CD47 is a transmembrane glycoprotein with a high expression in both, healthy and cancer (stem) cells. Level of the CD47 expression is negatively correlated with survival of cancer patients. Binding of CD47 to SIRPα, localized on a phagocyte, triggers intracellular signaling cascade. The final effect of this cascade is dephosphorylation of nonmuscle myosin-IIA, which disrupts its function and accumulation to phagocytic synapse. The blockage of CD47-SIRPα signaling pathway in a presence of the pro-phagocytic signal induces phagocytosis of cancer cells. Afterwards, phagocytes can serve as the antigen presenting cells and prime T cell response. Role of CD47-SIRPα signaling pathway in immunity has established this pathway as a target of cancer therapy testing. Preclinical research has identified a positive therapeutic effect of blocking this signaling pathway. Nowadays, the first phase of clinical trials is being conducted. The most prevalent approach of blocking CD47-SIRPα signaling pathway in therapy is the use of anti-CD47 blocking monoclonal antibodies, which cause mild anemia. However, alternative approaches of blocking this pathway are also being developed. In this bachelor thesis, I have summarized the research related to the blockage of CD47-SIRPα signaling pathway as a cancer therapy.