Paroxysmal supraventricular tachycardia: improving diagnosis and management within the accident and emergency department

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METHOD

This study was conducted in the A&E department of the Leicester Royal Infirmary serving a population of over one million and with an attendance of about 90 000 adults each year. All senior house officers (SHOs) within the department undergo a two week induction programme, which includes a lecture on rhythm recognition and an abbreviated Advanced Life Support (ALS) course. The opinion of senior medical staff is available within the department at all times. Middle grade staff are all ALS providers or instructors. A prospective audit was performed over a 15 month period of all adults who presented with a presumed diagnosis of PSVT based on a 12 lead ECG that was repeated after treatment. A total of 48 SHOs worked in the department over this time period.

The use of vagal manoeuvres and all drugs were recorded within the audit sheet and the peri-arrest guidelines of the Resuscitation Council UK were clearly visible within the resuscitation room. To ensure all patients were included a search was performed of the resuscitation room log and the patient administration system.

In the subsequent six months a standard protocol sheet was introduced (available to view on line at http://www.emjonline.com/supplemental) and prospectively audited. No additional training was given during the study period that involved 32 SHOs. The protocol sheet instructed the doctor to carefully inspect the ECG for regularity, rate, absence of P waves, and width of the QRS complex. If the diagnosis was considered to be PSVT treatment was started and recorded on the protocol sheet. ECGs were assessed by a cardiologist blinded to patient treatment. Ethical approval was not required as this was an audit of current practice. Comparison of categorical data was carried out using the $\chi^2$ test with a p value <0.05 considered significant.

RESULTS

Altogether 105 episodes of suspected PSVT were identified in the first audit. In eight patients no ECG could be found and four had spontaneously reverted before full assessment. Of the remaining 93 episodes the final diagnosis was PSVT in 58 (63%), atrial fibrillation in 22 (24%), atrial flutter in 7 (7%), ventricular tachycardia in 2 (2%), and sinus tachycardia in 4 (4%) (table 1).

After the introduction of the standard protocol sheet, a further 48 episodes of suspected PSVT were treated. Two patients spontaneously converted before treatment and no ECGs were not found in a further three patients. Of the remaining 43 cases the final diagnosis was PSVT in 37 (86%), atrial fibrillation in 3 (7%), atrial flutter in 2 (5%), and ventricular tachycardia in 1 (2%) (table 1). There was therefore a 23% improvement in arrhythmia diagnosis (95% confidence intervals: 9% to 38%). The baseline characteristics of each group of patients were similar in terms of age and sex. Sixty five of the 93 patients in the first audit and 27 of the 43 in the second audit had a past history of palpitations/PSVT (p = 0.72).

In the first arm of the audit loop 84 of the 93 patients received adenosine. In 32 cases (38%) the diagnosis was incorrect. Of the 43 patients in the second study group 6 of the 37 patients who received adenosine were not in PSVT (14%). When both groups of patients are considered together a total of 95 patients had a final diagnosis of PSVT. Ten patients (11%) responded to vagal manoeuvres, 74 (78%) to adenosine and the remaining 11 (11%) to verapamil or DC cardioversion (table 2).

The common side effects of flushing, dyspnoea, and chest pain were reported in 17% of cases. Adenosine was administered to eight asthmatic patients with no adverse

Abbreviations: SHO, senior house officer; PSVT, paroxysmal supraventricular tachycardia; ECG, electrocardiogram
effects. No patient given adenosine for atrial fibrillation or atrial flutter experienced proarrhythmic side effects. One patient given adenosine for PSVT developed ventricular standstill for six seconds without complication.

DISCUSSION

The results of this study illustrate the difficulties encountered in the diagnosis of narrow complex tachycardias by doctors in the A&E department. It also shows the value of audit and closing the audit loop. Although a mis-diagnosis rate of 37% would seem to be high it is consistent with a prospective study by Marco et al in the US. Of 79 patients treated with adenosine for presumed PSVT within their A&E department, 46 (58%) had this as their final diagnosis. The commonest difficulty was in patients with fast atrial fibrillation (14%) or atrial flutter (7%). An in-hospital study of 100 patients found only 33 with a final diagnosis of PSVT and 42 with atrial fibrillation or atrial flutter.

Prehospital based studies demonstrate that paramedics with appropriate training and following set protocols are more successful at making the correct diagnosis using “Lead II” rhythm strips. Brady et al identified 139 patients treated with adenosine by paramedics for presumed PSVT. In 105 episodes (75.5%) the diagnosis was correct though 18 (13%) were in atrial fibrillation. In a study of similar design of 85 of 107 patients (79%) were correctly diagnosed and 15 (14%) were in atrial fibrillation/flutter. The success of close adherence to the treatment protocol, designed to ensure those with fast atrial fibrillation were excluded, explains the 23% improvement in diagnosis found in this study.

Adenosine has a comparatively high frequency of side effects. Peripheral vasodilatation results in flushing, carotid body chemoreceptor activation causes dyspnoea, and chest pain may result from activation of adenosine pain receptors in the heart. These unpleasant effects are brief reflecting the drugs short half life as it is deaminated to inosine in the plasma and taken up by red blood cells. Though rare, there are many case reports of adenosine induced arrhythmias. In atrial fibrillation transient slowing of AV nodal conduction after administration of the drug may be of diagnostic benefit but enhanced conduction with a 1:1 ventricular response may occur. A similar response has been described in atrial flutter. This “rebound” phenomenon may result from an increase in sympathetic nerve activity and plasma catecholamine levels.

Table 1: Comparison of final rhythm diagnosis

<table>
<thead>
<tr>
<th>Rhythm</th>
<th>Group 1</th>
<th>Group 2*</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSVT</td>
<td>29 (31)</td>
<td>5 (12)</td>
</tr>
<tr>
<td>AF/Atflu</td>
<td>10 (3)</td>
<td>3 (8)</td>
</tr>
<tr>
<td>VT</td>
<td>4 (4)</td>
<td>1 (2)</td>
</tr>
<tr>
<td>ST</td>
<td>9 (17)</td>
<td>6 (18)</td>
</tr>
<tr>
<td>Total</td>
<td>43</td>
<td>43</td>
</tr>
</tbody>
</table>

*After the introduction of a standard protocol sheet. Percentages shown in parentheses.

Table 2: Treatment of patients with PSVT

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Group 1</th>
<th>Group 2*</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSM</td>
<td>4 (7)</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Valsalva</td>
<td>2 (3)</td>
<td>3 (8)</td>
</tr>
<tr>
<td>Adenosine</td>
<td>44 (76)</td>
<td>30 (81)</td>
</tr>
<tr>
<td>Other</td>
<td>8 (14)</td>
<td>3 (8)</td>
</tr>
<tr>
<td>Total</td>
<td>58</td>
<td>37</td>
</tr>
</tbody>
</table>

*After the introduction of a standard protocol sheet. CSM, carotid sinus massage. Percentages shown in parentheses.

REFERENCES


