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Oral Presentation

Thrombo-Inflammation in Acute Ischemic Stroke

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Abstract

Ischemic stroke has been classified as a merely thrombotic disease, so the main goal of its treatment is the recanalization of the occluded vasculature. However, despite fast restoration of blood circulation, progressive stroke still develops in many patients, which has led to the concept of reperfusion injury. The underlying mechanism is only partly known. Though, it is accepted now, that thrombotic and inflammatory pathways are key contributors to ischemic stroke, leading to the concept of thrombo-inflammation. In the acute phase after stroke, thrombo-inflammation occurs at the site of ischemic vascular injury, where platelets bind to von Willebrand factor and become activated. Downs tream cascades lead to the activation of the contact-kinin system, resulting in endothelial cell damage, expression of pro-inflammatory cytokines, recruiting immune cells to the side of damage, and supporting immune cell migration into the brain. Activated platelets, the damaged vasculature and attracted immune cells conspire together inducing secondary thrombotic events. In our stroke studies, we significantly improved the stroke outcome in rodents after blocking members of the thrombo-inflammation system (e. g. platelet receptors (GP1b), blood coagulation proteins (Kinin, F12), or immune cells (T cells)) in the acute phase. Therefore, anti-thrombo-inflammatory strategies could become novel treatment approaches in the future.

Keywords: Ischemic Stroke, Thrombo-Inflammation, Pro-Inflammatory Cytokines

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