

35. Aspirin, Plavix®, Coumadin® - when to use what

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Q1: "What is the difference between using coumadin and Plavix for treating clotting disorders? Do you need to be tested for your INR with Plavix? If not, why do so many of us get placed on coumadin instead of Plavix? Is Plavix also influenced by vitamin K food intake?"

A1: Plavix, similar to aspirin, is effective in treating arterial clots (stroke, heart attack, peripheral arterial disease), whereas coumadin is effective in treating venous clots (deep vein thrombosis or pulmonary embolism). Plavix does not influence the INR. Plavix' action is not influenced by vitamin K intake.

Q2: "I don't really understand the issue about aspirin helping prevent arterial clotting but not venous clots. Can anyone shed any light?"

A2: When we injure ourselves and bleed, the normal function of platelets is to clog up the hole in the blood vessels. Platelets do that by sticking together and forming a platelet plug. Medications such as aspirin, Plavix, Ticlid®, and aggrenox® impair platelet function, so that platelets do not stick together as well. In laymen's terms this is called making "platelets less sticky"; in medical terms this is called "inhibiting platelet aggregation". The effect is, that people do not clot as easily. This effect is mainly seen in arteries (see explanation below).

Q3: "My mother called today and said she's been diagnosed with fibromuscular dysplasia. The medication they placed her on was Plavix. I was wondering why they didn't put her on coumadin."

A3: Fibromuscular dysplasia is an uncommon disorder, in which for mostly unknown reasons blood vessel walls thicken and thereby narrow. Since it is mainly an arterial problem, anti-platelet drugs, such as Aspirin or Plavix, are the medications of choice.

Q4: "Does Aspirin therapy help prevent deep vein thrombosis?"

A4: In most cases probably not.

When we injure ourselves and bleed, platelets clog up the hole in the blood vessels. Platelets do that by sticking together and forming a platelet plug (= platelet aggregation). The plug is then strengthened by our clotting factors, which form a meshwork consisting of fibrin. One can prevent the formation of blood clots by either impairing platelet function, or by decreasing the activity of the clotting factors and thereby the fibrin-meshwork formation. Arteries are thick blood vessels with fast flowing blood. Blood clots in arteries are typically triggered by underlying roughening of the artery wall (= arteriosclerosis; atherosclerosis); blood platelets get stuck to the roughened blood vessel wall and form a clot. Thus, the medication of choice in trying to prevent thrombosis in arteries (i.e. stroke, heart attack, peripheral arterial disease, retinal artery thrombosis, etc.), are medications that act against platelets. The following medications are anti-platelet drugs:

- Aspirin (= ASA)
- Plavix (= Clopidogrel)
- Ticlid (= Ticlopidine)
- Aggrenox (= aspirin plus dipyridamole)

By interfering with platelet function, these drugs increase the patient's risk of bleeding, even though to a lesser degree than coumadin. The INR is not influenced by these drugs and vitamin K intake does not influence their effect.

Veins are made up very differently compared to arteries. Veins are thin blood vessels with slow flowing blood. Blood clots that form in veins (deep vein thrombosis, pulmonary embolism) are mainly made up of clotting proteins; platelets do not play a big role in venous clots. Coumadin (= warfarin) is an effective "blood thinner" by preventing the production of clotting factors in the liver, increasing the INR. It is therefore the drug of choice in venous thrombosis. Anti-platelet drugs do not play much of a role in preventing venous clots.

Occasionally, clots in arteries originate from one of the two left heart chambers (= left atrium, left ventricle) and travel from there with the blood stream to the brain, the retina, or the extremities. This typically happens in atrial fibrillation (= irregular heart beat). Such a clot is called an arterial embolism (= arterial thromboembolism; plural: "emboli"). These arterial emboli resemble the type of clots seen in veins, i.e. they have little platelet participation. They are therefore best

treated with coumadin, not with anti-platelet drugs, even though they are clots in arteries.

Personal comment: If a patient with venous blood clots comes off coumadin and asks whether he/she should take aspirin to prevent further clots I usually tell him/her that aspirin is unlikely going to prevent any venous clots. However, I typically do not strongly advise against them.