Acute inflammation – Immediate and early response to tissue injury (physical, chemical, microbiological, etc.) – Vasodilation – Vascular leakage and oedema – Leukocyte emigration (mostly PMNs) Cardinal Signs These signs are: – rubor (redness) – tumour (swelling) – calor (heat) – dolor (pain) – functio laesa, or loss of function (In the second century AD, the Greek physician Galen added this fifth cardinal sign).Examples: Urticaria, allergic rhinitis and bronchial asthma.Pathogenesis: a–increased capillary hydrostatic pressure b–increase capillary permeability c– increase osmotic pressure of tissue spaces – Function: 1–dilute bacterial toxins MPT–104 PATHOLOGY PATHOLOGY 14 2–contains antibody and antitoxins 3–block lymphatics by compression so limiting spread of infection by lymphatics 5) Dilatation of lymphatic vessels: –dilated at inflammed area –Some lymphatics may be blocked by fibrin thrombi this increase oedema but limits spread of infection –Draining lymph nodes develop lymphadenitis Cellular response – The cellular response of acute inflammation is marked by movement of phagocytic white blood cells (leukocytes) into the area of injury. Phagocytosis involves three distinct steps: – Adherence plus opsonisation – Engulfment – Intracellular killing through enzymes, toxic oxygen and nitrogen products produced by oxygen–dependent metabolic pathways (nitric oxide, peroxyonitrites, hydrogen peroxide, and hypochlorous acid) – If the antigen is coated with antibody or complement, its adherence is increased because of binding to complement.5– Pseudomembranous (Membranous) Inflammation: Characteristics & pathogenesis: Severe form of acute inflammation of mucous membranes caused by bacteria that produce exotoxins leading to mucosal necrosis and marked submucosal inflammation resulting in formation of false membrane composed of necrotic mucosal patches and excessive fibrin.The sequence of events in the cellular response to inflammation includes: S Pavementing S Emigration S Chemotaxis S Phagocytosis 1– Pavementing – The release of chemical mediators (i.e., histamine, leukotrienes and kinins) and cytokines affects the endothelial cells of the capillaries and causes the leukocytes to increase their expression of adhesion molecules.– As this occurs, the leukocytes slow their migration and begin to marginate, or move to and along the periphery of the blood vessels 2– Emigration and chemotaxis – Emigration is a mechanism by which the leukocytes extend pseudopodia, pass through the capillary walls by ameboid movement, and migrate into the tissue spaces.– It only occurs in special sites where the skin and subcutaneous tissues are thick and tough due to dense fibrous septa that extend between the deep fascia and the dermis; thus dividing the subcutaneous fat into compartments, therefore infection reaching these multiple compartments leads to multiple suppurative foci.MPT–104 PATHOLOGY PATHOLOGY 18 4– Catarrhal Inflammation: Characteristics: A mild form of acute inflammation of mucous membranes characterized by an exudate mixed with mucous secreted by the irritated mucous membrane.Characters and composition of pus: Pus is a non–coagulable (no fibrinogen) creamy alkaline yellowish or yellowish green fluid composed of: 1– Fluid exudate without fibrin (liquefied).It is worth noting that serous membranes may be affected by other types of inflammation other than serous and fibrinous, e.g. suppurative inflammation Gross Picture: The visceral and parietal layers of the serous membranes are thickened, reticulated, opaque and greyish in case of fibrinous type.Progressively fluid move into the tissues (increased vascular permeability and structural alteration of blood vessels) and cause swelling (tumour), pain, and impaired function.– Once they have exited the capillary, the leukocytes move through the tissue guided by secreted cytokines,

bacterial and cellular debris, and complement fragments (C3a, C5a).II- Diffuse: Caused by Streptococcus haemolyticus bacteria, which produce hyaluronidase (the spreading factor) and streptokinase (fibrinolysin) which dissolves fibrin.iii- Pyaemia: Multiple small abscesses caused by septic emboli derived from septic thrombi due to septic inflammation of veins near the abscess (septic thrombophlebitis).Carbuncle Is a special type of localised suppurative inflammation characterised by multiple communicating deep subcutaneous abscesses, each of which opens onto the surface by a sinus i.e. multiple sinuses occur.- The process by which leukocytes migrate in response to a chemical signal is called chemotaxis 3- Phagocytosis - During the next and final stage of the cellular response, the neutrophils and macrophages engulf and degrade the bacteria and cellular debris in a process called phagocytosis.Acute Suppurative Inflammation I- Suppurative Inflammation (Septic, Purulent or Pyogenic Inflammation) Caused by pyogenic organisms as Staphylococcus aureus, Streptococcus haemolyticus and others.Types of suppurative inflammation: Localised: Caused by Staphylococcus aureus bacteria, which produce coagulase enzyme that leads to fibrin deposition leading to localisation.MPT-104 PATHOLOGY PATHOLOGY 17 Site Occurs in loose connective tissues as subcutaneous tissues, areolar tissue of orbit, scrotum and pelvis. 4) inflammatory fluid exudate: It is the passage of protein rich fluid through the dilated capillary walls into the interstitial tissue at site of irritation.Microscopic Picture: The false membrane consists of necrotic patches, fibrin, bacteria and acute inflammatory cells.6- Other Types of Acute Non Suppurative Inflammation: Haemorrhagic Inflammation: Characterised by excessive erythrocytes within the exudate due to associated vascular damage.Abscess Abscess is a type of localised suppurative inflammation characterised by the formation of a cavity containing pus.c. Fistula: It occurs if evacuation of a deep abscess results in a tract communicating between two surfaces or hollow organs, this tract with two openings is called fistula.Differences between cellulitis and abscess Criteria Cellulitis Abscess Definition Acute diffuse suppurative inflammation.Microscopic Picture: Epithelial cells of the mucosa are swollen and vacuolated due to excess mucin (mucoid degeneration).The underlying submucosa shows dilated capillaries, fibrin and acute inflammatory cells (PMNs and macrophages).The exudation or movement of the fluid out of the capillaries and into the tissue spaces dilutes the offending agent.Dead neutrophils (called pus cells) release their proteolytic enzymes, which liquefy necrotic tissue and fibrin.4- Bacteria and bacterial pigments which may be yellowish (as in staphylococcal infections) or greenish (as in pyocyaneous infections).2- Large abscess: Because absorption of pus occurs at a very slow rate, a big abscess undergoes pointing and rupture (spontaneous evacuation), followed by healing.Complications of abscess: 1- Complications of evacuation and healing: a. Ulcer: It is a local defect or excavation of the surface (discontinuation of the epithelium).Microscopic Features: The .subserosa shows dilated capillaries, fibrin and acute inflammatory cells.2