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Treatment with Non-vitamin K Antagonist Oral Anticoagulants in Patients with Chronic Kidney Disease and Kidney Transplant Recipients

Adetokunbo Taiwo*

Department of Nephrology, Stanford University, 300 Pasteur Drive MC5785, Stanford, CA 94305, USA

Editorial

The prevalence of cardiovascular (CV) occurrences in the general population has successfully decreased thanks to the widespread use of novel oral anticoagulants (NOACs) in patients at greater risk of developing conditions like atrial fibrillation (AF) and venous thrombo-embolic events (VTE). The likelihood of such occurrences rises in some patient populations, including as those with chronic kidney disease (CKD), where the chance of having AF ranges from 19 to 24 percent and can reach as high as 27 percent in individuals with end-stage renal disease (ESKD). In contrast to the general population's 2 percent, kidney transplant recipients (KTRs) experience 7.3 percent within 36 months of the treatment.

predominantly warfarin, in terms of effective anticoagulation and safety of usage, including the risk of bleeding, frequency of thromboembolic events and drug-to-drug interactions. Although not legally prohibited, the use of NOACs in KTRs needs renal function testing and therapeutic drug monitoring of immunosuppressive medications due to their predominate renal excretion and potential pharmacokinetic interactions between two groups. Due to their wider therapeutic window, lower frequency of intracranial bleeding and lack of regular monitoring, NOACs are successfully used as thromboembolic event prevention in patients with AF and as treatment of conditions like deep vein thrombosis and pulmonary embolism in the general population. Acute ischemic stroke individuals who have atrial fibrillation have a fivefold increased chance of having an ischemic stroke [1].

It has been demonstrated that warfarin, a popular vitamin K antagonist (VKA), lowers the incidence of ischemic stroke in both CKD and KTR patients. In patients with CKD, warfarin-based anticoagulation reduced the risk of stroke from 26% to 9%, which is comparable to the relative reduction in stroke risk in the general population. KTR studies have also shown a trend towards a decline in the composite endpoints of death, stroke and gastrointestinal bleeding.

As a direct Xa inhibitor, rivaroxaban is also recommended for the treatment of VTE, the prevention of stroke and systemic embolism in NVAF and the prevention of VTE and CV events in atherosclerotic cardiovascular disease (ASCVD). Rivaroxaban had comparable results to warfarin in preventing stroke and systemic embolism in the randomised controlled trial ROCKET-AF (Rivaroxaban Once Daily Oral Direct Factor Xa Inhibition Compared with Vitamin K Antagonism for Prevention of Stroke and Embolism Trial in Atrial Fibrillation), which included 14,264 patients with AF at moderate to high risk for stroke (mean CHADS2 score 3.5 0.9) and all of Furthermore, the study found that there was no discernible difference between the two groups' rates of serious bleeding [2,3].

Almost total absorption (80 to 100%) is attained with a dosage of 10 mg, but this drops to 66 percent at a dose of 20 mg, indicating that absolute bioavailability is dose dependent. Peak concentrations are seen 2 to 4 hours after oral ingestion, with the majority of absorption taking place in the proximal small intestine. Approximately 92–95 percent of the plasma proteins in humans, primarily albumin, are heavily bound by rivaroxaban. With a steady-state volume of distribution (Vss) of around 50 litres, the distribution volume is modest. The metabolised portion of the rivaroxaban dosage, which is about 2/3 of the total dose, is excreted in the faeces and the kidneys, respectively.

A direct thrombin inhibitor called dabigatran is also advised for treating and preventing primary VTE as well as preventing stroke and systemic embolism in patients with NVAF [4]. The RE-LY study found that, when administered in doses of 110 mg twice daily, dabigatran-based treatment in patients with NVAF reduced stroke risk similarly to warfarin while also posing a lower risk of bleeding. When administered in doses of 150 mg twice daily, however, the reduction in stroke risk was superior but the risk of bleeding was similar. Furthermore, research on the pharmacokinetic characteristics of dabigatran has demonstrated that individuals with CrCl of 15–30 mL/min may benefit from a twice-daily 75 mg dosage schedule in order to get a similar level of drug exposure.

The oral prodrug dabigatran etexilate, having a mean bioavailability of 6.5 percent, is used to give dabigatran. Non-specific hydrolases totally transform it into the active product, which reaches maximal concentration 1.5 to 3 hours after injection with a distribution volume of 50 to 70 L. The bodily tissues quickly distribute dabigatran, which causes the plasma concentration to drop to 30% Cmax after 4-6 hours of treatment and then enter an elimination phase [5].

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Conflict of Interest

The author shows no conflict of interest towards this manuscript.

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***Address for Correspondence:** Adetokunbo Taiwo, Department of Nephrology, Stanford University, 300 Pasteur Drive MC5785, Stanford, CA 94305, USA; E-mail: ade_taiwo@stanford.edu*

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