Mide Cerrahi Hastalıkları

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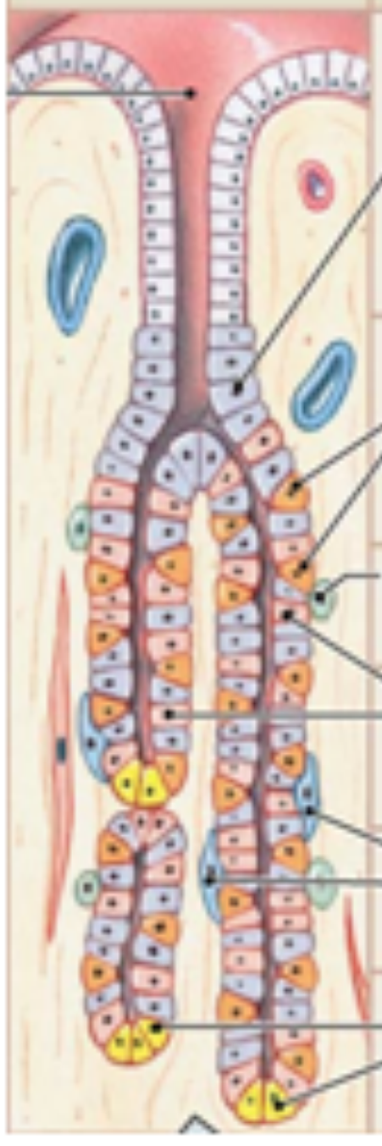
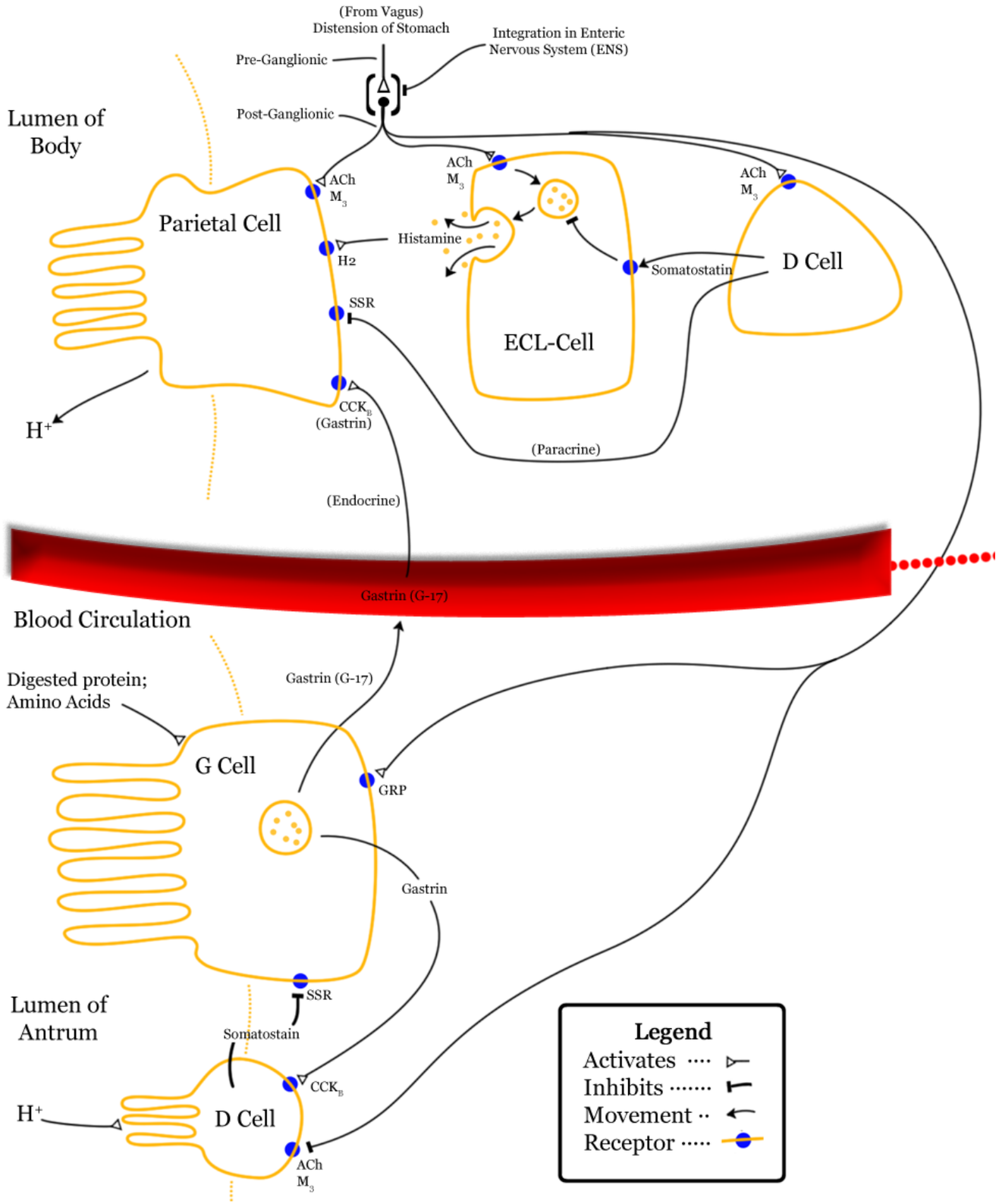
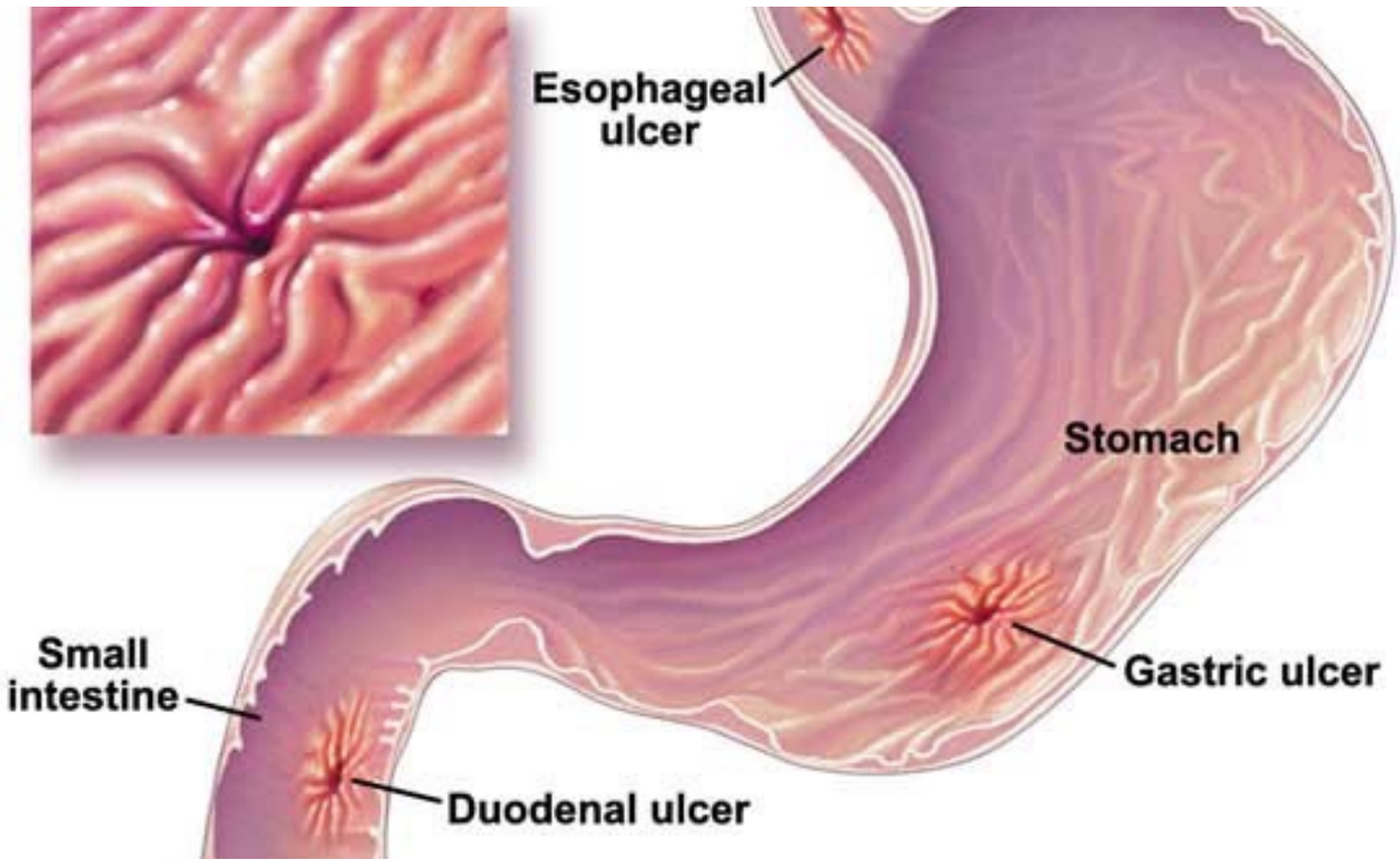
Historic milestones in gastric surgery

DATE	EVENT
350 B.C. – 201 A.D.	Existence of gastric ulceration was acknowledged by Diocles of Carystos (350 B.C.), Celsus, and Galen (131–201 A.D.).
1363	Guy de Chauliac describes closure of gastric wound.
1586	Marcellus Donatus of Mantua describes gastric ulcer at autopsy.
1600–1700	Reports of surgeons cutting stomach to remove foreign bodies.
1688	Muralto describes duodenal ulcer at autopsy.
1737	Morgagni describes both gastric and duodenal ulcer at autopsy.
1833	William Beaumont reports data recorded during his care of Alexis St. Martin who developed a gastric fistula from a left upper quadrant musket wound.
1869	Maury reportedly performs feeding gastrostomy to palliate esophageal stricture following consultation with Samuel D. Gross.
1875	Sidney Jones in London publishes the first successful gastrostomy for feeding.
1879	Paen performed distal gastrectomy and gastroduodenostomy. The patient died 5 d later.
1880	Rydygier resected a distal gastric cancer, and the patient died 12 h later.
1880	Billroth resects distal gastric cancer and performs gastroduodenostomy (Billroth I). Patient Therese Heller recovers and survives 4 mo.
1881	Anton Wolfler performs loop gastrojejunostomy to palliate an obstructing distal gastric cancer.
1884	Rydygier reports an unsuccessful gastrojejunostomy for benign gastric outlet obstruction.
1885	Billroth performs a successful distal gastrectomy and gastrojejunostomy (Billroth II) for gastric cancer.
1886	Heineke performs pyloroplasty.
1888	Mikulicz performs similar operation.
1892	Jaboulay describes bypassing the intact pylorus with gastroduodenostomy.
1902	Finney from Baltimore describes pyloroplasty technique.
1891–1913	Different techniques of gastrostomy are described by Witzel (1891), Stamm (1894), and Janeway (1913).
1920–1950	Subtotal gastrectomy grows popular as an operation for peptic ulcer. Von Haberer and Finsterer proponents.
1943	Dragstedt and Owen describe transthoracic truncal vagotomy to treat peptic ulcer disease. By the early 1950s, it is well recognized that some patients developed gastric stasis after this procedure, and transabdominal truncal vagotomy and drainage (pyloroplasty or gastrojejunostomy) become a standard ulcer operation.
1952	Farmer and Smithwick describe good results with truncal vagotomy and hemigastrectomy for peptic ulcer.
1953	Edwards and Herrington (Nashville) describe truncal vagotomy and antrectomy for peptic ulcer.
1955	Zollinger and Ellison describe the eponymous syndrome.
1957	Griffith and Harkins (Seattle) describe parietal cell vagotomy (highly selective vagotomy) for the elective treatment of peptic ulcer disease.
1980–2000	Japanese surgeons and other surgical groups from East Asia demonstrate that more aggressive lymphadenectomy may improve survival in patients with gastric cancer.
1990–current	Evolving role of laparoscopic techniques in the treatment of surgical gastric disease.
1995–current	Dramatic increase in bariatric operations.
2000–current	Development of natural orifice translumenal endoscopic surgery.

GASTRIC MUCOSA	CELL TYPES	SUBSTANCE SECRETED	STIMULUS FOR RELEASE	FUNCTION OF SECRETION
	Mucous neck cell	Mucus	Tonic secretion; with irritation of mucosa	Physical barrier between lumen and epithelium
		Bicarbonate	Secreted with mucus	Buffers gastric acid to prevent damage to epithelium
	Parietal cells	Gastric acid (HCl)	Acetylcholine, gastrin, histamine	Activates pepsin; kills bacteria
		Intrinsic factor		Complexes with vitamin B_{12} to permit absorption
	Enterochromaffin-like cell	Histamine	Acetylcholine, gastrin	Stimulates gastric acid secretion
	Chief cells	Pepsin(ogen)	Acetylcholine, acid secretion	Digests proteins
		Gastric lipase		Digests fats
	D cells	Somatostatin	Acid in the stomach	Inhibits gastric acid secretion
	G cells	Gastrin	Acetylcholine, peptides, and amino acids	Stimulates gastric acid secretion

