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Oral Anticoagulants: An Overview

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Editorial

The American College of Chest Physicians' Committee on Antithrombotic Medication and the National Heart, Lung, and Blood Institute examined the ideal therapeutic range for oral anticoagulant therapy. The advice made at the earlier conferences that the dosage of warfarin should be lowered for a variety of reasons is still relevant. Thus, the recommendations for the ideal treatment range for the various indications stay the same whenever a more intensive international normalised ratio is compared directly in a randomised study. Mechanical prosthetic heart valves, in particular, are an exception. The antiphospholipid syndrome and thrombosis in certain people may also necessitate a higher goal. Studies on atrial fibrillation have shown data that are consistent with past findings that the drug warfarin loses almost its entire efficacy. According to the Coumadin Aspirin Reinfarction Trial and a more recent combined hemotherapy and mortality prevention study, aspirin's effectiveness in the secondary prevention of acute myocardial infarction was not enhanced by the addition of low-dose warfarin.

Warfarin, on the other hand, was found to be beneficial in lowering myocardial ischemia events when administered at a target in the Thrombosis Prevention Trial, a primary prevention study in men free of ischemic heart disease at entrance. Low-dose aspirin therapy added to warfarin therapy produced a slight additional benefit but with the risk of more bleeding. Coumarins, which are vitamin K antagonists, prevent the cyclic interconversion of vitamin K and its vitamin K epoxide, which is how they work as anticoagulants. The posttranslational carboxylation of glutamate residues to g-carboxyglutamates on the N-terminal sections of vitamin K-dependent proteins requires vitamin K as a cofactor. For these coagulation factors to function biologically, g-carboxylation is necessary.

By preventing the vitamin K conversion cycle from occurring, coumarins cause the liver to create partly carboxylated and decarboxylated proteins with decreased procoagulant activity. The vitamin K antagonists have the potential to have a procoagulant impact in addition to their anticoagulant effects because they prevent the carboxylation of the regulatory anticoagulant proteins C and S. Coagulation proteins undergo a conformational shift during carboxylation in the presence of calcium ions that makes it easier for them to attach to cofactors on phospholipid surfaces. The carboxylation process, which is related to the oxidation of vitamin KH2 to vitamin K epoxide, calls for the reduced form of vitamin K, molecular oxygen, and carbon dioxide. After that, two reductase processes convert vitamin K epoxide back to vitamin KH2.

Vitamin K epoxide is converted to vitamin K1 by the first, which is sensitive to vitamin K antagonists, while vitamin K1 is converted to vitamin KH2 by the second, which is generally insensitive to vitamin K antagonists. Vitamin KH2 is depleted after treatment with vitamin K antagonists, which restricts the g-carboxylation of the coagulant proteins that are reliant on vitamin K. Vitamin K1 can mitigate the effects of coumarins since the second reductase step is particularly resistant to vitamin K antagonists.

Because vitamin K1 builds up in the liver and is accessible to the coumarin-insensitive reductase, patients receiving high doses of vitamin K1 may also become warfarin resistant for up to a week. Additionally, warfarin prevents the carboxylation of g-carboxyglutamate proteins produced in the bone. There is no proof that warfarin impacts bone metabolism when given to kids or adults, despite the fact that these side effects lead to foetal bone defects in mothers who take it during pregnancy [1-5].

Conflict of Interest

None.

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