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Opinion Article

Pathological Features of Drug-Eluting Stent Thrombosis: Implications for Clinical Practice

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DESCRIPTION

Drug-Eluting Stents (DES) have been widely used in Percutaneous Coronary Intervention (PCI) to treat Coronary Artery Disease (CAD) due to their ability to reduce the risk of restenosis compared to Bare-Metal Stents (BMS). However, the use of DES has been associated with an increased risk of Stent Thrombosis (ST), a potentially life-threatening complication. In this essay, we will discuss the pathological perspective of DES thrombosis, including the mechanisms of thrombosis, the pathological features of DES thrombosis, and the factors that contribute to the development of DES thrombosis.

Mechanisms of thrombosis

Thrombosis is the formation of a blood clot inside a blood vessel, which can obstruct blood flow and lead to ischemia and tissue damage. The development of thrombosis involves three main components: endothelial injury, altered blood flow, and hypercoagulability.

Endothelial injury can occur due to mechanical trauma from the stent implantation or due to the exposure of the vessel wall to inflammatory cytokines released during the PCI procedure. This injury triggers a cascade of events that result in platelet activation and the release of pro-inflammatory cytokines and chemokines. The activated platelets recruit more platelets and form a platelet plug at the site of injury, which is the first step in the formation of a thrombus.

Altered blood flow can also contribute to the development of thrombosis. The flow of blood in the coronary arteries is normally laminar, meaning that the blood flows in a smooth, orderly manner. However, the stent implantation can disrupt the laminar flow and create turbulent flow, which can cause platelet activation and thrombus formation.

Hypercoagulability is the third component that contributes to the development of thrombosis. Hypercoagulability can occur due to genetic factors or as a result of an acquired condition such as cancer, pregnancy, or immobilization. Hypercoagulability can also be induced by drugs such as hormonal contraceptives, hormone replacement therapy, and chemotherapy.

Pathological features of DES thrombosis

DES thrombosis has distinct pathological features that differentiate it from thrombosis in BMS. The main pathological features of DES thrombosis include the presence of fibrin-rich thrombi, delayed healing of the endothelium, and the presence of malapposed or uncovered stent struts.

Fibrin-rich thrombi: The thrombi that form in DES are fibrin-rich, meaning that they contain a high concentration of fibrin. Fibrin is a protein that is involved in the coagulation process and is responsible for the formation of a stable clot. Fibrin-rich thrombi are more resistant to the action of antiplatelet agents and are more difficult to dissolve compared to platelet-rich thrombi.

Delayed healing of the endothelium: The endothelium is the innermost layer of the blood vessel wall and plays a critical role in maintaining vascular homeostasis. The endothelial cells release nitric oxide, prostacyclin, and other vasodilators that regulate vascular tone and prevent platelet activation and thrombosis. The stent implantation can cause endothelial injury and delay the healing of the endothelium, which can promote thrombus formation.

Malapposed or uncovered stent struts: DES are coated with a drug that is released over time to prevent restenosis. The drug-coating can interfere with the healing process and promote the formation of malapposed or uncovered stent struts. Malapposed stent struts are those that are not in contact with the vessel wall, while uncovered stent struts are those that are not covered by endothelial cells.

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