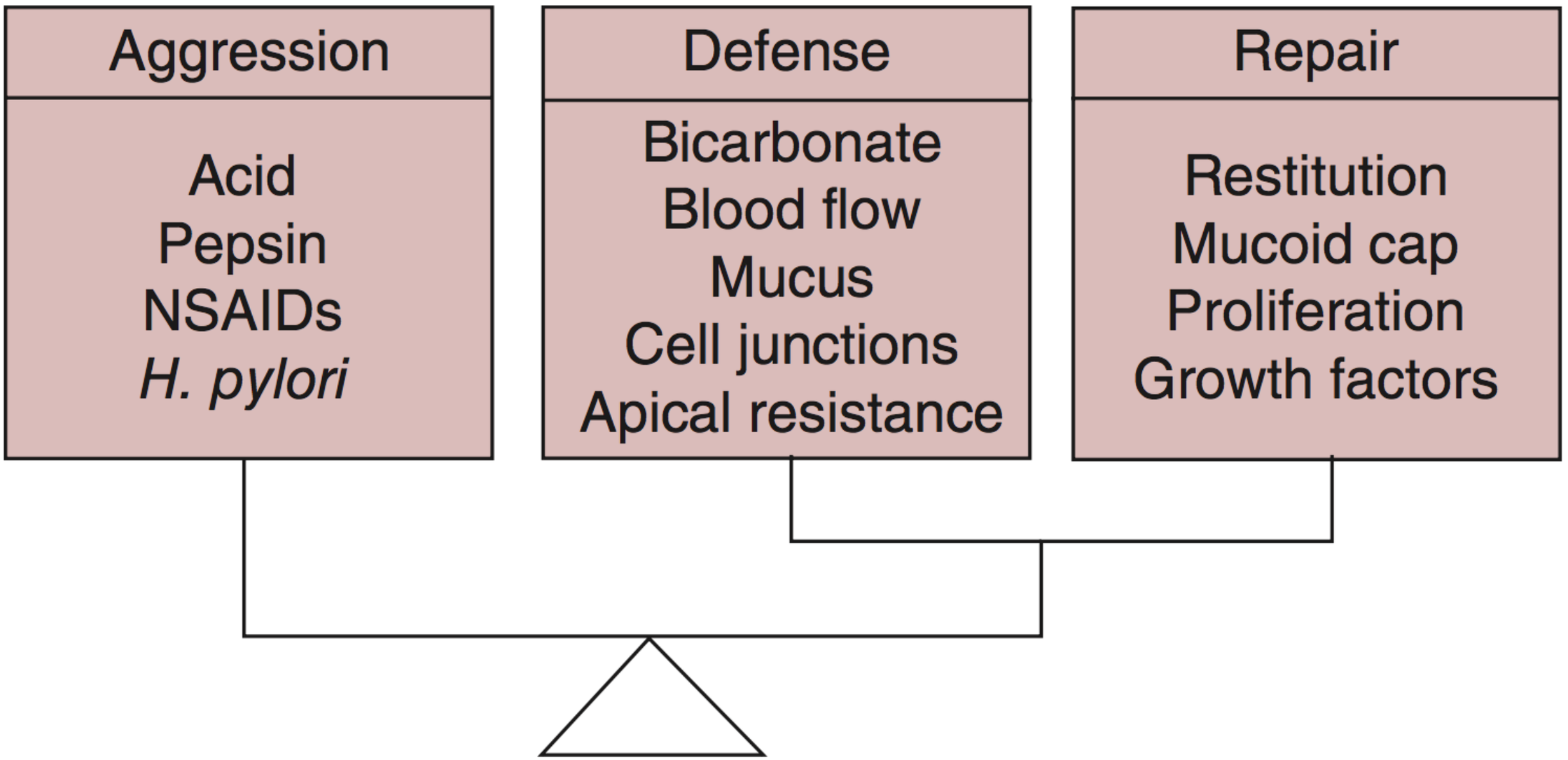

(From Vagus)
Distension of Stomach
Integration in Enteric
Nervous System (ENS)
Pre-Ganglionic
Post-Ganglionic
Lumen of
Body
ACh
M3
Parietal Cell
Histamine
H2
SSR
H+
CCK2
(Gastrin)
ACh
M3
ECL-Cell
Somatostatin
ACh
M3
D Cell
(Paracrine)
(Endocrine)
Gastrin (G-17)
Blood Circulation
Digested protein;
Amino Acids
Gastrin (G-17)
G Cell
GRP
Gastrin
SSR
Lumen of
Antrum
Somatostain
CCK2
H+
D Cell
ACh
M3
Legend
Activates
Inhibits
Movement
Receptor

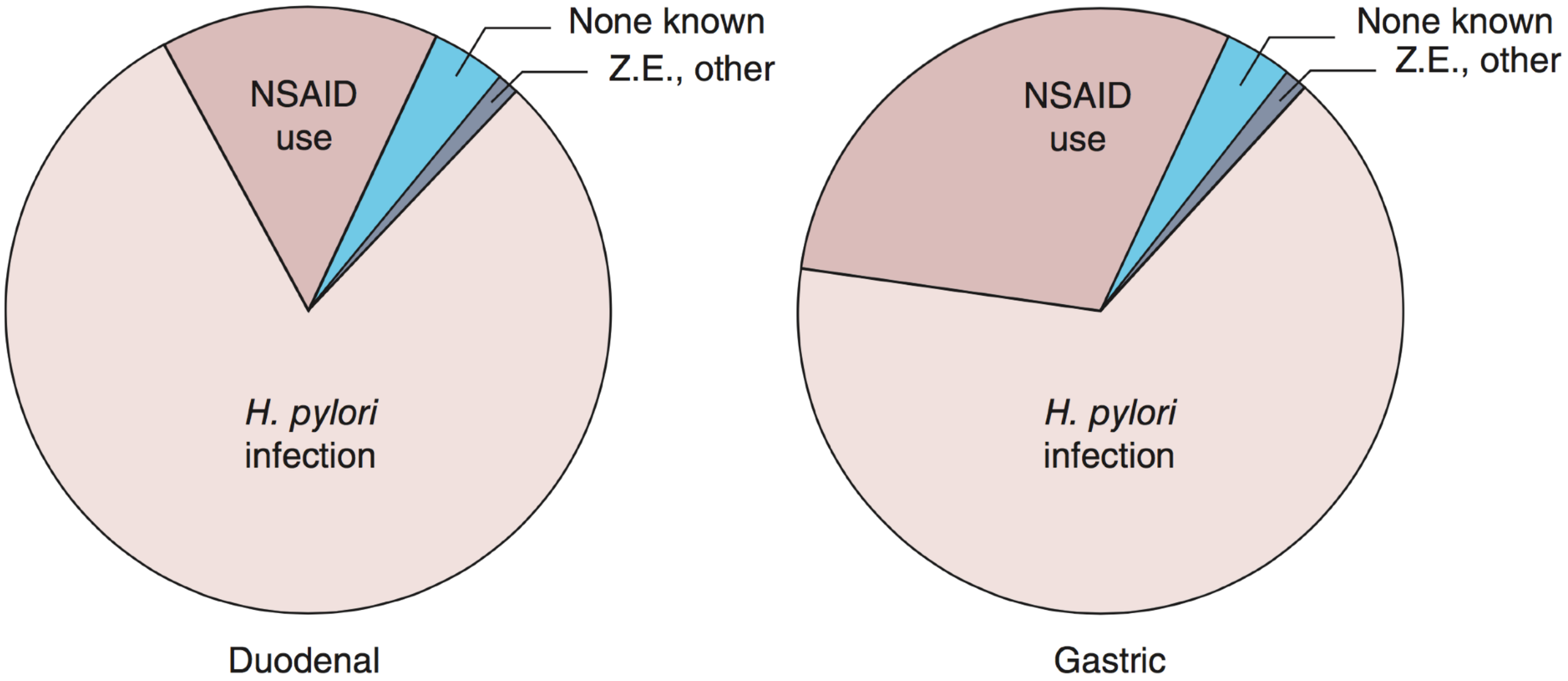
Peptik ülser hastalığı



- peptik aktiviteye bağlı ortaya çıkan ülser
- mide-özofagus-duodenum-jejenum-meckel

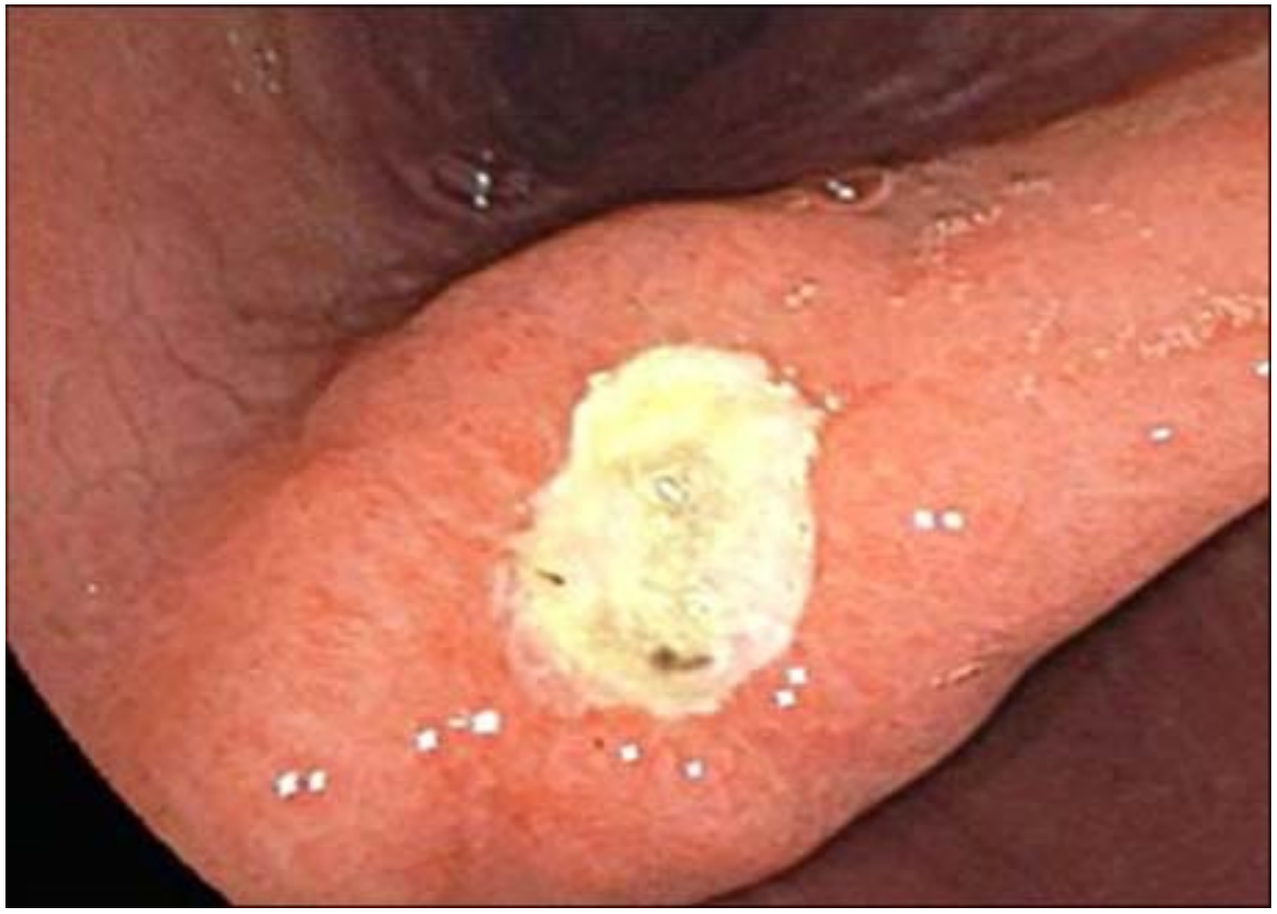
- “no acid, no ulcer”
- mide ülseri; savunma azalır
- duodenal ülser; saldırı artar

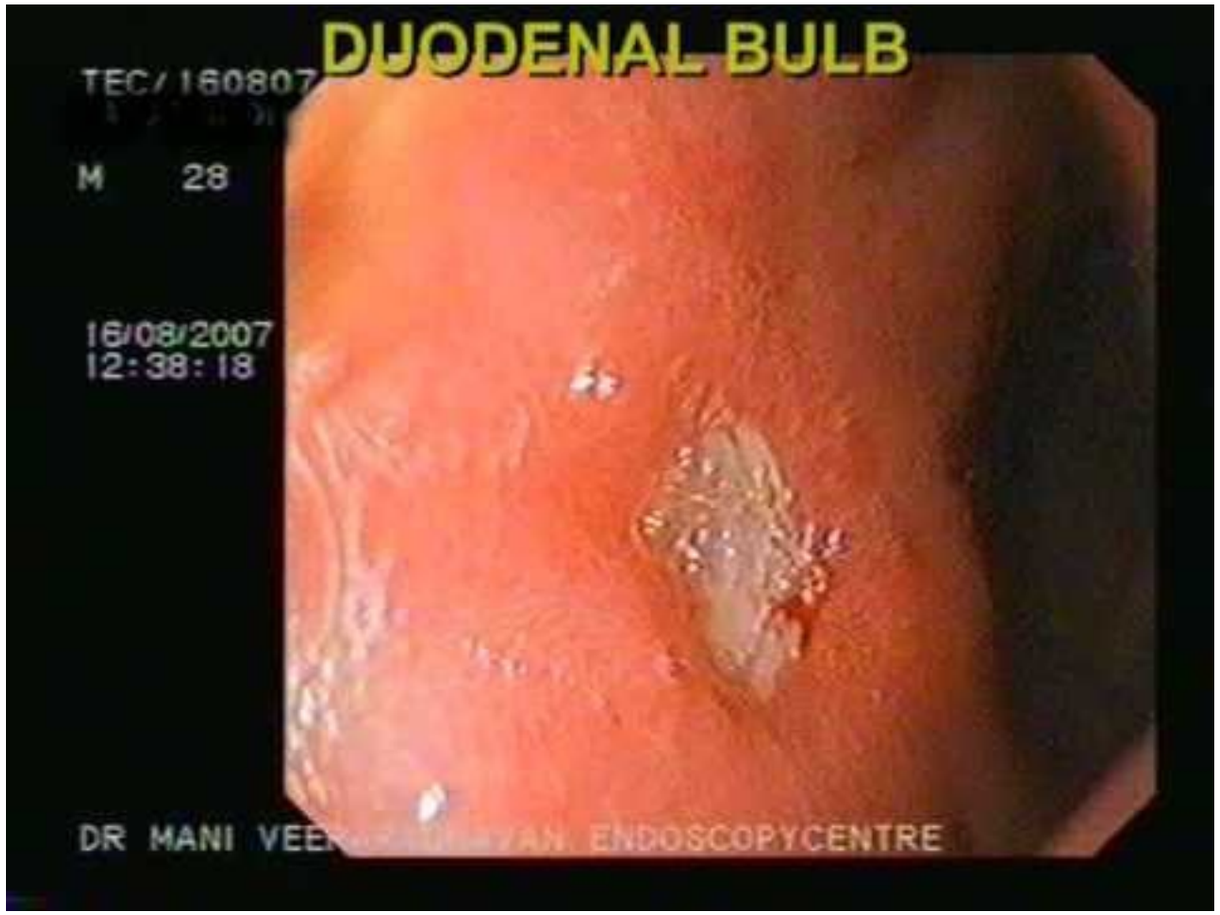

Aggression
Acid
Pepsin
NSAIDs
H. pylori
Defense
Bicarbonate
Blood flow
Mucus
Cell junctions
Apical resistance
Repair
Restitution
Mucoid cap
Proliferation
Growth factors

