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Dr. Shaziia Noor
Professor, Moalijat Kashmir Tibbia College Hospital Research Center Taleemabad Shilvath, Srinagar, Jammu and Kashmir, India

Corresponding Author:
Dr. Shaziia Noor
Professor, Moalijat Kashmir Tibbia College Hospital Research Center Taleemabad Shilvath, Srinagar, Jammu and Kashmir, India

Gastritis (Warm-e-meda) with Unani treatment a review

Dr. Shaziia Noor

Abstract
Gastritis is histologically documented inflamation of gastric mucosa. It is one of commonest problem among people. Unani physcian has mentioned gastritis by different names e.g Hurqat- e- meda, sozish- e- meda, warm- e- media& Iltehab- e- meda. Gastritis can be acute or chronic.Acute gastritis is a transient mucosal inflammatory process that may be asymptometic or cause inconsistant degree of epigastric pain, nausea & vomiting and is often erosive & haemarrhagic, neutrophil are predominant cells in superfical epithelium. Asprin or NSAID drugs intake result in gastritis in many cases. In unani system of medicine, drugs are used for the treatment of gastritis with minimum side effect.

Keywords: Gastritis, stomach, inflammation, Unani treatment Warm- emeda, usool- e ilaj, ilaj bid tadbeer

Introduction
The term gastritis should be reserved for histologically documented inflammation of gastric mucosa. Gastritis is not the mucosal erythema seen during endoscopy and is interchangable with dyspepsia [1].

Anatomy: Stomach-- it is j Shaped structure which lies obliqely in the epigastric, umblicus &left hypochondrium region. It is a muscular bag forming widest & most distensible part of digestive tube. Itact as reservior of food and help in digestion of protein and fats, with 10 inch long, mean capacity 30ml at birth, 1000ml at puberty,1500 -2000ml in adult. Stomach has 2 orifices (pyloric & cardic end)2 curvatures (lesser & greater curvature) & 2 surfaces (anterior &posterior surfaces, lies left 7^{th} costal cartilage 1 inch from its junction with the sternum, at the level of vertebre T11.The stomach has five anatomical regions as cardiac,fundus, body, antrum, plyrous [2].
Histologically the stomach is made of four layers (outer to inner):- (1).Serosa outer layer [2]. muscularis mucosa having 3 layers.

a) Outer longitudinal b) middle circular. C): Inner oblique, [3]. submucosaloose aerolar tissue [4]. Mucosa (inner layer) 2 layers superficial & deep, between the two layerslamina propria.

Blood supply: Left gastric artery (branch of coeliac), Right gastric artery (branch of hepatic artery), Right gastrioepiploic artery (branch of gastroduodonal artery), Left gastroepiploic artery (branch of spleenic artery. The venious drainage isportal vein, superior mesenteric vein, spleenic vein.

Nerve supply: sympatheticT6_T7 of spinal cord via greater splanchic nerve,coelioc& hepatic plexus. These nevers are a) vasomoter b) moter to pyloric sphincter but inhibitory to the root of gastric muscular c) chief pathway for pain sensation from stomach.para sympathetic_vagus through the oesphageal plexes &gastric nervre [3].
Gastric glands
The main gastric glands are simple or branched tubular glands that lie at right angles to the mucosal surface. The gland open into the gastric pit occupy the superficial one/ fourth or less of mucosa, the remaining thickness being closely packed gastric gland. Chief or peptic cell_secrete pepsin. Oxyntic or parietal cell_secretion of Hcl& intrinsic factor which combines with vitamin B12.mucous neck cell_ secrete mucus.G- cell_secretes gastrin hormone. Endochromffin like cell _secrete Histamine. D- cell_ secrete somatostatin [4].

Classification of gastritis_
Gastritis can be acute or chronic

Acute Gastritis
Acute gastritis is transient acute inflammatory involvement of stomach,mainly mucosa that may be asymtomatic or cause variable degree of epigastric pain, nausea vomiting.

Etiology
1. Diet and personal habits:- Highly spiced food,alcohol consumption,heavy smoking,malnutrition.
2. Infection: Bacterial e.g H. Pylori, diptheria, salmonellosis, pneumonia, staphyloccocal food posioning & viral infection as Viral hepatitis, influenza, infectious mononucleosis.
3. Drugs: Aspirin (NSAIDS), cortisone, prepartion of iron, chemotherpy
4. Chronic and physical agents: intake of corrosive chemicals as caustic soda, phenol, lysol, & gastric irraadiation, freezing.
5. Severe stress: emotional factor like shock, extensive burns, trauma, surgery [4].

