Caustic Ingestions / Corrosive Injury

Burn α Quantity Majority: intentional	Alkali pH > 7 More common	Acid pH < 7
Caustic Agents	Cleaning/draining agent, button battery	Swimming pool cleaners, battery fluid
Charecteristics	Tasteless, dourless, colurless more amount ingested	Pungent odour, unpleasant taste. Smaller amount ingested
Pathogenesis	**Liquefactive necrosis** Transmural damage in esophagus Gastric acid neutralises – limits the damage	**Coagulative necrosis** Coagulum →limits full thickness injury
Axiom	Bites the esophagus &licks the stomach Esophagus >> Buton battery- burn in 4Hr; perforates in 6 Hr	Licks the esophagus & bites the stomach. Stomach >> Upper airway injuries more common
Presentation	**Early symptoms – no correlation with extent or severity of burn**	
Symptoms	**Initial:** Oropharyngeal/retrosternal pain; Dysphagia/odynophagia; Hypersalivation Persistent pain, fever, shock, tachycardia, rebound tenderness → perforation	
Bleeding	3%; usually 3 weeks after ingestion	
Fistulisation	3% tracheoesophageal fistula - Cough on swallowing liquids; pneumonia 0.02% aortoenteric fistula- GI bleed	
Stricture	MC complication; 1/3rd develop strictures; α depth of injury **Esophageal:** 3 weeks/years to develop; dysphagia; Manometry → low amplitude, long duration waves **Stomach:** Less common; acid ingestion; Antrum- MC affected; Symps s/o GOO Cicatrization and pseudodiverticulae noted on barium	
Esophageal Squamous cell carcinoma (SCC)	30%; after 30 years of ingestion Better prognosis than other forms of SCC → early presentation; less lymphatic/direct spread; better response to surgery/RT in view of scar tissue	
Examination	**Corrosive Type, Time, Together with food/other drugs, tongue burn** **Oropharynx:** edema, erosions, burn **Neck, chest, abdomen:** respiratory distress, perforation	
Labs	High TC/CRP/lactic acidosis/creat → poor prognosis	
Imaging	**CXR:** perforation, foreign body,pneumonia CT - depth of necrosis; optional	
Management	Respiratory- ? intubation; airway support; supplemental oxygen Fluids - NPO No Ryles tube- Can cause retching → worsens injuries Pain control IV PPI- prevents stress ulcers Broad spectrum Antibiotics - ? perforation **Emetics, neutralising agents, NG tube- contraindicated** Steroids- ? role Emergency surgery- in perforation; resect necrotic tissues; Feeding Jejunostomy	
Endoscopy after ingestion	**< 24 Hr → extent of gastro-esophageal injury;** Endoscopic vaccum therapy with sponges - can maintain patency; > 48hr - endoscopic grading not correct due to submucosal edema	

ZARGAR et al. GASTROINTESTINAL ENDOSCOPY 1991

ZARGAR	DESCRIPTION	MANAGEMENT	SEQUALAE
GRADE 0 No	**No** Visible Damage	Pain control Liquid diet → regular diet in 48 hours	NO SEQUALAE
GRADE 1 **Edema**	**EDEMA**, HYPEREMIA		
GRADE 2A **Ulcers**	TRANSMURAL INJURY, SUPERFICIAL / **Focal ULCERS**		
GRADE 2B **Ulcers**	CIRCUMFERENTIAL INJURY, DEEP/ **Extensive ULCERS**	Monitor for 1 week Watch for perforation signs; if + needs CT Swallowing saliva → liquids; Not tolerate → NG/NJ tube TPN/FJ	Stricture in 70-100% of cases
GRADE 3A **Necrosis**	**Focal Necrosis AND** ESCHAR		
GRADE 3B **Necrosis**	**Extensive Necrosis**		
GRADE 4 **Perf**	**Perforation**	Surgery	

Stricture:

Endoscopy: 3-6 weeks later for fibrosis to set in; Perforation rate is higher and success rate for dilation is lower than other strictures; Multiple sessions needed.

