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Wolff-Parkinson-White (WPW) syndrome: what the critical care nurse needs to consider when administering antiarrhythmics

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Abstract: This paper discusses the importance of critical care and emergency nurses having an understanding of why pre-existing cardiac disorders can influence antiarrhythmic treatment. The patient with a pre-excitation syndrome is usually managed in a coronary care unit. However, these patients may be admitted to an intensive care unit (ICU) with complications of Wolff Parkinson White (WPW) syndrome; for example post cardiopulmonary arrest or WPW as a co-morbidity. It is common practice in critical care areas for registered nurses to administer antiarrhythmics without a doctor’s prescription in life-threatening situations. Therefore, the critical care nurse must have knowledge of the implications of administering standard antiarrhythmic agents if this patient reverts into a tachyarrhythmia. If antiarrhythmics are administered that are contraindicated in patients with WPW syndrome, then there is potential for deleterious effects. This case study highlights the different pharmacological agents for treating tachyarrhythmias in a patient with WPW syndrome. The paper outlines the correct treatment and discusses the deleterious effects of incorrect administration of drugs in WPW syndrome.


INTRODUCTION

Wolff, Parkinson and White first described a syndrome of paroxysmal tachycardia occurring in healthy young people in 1930. These young patients do not usually have a known history of cardiac disease and are asymptomatic; however, they may describe frequent episodes of increased heart rate. The incidence of WPW syndrome is 4 per 100,000 of the general population.

Pre-excitation refers to the ventricle being excited early and WPW is the most common of the pre-excitation syndromes. Patients in the ICU may have a pre-existing condition of WPW and therefore the critical care nurse must have thorough knowledge of the different pharmacological treatments required by these patients in the event of the patient developing a tachyarrhythmia.

LITERATURE REVIEW

After reviewing the literature, Robinson states that the clinical significance of the pre-excitation syndrome is its tendency to be associated with rapid supraventricular arrhythmias. Thirty-four per cent of patients with WPW syndrome are subject to episodes of atrial fibrillation (AF). AF can be potentially fatal in these patients when the fibrillatory impulses are conducted rapidly over the accessory pathway.

This view is supported by Auricchio et al.; they believe that WPW patients who have syncope attacks should be investigated for episodes of rapid AF. Xie et al. also stated that the reported incidence of patients with WPW who develop arrhythmias is between 12-80 per cent and most patients will have an onset of tachycardia before the age of 40.

Kirkton et al. investigated ICU patients who developed supraventricular tachycardia (SVT). They concluded that the majority of tachyarrhythmia episodes in patients in ICU required multiple pharmacological agents. Critically ill patients present difficult management problems because of the many complicated aspects of the client’s condition such as major trauma, surgery and complications. They proposed that clinicians in ICU follow a set treatment algorithm to assist them with terminating new onset tachyarrhythmias in the critically ill client.

Heinz suggests that clinicians must be very cautious in treating SVT with patients who have WPW syndrome. Calcium channel blockers and digitalis can have disastrous effects if used on these patients. They have the potential to enhance the likelihood of the patient developing AF because of the acceleration of conduction across the accessory pathway.

Adenosine is one of the commonly used antiarrhythmic drugs for treating SVT with WPW syndrome. However, there have been reports demonstrating that adenosine can predispose patients to AF. Xie et al. stated that adenosine is an effective and safe pharmacologic agent in treating SVT in the presence of WPW syndrome but has the potential to be dangerous in the pre-excited
patient with AF. Xie et al. also state that ventricular fibrillation (VF) has been reported after administration of adenosine in patients with WPW and AF.

Hoffmeister et al. also stated that antiarrhythmic drug treatment is complicated by proarrhythmias resulting from the antiarrhythmic drug itself. The proarrhythmia effects are of clinical significance in patients who are prone to developing arrhythmias. Al-Khatib et al. stated that a prospective clinical trial showed that adenosine terminated episodes of SVT in 29 out of 29 patients with an accessory pathway. He explained that the proarrhythmia effects of adenosine can induce atrial flutter in up to 12 per cent of patients, therefore the administration of adenosine to patients with WPW syndrome should be given in medical facilities that have resuscitation equipment available.

Robinson further adds that there are other options to treating SVT and AF in the patient with WPW syndrome. Vagotonic manoeuvres such as carotid sinus massage may be initially attempted; however, they have been reported to fail 50 per cent of the time. A patient who has frequent episodes of AF would benefit from surgical ablation of the accessory pathway. This result in the AF ceasing to be a life threatening problem. Al-Khatib agrees that radio frequency ablation is an effective treatment modality resulting in long-term success rates between 88-99 per cent.