Mechanism or pathology of gastritis
1) Reduced blood flow resulting in mucosal hypoperfusion due to ischemia.
2) Increased acid secretion and its accumulation due to H. Pylori resulting in damage to epithelial barrier
3) Decreased production of bicarbonate buffer.(3)

Clinical feature
Suden onset of epigastric pain, nausea, and vomiting
Mucosal histology studies marked infiltration of neutrophil with oedema and hyperemia. If not treated the picture will evolve into chronic gastritis [5].
Bacterial infection of stomach or phlegmonous gastritis isarare, potentially life threatening disorder characterized by marked diffuse acute inflammatory infiltrates of entire gastric wall, at times accompanies by necrosis.Patient may affected elderly individual, AIDS,alcoholics. Organism associated with this entity include streptococci, staphylococi, E. Coli, proteus and haemophillus spices. Failure of supportive measure & antibiotics may result in gastrectomy [5].

Chronic Gastritis
Ch. Gastritis is identified histologically by an inflammatory cell infiltrate consisting primarily of lymphocytes & plasma cell with scanty neutrophil involvement.Ch. Gastritis are classified according to histological characteristics.these includes, 1)Superfical Atrophic changes 2)Gastric Atrophy. The association of atrophic gastritis with the development of gastric concern has led to the development of endoscopic& serlogic markers of severity.some of these include of gross inspection &classification of mucosal abnormalities duringstandard endoscopy magnification endoscopy, endoscopy with narrow band imaging & autoflourence imaging&measurement of several bicarbonates including pepsinogen 1&2 level, gastrin 17&anti -H. Pyloric serologies.

Phases
1st Stage: Superfical gastritis Inflamatory change are limited to lamina propria of surface mucosa with oedema & cellular infiltration

2nd Stage: Atrophic gastritis: Inflamation to deeper into the mucosa with progressive distortion & destruction of glands.

3rd Stage: gastric atrophy: Glandular structure are loss & there is pacicity of inflammatory infiltrate. Endoscopically the mucosa may be substantially, then,permitting clear visulization of underlying blood vessel [1].

Classification of ch, gastritis
1. Type A (autoimmune gastritis): Body fundic predominent
2. Type B (H.Pylori gastritis): Antral predominent
3. Type AB (mixed environment gastritis): Antral body gastritis
4. Chemical (reflex) gastritis: Antral body predominent
5. Miscellaneous form of gastritis [4].

Type A (autoimmune gastritis)
Type A gastritis involves mainly the body fundic mucosa. It is also called as autoimmune gastritis due to presence of circulating antibodies against parietal cells intrinsic factor results in depletion of parietal cells &impaired secretion of intrinsic factor. The parietal cell antibody is directed aganist H+K+ATPase leading atrophic gastritis.The mechanism is thought to involve molecular mimicry between H. plyori& H+K+ATPase Autoimmune or atrophic gastritis (Antibodies are formed aganist parietal /oxyntic cell

HCL decreased
Achlorhydria (Hypoachlorhydria) With body defence mechanism weakened,Gastric acid play important role in feedback inhibition of gastrin realease by G- cell. Achlorhydria coupled with relective sparing of antral mucosa (site of G.cell) leads to hypergastrinomia. Paritel cell (Source of IF depeltion realease) IFdecreased VitaminB12 deficiency Pernicious Anemiaor megaloblastanemia [1].

Type B gastritis
Type B gastritis antral predominant gastritis, H. Pylori is the cause of type B gastritis. *H.PYLORI:- Spiral* – shaped, aerophillus, lophotrohus, gram- negative urease producing spectrum. It loves in the mucus layer of stomach adherent to mucosal cell. It has several acid resistance mechansim of which the most important acid is urease enzyme which catalyzes urea hydrolysis& produces buffering ammonia [6].

Pathogensis of H. Pylori
H. Pylori does not invades the mucosa. Instead it damage the mucosa by distrupting the mucous layers liberating enzymes &toxins and adhering to the gastric epithelium urea converts in to ammonia.Thus alkalinsing the surrounding acidic medium. So that H.pylori can survive. Butsimultaniously produces ammonia induced mucosal damage.
H. Pylori produce toxin Vac A(vacualating toxin)& Cag A (associated protein) as well as urease and adherent factors [7].
Diagnosis

Invasive test:-Endoscopic biopsy
a. histologicexamination

b. biopsy urease test
c. culture of microorganisms
d. H. pylori fecal antigen test.

Non Invasive test: a) serologic test (ELSA) b)) 14c urea breath test [5].

Unani concept

In Unani concept gastritis or warm -e-meda as Unani physician has mentioned the gastritis by different names e.g Hurqat-Meda, Sozish-Meda, Warm-Meda & Iltehab-Meda [8].

According to concept Warm -Meda is condition in which there is inflammation of mucous membrane of stomach.The ratio between male and female is same i. e 1:1. According to Humours:- Warm Meda is divided into 4 types

1. Warm Meda dammi (falgemuni).
2. Warm Meda safravi (Hamratemedi)
3. Warm Meda Balghami(warm rekhu)
4. Warm Meda saudavi (warm salb) [10].

