Debris

Scattered pieces of rubbish or remains.

Microglial debris

If microglial debris is not removed in a timely manner, accumulated debris may influence CNS function. Clearance of microglial debris is crucial for CNS homeostasis. However, underlying mechanisms remain obscure. Zhou et al. investigated how dead microglia are removed. They find that although microglia can phagocytose microglial debris in vitro, the territory-dependent competition hinders the microglia-to-microglial debris engulfment in vivo. In contrast, microglial debris is mainly phagocytosed by astrocytes in the brain, facilitated by C4b opsonization. The engulfed microglial fragments are then degraded in astrocytes via RUBICON-dependent LC3-associated phagocytosis (LAP), a form of noncanonical autophagy. Interference with C4b-mediated engulfment and subsequent LAP disrupt the removal and degradation of microglial debris, respectively. Together, we elucidate the cellular and molecular mechanisms of microglial debris removal in mice, extending the knowledge on the maintenance of CNS homeostasis [1].

Embolic debris

Maekawa et al., investigated possible associations among the presence of cholesterol crystals in embolic debris, the proportions of debris components, and postoperative cerebral embolism in patients undergoing carotid artery stenting (CAS).

Sixty-seven consecutive procedures were performed for internal carotid artery stenosis with CAS at the hospital between November 2015 and February 2018. Procedures for emergency CAS for stroke in evolution or crescendo transient ischemic attack were excluded (n = 12). The embolic debris from remaining procedures (n = 55) was stained with hematoxylin-eosin and the red blood cells, white blood cells, and fibrin were quantified by color-based segmentation. Cholesterol crystals and calcification were examined histopathologically. Diffusion-weighted imaging (DWI) was performed 1-3 days after CAS, and the images were used to classify procedures according to the presence of new lesions.

Of the 55 CAS procedures, new DWI lesions were identified after 32. One patient had symptomatic cerebral embolism. Higher proportions of patients with cholesterol crystals in embolic debris (17 vs. 78%, $p < 0.001$) and higher proportion of white blood cells (mean 2.3 [0-9.9] vs. 4.2% [0-29.9%], $p < 0.01$) were observed in the embolic debris of procedures with and without new DWI lesions.

Cholesterol crystals were common in the embolic debris from patients with postoperative ischemic lesions after CAS. These results suggest that inflammatory destabilization of the intraplaque lipid component is related to postprocedural DWI lesions [2].

1)
Zhou T, Li Y, Li X, Zeng F, Rao Y, He Y, Wang Y, Liu M, Li D, Xu Z, Zhou X, Du S, Niu F, Peng J, Mei X, Ji SJ, Shu Y, Lu W, Guo F, Wu T, Yuan TF, Mao Y, Peng B. Microglial debris is cleared by astrocytes via C4b-facilitated phagocytosis and degraded via RUBICON-dependent noncanonical autophagy in mice.

Nat Commun. 2022 Oct 24;13(1):6233. doi: 10.1038/s41467-022-33932-3. PMID: 36280666.
2)

Maekawa K, Shibata M, Nakajima H, Kitano Y, Seguchi M, Kobayashi K, Sano T, Yabana T, Miya F. Cholesterol Crystals in Embolic Debris are Associated with Postoperative Cerebral Embolism after Carotid Artery Stenting. Cerebrovasc Dis. 2019 Jan 2;46(5-6):242-248. doi: 10.1159/000495795. [Epub ahead of print] PubMed PMID: 30602147.