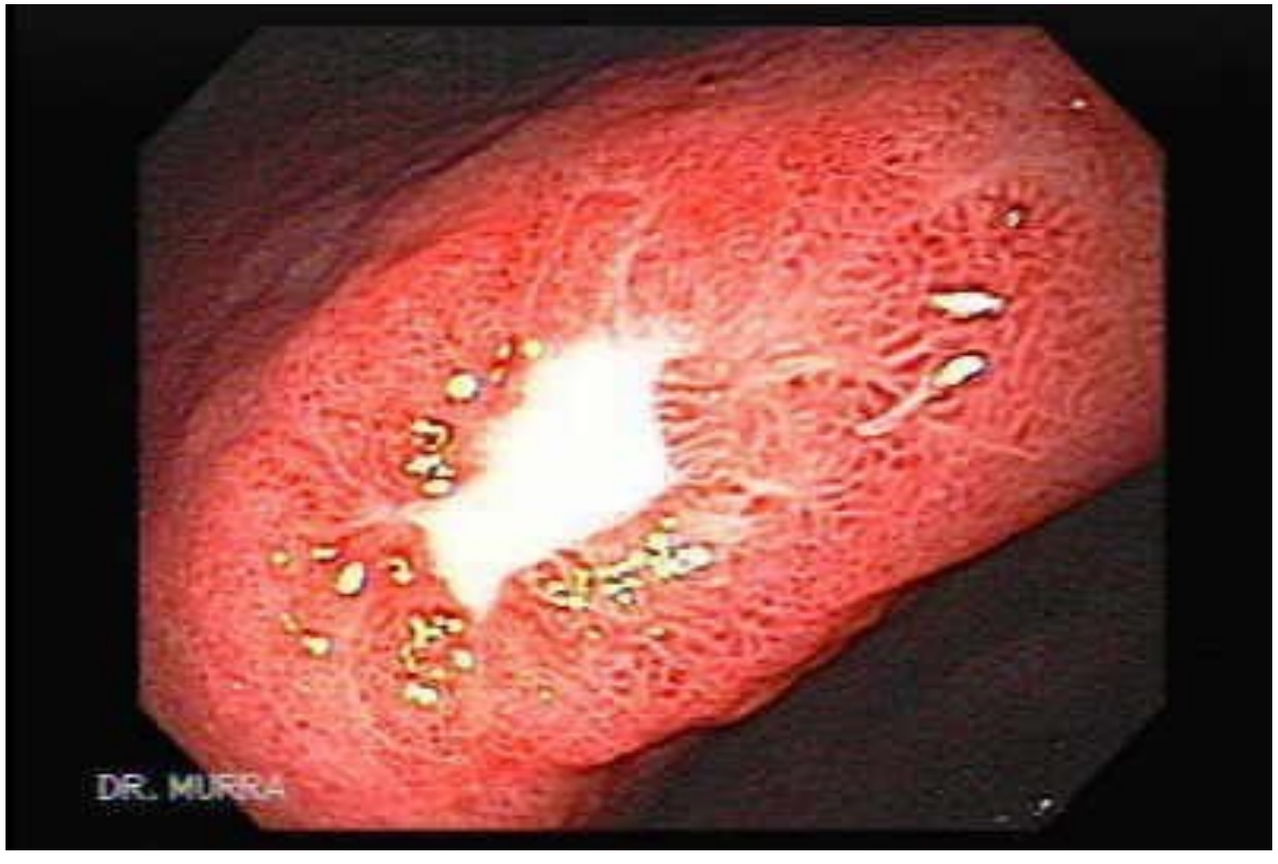

None known
Z.E., other
NSAID
use
H. pylori
infection
Duodenal
None known
Z.E., other
NSAID
use
H. pylori
infection
Gastric

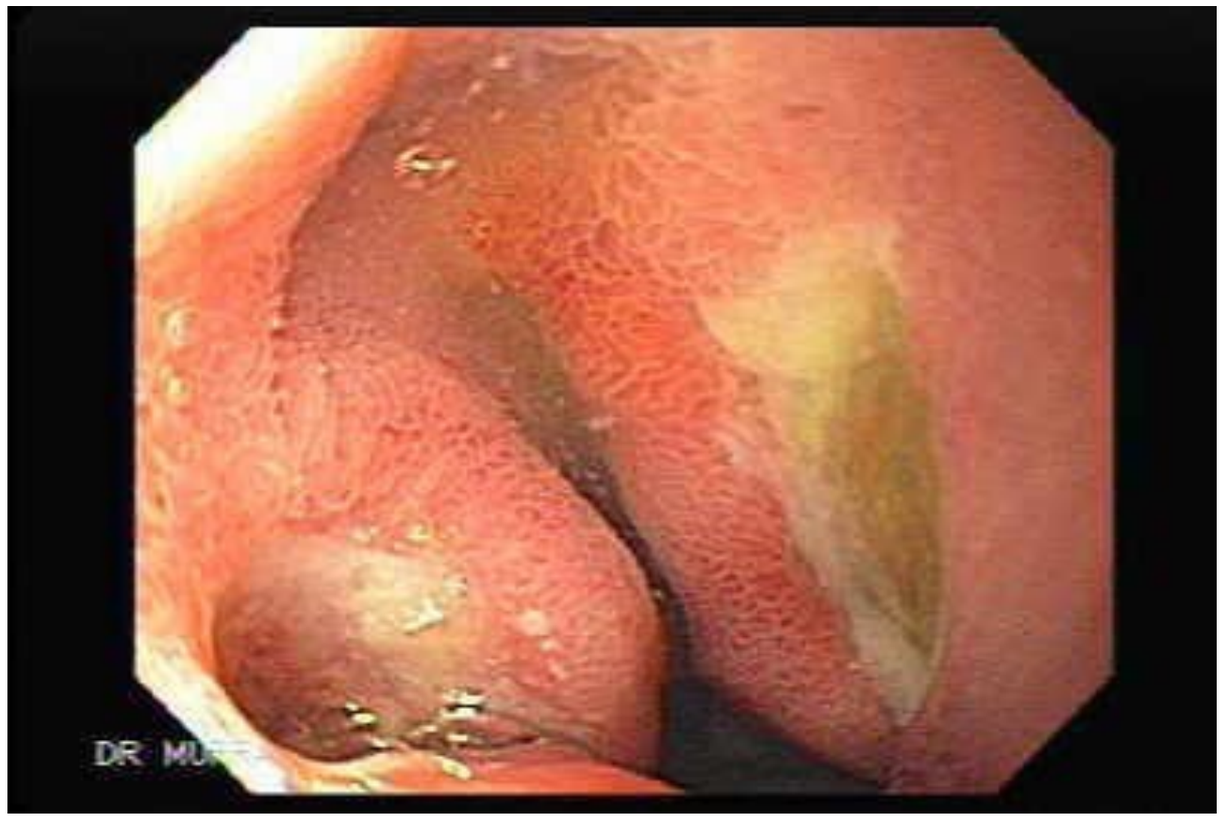
Tanı:

- Klinik:
  - epigastrik ağrı
  - komplikasyonlarına bağlı semptomlar
- Görüntüleme:
  - ÖMD
  - Endoskopi*




DUODENAL BULB
TEC/ 160807
M 28
16/08/2007
12:38:18
DR MANI VEE
ENDOSCOPYCENTRE


DR. MURRA


DR MU

Tedavi: Medikal

- H.pylori eradikasyonu/predispozan ilaç stoplanması
- Gastrik asit nötralizasyonu
  - antasit
- Gastrik asit salınımının inhibisyonu
  - H2RB
  - PPI
- Mukozal savunmanın güçlendirilmesi
  - Sukralfat
  - Bizmut
  - PG

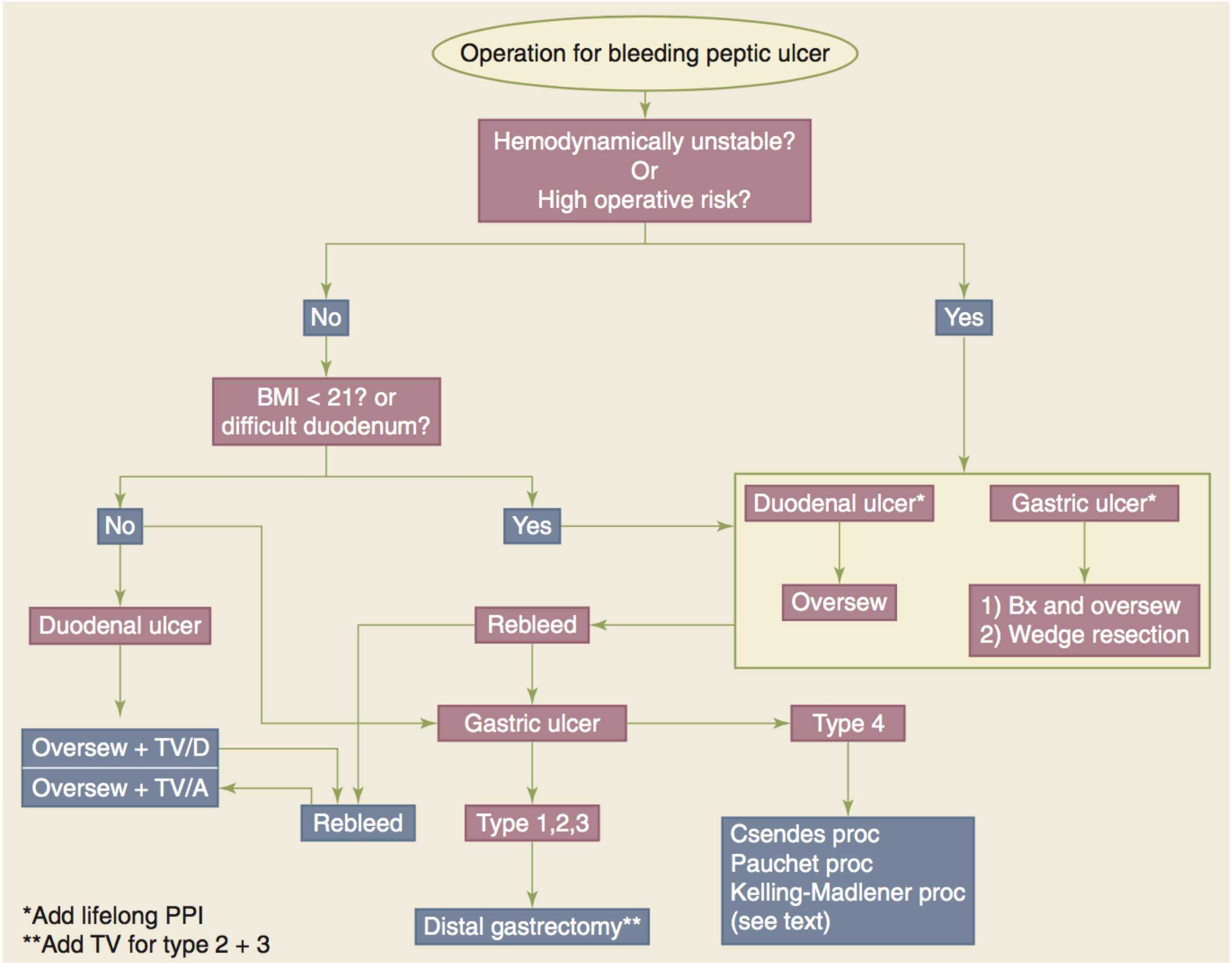
Cerrahi tedavi?
—ne zaman—

- Komplikasyon geliştiğinde
  - Kanama
  - Perforasyon
  - Obstruksiyon
- Intractibilite

General management of patients with complicated peptic ulcer disease

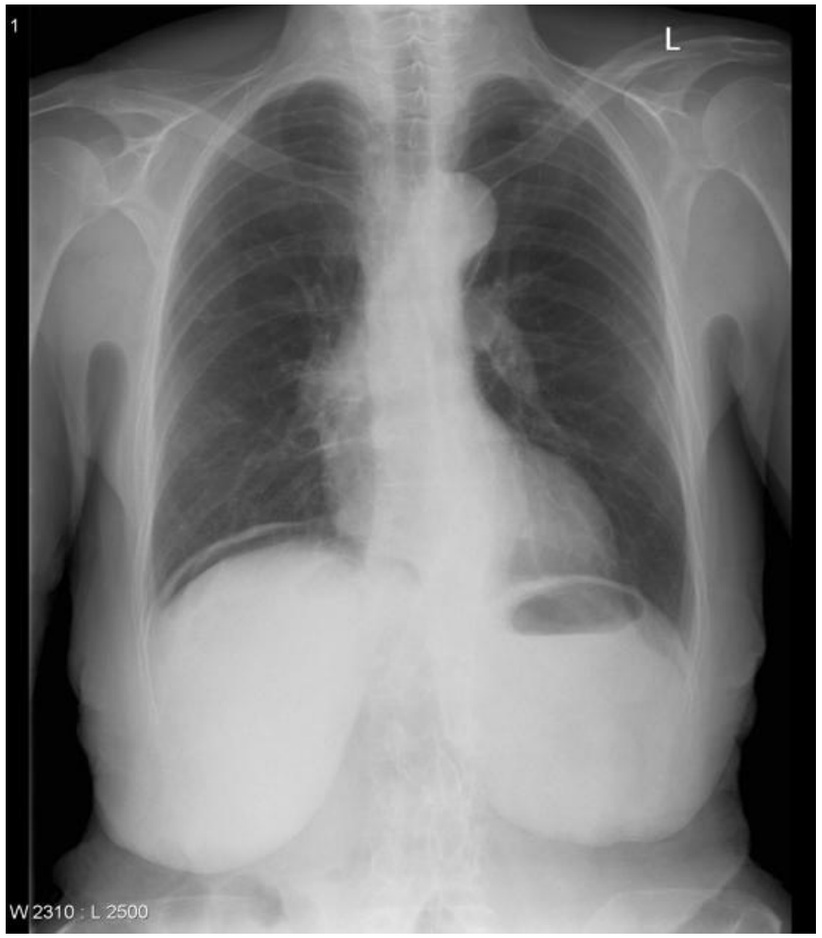
Initial management
1. Careful monitoring, preferably in an intensive care unit.
2. Fluid resuscitation, plus blood transfusions, as needed.
3. Give intravenous, full dose PPI (eg, 180 mg bolus followed by 8 mg/hour pantoprazole or esomeprazole).
4. Endoscopy for possible therapeutic intervention: • within 24 hours in most cases • as soon as patient is stable in severe hemorrhage
Once patient is tolerating oral intake
1. Switch to full dose oral PPI twice daily.
2. Treat for *H. pylori* if tests are positive*.
3. Ensure non-aspirin NSAIDs are stopped, if at all possible.
4. Stop aspirin, including low-dose aspirin, if cardiovascular status permits.
5. For gastric ulcers, at 8 to 12 weeks, perform upper endoscopy to evaluate healing and exclude neoplasia (unless there is confidence that the stomach and duodenum were adequately evaluated endoscopically or surgically during hospitalization). Giant duodenal ulcers also warrant follow-up endoscopy to ensure healing and exclude rare malignancy or other etiologies.
6. If initial tests for *H. pylori* are negative, at roughly four weeks (or later for large ulcers):
a. Switch to full dose H2 receptor antagonists and continue for two weeks
b. If an upper endoscopy will be performed, biopsy for *H. pylori* urease testing and histology
c. If upper endoscopy will not be performed, obtain an *H. pylori* breath test or stool antigen test

PPI: proton pump inhibitor; *H. pylori*: *Helicobacter pylori*; NSAID: nonsteroidal anti-inflammatory drug.
* With an established ulcer, *H. pylori* is only ruled out when two tests obtained while the patient is off of antibiotics, PPIs, and bismuth are negative.

