

ANTICOAGULANTS

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ABSTRACT

Anticoagulants are medicines that prevent the blood from clotting as quickly or as effectively as normal. Some people call anticoagulants blood thinners. However, the blood isn't actually made any thinner - it just doesn't clot so easily whilst you're taking an anticoagulant.

Anticoagulants are wont to treat and stop blood clots which will occur in your blood vessels. Blood clots can block blood vessels (an artery or a vein). A blocked artery stops blood and oxygen from going to a neighbourhood of your body (for example, to a neighbourhood of the guts, brain or lungs). The tissue supplied by a blocked artery becomes damaged or dies, and this leads to serious problems like a stroke or attack [1]. A blood clot in a large vein, such as a clot in a leg vein - a deep vein thrombosis (DVT), can lead to serious problems. For example, it can lead to a clot that travels from a leg vein to the lungs (a pulmonary embolism). Anticoagulants are used to prevent blood clots as well - the most common condition for this is atrial fibrillation (AF) [2].

Keywords: Anticoagulant, Atrial Fibrillation, Pulmonary Embolism

INTRODUCTION

List of anticoagulants

There are many anticoagulants, including:

- Heparin
- ➢ Warfarin (Coumadin)
- Rivaroxaban (Xarelto)
- Dabigatran (Pradaxa)
- Apixaban (Eliquis)
- Edoxaban (Savaysa)
- Enoxaparin (Lovenox)
- Fondaparinux (Arixtra)

Side effects and risks

There are side effects related to anticoagulant or antiplatelet drugs, and a few are often serious. Call your doctor if you notice any of the subsequent symptoms while taking any anticoagulant or antiplatelet drugs:

- Increased bruising
- Red or pink coloured urine

- Stools that are bloody or appear as if dregs
- More bleeding than normal during your menstrual period
- Purple toes

 \blacktriangleright Pain, change in temperature, or blackish areas in your fingers, toes, hands, or feet.

Because of the side effects of those sorts of drugs, certain people have an increased risk of complications when using them. Some people shouldn't use them at all. If you have bleeding disorder, diabetes, high blood pressure, balance problems, congestive heart failure, or liver or kidney problems, talk to your doctor. Warfarin may increase your risk of complications from these conditions. If you are pregnant or breastfeeding, do not use warfarin. Doing so can increase the risk of fetal death and harm to your baby [3].

The coagulation cascade is triggered by tissue factor release from tissue trauma or vascular injury. Tissue factor forms a posh with factor VIIa within the presence of calcium and cleaves clotting factors X and IX to their activated forms (factors Xa and IXa). The prothrombinase complex is then assembled on a phospholipid membrane and cleaves prothrombin (factor II) to factor IIa (thrombin). Thrombin is one among the foremost potent activators of primary (platelet-mediated) and secondary (clotting factor-mediated) hemostasis. Thrombin also can potentiate clot formation by fibrin polymerization, platelet receptor activation, endothelium activation, and activation of things V, VIII, XI, and XIII. Anticoagulant agents can inhibit thrombogenesis by altering

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various pathways within the clotting cascade or by targeting thrombin directly, attenuating thrombin generation. Indirect inhibitors, however, target and bind to present plasma cofactors, like antithrombin (AT), catalyzing their interaction with clotting enzymes.

New Oral Anti-Coagulants

Oral anticoagulants are widely used for long-term prevention and treatment of venous and arterial thromboembolism. Until recently, vitamin K antagonists, like warfarin, were the sole available oral anticoagulants. This situation changed with the recent introduction of the non-vitamin K antagonist oral anticoagulants (NOACs), which include dabigatran, rivaroxaban, apixaban, and edoxaban. Designed to beat the restrictions of warfarin, the NOACs have revolutionized oral anticoagulation because they seem to be a minimum of as effective as warfarin, but are more convenient to administer because the NOACs are often given in fixed doses without routine coagulation monitoring. Moreover, as a category , the NOACs are related to significantly less intracranial bleeding than warfarin. This is an important advantage because bleeding into the brain is the most feared complication of anticoagulation therapy. In the us, rivaroxaban and apixaban are licensed for prevention of venous thromboembolism (VTE) after elective hip or knee replacement surgery and dabigatran, rivaroxaban, apixaban, and edoxaban are approved for treatment of VTE and for stroke prevention in patients with atrial fibrillation (AF). Although not approved within the us for this indication, rivaroxaban is licensed in Europe for prevention of recurrent ischemia in stabilized patients with acute coronary syndrome (ACS). In this theme series, the role of NOACs for the prevention and treatment of VTE is reviewed by Messerschmidt and Friedman1 and Bacchus and Schulman2 respectively, whereas the evidence supporting their use for stroke prevention in AF will potentially be covered by Sharma et al [4]. Carreras and Mega3 discuss the potential role of the NOACs as adjuncts to antiplatelet therapy in patients with ACS and Crowther et al will potentially provide an update on the status of antidotes for the NOACs.

On the backdrop of those reviews, the aim of this introductory article is to

(1) Compare the pharmacological profiles of the NOACs thereupon of warfarin,

(2) Identify the doses of the NOACs for every approved indication,

(3) Provide a summary of the phase III clinical trial trials performed, to date, with the NOACs,

(4) Briefly discuss the continued studies with the NOACs,

(5) Review the emerging real-world data with the NOACs, and

(6) Highlight the potential opportunities for the NOACs and identify the remaining challenges [5].

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