**PATHOPHYSIOLOGY OF WPW**

WPW syndrome is caused by the abnormal conduction of electrical impulses to the ventricles. Electrical impulses generated by the intrinsic pacemaker, the SA node, arrive at the ventricles prematurely. These impulses travel through a shortcut (bypass tract), from the SA node to the ventricles, as the impulse is not slowed as normal by the atrioventricular node.

In patients with WPW syndrome, there is a congenital abnormality in the heart consisting of a small, extra muscle strand that connects the atrium to the ventricles. The abnormal pathway is referred to as the Kent bundle. In WPW the ventricles are pre-excited or conducted early and this predisposes the patient's heart to tachyarrhythmias and rhythm abnormalities. Conduction across an accessory pathway may be directed from the atria to the ventricles in an antegrade or forward conduction and also in a backward direction moving from the ventricles back into the atria – this is referred to as retrograde conduction. This results in a 're-entry' circuit where an atrial premature contraction finds the normal pathway receptive but the accessory pathway refractory. The impulse descends down the AV node then ascends via the receptive accessory pathway in a retrograde fashion. Thus a circular conduction pathway forming a loop is established causing a reentrant tachycardia.

Fusion beats may also result from the concurrent spread of an impulse through the normal and accessory pathways. The impulses enter the ventricle at different speeds causing fusion or joining together of the two impulses. A delta wave or a slurring at the beginning of the QRS complex results and this is due to the premature depolarisation of the ventricle creating the fusion complex (Figure 1).

**CASE STUDY**

The following is an example of how a patient may present to an emergency department with such a syndrome. A 42 year old woman presented via the ambulance to the emergency department with tachycardia and palpitations. Twelve months previously she had an acute episode of narrow complex tachycardia for 60 minutes where it resolved spontaneously and was documented at a rate of 230 beats per minute with no evidence of a short P-R interval or delta wave on the ECG. She was referred by her cardiac physician for electrophysiology studies to be performed at a tertiary hospital with a tentative diagnosis of WPW syndrome. Two days prior to this admission she had complained of a viral infection.

Continued cardiac monitoring showed a narrow complex tachycardia at a rate of 230 beats a minute (Figure 2), respiratory rate of 27 breaths a minute and blood pressure of 110/70mmHg. She stated that she had not followed up the referral for electrophysiology studies as she had not had any prolonged episodes of palpitations. She did admit to a few short episodes which occurred every few months but only lasting 30 seconds. An intravenous line was inserted, Adenosine 6mg was administered followed 3 minutes later with a further 12mg dose.

The patient reverted to sinus rhythm for 2 minutes then went back into narrow complex tachycardia at a rate of 230 bpm. Her blood pressure then dropped to a systolic of 70mmHg, diastolic was unrecordable, the patient began to complain of feeling unwell, nauseated and her oxygen saturation dropped to 90 per cent. She then became unresponsive with no respirations or cardiac output. Cardiopulmonary resuscitation was commenced and immediately the patient was administered a direct current (DC) shock of 50 joules, followed by 100 joules, then 100 joules.

She reverted to sinus rhythm for 5 minutes then went back into the narrow complex tachycardia of 230 beats a minute. Her blood pressure was now 100/50mmHg, oxygen saturation of 97 per cent, on 100 per cent oxygen via the rebreathing mask. The patient was sedated and again cardioverted twice with 100 joules, then given intravenous Flecainide 60mgs administered over 10 minutes. The patient reverted again to sinus tachycardia at a rate of 120 beats a minute. Blood pressure at this stage was 100/70mmHg and respirations at the rate of 20 breaths a minute.

A repeat ECG was performed, as well as a chest x-ray and a blood sample taken for routine bloods and cardiac enzymes. The patient was then admitted into the coronary care unit where she was commenced on oral Flecainide. Education was commenced in the coronary care unit to emphasise the importance of seeking

*Figure 1. Slurred 'R' wave characteristic of WPW.*
immediate medical attention if she has further episodes of palpitations. Explanation of the syndrome was given and the importance of future investigations and possible further treatment modalities were discussed. She was observed for 2 days with no more episodes of tachycardia. She was then discharged home and referred for urgent electrophysiology studies.

