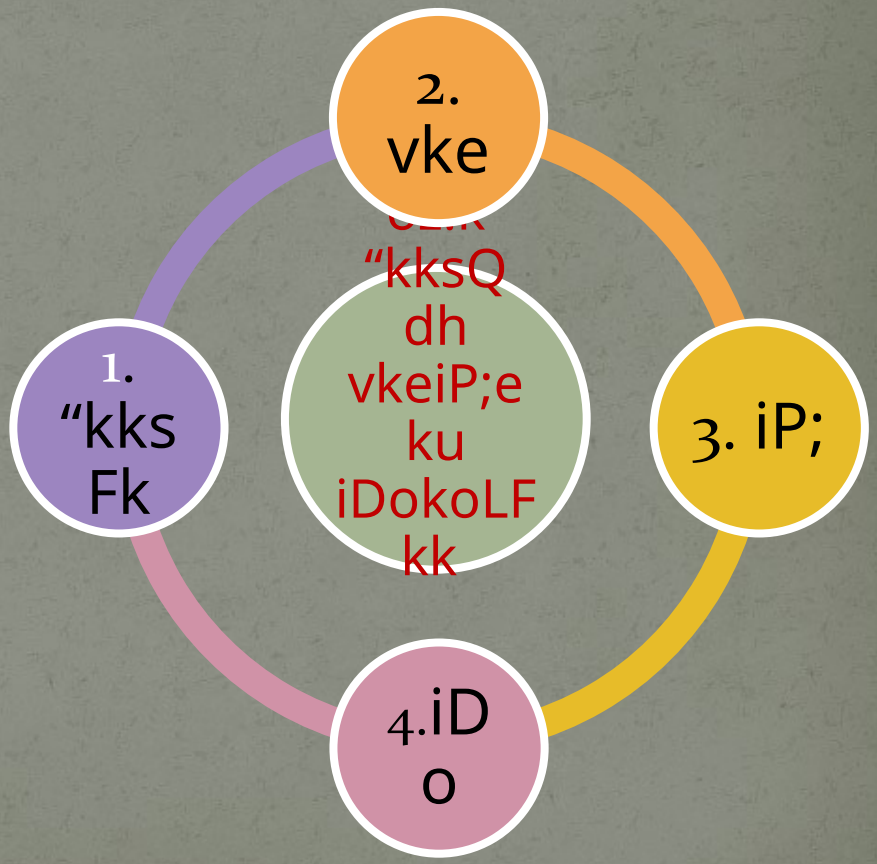
oz.k”kksFk(Inflammation)

- **fu#fDr** oz.kL;iwoZoz.k”kksQA
- **izdkj 6-** ok-] fi-] d-] j-] lfUu-] vkxUrqt
- **y{k.k** 1- lkekU; 2- fo”ks’k
- lxkkSjoa L;knuofLFkrRoa lksRls/keq’ek•Fk fljkruqRoe~A
- Lkykseg’kkZax foo.kZrk p lkekU;fyax “o;Fkks% izfn’VeAA p-fp-12@11
- **Okkrt “kksQ** d`’.k v#.k] i#’k] se`nq] rksn vkfn osnuk
- **firt “kksQ** ihyk] e`nq] yky] “kh?kz Qsyus okyk] nguor~ osnuk
- **dQt “kksQ** “osr] dBksj] fLuX/k] “khr] ean Qsyusokyk] d.Mw
- **lf™kikrt “kksQ** rhuks nks’kksa ds y{k.k

- **jät “kksQ -** firt “kksQ leku y{k.k] vR;f/kd d`”.k of.kZ A
- **vkxUrqd “kksQ** - firt “kksQ leku y{k.k] ykksfgrkoHkkl”p A
- **minzo** - oz.k”kksQ dk ikdkfHkeq[k gksukA lq-lw-17@5
- **uksV** - pjd us “kksQ ds 7 minzo crk,A
- NfnZ% “oklks•#fpLr`”.kk Tojks•frlkj ,o pA