No preventive measures; ? low rate in people received steroids;

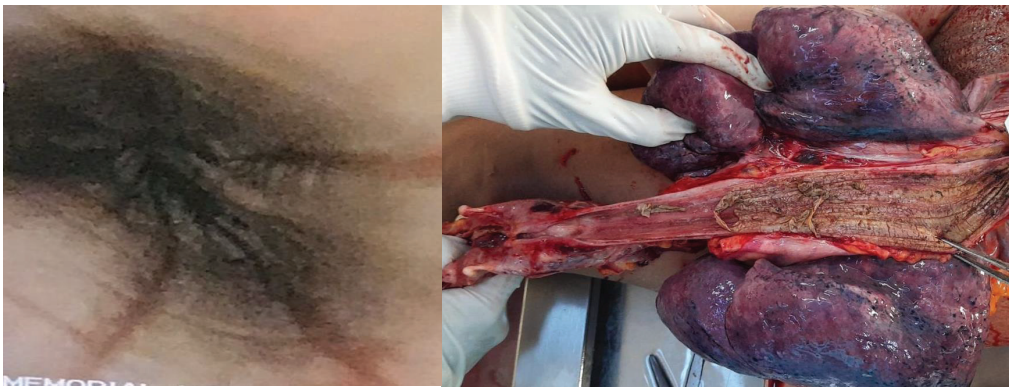
Reconstructive surgery after 6 months; Gastric pull up- transhiatal esophagectomy with cervical anastomosis

If stomach is involved- colonic transposition; either right or left colon can be used; retrosternal esophagocoloplasty

Native esophagus → risk of cancer and mucocele of retained esophagus

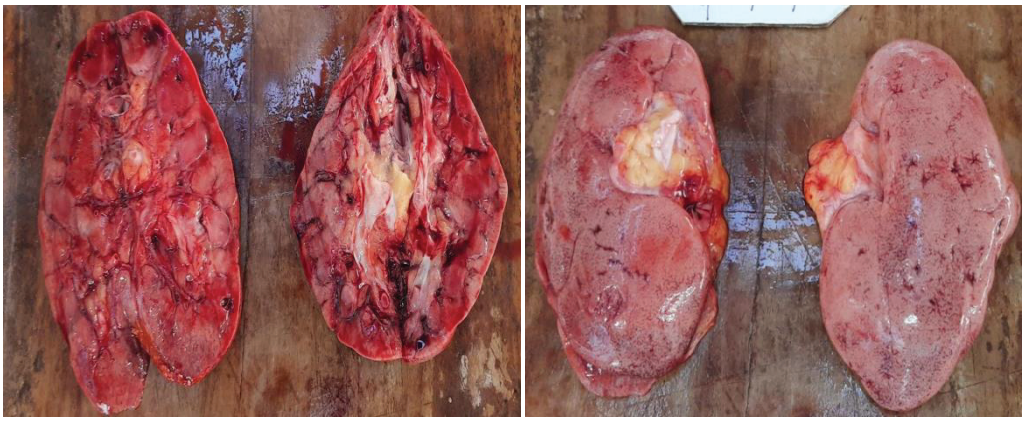
Endoscopic surveillance every two to three years beginning 10 to 20 years after the caustic ingestion due to the increased risk of esophageal cancer (**ASGE**)

Esophagus: Endoscopy and autopsy:



Esophagus: Endoscopy and autopsy:

Kidneys: Gross and Cut: Autopsy



CD Vs UC

Crohn's disease	Ulcerative colitis
NOD2/CARD15: chromosome 16 Chromosome 8, 13, 21	Chromosome 11, 20
Female: Male = 1.3:1 but not in children	1:1 ; slight male predominance
Later age of onset	Earlier (2nd - 3rd decade)
Second peak (@60-70 yrs) more pronounced	Second peak less pronounced
Prior misdiagnosis of IBS is common	Rare
Prodromal period seen	Not seen
Diagnosis delayed	Earlier diagnosis
Fecal occult blood in 50%	Frank blood is common
Pain more common (MC symptom diarrhoea)	Less common than in CD
Constitutional symptoms present	Absent
Tenismus less common if associated with colitis (because of rectal sparing)	More common
Right sided disease	Left sided
SI disease present	Absent
Fistulization seen (except ?)	Fistulisation not seen
Major perianal granulomas	?
Panmural disease	Not
ASCA ALCA/ACCA	pANCA in 40-70%
NOD-2 & ATLG-1 present	Absent
Predominant IgG2	IgG1 > IgG3
TH1 response	TH2 response
Pseudopolyps rarely seen	Characteristic
TPN and bowel rest help in management	Of no help
Pseudopolyps less common	More common
Malignancy is less common	Malignancy more common
Smoking is a risk factor	Smoking & appendectomy is protective