DISCUSSION

In the majority of patients, WPW usually presents on the ECG as a short P-R interval and a delta wave. The P-R interval is measured from the beginning of the P wave to the beginning of the QRS complex and reflects the time taken by the cardiac impulse to travel from the SA node to the ventricular muscle fibres. The normal duration of the P-R interval is 0.12-0.20 seconds. The P-R interval will be shortened when the impulse travels along an accelerated pathway such as the Kent bundle bypassing the AV node. Both the sinus beat with normal conduction and the pre-excitation conduction travel spontaneously down the pathways and create a fusion beat. However, the ventricles are activated by the accessory pathway first since there is no AV delay. It is this abnormal conducted impulse that causes the ventricular myocardium to depolarise earlier than normal (Figure 3).

Seventy per cent of patients with WPW have a finding of a shortened PR interval and a delta wave on ECG. These findings are not present in the remaining 30 per cent and this results in what is known as a concealed WPW syndrome. It is the location of the accessory pathway that influences its ability to be identified on the electrocardiography and in this case of concealed WPW diagnosis can only be made by electrophysiological studies. Patients may have single or multiple accessory pathways and the most common locations are the right wall, left lateral, posteroseptal and anteroseptal. The axis of the delta wave on the 12 lead ECG is helpful in indicating the location of the accessory pathway(s).

Arrhythmias associated with the WPW syndrome are often paroxysmal (that is, they begin and end abruptly), supraventricular (the complexes originate above the ventricles) and tachycardias (rapid heart rhythm) — they are abbreviated as PSVT. A patient experiencing an episode of PSVT often describes the arrhythmia as a feeling of rapid palpitations in the chest. However, other patients may be asymptomatic. In PSVT, the patient’s heart rate rises commonly from 130 to 240 per minute. Patients with WPW syndrome also have an increased risk of re-entrant SVT that utilises the accessory pathway as part of the tachycardia circuit. Premature atrial or ventricular contractions may trigger the re-entrant tachycardia. It commonly utilises the normal AV conduction for antegrade conduction and the accessory pathway for retrograde conduction.

AV re-entrant tachycardia with antegrade conduction which occurs through the normal conduction pathways in the patient with WPW syndrome is referred to as orthodromic conduction. Antidromic conduction, on the other hand, refers to conduction in the opposite direction of the normal current and this can occur when there is antegrade conduction through the accessory pathway with retrograde conduction through the normal conduction pathway. It occurs less commonly in WPW syndrome because it requires the presence of multiple accessory pathways.

Another complication for patients with WPW is the increased risk of developing AF. Marriott and Conover’s study of 157 patients with WPW indicated that 41 of the patients had episodes of AF. AF is a common occurring arrhythmia in WPW and when it occurs in these patients the ventricle may be stimulated 300 or more times per minute and this can cause deterioration into VF.

The AV node in a person without WPW can usually control the ventricular rate of AF, despite the increase in number of impulses from the atria. But an accessory pathway simply conducts all the impulses from the rapidly fibrillating atria and this can lead to VF. The accessory bypass tract does not have the same delaying conducting properties as the AV node, therefore the ventricular
responses during atrial flutter or fibrillation may be extremely rapid and this can result in VF. Drugs that prolong the refractory period of the AV node should not be used as they encourage the conduction down the accessory pathway. It is essential that the critical care nurse recognises that there is the potential for deterioration if Digitalis and calcium channel blockers are administered to patients with WPW syndrome.

When a patient with WPW syndrome develops a wide tachyarrhythmia, the critical care nurse must ensure that diagnosis and treatment is accurate. The potential for misdiagnosis can occur when the patient develops a widened SVT or AF and the arrhythmia is treated with intravenous Lidocaine. Lidocaine is an example of a drug which shortens the refractory period of the accessory pathway and therefore may cause an accelerated ventricular rate.

A patient with WPW syndrome who develops AF is treated cautiously with pharmacological agents. Adenosine is recommended for treatment of SVT, however, it is contraindicated in the treatment of AF in WPW syndrome. Drugs which shorten the refractory period of the accessory pathway or prolong the refractory period of the AV node can cause an increased ventricular response and lead to deterioration into VF. Adenosine has been reported to accelerate accessory pathway conduction in patients with AF and cause degeneration into VF. Marriott and Conway recommend cardioversion as the first line emergency treatment for circulatory impairment and the administration of procainamide. Procainamide will block the accessory pathway and slow the heart rate (Table 1).

