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Short Communication

The Impact of Inflammatory Factor on Topographic Distribution in a Healing Aneurysm

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DESCRIPTION

The development of an endothelialzed neo intima and thrombus organization is essential for aneurysm healing after Endovascular Therapy (EVT). The procedure is mostly facilitated by the migration of myofibroblast cells, which are derived from the wall of the aneurysm and the surrounding vessel. Cell migration is initiated and directed in large part by the activation and deactivation of the inflammatory cascade [1]. The issue is complex since the pathophysiology of Intracranial Aneurysms (IA) is linked to the loss of mural cells, which in turn causes persistent inflammation of the aneurysm wall. Moreover, bioactive and especially inert endovascular materials (stents) have the potential to cause a protracted local inflammatory response that hinders biological IA healing. While some localized acute inflammation is required to start the healing process of aneurysms, persistent and excessive inflammation can cause thrombus remodeling without thrombus maturation, which can result in aneurysm development or residual aneurysm perfusion. EVT-related healing and permanent occlusion are particularly difficult in IAs that is prone to rupture and have severely deteriorated walls. Indeed, in hypo cellular IAs following EVT, both human histopathology and experimental investigations have demonstrated variable neo intima production and a heterogeneous pattern of thrombus structure between the aneurysm dome and its neck [2]. Therefore, it is believed that a spatial-topographic distribution of inflammatory components is essential for effective IA healing, in addition to timely evolution. Following EVT, loco regional inflammation is essential to the healing of aneurysms. This biological process does not significantly involve the aneurysm wall. In contrast, Day 7 inside the thrombus is when humoral inflammation cells and markers peak and Day 7 inside the early neo intima is when they peak even more. Seven days of healing results in an increasing release of VEGF. Concurrently, endothelial cells expressing CD31 create a continuous layer of cells extending from the nearby vessel along the neo intima, a process linked to full aneurysm healing. Prior research has demonstrated that a chronic mural inflammatory response resulting from the rarefication of aneurysm wall cells causes additional aneurysm growth and rupture [3]. For example, research has shown that the walls of human cerebral aneurysms that have ruptured exhibit a significant overexpression of M1 macrophages and mast cells in comparison to those that have not. Aneurysm formation and rupture have also been linked to M1/M2-macrophage imbalance, mural leukocyte infiltration, phenotypic modulation and loss of smooth muscle cells, endothelial dysfunction, and cell death. However, the thrombus and the neo intima are the primary locations where the same chemokines and cell types accumulate in endovascularly treated aneurysms. However, these mediators trigger distinct mechanisms in this area, encouraging the formation of new endothelialized neo intima, thrombus organization, smooth muscle cell invasion, and myofibroblast differentiation into secretory and contractile phenotypes. Similar findings were observed in a study that looked at the histology and molecular healing of swine experimental aneurysms [4]. At day 3, leukocytes and macrophages were found to be infiltrating the thrombus, and days 7-14 saw the invasion of myofibroblasts. It seemed that macrophage presence was essential for thrombus organization. In terms of genetics, an analysis of different gene expressions between the neck and dome of experimental aneurysms following coil embolization in a rabbit model revealed that good healing is associated with overexpression of genes encoding adhesion molecules, chemo attractants, and proteases (but not structural molecules, like collagen). Another study of ruptured and unruptured human aneurysms also reported the dual and opposing actions of inflammatory factors, which can be either stimulation or destruction depending on the site within the aneurysm complex. The authors discovered that increased luminal thrombosis organization and mural T-cell and macrophage infiltration are linked to the expression of VEGF-receptors in the aneurysm fundus [5].

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