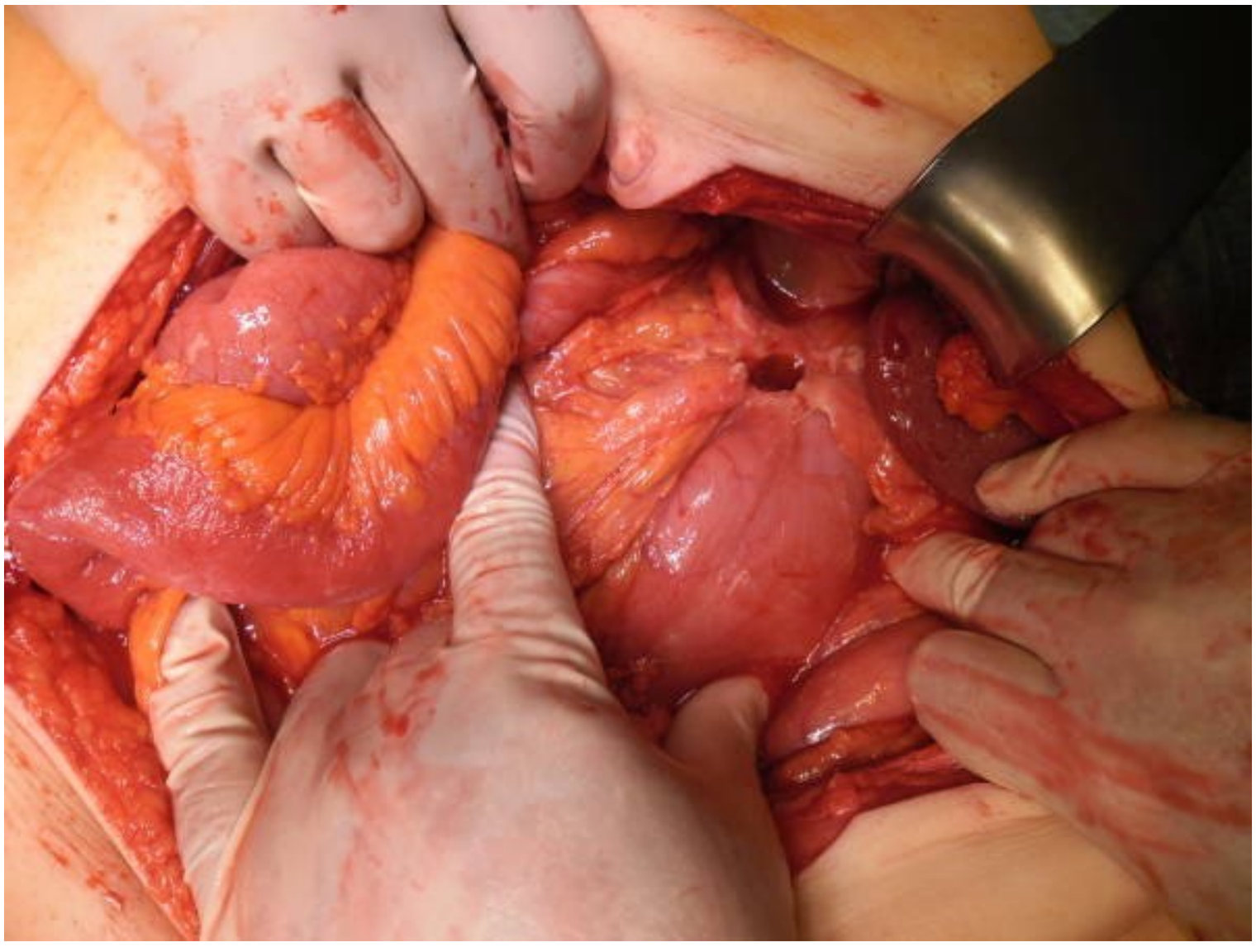

Operation for bleeding peptic ulcer
Hemodynamically unstable?
Or
High operative risk?
No
Yes
BMI < 21? or
difficult duodenum?
No
Yes
Duodenal ulcer*
Gastric ulcer*
Oversew
1) Bx and oversew
2) Wedge resection
Duodenal ulcer
Rebleed
Oversew + TV/D
Oversew + TV/A
Gastric ulcer
Type 4
Rebleed
Type 1,2,3
Csendes proc
Pauchet proc
Kelling-Madlener proc
(see text)
Distal gastrectomy**
*Add lifelong PPI
**Add TV for type 2 + 3

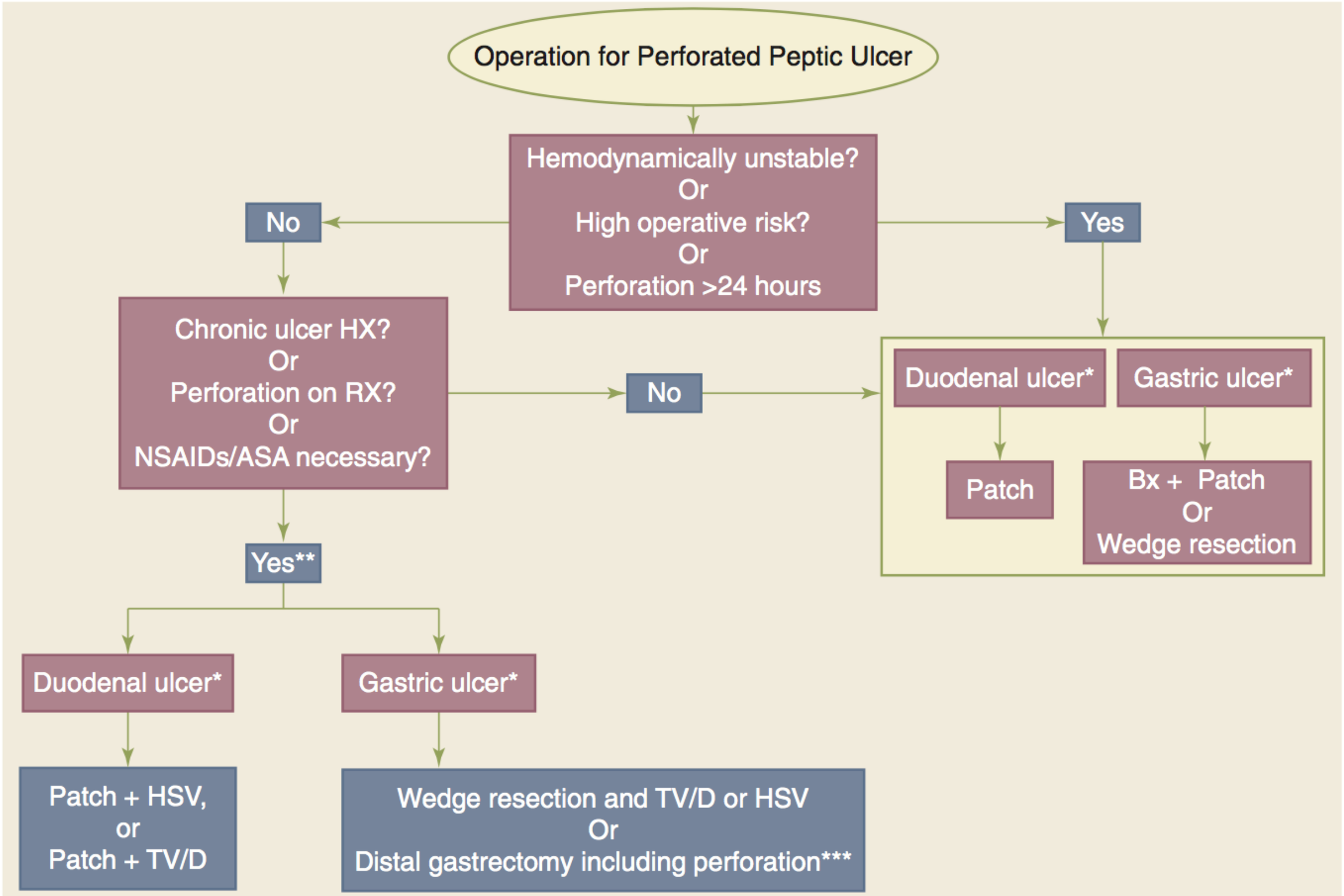
PUP klinik-tanı

- ani başlayan epigastrik-yaygın şiddetli ağrı
- tahta karın



tedavisi: Cerrahi
primer tamir+omentoplasti/omentopeksi (Graham)





* In all patients, test and treat for *H. pylori*, and if vagotomy not performed (most patients today) consider lifelong PPI.

** Avoid truncal vagotomy and avoid gastrectomy if BMI<21

*** Consider adding vagotomy for type II and type III gastric ulcer

Obstruksiyon

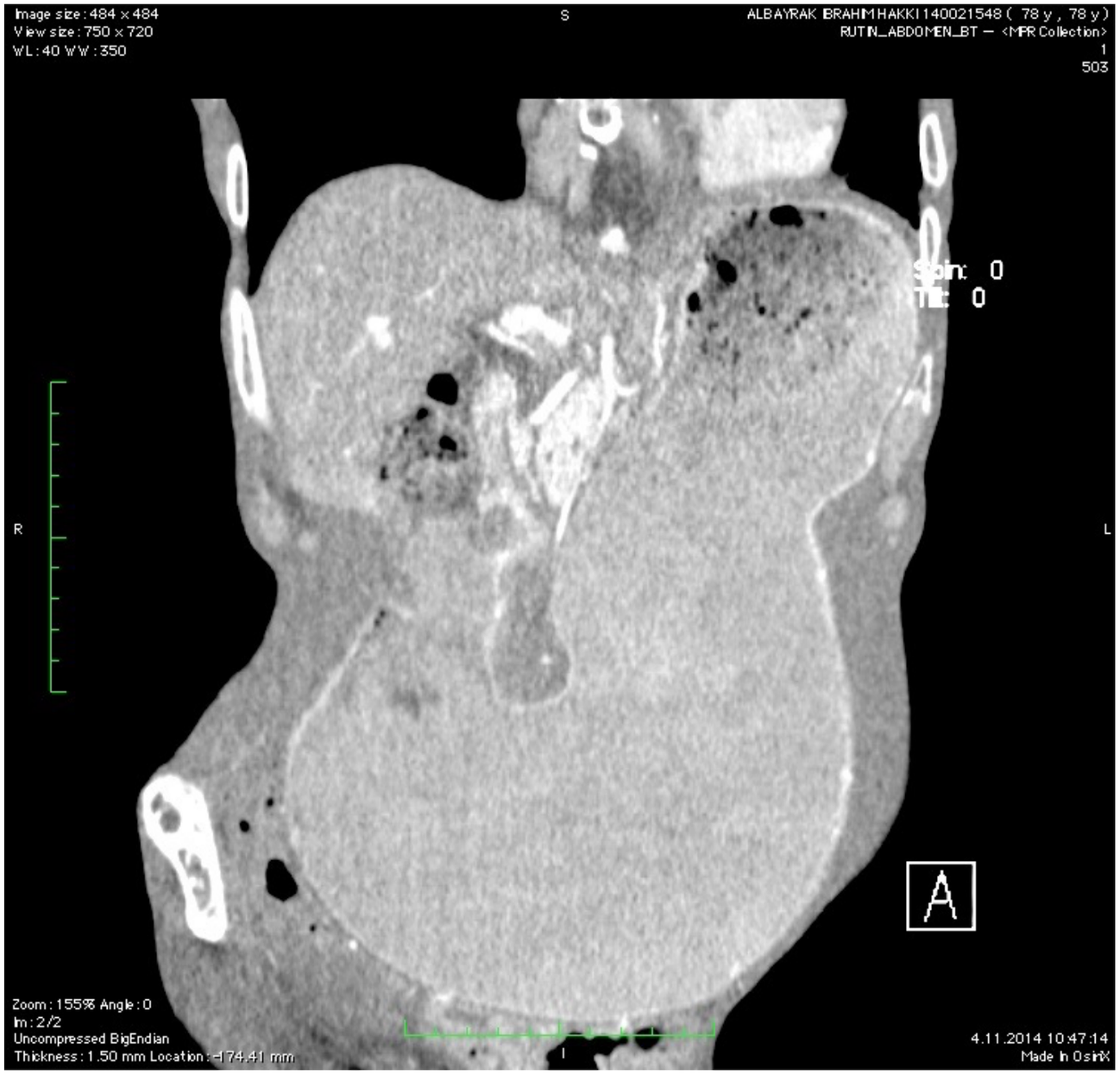


Table 26-13

Differential diagnosis of intractability or nonhealing peptic ulcer disease

Cancer
- Gastric
- Pancreatic
- Duodenal

Persistent *Helicobacter pylori* infection
- Tests may be false-negative
- Consider empiric treatment

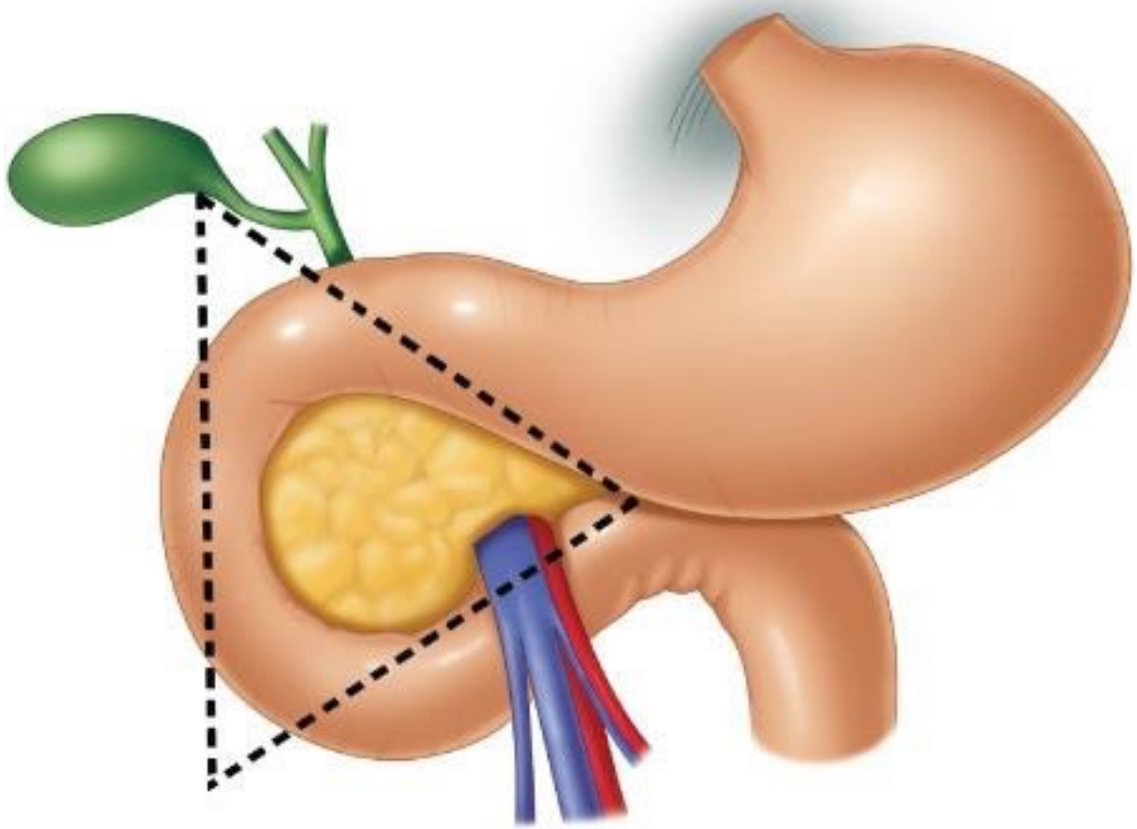
Noncompliant patient
- Failure to take prescribed medication
- Surreptitious use of NSAIDs

