

## Lec 2

In **Acute inflammation**, **histamine** and **cytokines** like **TNF-alpha** and **IL-1** are released. Histamine induces **vasodilation** and **increases the permeability** of endothelial cells (**transudate** followed by **exudate** state including purulent exudate if infection) resulting in **stasis** (congestion, and erythema) in the affected area. Cytokines activate selectins and integrins on endothelial cells, facilitating the accumulation of white blood cells (WBCs) (**neutrophils** followed by macrophages) on the endothelium (**margination, rolling**(selectin decr.velocity), **adhering**(**selectin**(weak) followed by **integrin** (firmly))). Also, **CD31** will promote WBC **transmigration** across the endothelium (activate enzymes that digest basement membrane) and directs them to the site of inflammation. Excess fluid and microbes can drain into the lymphatic vessels, causing **lymphangitis** which may result in **reactive lymphadenitis** or **inflammatory lymphadenitis** or severe (**resistance lymphadenitis**) when processed by lymph nodes.

**Exudate:** Caused by inflammation due to histamine release and vasodilation, leading to high vascular permeability. Contains high protein, white blood cells, and high specific gravity -> (**Purulent exudate** indicates infection, with accumulated white blood cells, debris, and microbes(pus)).

**Transudate:** Results from imbalances in hydrostatic or colloidal osmotic pressure (e.g., in heart failure or liver disease). Contains low protein, few white blood cells, and low specific gravity.