

Leukocyte

Leukocyte is another term for [white blood cell](#).

There are several different types of white blood cells, each with specific functions:

Neutrophils: Neutrophils are the most abundant type of white blood cell and are the first responders to infections. They engulf and destroy bacteria, fungi, and other pathogens through a process called phagocytosis.

Lymphocytes: Lymphocytes are involved in specific immune responses. There are two main types of lymphocytes:

T cells (T lymphocytes): T cells play a central role in cell-mediated immunity. They can directly attack infected or abnormal cells and help coordinate immune responses. **B cells (B lymphocytes):** B cells are responsible for antibody-mediated immunity. When activated, they produce antibodies that can recognize and neutralize pathogens like bacteria and viruses. **Monocytes:** Monocytes are phagocytic cells that help remove dead cells and pathogens from the body. They can also differentiate into macrophages and dendritic cells, which are important for presenting antigens to T cells and initiating immune responses.

Eosinophils: Eosinophils are primarily involved in the immune response against parasites and are also implicated in allergic reactions and asthma.

Basophils: Basophils release chemicals such as histamine during allergic reactions and inflammatory responses. They play a role in the body's response to allergens and certain infections.

[Monocytes](#) are a type of [white blood cell](#) ([leukocytes](#)). They are the largest of all leukocytes.

[Neutrophils](#) (also known as neutrocytes) are the most abundant type of [granulocytes](#) and the most abundant (40% to 70%) type of [white blood cells](#) in most mammals.

What is the difference between monocytes and neutrophils?

A. Broadly, the similarities are: that neutrophils and monocytes are both [phagocytes](#), and they both work to fight infections. But monocytes can turn into [macrophages](#) (when they get into tissues), which are very good at eating things, as well as presenting antigens. Neutrophils eat, but don't present, antigens

see [Leukocyte in cerebrospinal fluid](#).

White [blood cells](#) (WBCs), also called [leukocytes](#) or [leucocytes](#), are the [cells](#) of the [immune system](#) that are involved in defending the body against both [infectious disease](#) and foreign invaders. All [leukocytes](#) are produced and derived from a multipotent cell in the bone marrow known as a hematopoietic stem cell. Leukocytes are found throughout the body, including the blood and lymphatic system.

One classification system for [lymphomas](#) divides the diseases according to the size of the [white blood cells](#) that has turned cancerous. The large-cell lymphomas have large cells. A large cell, in this context, has a diameter of 17 to 20 μm .

see [White blood cell count](#).

In a study of Senders et al. from [Boston](#) and [Utrecht](#), patients were extracted from the [National Surgical Quality Improvement Program](#) registry (2005-2015) and analyzed using [multivariable logistic regression](#).

A total of 7376 [patients](#) were identified, of which 948 (12.9%) experienced a major [complication](#). The most common major complications were [reoperation](#) (5.1%), [venous thromboembolism](#) (3.5%), and [death](#) (2.6%). Furthermore, 15.6% stayed longer than 10 d, and 11.5% were readmitted within 30 d after surgery. The most common reasons for reoperation and [readmission](#) were [intracranial hemorrhage](#) (18.5%) and [wound](#)-related complications (11.9%), respectively. Multivariable analysis identified older [age](#), higher [body mass index](#), higher American Society of Anesthesiologists ([ASA](#)) classification, dependent [functional](#) status, elevated preoperative [white blood cell](#) count (white blood cell count [WBC](#), $>12\,000$ cells/mm³), and longer operative time as predictors of major complication (all $P < .001$). Higher ASA classification, dependent [functional](#) status, elevated [WBC](#), and [ventilator](#) dependence were predictors of extended length of stay (all $P < .001$). Higher ASA classification and elevated WBC were predictors of reoperation (both $P < .001$). Higher ASA classification and dependent functional status were predictors of readmission (both $P < .001$). Older age, higher ASA classification, and dependent functional status were predictors of death (all $P < .001$).

This study provides a descriptive analysis and identifies predictors for short-term complications, including death, after craniotomy for primary malignant brain tumors ¹⁾.

Leukocytes are recruited into the cerebral microcirculation following an ischemic insult. The leukocyte-endothelial cell adhesion manifested within a few hours after ischemia (followed by reperfusion, I/R) largely reflects an infiltration of neutrophils, while other leukocyte populations appear to dominate the adhesive interactions with the vessel wall at 24 h of reperfusion. The influx of rolling and adherent leukocytes is accompanied by the recruitment of adherent platelets, which likely enhances the cytotoxic potential of the leukocytes to which they are attached. The recruitment of leukocytes and platelets in the postischemic brain is mediated by specific adhesion glycoproteins expressed by the activated blood cells and on cerebral microvascular endothelial cells. This process is also modulated by different signaling pathways (e.g., [CD40/CD40L](#), Notch) and cytokines (e.g., RANTES) that are activated/released following I/R. Some of the known risk factors for cardiovascular disease, including hypercholesterolemia and obesity appear to exacerbate the leukocyte and platelet recruitment elicited by brain I/R. Although lymphocyte-endothelial cell and -platelet interactions in the postischemic cerebral microcirculation have not been evaluated to date, recent evidence in experimental animals implicate both CD4+ and CD8+ T-lymphocytes in the cerebral microvascular dysfunction, inflammation, and tissue injury associated with brain I/R. Evidence implicating regulatory

T-cells as cerebroprotective modulators of the inflammatory and tissue injury responses to brain I/R support a continued focus on leukocytes as a target for therapeutic intervention in ischemic stroke.

Rashad et al., from Sendai, Japan showed the intense activation of immune cells, particularly the microglia, along with the increase in macrophage activity and NLRP3 inflammasome activation that is indicated by NLRP3, Interleukin 1 beta (IL-1 β), and Interleukin 18 gene and Caspase-1 upregulation and cleavage as well as pyroptosis.

Leukocytes were observed in the brain parenchyma, indicating a role in cerebral venous thrombosis (CVT)-induced inflammation. In addition, astrocytes were activated, and they induced glial scar leading to parenchymal contraction during the subacute stage and tissue loss. MMP9 was responsible primarily for the BBB breakdown after CVT and it is mainly produced by pericytes. MMP9 activation was observed before inflammatory changes, indicating that BBB breakdown is the initial driver of the pathology of CVT. These results show an inflammation driven pathophysiology of CVT that follows MMP9-mediated BBB breakdown, and identified several targets that can be targeted by pharmaceutical agents to improve the neuroinflammation that follows CVT, such as MMP9, NLRP3, and IL-1 β . Some of these pharmaceutical agents are already in clinical practice or under clinical trials indicating a good potential for translating this work into patient care ²⁾.

¹⁾

Senders JT, Muskens IS, Cote DJ, Goldhaber NH, Dawood HY, Gormley WB, Broekman MLD, Smith TR. Thirty-Day Outcomes After Craniotomy for Primary Malignant Brain Tumors: A National Surgical Quality Improvement Program Analysis. Neurosurgery. 2018 Dec 1;83(6):1249-1259. doi: 10.1093/neuros/nyy001. PubMed PMID: 29481613.

²⁾

Rashad S, Niizuma K, Sato-Maeda M, Fujimura M, Mansour A, Endo H, Ikawa S, Tominaga T. Early BBB breakdown and subacute inflammasome activation and pyroptosis as a result of cerebral venous thrombosis. Brain Res. 2018 Jul 4. pii: S0006-8993(18)30362-7. doi: 10.1016/j.brainres.2018.06.029. [Epub ahead of print] PubMed PMID: 29981290.

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