Angioplasty with Coronary Stents: Risk of Thrombosis

Minimally invasive techniques to revascularize coronary muscle have become standard of care for many patients with events related to coronary artery disease. Stenting procedures are also being used to help revascularize other areas as well such as the brain. These patients require specific considerations while undergoing certain procedures in the dental office.

New studies have shown increased risk of stent thrombosis, myocardial infarction and death caused by inappropriate discontinuation of antiplatelet therapy after coronary artery stenting using stents treated with certain medications. The dental community must be aware of the potentially severe consequences of discontinuing antiplatelet therapy in this group of patients.

Coronary artery stents have been used for three decades to prevent artery collapse and restenosis after Percutaneous Transluminal Coronary Angioplasty. While the initial bare-wire stents were shown to be effective in eliminating the complication of abrupt artery closure, restensois persisted to be a problem in up to 25% of cases. This restenosis was found not to be caused by recurrent coronary artery disease, but rather by growth of smooth muscle cells in the area of angioplasty.

Drug eluting stents (DES) were developed which are a normal metal stent that has been coated with a pharmacologic agent that is known to interfere with the biologic process responsible for restenosis. The DES have been extremely successful in reducing restenosis from the 20-30% range to single digits when combined with appropriate anti-platelet therapy. Currently only two drug-eluting stents, the CYPHER™ sirolimus-eluting stent and the TAXUS™ paclitaxel-eluting stent system, have received FDA approval for sale in the United States (the Cypher stent in April 2003; the Taxus stent was approved a year later in March 2004).

One known complication of the coronary stents is the risk of stent thrombosis after placement. The risk with bare-metal stents (stents not coated with a drug) decreases rapidly after two to four weeks, and patients typically need antiplatelet for only about one month. In comparison, with drug-eluting stents the risk of stent thrombosis lasts for a longer period of time and patients with DES require prolonged antiplatelet therapy (up to one year). The current studies show evidence that drug-eluting stents may be susceptible to events known as “late” and “very late” stent thrombosis, where the blood-clotting inside the stent can occur one or more years post-stent placement. While this has been seen rarely in both the Taxus and Cypher stent, thrombosis is extremely dangerous, fatal in over one third of cases. To prevent thrombosis, post-stent antiplatelet therapy is very important and patients should not be advised to stop taking aspirin, Plavix or Ticlid without first consulting with their interventional cardiologist.

One of the biggest concerns of cardiologists has been what type and duration of antiplatelet therapy is optimal after placement of DES. Early on in the bare metal stent experience, it became clear that the stent surfaces were thrombogenic: the foreign metal object attracted platelets, which tended to congregate and clot, forming a thrombus -- this thrombus could suddenly close off a major coronary artery, causing a myocardial infarction.

Aspirin, combined with ticlopidine (Ticlid) and more recently clopidogrel (Plavix) have become the primary antiplatelet drugs prescribed after stenting. These medications kept the platelets "slippery" while the artery healed and formed a thin layer of endothelial cells over the metal stent, removing the threat of thrombus formation. Four to six weeks seemed to suffice in the era of bare metal stents. But DES work specifically by inhibiting or slowing cell growth, so the length of time for patients to be on antiplatelet therapy was lengthened. Currently the American College of

Cariology/American Heart Association recommends 3 months for the Cypher stent, and 6 for the Taxus, and ideally, up to 12 months if the patient is not at high risk for bleeding. However, because of their concerns, most cardiologists prescribe Plavix for a year and aspirin for life; some even prescribe Plavix for life.

The problem that arises in the dental community is a misguided understanding by both the patients and the providers that these blood-thinning medications should be discontinued if the patient requires oral surgery of any sort. Both patients and dentists are very concerned about the perception that uncontrolled bleeding that might ensue on these medications. In fact, patients are often told by their dentists to stop their Plavix and aspirin prior to oral surgery, with sometimes unintended and life-threatening consequences. In almost all oral surgery procedures the bleeding can usually be controlled with local measures such as absorbable gelatin sponge/packing/sutures and certain antifibrinolytic medications, so there is little or no indication to interrupt antiplatelet drugs for almost all oral surgery procedures.

It is crucial that dental providers advise their patients to continue taking their prescribed anti-clotting (antiplatelet) medications. Most cardiologists have been recommending clopidogrel (Plavix) or ticlopidine (Ticlid) for twelve months and aspirin for life. These blood-thinning medications are critical in preventing blood-clots, a known risk that accompanies stent implantation. If you are planning oral surgery on a patient receiving antiplatelet therapy and feel that it is vital to alter the patients antiplatelet therapy, it is important to consult with the patient's interventional cardiologist before discontinuing any antiplatelet therapy. Most oral surgery procedures can be done without altering the patient's antiplatelet therapy as there is little or no change in the intraoperative or postoperative hemostasis.

References:

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