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| **Pathology Question:** |
| How does healing occur after implant placement and how does diabetes effect this? |
| **Report:** |
| Healing after implant placement occurs between the edge surface of the implant and the prepared osteotomy edge and occurs in four overlapping stages, beginning with hemostasis. The hemostasis stage begins with vasoconstriction and platlet aggregation to form a clot mainly at the inner side of the implant grooves within 24 hours. Platelets play an important role regulating hemostasis and produce biologically active products including vasoactive mediators and chemotactic factors such as proteases, cytokines, and growth factors. Cytokines initiate an inflammatory reaction that serve to remove debri and damaged tissues. Following hemostasis, is inflammation, the second stage in wound healing. Granulocytes and macrophages begin to infiltrate the wound during the inflammation stage. Neutrophils are at the site in the early part of this stage to protect against bacterial invasion, and macrophages come later to initiate collagen synthesis. The inflammatory phase continues for up to 4 to 6 days after. Late in the inflammation stage and overlapping with the following stage, granulation tissue formation begins. The granulation tissue is a soft tissue rich in newly formed vascular structures, inflammatory cells, erythrocytes, and immature fibroblasts. The third stage in healing, is proliferation. Angiogenesis occurs during this stage to restore oxygen supply and start bone healing. Additionally, the formation of the provisional connective tissue matrix occurs. This matrix consists of densely packed mesenchymal cells, collagen fibers and vessels but no or limited inflammatory cells within the soft tissue. The cells within the tissue begin to differientiate into osteoblasts, creating bone. The proliferation stage ends with woven bone formation; this consists of fingerlike projections of immature bone embedded in a primary spongiosa. The fourth and final stage of healing is remodeling, which entails lamellar bone formation and vascular maturation and regression. Lamellar bone consists of lamellae surrounded by marrow spaces rich in vessels, adipocytes, mesenchymal cells, and inflamatory cells. Bone formation starts 4 days after placement of the implant and depending on mechanical stress caused by occlusal forces, remodeling around the implant can persist for atleast 1 year.  Diabetes may affect implant healing in several ways including by lack of vascularization, persistant hyperglycemia, immunosuppression, and by psychological stresses. To begin, diabetic patients tend to have vascular disorders, such as obstructed vascular sclerosis, leading to decrease of vascular circulation. This decrease in vascular circulation can lead to hypoxia which enhances initial inflammatory reactions and increases oxidant free radicals delaying would healing. This happens because oxidant free radicals induce incomplete formation of gap junctions in blood vessels. Also, in terms of vascularization, elevated blood-sugar promotes the production of TNF-alpha which is involved in the inhibition of hemangiogensis, thus impairing healing. Secondly, persistant hyperglycemia found in diabetic patients can inhibit osteoblast activity and alter the response of the parathyroid hormone that regulates metabolism of calcium and phosphorus. As a result of the altered parathyroid hormone, there is a decrease in collagen formation during callus formation, apoptosis of bone lining cells occurs, and there is an increase in osteoclastic activity due to the persistant inflammatory state mentioned earlier. Additionally, diabetic patients are immunosuppressed which causes them to be more susceptible to infection. Infections at the implant site will impair and delay wound healing. Lastly, diabetic patients can be under psychological stress which can cause a substainial delay in wound healing. |
| **References:** |
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