interleukin 17

Interleukin 23 and interleukin 17 are pro-inflammatory cytokines. IL-23 is secreted by activated macrophages and dendritic cells, while IL-17 by Th17 cells. Serum IL-23 and IL-17 are known to be elevated in numerous inflammatory diseases including neurodegenerative diseases. The role of serum IL-23 and IL-17 in aneurysmal subarachnoid hemorrhage (aSAH) has been investigated.

In a study, 80 patients with aSAH (Hunt and Hess grade I-V) were prospectively recruited. Chaudhry et al. enrolled 24 control patients with lumbar spinal stenosis. Peripheral venous blood was withdrawn from controls and from aSAH patients at day 1 and day 7, allowed to clot and centrifuged to obtain serum. Enzyme linked immunoassay kits were employed to quantify the serum levels of IL-23 and IL-17 by applying 50µL of serum samples. Post hemorrhagic complications and clinical outcome were documented prospectively from patient's hospital record.

Serum IL-23 and IL-17 levels were significantly elevated in aSAH patients at day 1 and day 7 (n=80) as compared to control patients (n=24). Further analysis after dichotomy of patients who suffered from post hemorrhagic complications including cerebral vasospasm, chronic hydrocephalus, seizures, cerebral ischemia, delayed neurological deficits showed differential correlations with different post hemorrhagic complications

Serum IL-23 and IL-17 levels did not correlate with clinical outcome.

Serum IL-23 and IL-17 levels were elevated in patients with aSAH showing upregulation of IL-23/IL-17 inflammatory axis after aSAH. Serum IL-23 and IL-17 showed differential correlations with post hemorrhagic complications and no correlation with clinical outcome [1)].

IL-17A has been identified to promote microglia activation, but the role in the pathology following ICH remains unclear. Autophagy is involved in modulation of cell metabolism, cell survival, and immune response. However, the role of IL-17A in autophagy following ICH has not been well defined. In this study, we assessed the role of IL-17A in microglial autophagic activity following ICH. The microglia were treated with IL-17A, and then autophagy and inflammation were detected. In addition, RNA interference in essential autophagy genes (ATG5 and ATG7) was also utilized to analyze microglial autophagy in vitro. Furthermore, ICH mice were made by injection of autologous blood model in vivo. And the IL-17A-neutralizing antibody was utilized to assess the neurological scores and brain edema. These data demonstrated that IL-17A promoted microglial autophagy and microglial inflammation. The suppression of autophagy using RNA interference in essential autophagy genes (ATG5 and ATG7) decreased microglial autophagy and inflammation. Moreover, IL-17A Ab significantly reduced brain water content and improved neurological function of ICH mice. Taken together, these data demonstrated that IL-17A promoted microglial autophagy and microglial inflammation, and IL-17A-mediated activation of autophagy might represent novel clues in ICH therapy [2)].

1)
Chaudhry SR, Güresir E, Vatter H, Kinfe TM, Dietrich D, Lamprecht A, Muhammad S. Aneurysmal subarachnoid hemorrhage lead to systemic upregulation of IL-23/IL-17 inflammatory axis. Cytokine. 2017 Jun 10;97:96-103. doi: 10.1016/j.cyto.2017.05.025. [Epub ahead of print] PubMed PMID: 28609751.
2)
Shi H, Wang J, Wang J, Huang Z, Yang Z. IL-17A induces autophagy and promotes microglial neuroinflammation through ATG5 and ATG7 in intracerebral hemorrhage. J Neuroimmunol. 2017 Jul

25. pii: S0165-5728(17)30325-9. doi: 10.1016/j.jneuroim.2017.07.015. [Epub ahead of print] PubMed PMID: 28778418.