Inflammation and Homoeopathy

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Article outline

Definition, Cardinal signs of Inflammation, Acute inflammation, Release of preformed mediators, Release of preformed mediators, Activation of soluble reaction cascades, Acute phase response, The complement cascade, Synthesis of new inflammatory mediators, Lipooxygenase pathway, Cyclooxygenase pathways, Chronic inflammation

Definition

- A response of body tissues to injury or irritation; characterized by pain and swelling and redness and heat.

(wordnet.princeton.edu/perl/webwn)

- Inflammation is the complex biological response of vascular tissues to harmful stimuli, such as pathogens, damaged cells, or irritants. It is a protective attempt by the organism to remove the injurous stimuli as well as initiate the healing process for the tissue.

(en.wikipedia.org/wiki/Inflammation)

- The nonspecific immune response that occurs in reaction to any type of bodily injury. It is a stereotyped response that is identical whether the injurious agent is a pathogenic organism, foreign body, ischemia, physical trauma, ionizing radiation, electrical energy or extremes of temperature.

(www.als.net/als101/glossary.asp)

- A localized protective reaction of tissue to irritation, injury, or infection, characterized by pain, redness, swelling, and sometimes loss of function.

(www.apluspetgoods.com/petsupplies/cat-glossary.php)

- A localized protective response elicited by injury or destruction of tissue, which serves to destroy, dilute or wall off both the injurious agent and the injured tissue.

(www.streamlinelaser.com/definitions.html)

- The body's reaction to trauma, infection, or a foreign substance, often associated with pain, heat, redness, swelling, and/or loss of function.

(www.cataractmd.ca/eye-dictionary.php)

- A response to injury that involves swelling, redness, heat, and pain that serves to rid the body of a toxic substance or damaged tissue.

(www.beincharge.com/bic/application)

- A response to injury that is marked by redness, heat, pain, swelling, and often loss of function.

(www.oiaustralia.org/information/dictionary.html)

- Inflammation is the redness, swelling, heat and pain in a tissue due to chemical or physical injury, or to infection. It is a characteristic of allergic reactions in the nose, lungs, and skin.

(www.celebratelove.com/asthmaglossary.htm)

- A condition in which tissue reacts to injury and undergoes changes during the healing process.

(www.peteducation.com/dict_alpha_listing.cfm)

- A basic way in which the body reacts to infection , irritation or other injury, the key feature being redness, warmth, swelling and pain . Inflammation is now recognized as a type of nonspecific immune response.

(www.copdchicago.org/index.php)

- A typical reaction of tissues to injury or disease, usually marked by four signs: pain, swelling, redness, and heat. It may be acute (as in a bum or in gouty arthritis) or chronic (as in rheumatoid arthritis or chronic infections such as tuberculosis).

(www.spondylitis.org/patient_resources/glossary.aspx)

- A process in which part of the body can become hot and swollen, not necessarily directly due to an infection.

(www.encephalitis.info/Shared/Glossary.html)

Over all the inflammation is a protective response that involves the interaction of a number of cellular and molecular components. These interacting elements must be coordinated and controlled in order to deliver an appropriate response to injury or infection.

Cardinal signs of Inflammation

Acute inflammation is characterized by four cardinal physical signs-

- rubor (redness) (Psora)
- calor (heat) (Psora/ Sycosis)
- tumour (swelling) and (Sycosis)
- dolor (pain). (Psora/ Sycosis/ Pseudopsora)

It occurs rapidly after injury and is caused chiefly by components of the intrinsic immune system (Psora). The purpose of acute inflammation is-

- the localisation and eradication of any infective agent or
- the healing of injury.

Inflammatory responses, however, can also harm normal tissue (Syphilis) and, therefore, these responses must be controlled in both their intensity and extent to prevent inappropriate inflammation (Pseudopsora) and potential inflammatory disease (Sycosis). Furthermore, some microorganisms have developed very effective strategies to evade the inflammatory response. Infection with these agents can lead to chronic inflammation because there is continued immunological activation, but a failure to eradicate the microorganism.

Acute inflammation

Inflammation is seen when an immunogen is injected into the skin (Psora). Underlying this is-

- An invasion of the tissues by polymorphonuclear neutrophils (Psora)
- macrophages
- Later on lymphocytes and
- An increase in vascular permeability (Sycosis), causing fluid exudation (Sycosis).

There may also be local tissue effects including increased glandular mucus production (Sycosis) and tissue remodelling (Psora) mediated by fibroblasts and endothelial cells, ultimately causing scar formation (Sycosis).

