pendular motion of the heart**

The air between the heart and adjacent areas may alter the pendular motion of the cardiac contraction and contribute to ECG changes. During systole, the heart moves towards the sternum, and away from the sternum during diastole [9]. The positive wave generated during systole corresponds to ST elevation in the precordial lead.
leads. During diastole, the inferior wall of the heart is pushed up by the air into the thoracic cavity, and moves towards the caudal end resulting in a positive wave in the inferior leads during diastole [8].

**Rotation of the heart**

Clockwise rotation of the heart on the longitudinal axis and posterior shifting have been suggested as other possible reasons for such ECG changes [10].

**Effect of the pleural pressure on coronary circulation**

Air in intrapleural space may restrict cardiac contractility and cause compression of the coronary vessels. Also, increased intrapleural pressure may decrease venous return and stroke volume, resulting in tachycardia. Tachycardia further increases the oxygen demand, shortens diastolic perfusion, and may lead to ischemia of coronary vessels.

Several reports cite that the patients presenting with pneumothorax being initially diagnosed as MI. Some patients have received thrombolytic therapy or urgent coronary angiography before the exact diagnosis of pneumothorax has been made [11]. Also, a case of MI in a patient with tension pneumothorax has been reported [12,13] and the authors postulated that the acute stress of pneumothorax with its associated sympathetic surge, hypoxia, and tachycardia could be a stimulus to initiate plaque rupture in a susceptible individual.

Physicians should be aware that reversible ECG changes, specifically ST segment depression and elevation, are not uncommon in patients with spontaneous pneumothorax. A complete history and physical examination are mandatory prior to initiation of therapy for acute coronary syndrome, in order to avoid premature action on the basis of the ECG alone.

**References**


Table 1

Clinical characteristics of patients.

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Risk factors for CAD/ pneumothorax</th>
<th>Heart rate (bpm)</th>
<th>Blood pressure (mm Hg)</th>
<th>Chest X-ray (percentage of pneumothorax)</th>
<th>Electrocardiogram pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>33</td>
<td>M</td>
<td>Smoker</td>
<td>78</td>
<td>110/70</td>
<td>Left (42)</td>
<td>ST elevation in II, III, aVF</td>
</tr>
<tr>
<td>35</td>
<td>M</td>
<td>Smoker, HTN</td>
<td>81</td>
<td>110/80</td>
<td>Left (35)</td>
<td>ST elevation in I, V2, V5, V6</td>
</tr>
<tr>
<td>38</td>
<td>F</td>
<td>DM, OCP</td>
<td>67</td>
<td>120/70</td>
<td>Right (45)</td>
<td>ST elevation in I, V2, V5, V6</td>
</tr>
<tr>
<td>32</td>
<td>M</td>
<td>None</td>
<td>92</td>
<td>90/60</td>
<td>Left (42)</td>
<td>ST elevation in I, V2, V5, V6</td>
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<tr>
<td>42</td>
<td>F</td>
<td>HRT</td>
<td>77</td>
<td>130/80</td>
<td>Left (60)</td>
<td>ST depression in V2, V3</td>
</tr>
<tr>
<td>38</td>
<td>M</td>
<td>Weight lifter</td>
<td>84</td>
<td>120/90</td>
<td>Left (42)</td>
<td>ST depression in V2, V3</td>
</tr>
<tr>
<td>40</td>
<td>M</td>
<td>None</td>
<td>89</td>
<td>90/40</td>
<td>Right (55)</td>
<td>ST elevation in I, V2, V5, V6</td>
</tr>
<tr>
<td>38</td>
<td>M</td>
<td>Type A personality</td>
<td>110</td>
<td>100/70</td>
<td>Left (45)</td>
<td>ST elevation in II, III, aVF</td>
</tr>
<tr>
<td>34</td>
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<td>None</td>
<td>82</td>
<td>110/60</td>
<td>Left (45)</td>
<td>ST elevation in II, III, aVF</td>
</tr>
<tr>
<td>36</td>
<td>F</td>
<td>Family history of CAD</td>
<td>77</td>
<td>110/90</td>
<td>Right (55)</td>
<td>ST elevation in V1-V6</td>
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<tr>
<td>54</td>
<td>M</td>
<td>None</td>
<td>89</td>
<td>100/60</td>
<td>Left (45)</td>
<td>ST elevation in I, V2, V5, V6</td>
</tr>
<tr>
<td>40</td>
<td>F</td>
<td>DM, HTN, OCP</td>
<td>86</td>
<td>140/100</td>
<td>Right (45)</td>
<td>ST depression in V2, V3</td>
</tr>
<tr>
<td>45</td>
<td>M</td>
<td>DM, HTN, Smoking</td>
<td>68</td>
<td>150/100</td>
<td>Left (45)</td>
<td>ST depression in V2, V3</td>
</tr>
<tr>
<td>40</td>
<td>F</td>
<td>None</td>
<td>71</td>
<td>130/90</td>
<td>Left (45)</td>
<td>ST depression in V2, V3</td>
</tr>
<tr>
<td>35</td>
<td>M</td>
<td>None</td>
<td>81</td>
<td>120/80</td>
<td>Right (45)</td>
<td>ST elevation in I, V2, V5, V6</td>
</tr>
</tbody>
</table>

CAD — coronary artery disease, M — male, F — female, DM — type 2 diabetes, HTN — hypertension, OCP — oral contraceptive pills, HRT — hormone replacement therapy, HR — heart rate, BP — blood pressure in mm Hg, bpm — beats per minute.