- vkea foiP;ekua p lE;d iDoa p ;ks fHk’kd~ A Tkkuh;kr l Hkosn~ oS| “ks’kkLrLdjo`r;% AA lq-lw-17@7
- **vke oz.k”kksQ y{k.k**
- r= eUnks’erk Rod~lo.kZrk “khr”kksQrk LFkS;Z eUnosnurk•Yi”kksQrk pke y{k.keqfn’Ve~ AA lq-lw-17@5
- **iP;eku oz.k”kksQ y{k.k**
- lqfpfHkfjo fuLrq|rs] n”;r bo fiihfydkfHkLrkfHk”p lalI;Zr bo] fN|r bo “kL=s.k] fHk|r bo “kähfHkLrkM;r bo n.Msu] ihM;r bo ikf.kuk] nãrs iP;r bo pkfXu{kkjkH;keks’kpks’k ifjnkg”p HkofUr% o`f”pdfo) bo p LFkkuklu”k;us’kq u “kkfUreqiSfr vk/ekr cfLrfjookr”p “kksQks Hkofr] RoXoSo.;Z “kksQkfHko`)h Toj nkg fiikl k Hkäk#fp”p iP;ekufyax

- **iDo oz.k”kksQ y{k.k**
- Oksnuksi”kkfUr% ik.Mqrk•Yi”kksQrk oyhizknqHkkZo Lrod~ ifjiqVua fuEun”kZue^{3}~xqY;k•oihfM;rs izR;q™keua cLrkfooksndlapj.ka iw;L; izihM;R;sdeUreUrs pkoihM;rs] eqgqeqZgqLrksn% d.Mq#™krrk O;k/ks#inzo”kkfurHkZäkfHkdka{kk p iDofyaxa A lq-lw-17@5
- **dQt “kksaQ** dQt “kksQ@ vfHk?kkrt “kksQ eas nks’k dh xfr xaHkhj gksus ds dkj.k] “kksQ ds iDo y{k.k fn[kkbZ ugha nsrs vkSj fpfdRld iDo”kksQ dks viDo le>us dk izekn djrk gSA
- **dQt “kksQ ds y{k.k** Rod~ lo.kZrk “khr”kksQrk LFkS;Ze•Yi#trk•”eoPpA

Inflammation -

- Inflammation means burning & inflammation is defined as local response of living tissue to injury due to any agent.
- **Types** – 1. Acute 2. Chronic

Chemical mediators of acute inflammation –

- There are number of chemical mediators which take part in the processs of acute inflammation like vasodilation. Chemotaxis ares of inflammation in response to release of chemical mediators by neutrophils, monocytes & injured tissue.
- These chemical mediators released from the cells, plasma & damaged tissue, they are divided into 2 heads.

- **Cell derived –**
- Mast cells - histamine
- Platelets - serotonin
- Inflammation cells - prostaglandins , leukotriene platelet, activating factors.
- **Plasma derived –**
- Clotting & fibrinolytic system
- Kinin system
- Complement system
- From above chemical mediators two important mediators have role in early inflammatory response – histamine & serotonin

- The main action of histamine are increased vascular permeability, vasodilatation, itching, pain, edema while serotonin having similar action to histamine but it is ales potent than histamine.

➢ **Systemic effects of acute inflammation –**

- Fever 2) lymphangitis 3) toxemia 4) leucocytosis

➢ **Local effects of acute inflammation –**

1. Rubor 2. Color 3. Dolor 4. Tumor

5. Loss of function.

- **Fact of acute inflammation –**
- **Resolution –** complete returns to normal tissue following acute inflammation.
- **Healing by scarring –** when severe tissue destruction is excessive then healing occurs by fibrosis.
- **Progression to suppuration –** bacteria causes serve tissue necrosis & this process finally results in suppuration.
- **Progression to chronic inflammation –** if abscess not trained properly may get organized by dense fibrous tissue & cavity get calcified termed as chronic inflammation.

- **Treatment –**

1. Analgesic & anti-inflammatory
2. Antibiotic
3. Mgso4 dressing
4. Multivitamin
5. Tight bandage
6. I&D if suppuration.

Inflammation ---- increase tissue permeability ----- exudation of protein ----- fibrin formation ----- pyogenic membrane ----- entry of bacteria ----- release toxin & enzymes ----- tissue destruction ----- pus.