Ablation is another treatment successfully used in the management of WPW syndrome. Ablation refers to the partial or complete

Table 1. The assessment of possible drugs for WPW syndrome.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Comment</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flecainide</td>
<td>Indicated for treatment of tachyarrhythmias in WPW syndrome</td>
<td>• proarrhythmic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• conduction disturbances</td>
</tr>
<tr>
<td>Procainamide</td>
<td>Indicated for treatment of tachyarrhythmias in WPW syndrome</td>
<td>• conduction disturbances</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• hypotension</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Indicated for treatment of tachyarrhythmias in WPW syndrome</td>
<td>• toxicity</td>
</tr>
<tr>
<td></td>
<td>Precaution: in treating AF in the presence of WPW</td>
<td>• proarrhythmic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• bradycardia</td>
</tr>
<tr>
<td>Adenosine</td>
<td>May be administered to patients with SVT in the presence of WPW syndrome</td>
<td>• bradycardia</td>
</tr>
<tr>
<td></td>
<td>Precaution: not effective in terminating atrial fibrillation/flutter in</td>
<td>• ventricular excitability</td>
</tr>
<tr>
<td></td>
<td>the presence of WPW syndrome</td>
<td>• can induce AF</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• may cause VF</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Contraindicated in the treatment of atrial fibrillation/flutter with WPW</td>
<td>• may facilitate conduction down the accessory pathway and increase risk of</td>
</tr>
<tr>
<td></td>
<td>syndrome</td>
<td>VF</td>
</tr>
<tr>
<td>Verapamil</td>
<td>Contraindicated in the treatment of atrial fibrillation/flutter with WPW</td>
<td>• may facilitate conduction down the accessory pathway and increase risk of</td>
</tr>
<tr>
<td></td>
<td>syndrome</td>
<td>VF</td>
</tr>
<tr>
<td></td>
<td>Precaution: with treating SVT in the presence of WPW syndrome</td>
<td>• may shorten the refractory period of the accessory pathway and therefore</td>
</tr>
<tr>
<td></td>
<td></td>
<td>increase conduction</td>
</tr>
<tr>
<td>Lignocaine</td>
<td>Precaution: with treating tachyarrhythmias in the presence of WPW</td>
<td></td>
</tr>
</tbody>
</table>
destruction of the conduction structures. The radio frequency current has graced amounts of energy which is delivered to a highly focused area. Electrophysiological mapping is performed to determine the location of the accessory pathway and to guide the catheter in the correct position for ablation. Catheter ablation can be performed in conjunction with standard electrophysiological studies combining diagnosis and therapeutic intervention into one study.

A cost effectiveness study of radio frequency ablation by Hogenhuis et al. in Knight compared with other treatment strategies in WPW syndrome supported the practice of performing ablation in patients who experience paroxysmal SVT or AF. Radio frequency catheter ablation is an effective treatment for preventing arrhythmias related to accessory atrioventricular connections. There is a 0.15 per cent risk of sudden death in untreated symptomatic patients with the WPW syndrome who sustain an arrhythmia. It is therefore considered appropriate to perform radio frequency ablation in all symptomatic patients. Ablation is considered to be more effective in preventing arrhythmias, is more cost effective and is associated with lower risk of proarhythmia.

CONCLUSION

Increasing knowledge and understanding of arrhythmias in critical care and emergency nurses may aid in the rapid and accurate diagnosis of life threatening arrhythmias which will ensure prompt intervention and appropriate pharmaco logical treatment. It is imperative that critical care nurses have an understanding of WPW syndrome and the implications it has on the treatment of life-threatening arrhythmias. These patients are predisposed to episodes of paroxysmal tachycardia.

Failure to understand the significance of the different treatment modalities between SVT without WPW and SVT with WPW risks inappropriate treatment with traditional antiarrhythmic blocking drug such as venepamal or digoxin. These drugs have the potential in the setting of WPW to enhance the likelihood of the patient developing AF because of the acceleration of conduction across the accessory pathway and this has the potential to cause rapid deterioration of the patient.

Another aspect that critical care nurses must be aware of is the resemblance of AF in a patient with WPW syndrome to VT. This is due to the delta wave giving an appearance of a wide QRS, however, on close examination, the rhythm is always irregular. Intensive and coronary care units usually have standing orders for pharmacological treatment initiated by nurses in a cardiac arrest. It is imperative, therefore, that critical care nurses have understanding of the dangers of administering certain antiarrhythmic drugs to a patient with a pre-excitation syndrome.

The consequences of incorrect diagnosis of AF with WPW and inappropriate treatment of lignocaine are not reinforced in the education of critical care nurses. It is recommended that further education be included in the orientation and education programs of critical care nurses to include the consequences of treatment of tachyarrhythmias in a patient with WPW syndrome. Research on critical care nurses' knowledge of interpretation of arrhythmias in the setting of WPW syndrome and the associated treatment is recommended.

REFERENCES