Motility disorder

Zollinger-Ellison syndrome

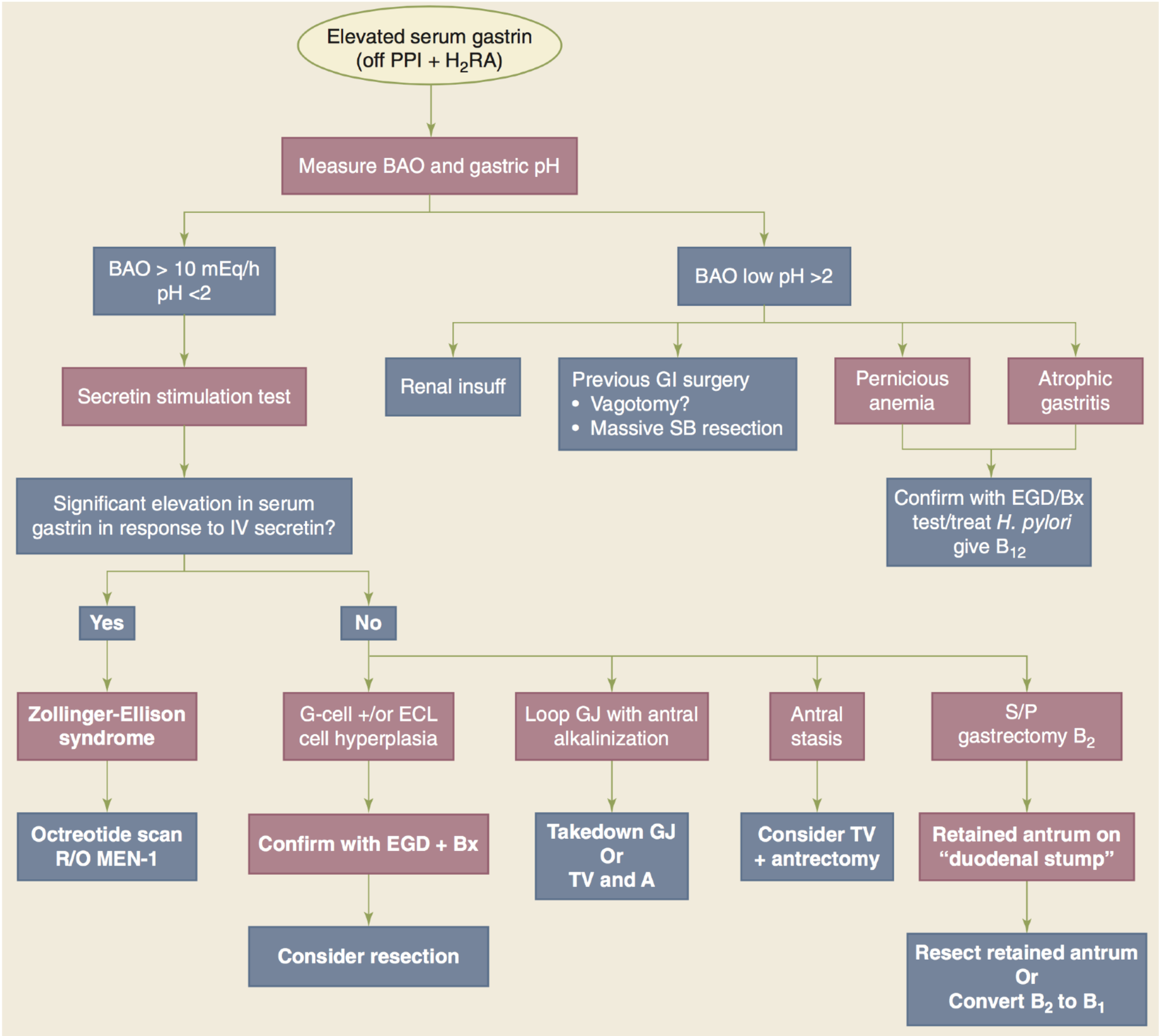
Zollinger-Ellison Sendromu (Gastrinoma)

- gastrin salgılayan tm (sporadik ya da MEN)
- duodenum,pankreas
- tedaviye dirençli ülser
- atipik yerleşimli ülser
- diare



- Bazal asit salınımı artmıştır.
- Tanı; serum gastrin düzeyi
- sekretin stimülasyon testi
- lokalizasyon; SS reseptör sintigrafisi

- Tedavi; cerrahi enükleasyon
- medikal-PPI

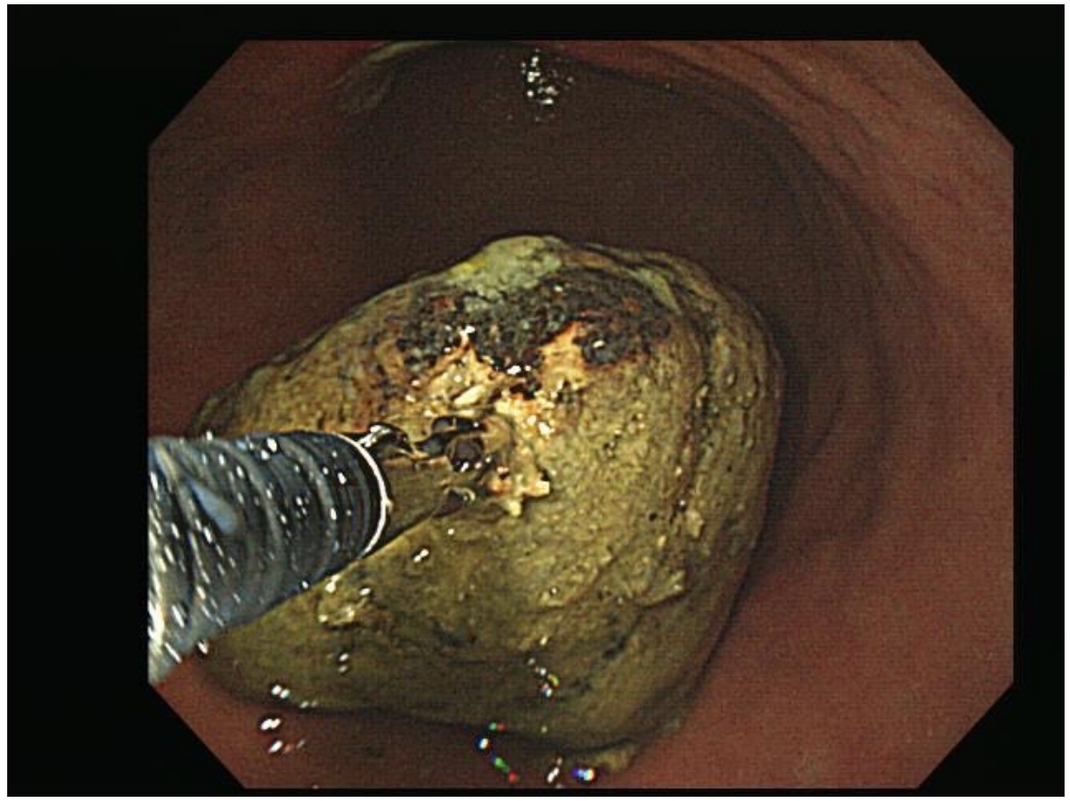

Elevated serum gastrin
(off PPI + H_2RA)
Measure BAO and gastric pH
BAO > 10 mEq/h
pH <2
BAO low pH >2
Secretin stimulation test
Renal insuff
Previous GI surgery
• Vagotomy?
• Massive SB resection
Pernicious anemia
Atrophic gastritis
Significant elevation in serum gastrin in response to IV secretin?
Confirm with EGD/Bx
test/treat H. pylori
give B_{12}
Yes
No
Zollinger-Ellison syndrome
G-cell +/or ECL cell hyperplasia
Loop GJ with antral alkalinization
Antral stasis
S/P gastrectomy B_2
Octreotide scan
R/O MEN-1
Confirm with EGD + Bx
Takedown GJ
Or
TV and A
Consider TV
+ antrectomy
Retained antrum on
"duodenal stump"
Consider resection
Resect retained antrum
Or
Convert B_2 to B_1

Bezoar

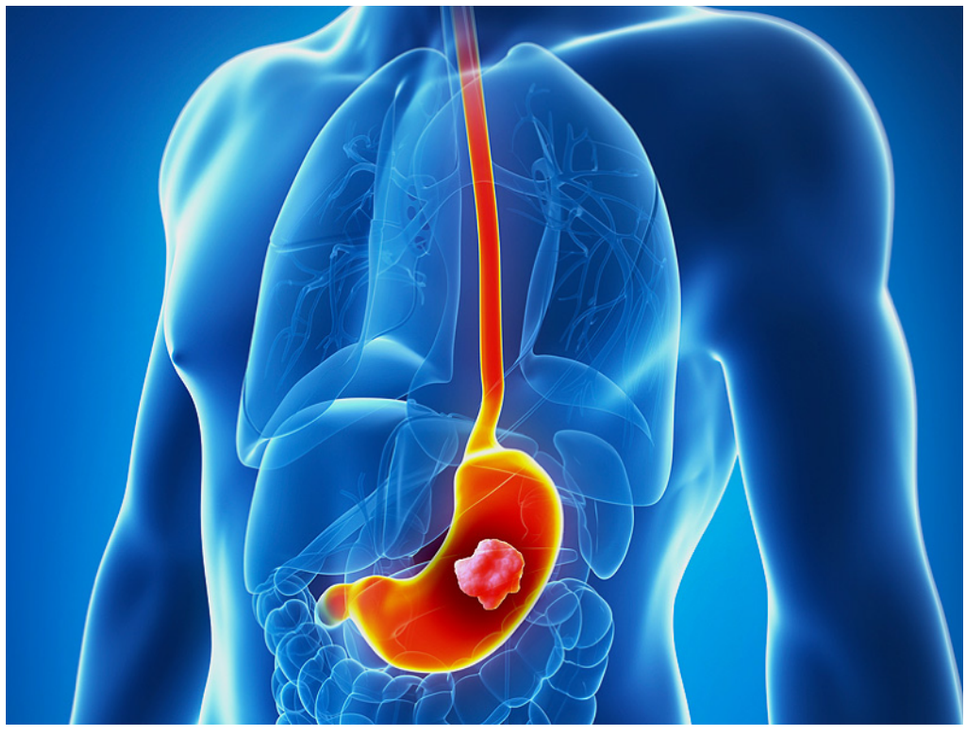
Classification of gastric bezoars

Phytobezoar
Persimmon (diospyrobezoar)
Psyllium
Fruit
Vegetables
Trichobezoar
Pharmacobezoar
Enteric coated aspirin
Extended release capsules (nifedipine, theophylline)
Sucralfate
Others
Other
Styrofoam
Fungi (candida)
Lactobezoar
Cement
Furniture polish
Shellac

