CD47 and M2 macrophage in pancreatic neuroendocrine tumor: from pathology to target therapy

Targeting immunosuppressive checkpoints has been one of the most significant advances in cancer treatment. CD47 interacts with SIRPα on macrophages to function as a negative immune checkpoint to send a “don’t eat me” signal to ensure that cells are not inappropriately phagocytosed. Since the higher expression of CD47 is believed to bestow cancer cells to evade phagocytic elimination by innate immune cells, targeting CD47 has become more intriguing for a novel cancer treatment. However, the potential therapeutic efficacies of CD47 in a variety of cancers are inconsistent, though the data resulted from animal models, pre-clinical and even clinical trials are mostly encouraging. Macrophages infiltrating cancer tissues, tumor-associated macrophages (TAMs), are an important component in the tumor microenvironment. TAM seems to act as an informative prognostic biomarker in pancreatic neuroendocrine tumor (PanNET). Further understanding of the role of CD163+ macrophage (M2-TAM) in PanNET prognosis may accelerate to improve target treatment outcome. Here, CD47 expression and CD163+ TAM infiltration in human normal pancreatic tissue and PanNETs were studied. We not only analyzed the correlation of CD47 and CD163+ macrophage with clinicopathologic features and prognosis, but also exploded the association of CD47 expression and TAM infiltration in PanNETs. The findings of this study may provide new evidence to aid further understanding the roles of CD47 and TAM in PanNET progression, as well as to develop effective target therapy.