The mechanisms underlying the local acute inflammatory changes can be classified as-

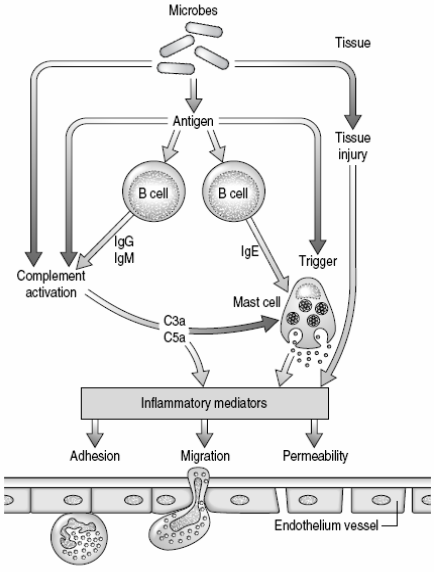
A. Release of preformed mediators from tissues and immune cells (Psora).
B. Activation of soluble reaction cascades (Psora).
C. Synthesis of new inflammatory mediators (Sycosis).

A- Release of preformed mediators

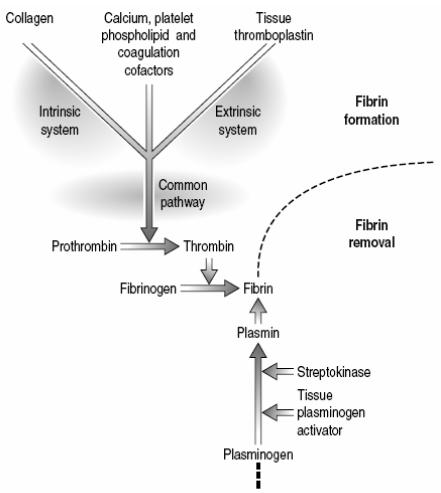
This is one of the first tissue responses to injury. The blood vessel damage is associated with immediate aggregation of platelets (Psora). It is accompanied by release of serotonin (5-

hydroxytryptamine), which promotes vasoconstriction (Psora), further platelet aggregation and the formation of a platelet plug (Sycosis).

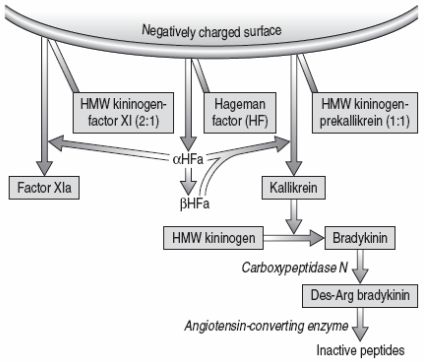
Other preformed mediators released include histamine, heparin, lysosomal enzymes and proteases, neutrophil chemotactic factor and eosinophil chemotactic factor. These factors subsequently induce vasodilatation (Psora), increasing blood flow to the site of injury (Psora), and recruit specific inflammatory cells to the area (Psora).



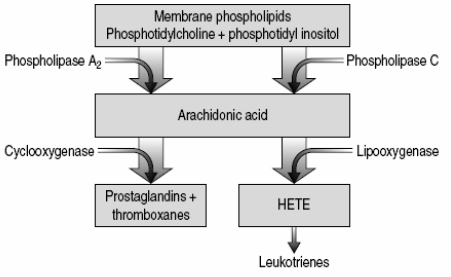
The immune system in acute inflammation. The interactions of a range of innate and adaptive components contribute to acute inflammatory processes.



The fibrin and plasmin pathways interact to maintain a balance in clot formation and breakdown.



The kinin pathway generates important mediators (e.g. bradykinin) that contribute to vasodilatation. HMW, high molecular weight.



Arachidonic acid metabolism is mediated by two distinct pathways, catalysed by lipooxygenase and cyclo-oxygenase, which generate distinct sets of late mediators of inflammation. HETE, hydroxyeicosatetraenoic acid.

B- Activation of soluble reaction cascades

Vascular endothelial cell damage (Syphilis) activates (Psora) plasma clotting factor XII (Hageman factor), which, in turn, activates the fibrin, fibrinolytic and kinin cascades. The fibrin cascade results in the conversion of prothrombin to thrombin (Pseudopsora) (resulting in platelet aggregation) and subsequently fibrinogen to fibrin (resulting in clot formation).

The counterbalancing (Psora) fibrinolytic cascade causes the conversion of plasminogen to plasmin, which breaks down clots and releases other inflammatory mediators. The kinin cascade converts prekallikrein to kallikrein, which, in turn, converts kininogen to bradykinin (resulting in vasodilatation, increased vascular permeability and pain induction).

The effects of these three cascades are essential for controlled acute inflammation:

- Clot formation limits blood loss and acts as a barrier to infection.

- Fibrinolysis limits the degree of clot formation, preventing inappropriate thrombus formation and vascular obstruction, and it allows the release of inflammatory mediators from cells trapped in the clot (Psora).
- Bradykinin causes increased blood flow, with increased delivery of inflammatory cells and exudation of mediators into the area of damaged tissue (Psora).

