

The Significance of Anticoagulant Main Categories

Joice Grace*

Department of Bioscience, University of Sheffield, Sheffield, UK

DESCRIPTION

These medications are frequently used to stop blood clots (Thrombi) from forming in veins or arteries or from growing while in the circulation. The medicine which, when administered in blood, stops it from clotting is an anticoagulant. Anticoagulants work by inhibiting the production and activity of certain clotting factors that are naturally found in blood. Thrombosis has been treated and prevented with anticoagulant medications. But, medications may increase the risk of haemorrhage. Therefore, it is crucial to evaluate the risk of bleeding and thrombosis before their clinical use. Due to their limited therapeutic ranges, older anticoagulant medicines like heparin and warfarin require dose adjustment. Deep vein thrombosis, in which clots form in so-called deep veins, such as those in the thighs; pulmonary embolism, in which a clot inhibits the pulmonary artery or one of its branches; coronary thrombosis, in which a clot obstructs a coronary artery in the heart; and disseminated intravascular coagulation, a systemic activation of the coagulation system. Blood storage and blood collection both require anticoagulants. Heparin, which would be administered intravenously, and coumarin- or indandione-based derivatives, which are taken orally, are the two main categories of anticoagulants.

Heparin

Heparin is a combination of mucopolysaccharides that promotes the action of antithrombin, a blood plasma protein that denatures thrombin. It is mostly used in inpatients (an enzyme that promotes clotting). Heparin is administered subcutaneously or intravenously to quickly inhibit coagulation as it is poorly absorbed from the gastrointestinal tract. Heparin does not pass through the placenta, is not secreted into breast milk, or interact to plasma proteins. The kidneys excretion and the liver metabolism put a stop to the drug's activity. Hemorrhage is the major adverse event of heparin; thrombocytopenia (lower number of circulating platelets) and hypersensitivity reactions are

also prevalent. Heparin administration enhances the anticoagulant effects of oral anticoagulants. With the antagonist protein protamine, which has a strong affinity for the negatively charged molecules of heparin, it is easy to block heparin-induced haemorrhage and counteract the anticoagulant effects of medication.

Oral anticoagulants

The coumarin derivatives resemble vitamin K, a vital element in the formation of several clotting factors, structurally. Coumarin derivatives interact with the metabolism of vitamin K in the liver, causing the production of faulty clotting factors that cannot bind calcium ions. The synthetic indandione derivatives (such as anisindione), which make up the other family of oral anticoagulants, are believed to have a comparable mode of action. Oral anticoagulants take a long time to start working because bioactive clotting factors must be eliminated from the blood as well as for the anticoagulant to be absorbed from the gastrointestinal system. The most common oral anticoagulant, warfarin, a coumarin derivative, is rapidly and virtually completely absorbed. The primary way that oral anticoagulants and heparin differ from each other is in the amount of time that they remain active. This is because oral anticoagulants have comparatively prolonged plasma half-lives due to their substantial interaction to plasma proteins. The liver metabolizes oral anticoagulants, which are then excreted in the urine and faeces. They might pass through the placenta and produce haemorrhages or foetal defects in neonates, but their appearance in breast milk doesn't seem to have any negative effects on lactating infants. The main adverse effect of oral anticoagulant treatment is hemorrhage. After several hours, bleeding stops when vitamin K is administered intravenously to assist with the production of functional clotting components. To stop excessive bleeding, plasma with natural clotting factors is administered. Oral anticoagulants and other medications that bind to plasma proteins or are metabolised by the liver may function inadequately.

Correspondence to: Joice Grace, Department of Bioscience, University of Sheffield, Sheffield, UK, Email: racemariieg@gmail.com

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