Warm-e Meda is also called as Warm Nazli asgastrits affects mostlyin mucous lining of stomach which result in excess production of white mucoid discharge. Thus warm Meda is divided in to 2 types:

1. Warm – e haad (Dammi & safravi)
2. Warm- e muzmin (Balghami & Saudavi) [9].

According to tibb- Jadeedwarm meda is divided into 2 types

1. Warm-e-mada Haad (acute gastritis)
2. Warm-e-meda muzamin (chronic gastritis) [10].

According to unani concept gastritis is the condition in which there is inflammation of mucous membrane of stomach. Beside this, there is inflammation of glands. Mucous membrane of stomachnot only become thick but its colour becomes grey & there occur execessive secreation from lining of stomach,that remains continouslyuntil not treated properly [11].

Causes

1. Ingestion of fatty food
2. Intake of spicy food
3. Sweetish or bitter things
4. Alcohol consumption
5. Overeating

These all are responsible for acute gastritis in some cases, however drinking of hot water may also cause inflammation of mucous membrane of stomach. If warm-e-haad persist for long duration of time,then warm-e- haad leads to change in to warm-e- muzamin [12].

In the third book of canon of medicine Avicenna describes the diseases of internal organ, in particular to detailed description of symptoms of gastritis and gastiric ulcer, close to the modern description i.e vomiting,pain,heart burn & in some cases bleeding [13]. Avicena provide data on disease of stomach as reaction of the organism to changing condition &violation of specific form of adaptability of organism [14, 15].

Usool-e-Ilaj

1. Taqleel-e-ghiza (Diet restriction)
2. Islah-e-Mizaj-e -Medawa Taqwiyat -e-Meda
3. Taleel -e -waram in case of waram -e-haar
4. Ishaal through enema in case of warm -e- haar
5. Rad'e mawaad in case of waram -e -haar
6. Istifraag by mushil-e-sauda in case of waram sulb

Ilaj Bil Tadbeer

Fasad through wareed-e- Akhal in case of waram -e- haar Meda
Light exercise
Long duration of sleep.
Massage with oil &vinegar in case of waram -e- rakhu
Qai is also adiviseble to make stomach empty.

Diet recommendation

Aghziya Lateefa or zo'od Hazim (easily digestable light diet) should be taken.

Diet restriction

Chilled water to be avoided in waram -e- rakhu.
Spicy or oil diet to be avoided.

Treatment in unani

Till date, We have so many treatment regimes in allopatheic as well as alternative medicine including use of antiacid, antibiotic, proton pump inhibitors, H2 blockerswhich provide the relief to patients. Likewise, In unani system of medicine plants, animals as well as minerals origin, drugs are being used for the treatment of gastritis with negligible side effect.

Some drugs are used as single drug called as mufridat adiviya as
asl- us- soos(Glycyrrhizaglabra linn),
Adrak (zingeber officinale Rosa)
Asgand (withania somnifera).
Khulanjan (Alpinia galanga (linn) wild).
Khatmi (Alehara officinalis linn).
Gaozban (onosma bracteatum)
Elva (Aloe barbadensis Mill)
Amla (emblica officinalis)
Kutki (picrorrhiza kurcoa)
Kalonji (Nigella sativa linn)
Haldi (curcuma longa)
Dar hald (Berberus aristale)
Aspghal (plantago ovata forsk)
Tabasheer (Bambusa Roxb)
Mastigi (pistacia lentiscus linn)
Satawar (Asparagus racemosus wild)
Samagh-e-arabi (Acacia)
Bael (Aegle Marmelos correa ex Roxb)
Kerala (Monordica charmlia linn)
Bhul neem (Andrographis paniculata wall)
Jaiphal (Myristica fragrans Hiutt)

Some drugs are used as compound drug called as murakabaat or compound drugs as Majoon zingber, Jawarish Mastigi, Jawarish Anarian Qurs satawar etc. These drugs of there mode of action are to help in soothing effect in inflamatory cells of stomach, provide ground material for healing, antiseptic as well as antibacterial effect. As Aslus–soos (Glycirrhiza glabra)/ licoricecontains flavoinoid that reduce gastric secreation, produce thick mucous that protect stomach linning for inflammation& ulceration [16, 17]. Carbenoxolene, isolated from rhizome was found to be quite

effective in the management of peptic ulcer. Dry powdered of this drug 3(g) is given twice a day in empty stomacha significicantresult is seen [18]. Amla (emblica officinalis) contains butanolic extract of water fraction of fruits posses an antioxidant property. The alcoholic extracts of fruits also indicated significiant reduction in acidity [19].

Conclusion

Gastritis is actually inflamatiory involvement of stomach, mainly mucosa.It can be acute or chronic. A wide range of population is affected worldwide by gastritis.In unani medicine the stength of stomach has principla significance & disease related to stomach are described in detail intheir literature,&by accentuate in functioning of Meda. Almost all unani physcians has contributed towards the information of strength & ailment of Meda.

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