Angiotensinconverting enzyme is an important factor in the degradation of bradykinin (Pseudopsora).

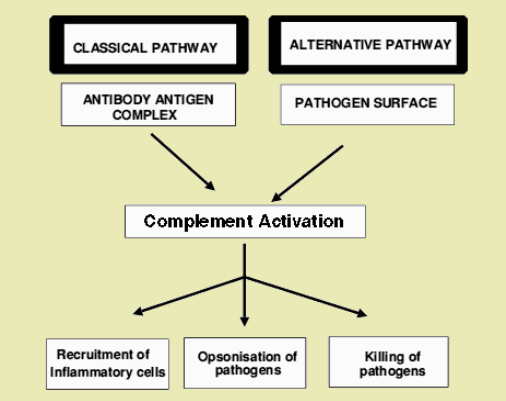
The complement cascade

The complement cascade system is composed of heat labile substances (proteins) that combine with antibodies or cell surfaces. This complex, multicomponent system is composed of about 26 proteins. This "complement cascade" is constitutive and non-specific but it must be activated in order to function.

The functions of complement include:

- making bacteria more susceptible to phagocytosis (Pseudopsora)
- directly lysing some bacteria and foreign cells (Syphilis)
- producing chemotactic substances (Psora)
- increasing vascular permeability (Psora)
- causing smooth muscle contraction promoting mast cell degranulation (Psora)

The complement system can be activated via two distinct pathways; the classical pathway (e.g. by binding to immune complexes) and the alternate pathway (e.g. by binding to foreign antigen).



C- Synthesis of new inflammatory mediators-

Damage to phospholipid cell walls (Syphilis) causes release of arachidonic acid, which is subsequently metabolised via the lipooxygenase and cyclooxygenase pathways (Pseudopsora). This tends to occur about 4–6 hours after injury.

Lipooxygenase pathway-

The lipooxygenase pathway generates leukotrienes, chemotactic and activating factors (Sycosis).

Cyclooxygenase pathways-

The cyclooxygenase pathways generates prostaglandins including prostacyclin (prostaglandin I2), and thromboxanes (Sycosis).

Acute phase response

Acute inflammation is associated with the production of proinflammatory cytokines including IL-1, IL-6 and IL-8 (Sycosis). These cytokines stimulate the liver to produce a series of proteins (Psora) that are collectively known as the acute phase proteins.

These include-

- α1- antitrypsin- (enzyme inhibition) (Psora)
- Complement components- (C3 and C4) (opsonisation) (Pseudopsora)
- C-reactive protein (CRP)- (CRP binds to the C polysaccharide of Streptococcus pneumoniae) (Syphilis)
- fibrinogen and haptoglobins- (scavenging) (Syphilis)

In clinical terms, measurement of acute phase proteins is useful to assess the degree of inflammation in an individual and also to assess the response to therapy. Measurements of serum CRP are particularly useful in this respect as it has a short half life (approximately 6 hours) and a response to should be quickly reflected in a falling CRP level.

The physiological effects of early- and late-phase mediators in type I hypersensitivity reactions

Mediators	Effect
Preformed (early)	
Histamine	Vasodilatation increased, vascular permeability, bronchoconstriction
Heparin	Anticoagulation
Lysosomal enzymes	Proteolysis
Neutrophil chemotactic factor	Chemotaxis of neutrophils
Eosinophil chemotactic factor	Chemotaxis of eosinophils
Newly synthesised (late)	
Leukotrienes LTC4, LTD4	Vasodilatation
Leukotriene LTB4	Bronchoconstriction, chemotaxis
Prostaglandins, thromboxanes	Vasodilatation, platelet activation, bronchoconstriction
Platelet-activating factor	Platelet activation

Chronic inflammation

The purpose of an acute inflammatory response is the eradication of the agent or microorganism that triggered the initial response. In some circumstances that eradication is ineffective or incomplete. To meet this, a phase of chronic inflammation ensues.
The nature of chronic inflammatory damage is dependent on –

- the triggering agent
- the affected site and
- the dominant immune response.

Homoeopathic approach to Inflammation

Top twenty remedies for inflammation in general, repertorized among 522 rubrics pertaining to inflammation found in various repertories.

Remedy	bell.	sulph.	merc.	acon.	puls.	ars.	rhus-t.	sil.	hep.	phos.
Rep. Marks	134	129	128	126	112	111	102	89	86	82
Remedy	lyc.	bry.	canth.	nux- v.	calc.	apis	lach.	nit-ac.	sep.	thuj.
Rep. Marks	81	80	77	77	73	71	68	68	58	57

Prevalence of miasms in inflammation

Psora	86 %
Sycosis	84 %
Tubercular	76 %
Syphilis	75 %
Cancerous	74 %

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