UpToDate®

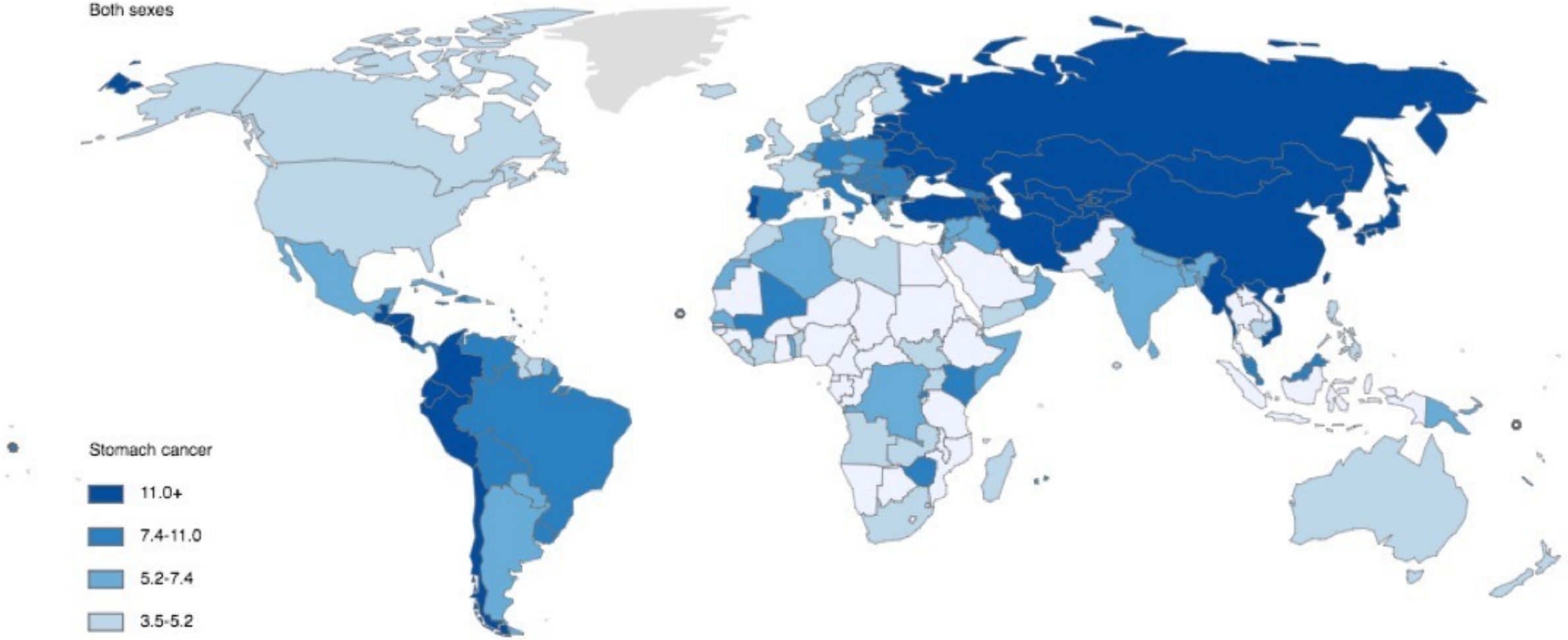


Mide Kanserleri



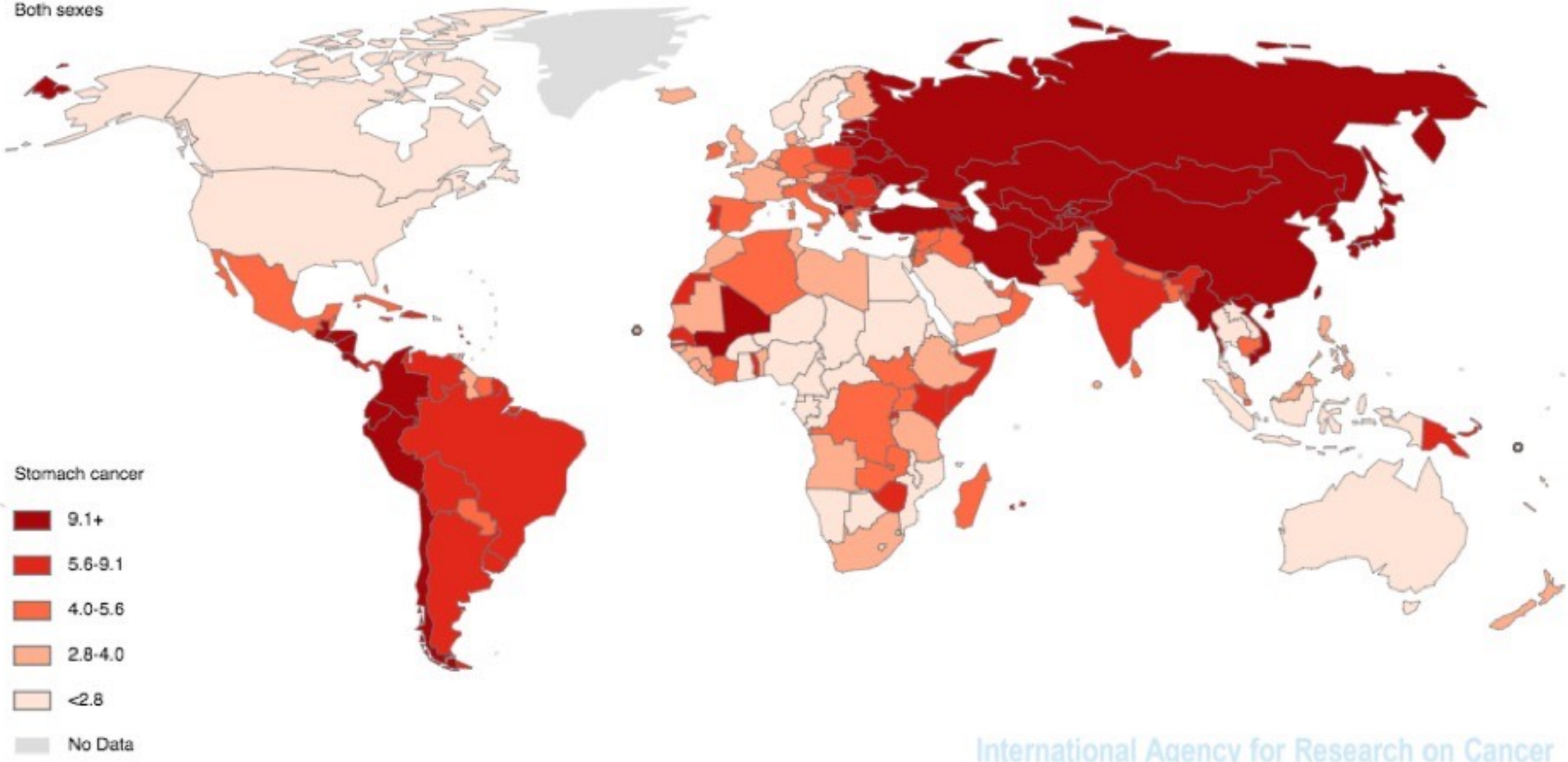
Incidence ASR

Both sexes



Mortality ASR

Both sexes



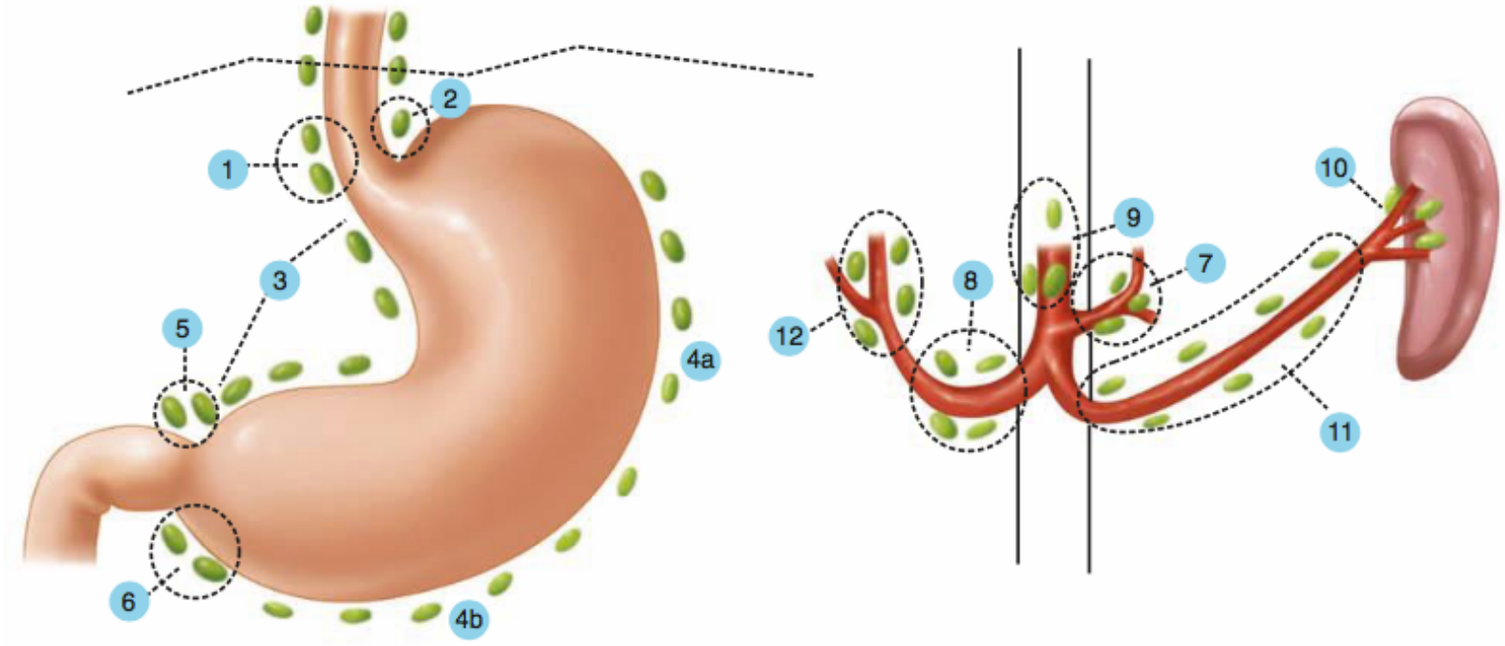


Table 5 Anatomical definitions of lymph node stations

No.	Definition
1	Right paracardial LNs, including those along the first branch of the ascending limb of the left gastric artery.
2	Left paracardial LNs including those along the esophagocardiac branch of the left subphrenic artery
3a	Lesser curvature LNs along the branches of the left gastric artery
3b	Lesser curvature LNs along the 2nd branch and distal part of the right gastric artery
4sa	Left greater curvature LNs along the short gastric arteries (perigastric area)
4sb	Left greater curvature LNs along the left gastroepiploic artery (perigastric area)
4d	Rt. greater curvature LNs along the 2nd branch and distal part of the right gastroepiploic artery
5	Suprapyloric LNs along the 1st branch and proximal part of the right gastric artery
6	Infrapyloric LNs along the first branch and proximal part of the right gastroepiploic artery down to the confluence of the right gastroepiploic vein and the anterior superior pancreatoduodenal vein
7	LNs along the trunk of left gastric artery between its root and the origin of its ascending branch
8a	Anterosuperior LNs along the common hepatic artery
8p	Posterior LNs along the common hepatic artery
9	Celiac artery LNs
10	Splenic hilar LNs including those adjacent to the splenic artery distal to the pancreatic tail, and those on the roots of the short gastric arteries and those along the left gastroepiploic artery proximal to its 1st gastric branch
11p	Proximal splenic artery LNs from its origin to halfway between its origin and the pancreatic tail end
11d	Distal splenic artery LNs from halfway between its origin and the pancreatic tail end to the end of the pancreatic tail
12a	Hepatoduodenal ligament LNs along the proper hepatic artery, in the caudal half between the confluence of the right and left hepatic ducts and the upper border of the pancreas
12b	Hepatoduodenal ligament LNs along the bile duct, in the caudal half between the confluence of the right and left hepatic ducts and the upper border of the pancreas
12p	Hepatoduodenal ligament LNs along the portal vein in the caudal half between the confluence of the right and left hepatic ducts and the upper border of the pancreas
13	LNs on the posterior surface of the pancreatic head cranial to the duodenal papilla
14v	LNs along the superior mesenteric vein
15	LNs along the middle colic vessels
16a1	Paraaortic LNs in the diaphragmatic aortic hiatus
16a2	Paraaortic LNs between the upper margin of the origin of the celiac artery and the lower border of the left renal vein
16b1	Paraaortic LNs between the lower border of the left renal vein and the upper border of the origin of the inferior mesenteric artery

- Malign mide tümörleri
  - Adenokarsinom (95%)
  - Lenfoma (4%)
  - GIST (1%)

Frequency of gastric tumors

TUMOR TYPE	NO. OF CASES	PERCENT
Malignant tumors	4199	93.0
Carcinoma	3970	87.9
Lymphoma	136	3.0
Leiomyosarcoma	77	1.7
Carcinoid	11	0.3
Others	5	0.1
Benign tumors	315	7.0
Polyp	140	3.1
Leiomyoma	92	2.0
Inflammatory lesions	30	0.7
Heterotopic pancreas	20	0.4
Others	33	0.8

Factors increasing or decreasing the risk of gastric cancer

Increase risk
- Family history
- Diet (high in nitrates, salt, fat)
- Familial polyposis
- Gastric adenomas
- Hereditary nonpolyposis colorectal cancer
- *Helicobacter pylori* infection
  - Atrophic gastritis, intestinal metaplasia, dysplasia
- Previous gastrectomy or gastrojejunostomy (>10 y ago)
- Tobacco use
- Ménétrier's disease

Decrease risk
- Aspirin
- Diet (high fresh fruit and vegetable intake)
- Vitamin C

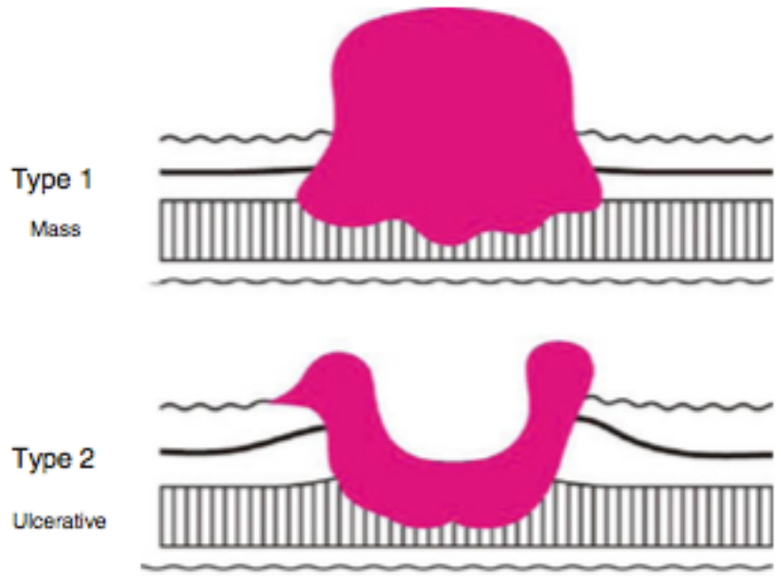
Genetic abnormalities in gastric cancer

ABNORMALITIES	GENE	APPROXIMATE FREQUENCY %
Deletion/suppression	*p53*	60–70
	FHIT	60
	APC	50
	DCC	50
	E-cadherin	<5
Amplification/ overexpression	*COX-2*	70
	HGF/SF	60
	VEGF	50
	c-met	45
	AIB-1	40
	β-*catenin*	25
	k-sam	20
	ras	10–15
	c-erb B-2	5–7
Microsatellite instability		25–40
DNA aneuploidy		60–75

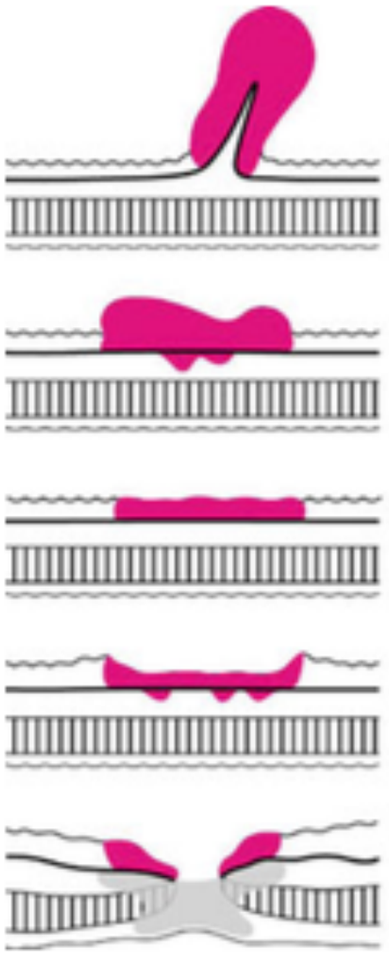
1900 Cases

Precancerous lesion	Number of cases	%
Hyperplastic polyp	10	0.53
Adenoma	47	2.47
Chronic ulcer	13	0.68
Atrophic gastritis	1802	94.84
Verrucous gastritis	26	1.37
Stomach remnant	2	0.11
Aberrant pancreas	0	0
	Total 1900	100

N.C.C.H., Tokyo April 1988



Type 0 (superficial)	Typical of T1 tumors.
Type 1 (mass)	Polypoid tumors, sharply demarcated from the surrounding mucosa.
Type 2 (ulcerative)	Ulcerated tumors with raised margins surrounded by a thickened gastric wall with clear margins.
Type 3 (infiltrative ulcerative)	Ulcerated tumors with raised margins, surrounded by a thickened gastric wall without clear margins.
Type 4 (diffuse infiltrative)	Tumors without marked ulceration or raised margins, the gastric wall is thickened and indurated and the margin is unclear.
Type 5 (unclassifiable)	Tumors that cannot be classified into any of the above types.



