PATHOGENESIS OF PEPTIC ULCER

H.pylori INDUCED ULCER

Gram negative bacteria produced heat shock proteins

Cytokines, histamine, lipopolysaccharides, certain enzymes

Phospholipase

Urease, protease, fucosidase etc.

- Urease convert in acidic media urea into ammonia and carbon dioxide. Ammonia itself cause destruction of mucosal lining.

- Ammonia cause infection of mucosal lining and ultimately inflammatory mediators release.

- Cytokines → Leukocytes adhesion and inflammatory reactions starts

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Damage mucosa of GIT

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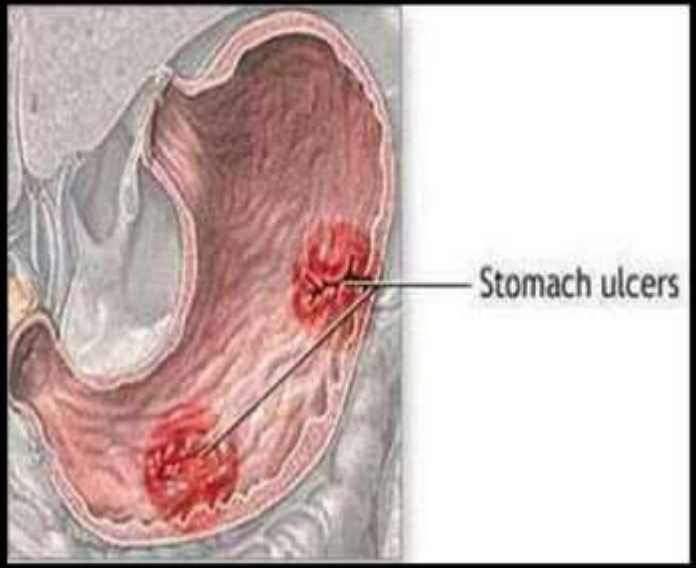
Ulcer occurs


Stomach ulcers


H.pylori as seen under an electron microscope

DRUG INDUCED ULCER

Drugs for example NSAIDS as aspirin(non selectively inhibit cox1 and cox2 in human body

Arachidonic acid → cox1,2 → Prostaglandins

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Controls gastric juice secretions

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Damage mucosal lining lead to ulcer

STRESS INDUCED ULCER

In stress energy consumption increase so increase glycolysis which is usually done by cortisol hormone

This hormone inhibit phospholipase A2

No arachidonic acid formation no prostaglandin increase gastric juice secretions

Cause ulcer

STEROIDS INDUCED ULCER

Steroids acts on cell membrane (phospholipid)

Inhibit phospholipase

Inhibits arachidonic acid no prostaglandins and damaging of mucosal lining

ULCER DUE TO GENETIC DEFECT

Rare genetics occurs some time having blood group O positive the size of parietal cell is increase

Increase cell demand as HCL secretions increase

Cause destruction of mucosal lining leading towards ulcer

ZES(Zollinger-Ellison Syndrome)

In this syndrome tumor of goblet cell occurs

Abnormal mucus secretions(gastrin acts on parietal cells)

Increase secretions of gastric juice

Mucosal lining damage