Learning Objectives

- Pulmonary hemorrhage following synchronized cardioversion is exceedingly rare
- The relevance of shock magnitude, duration, as well as preprocedural anticoagulation, remain unclear
- Endotracheal intubation, bronchoscopy and reversal of anticoagulation, if possible, remain key initial temporization measures

Introduction

Electrical cardioversion remains an effective technique to restore sinus rhythm in patients with atrial fibrillation. The procedure is relatively safe, with major bleeding events being quite rare. Based on currently existing data, only one reported case of pulmonary alveolar hemorrhage immediately following a synchronized cardioversion for nonvalvular atrial fibrillation (NVAF) has been reported. [1] As such, very little is known about this potential complication, specifically regarding its etiology and pathophysiology. This is only the second reported case of pulmonary hemorrhage following elective synchronized cardioversion for paroxysmal atrial fibrillation.

Case

A 71-year-old male with persistent nonvalvular atrial fibrillation (NVAF) on dabigatran for anticoagulation and congestive heart failure presents with shortness of breath and large volume hemoptysis within 24 hours of an elective synchronized cardioversion; during that procedure, two shocks of 360J each were administered with minor skin burning being the only immediate complication.

The patient was significantly hypoxic on arrival, in significant respiratory distress, with crackles and decreased breath sounds noted at the left lung base on physical examination. Chest X-ray and CT showed patchy and confluent ground glass opacities in the left lung concerning for alveolar hemorrhage (Figure 1). Dabigatran was immediately reversed with idarucizumab and the patient underwent protective lung ventilation with selective right mainstem endotracheal intubation.

Table 1. Serologies, Titers and Work-up

<table>
<thead>
<tr>
<th>Serology</th>
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</thead>
<tbody>
<tr>
<td>ANA</td>
<td>Negative</td>
<td>BAL Culture</td>
<td>Negative</td>
</tr>
<tr>
<td>C-ANCA</td>
<td>Negative</td>
<td>ESR</td>
<td>111 mm/hr</td>
</tr>
<tr>
<td>P-ANCA</td>
<td>Negative</td>
<td>CRP</td>
<td>143.5 mg/L</td>
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Case Continued

Subsequent bronchoscopy with alveolar lavage demonstrated copious amounts of bloody secretions from the entire left lung with no significant endobronchial lesions. Cultures, including alveolar lavage cultures, remained negative. Subsequent workup ruled out autoimmune or vasculitis etiologies (see Table 1). Significant bleeding stopped within 24 hours and the patient was discharged home on dabigatran, with repeat imaging showing interval resolution of his left lung alveolar opacities (Figure 1).

Discussion

Electrical cardioversion remains a safe effective method of achieving sinus rhythm, particularly in patients with persistent atrial fibrillation either resistant to, or intolerant of other forms of rhythm control. Major adverse events include arrhythmias, thromboembolic events, skin burns and transient hypotension. [2] Only one prior case of PAH following electrical synchronized cardioversion has been reported; it involved lower energy delivery (70J) and failed to mention use of preprocedural anticoagulation. [1]

Electricity-induced pulmonary damage is rare, usually occurring through accidental exposure to large voltage power sources. Mechanisms include electrical burns, cardiogenic pulmonary edema, as well as pulmonary hemorrhage. [3-6] The pathophysiology of electricity-induced cellular damage may involve electroproportion of cellular membranes or heat-induced macromolecule denaturation. [7,8] It remains unclear how the type and proximity of an electrical source as well as coexisting therapeutic anticoagulation play a role in the condition’s pathogenesis.

References

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