Type 0-I (protruding)[a]	Polypoid tumors.
Type 0-II (superficial)	Tumors with or without minimal elevation or depression relative to the surrounding mucosa.
Type 0-IIa (superficial elevated)[a]	Slightly elevated tumors.
Type 0-IIb (superficial flat)	Tumors without elevation or depression.
Type 0-IIc (superficial depressed)	Slightly depressed tumors.
Type 0-III (excavated)	Tumors with deep depression.

[a] Tumors with less than 3mm elevation are usually classified as 0-IIa, with more elevated tumors being classified as 0-I

lauren

intestinal tip (53%)

- Yaşlı
- Erkek>Kadın
- Endemik
- intestinal metaplazi
- Distal
- Hematojen

diffuz tip (33%)

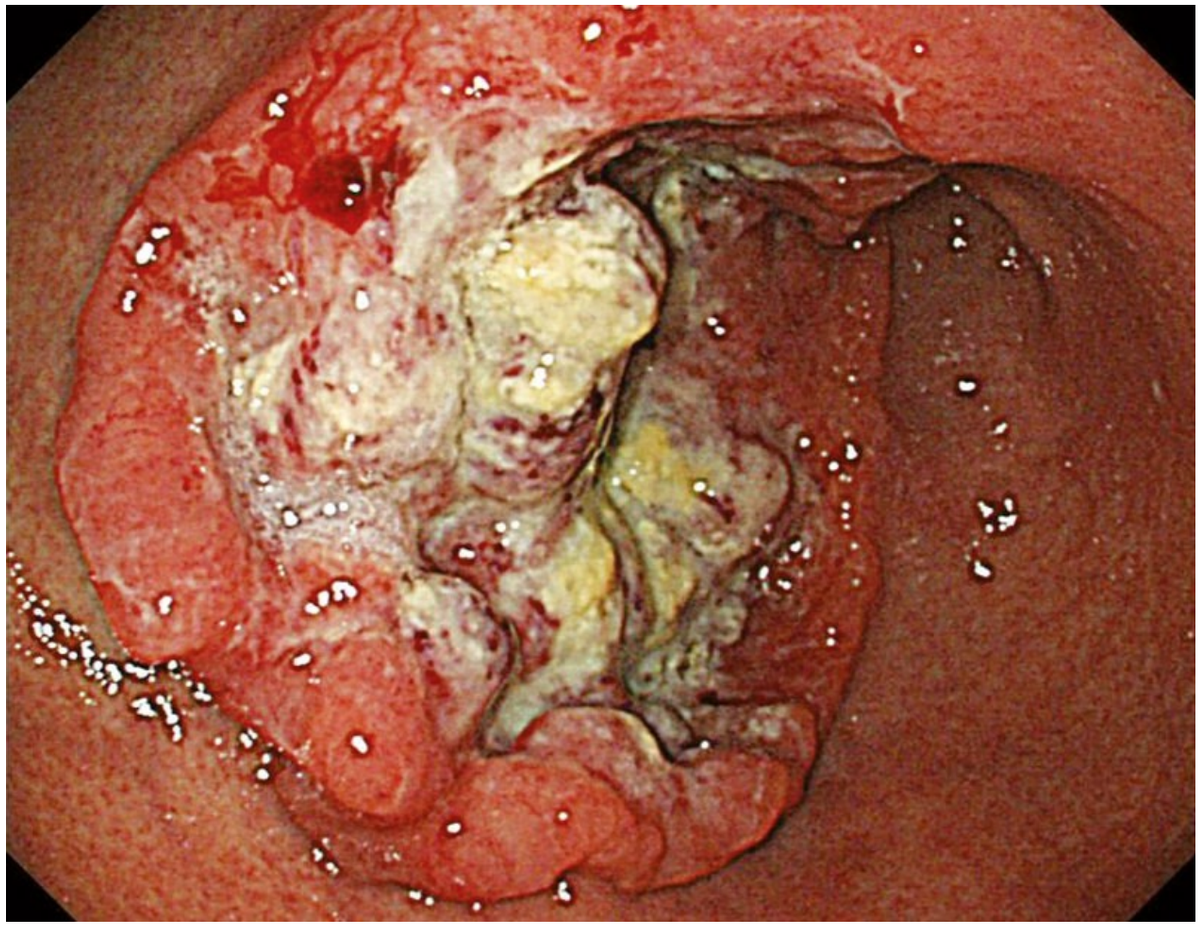
- Genç
- Cinsiyet eşit
- Sporadik
- Kötü differansiye
- Proksimal
- Lenfatik

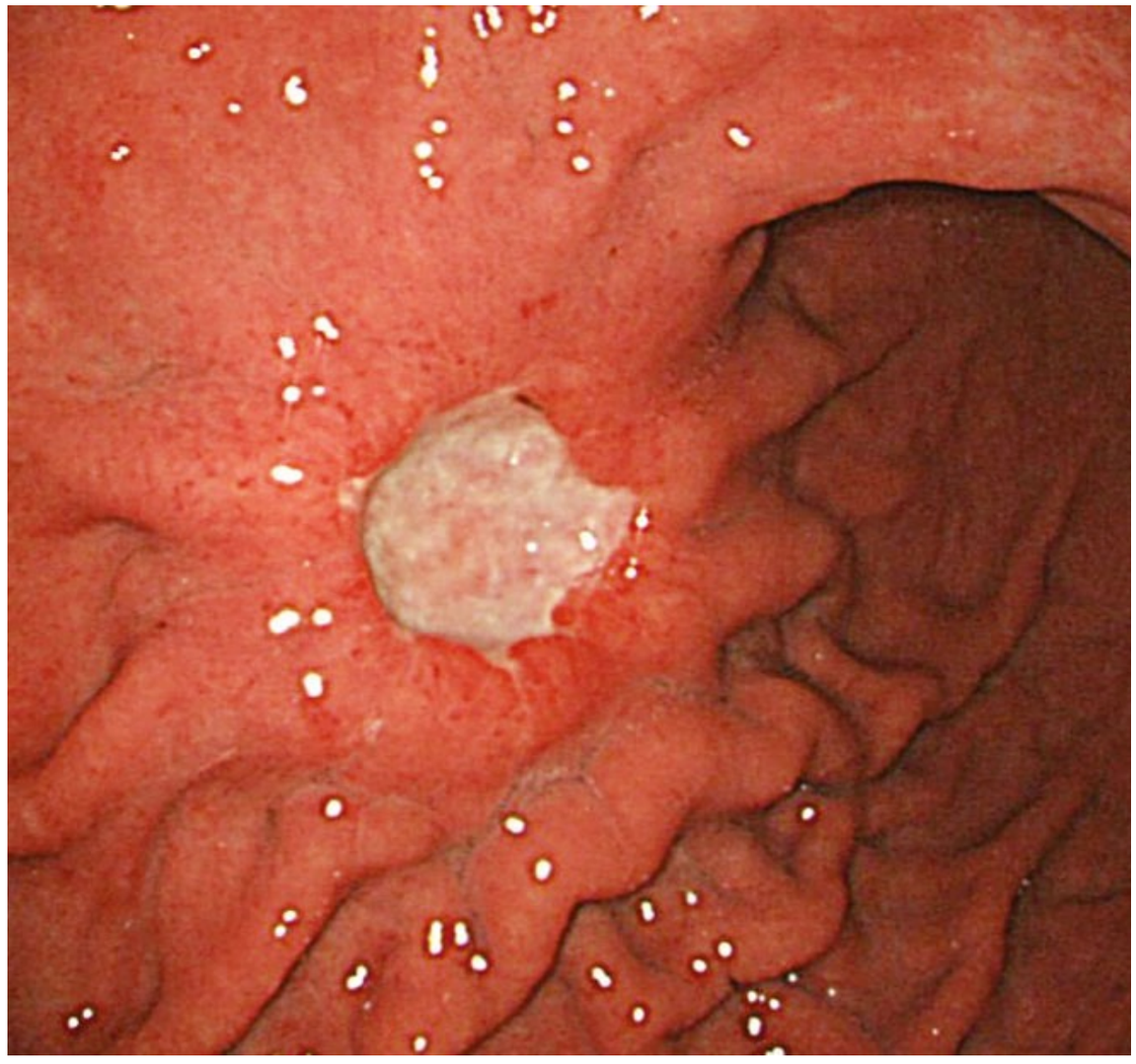
Semptom:

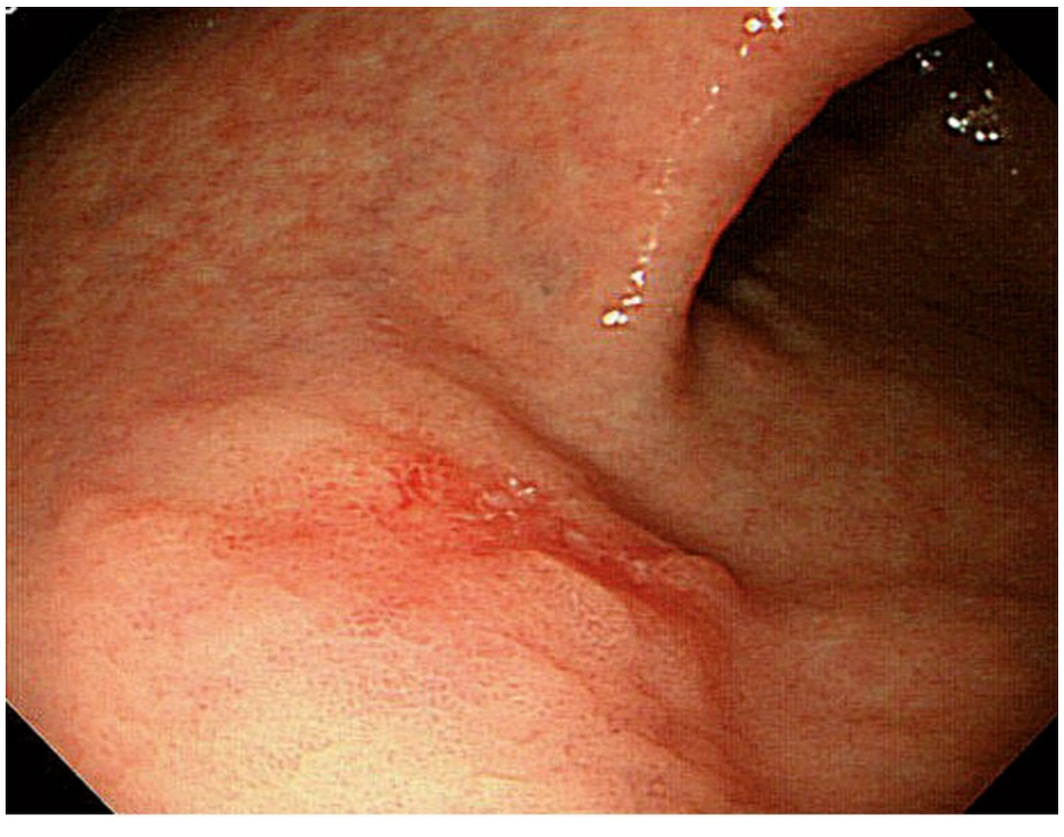
- Kilo kaybı
- erken doyma
- ağrı
- kusma
- GİS kanama-anemi
- disfaji

FM:

