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Non-Valvular Atrial Fibrillation and Stroke: Novel Oral Anticoagulants versus Vitamin K Antagonists

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Introduction

Atrial fibrillation (afib) is the most common sustained cardiac arrhythmia and second most common cardiovascular condition in adults in the United States (Zarraga & Kron, 2012). 2.2 million affected, increases risk of stroke 4 to 6 times (National Institute of Neurological Disorders and Stroke, 2015). Vitamin K antagonists (VKAs) have been mainstay of therapy for over 60 years (King, Holley, & Moore, 2013). Limitations with VKAs such as variability in response, need for frequent lab draws, food and drug interactions, increases patient adherence and under prescribing by physicians (Halperin & Goyette, 2012). Novel oral anticoagulants (NOACs) have been approved by FDA over last 5 years for management of non-valvular afib and stroke prevention. Non-valvular afib is in the absence of rheumatic aortic stenosis, a mechanical or bioprosthetic heart valve, or mitral valve repair (Kron, 2014). NOACs are direct thrombin inhibitors and Xa inhibitors that intend to improve patient adherence, simplify anticoagulation, and overcome barriers to long-term therapy.

Signs and Symptoms

Atrial fibrillation: often have, some asymptomatic in others, difficult to diagnose (Halperin & Goyette, 2012). Heart rate changes, fatigue, palpitations, shortness of breath, exercise intolerance, chest pain, edema, dizziness, and confusion (National Heart, Lung and Blood Institute [NIH], 2014a). Signs and symptoms

Blood supply is interrupted or reduced and oxygen and nutrients cannot get to brain tissue, leading to brain cell death.

Two types are ischemic and hemorrhagic.

Ischemic stroke includes transient ischemic attacks (TIAs) which are temporary blockages that do not leave permanent symptoms (NIH, 2014).

Ischemic stroke from plaque build up and rupture in arteries, leading to blood supply is interrupted or reduced.

Embolic stroke is a type of ischemic stroke that is caused either from plaque breaking off of an artery and goes to artery in the brain (NIH, 2014).

Hemorrhagic stroke is secondary to increase pressure on the walls of the arteries of the brain.

Major complication of afib are aneurysms, arteriovenous malformations (AVMs) are a cause of hemorrhagic strokes (NIH, 2014).

Significance of Pathophysiology

Non-Valvular Atrial Fibrillation

Important to know the pathophysiology of afib and stroke to understand why anticoagulation is needed.

Non-valvular afib independently increases the risk of embolic stroke five fold (Deswalina & Huang, 2011). Risk of ischemic stroke and systemic embolism increases 4-5% per year without antithromboprophylaxis (Halperin & Goyette, 2012).

Specifically for non-valvular afib, two categories of anticoagulants can be used, VKA and NOACs.

In relation to clot formation from the pathophysiology of afib and stroke, health care provider should understand how each medication works in the coagulation cascade.

VKAs

VKAs such as warfarin, interrupt the conversion of vitamin K, which then inhibits the formation of vitamin K-dependent clotting factors in the coagulation cascade.

Coagulation factors II, VII, IX, and X are vitamin K- dependent proteins in the blood.

Interception of the clotting factors prevents conversion of V, VII, IX, and X to factor Xa, and factor II to thrombin.

NOACs

Most studied are factor Xa inhibitor, rivaroxaban, apixaban, and direct thrombin inhibitor, dabigatran.

Factor Xa and co-factor Ya form prothrombinase complex that activates prothrombin to thrombin, which is a major part of clotting process (King, Holley & Moore, 2013).

Thrombin converts fibrinogen to fibrin, as well as other factors that lead to further strengthening of the clot.

Thrombin has actions but it can be bound. Heparins require binding to all three sites to inactivate thrombin, therefore bound thrombin is not affected by heparin (King, Holley & Moore, 2013).

Direct thrombin inhibitors block directly to thrombin, and inhibit both soluble and clot bound thrombin, and factor Xa inhibitors can act on both clot and free thrombin.

Have ability to improve accuracy of treatment for afib and stroke.

Implications in Nursing

Non-Valvular Atrial Fibrillation

Non-Valvular afib increases in prevalence with age and often requires the use of oral anticoagulants to prevent ischemic stroke (Halperin & Goyette, 2012).

Will need to see patients with non-

valvular afib and stroke need education about potential benefit between VKAs and NOACs.

Clinical trials associated with need to determine risk for stroke with afib.

The higher the individual's score, the higher the odds of stroke (Zarraga & Kron, 2013).

Direct-Acting Oral Thrombin Antagonists

Dabigatran study showed 32% lower risk of stroke, systemic embolism, hemorrhagic stroke and acute myocardial infarction, no relation between dabigatran and harm outcomes except for gastrointestinal (GI) bleeding (Lancet, 2008; Fuster, G. Koh, Rho, Broshar, & Fang, 2015).

Study used a large database of participants in real world practice, 2 years after dabigatran became available.

Precaution and dose adjustment for renal impairment and elderly at increased risk for GI tract bleeding.

(Adam, McDuffie, Ortel, & Williams, 2012).

Stopped more often than other NOACs due to adverse effects in GI system (Adam et al., 2012).

Factor Xa inhibitors (rivaroxaban and apixaban)

Rivaroxaban found to be non-inferior to VKA for prevention for stroke and systemic embolism but was found to have a higher risk of major and fatal bleeding (Ogbonna & Jeffery, 2013).

Apixaban found to be non-inferior to VKA warfarin in prevention of stroke or systemic embolism as well as lower rates of major and intracranial bleeding (Ogbonna & Jeffery, 2013).

Dabigatran

Newest direct factor Xa inhibitor being studied for non-valvular afib and stroke prevention.

Found to be non-inferior to VKA warfarin in reducing the rate of stroke and systemic embolism, with a lower incidence of bleeding and cardioembolic deaths (Achariya & Deswalina, 2015).

References


The coagulation cascade and basic mechanisms of action of NOACs (King, Holley, & Moore, 2013)