Celiac Vs Tropical Vs Whipples

FEATURE	CELIAC DISEASE	TROPICAL SPRUE	WHIPPLE'S DISEASE
Age	Infants – 3 months, **around weaning** **Mean age of presentation now is 45 yrs** 25% diagnosed in age > 60 yrs **Prevalence 1%**	**Adults (typical)** Children (sometimes, no epidemic form in children)	**Not** found in children **White adults (55 yrs)** Very rare disease, 2000 cases Wrongly named intestinal lipodystrophy
Causative factors	**Barley, Rye, Oat, Wheat** CMI and ABMI both play important role (Gluten = prolamine + glutenin-insoluble) 31-49 aa of α-gliadine (QQQPF) High glutamine & prolamine content in gliadine tTG deamidates glutamine into –ve epitopes **HLA-DQ2 (α1*02 <α1*05 + β1*02) > DQ8** Gliadine's glutamine → -- ve glutamic acid **IL-4, IL-15 & IFN gamma** play important role IL15 is a mediator, regulates IEL homeostasis IL15 triggers adaptive immune response in LP No peripheral lmphadenopathy **Associated with Down's syndrome** **Gamma-delta lymphocytes # increased**	Post infection Malabsorption • EPEC • Giardia • Cyclospora Bacterial and mycotoxins involved **Damage to stem cells** Exaggeration of ILEAL BREAK **SI transit time is increased** SIBO (E.coli, klebsiella) Increased IEL (nonspecific) **Functional** pancreatic insufficiency But stool contains FFA	**T. whippeli (fastigious); doubles in 1-4 day** Gram positive bacilli (actinomycetes) Commensal bacteria,only humans Relatively amall genome = 9,26,000 bp 0.25 by 2.5 µm (electron dense outer layer) Variation is due to WiSP (VNTR sequences) Slightly more common in farm workers -10% **HLA DRB1*13 & DQB1*06** **Reduced CD4/CD8 ratio in LP & circulation** Reduced CMI & ABMI, CD11b, IL-12 (But ↑ IgG response in asympto. carriers) Increased IL-16 & nucleosomes → apoptosis TH1 → TH2 **Defective monocyte / macrophage function** **Defective chemotaxis of cells** **M2 / alternative activation phenotype** **Intracellular glycoprotein deposits**
Sex	F > M, slight (except in DH)	No sex predliction	M > F ; 3:1
Symptoms	Most are asymptomatic Diarrhea, steatorrhea (in extensive disease), Vague Abdominal pain/ Discomfort, Bloating (severe nausea,vomiting and pain not seen)	Borborygmi, Sore Tongue, Leukonychia, Aguesia Nocturia !!!	**Arthralgia usually precede abdo symptom** **Abdominal pain is the dominant symptom** **Low grade fever, wt loss, diarrhea Giardia infection seen in 10%**
Diarrhoea	Episodic **(nocturnal, early morning, postprandial)**	Cronic watery/rare bloody diarrhoea **Steatorrhoea in 90%** **Stool fat is largly free fatty acid**	+ may be associated with occult beeding Sometimes gross GI bleding can occur
Fever	-	+ (in 25 %)	+ (chronic low grade intermittent fever)
Dementia	-	-	+ (CNS manifestations) – 10-40% of GI pts More common in refractory clinical relapse
CNS manifestations	Seizures **B/L cerebroparieto occipital calcification**		Oculomasticatory myorythmia Oculofacial skeletal myorhythmia
Clubbing	+ Similar to in UC	- (present in IPSID)	+