- kilo kaybı
- kitle
- asit
- Virchow
- Irish
- Sister-Mary Joseph







Küratif tedavinin temeli; Cerrahi

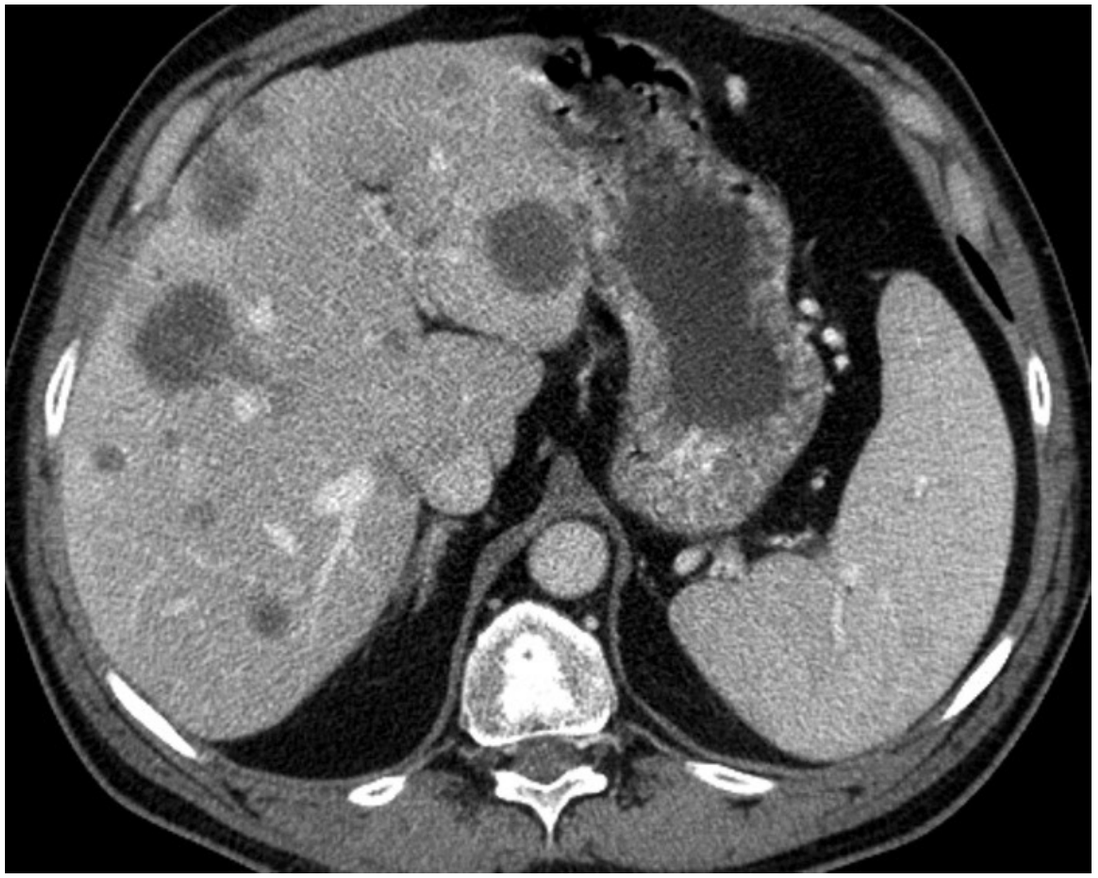
Küratif cerrahi

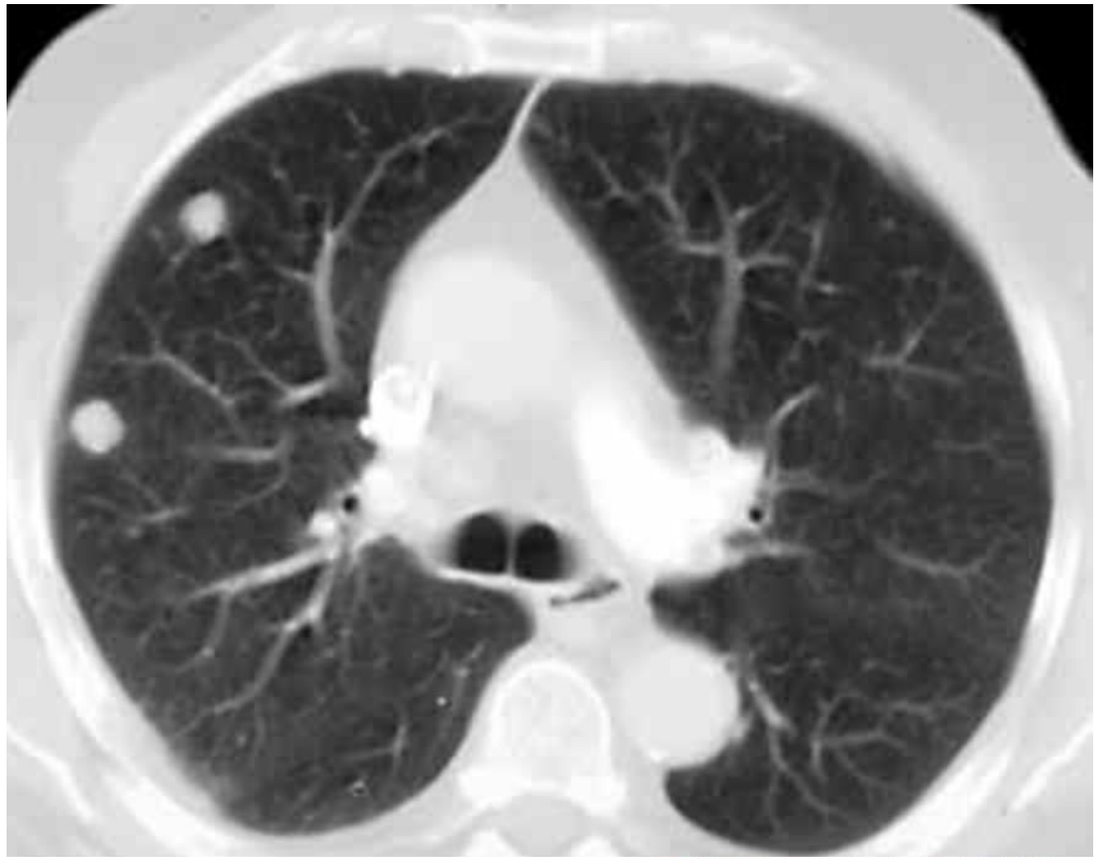
Palyatif cerrahi

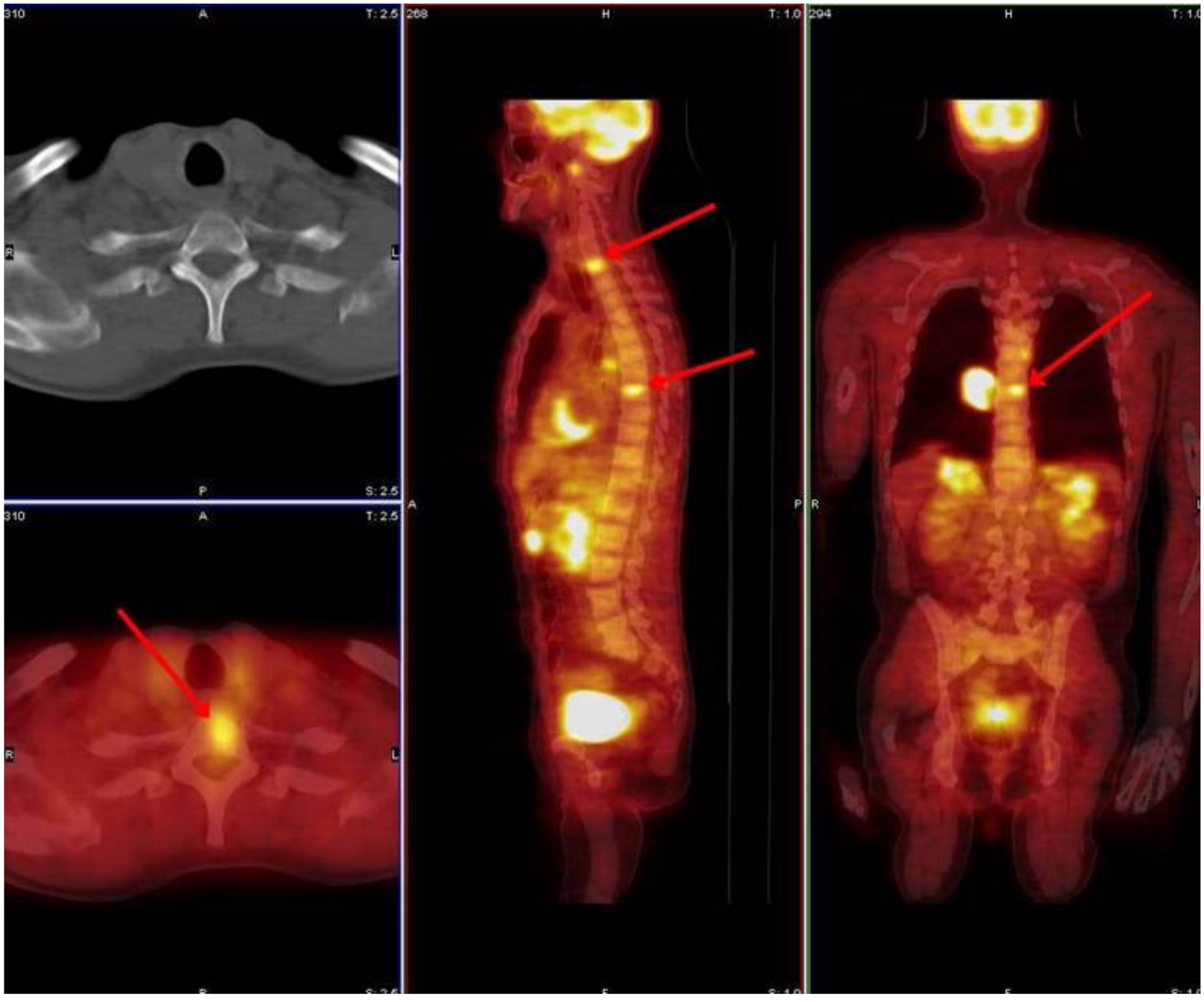
geride tümör bırakmayan cerrahi..

muhtemel metastaz yerleri;

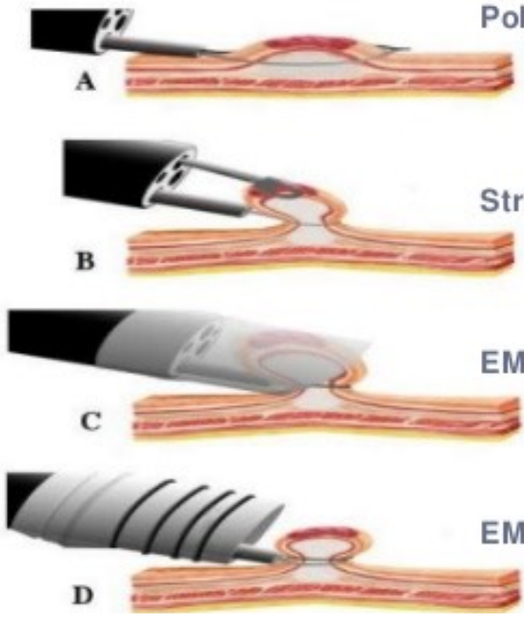
lenf nodları!!
karaciğer
akciğer
periton
kemik
.....






310
A
T: 2.5
R
L
P
S: 2.5
310
A
T: 2.5
R
L
268
H
T: 1.0
A
P
294
H
T: 1.0
R

Standard EMR procedure



Polypectomy; Deyhle et al., *Endoscopy*, 1973

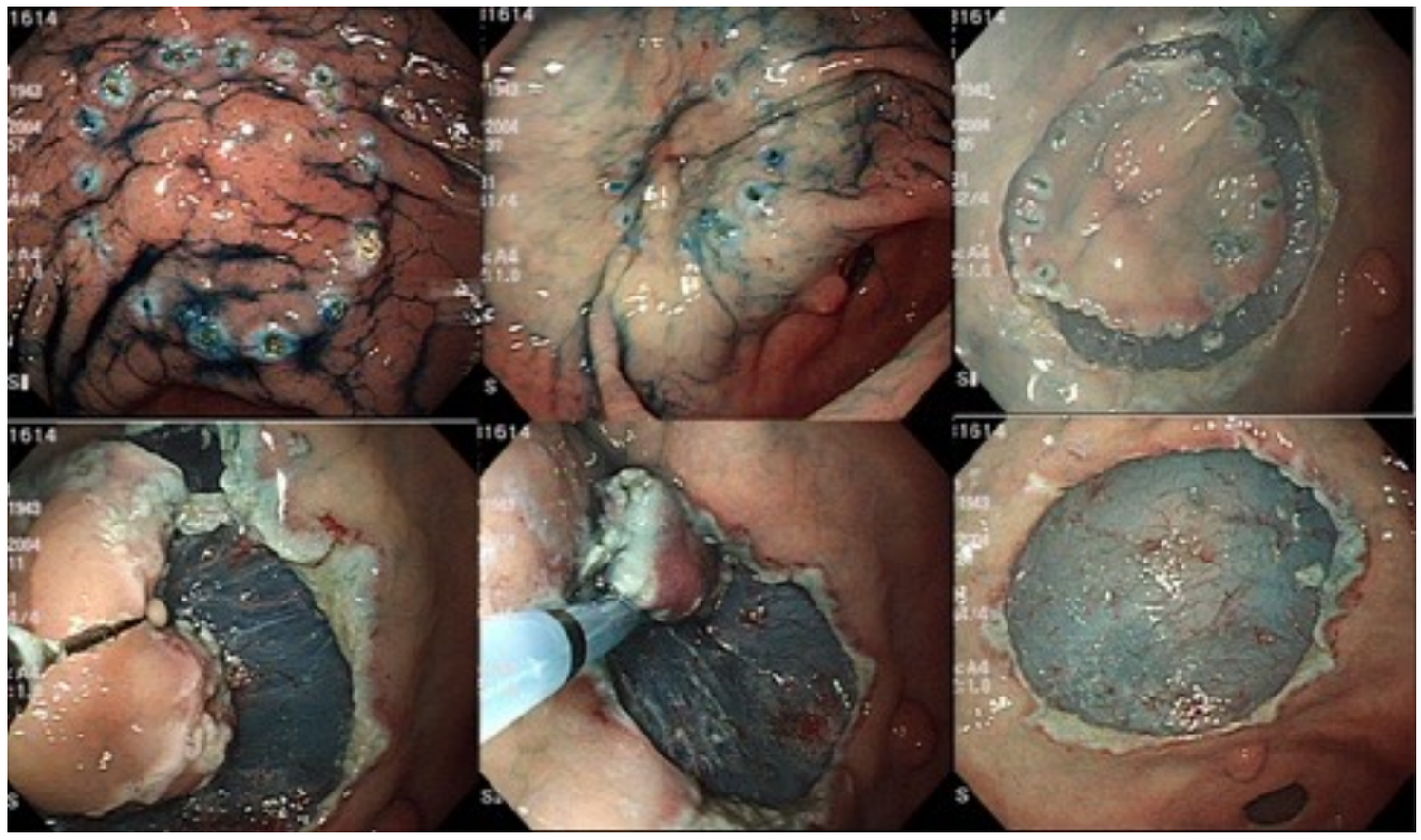
Strip Biopsy; Tada et al., *Gastroenterol Endosc*, 1984

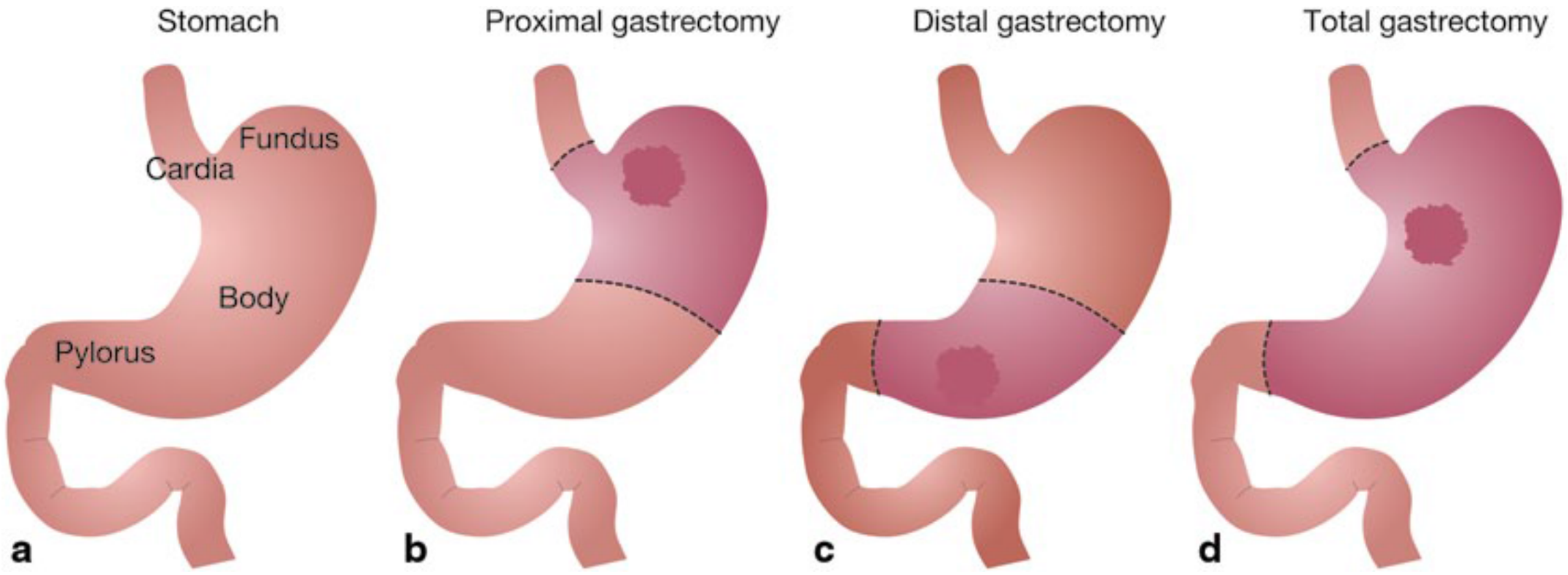
EMR-C; Inoue et al., *Gastrointest Endosc*, 1993

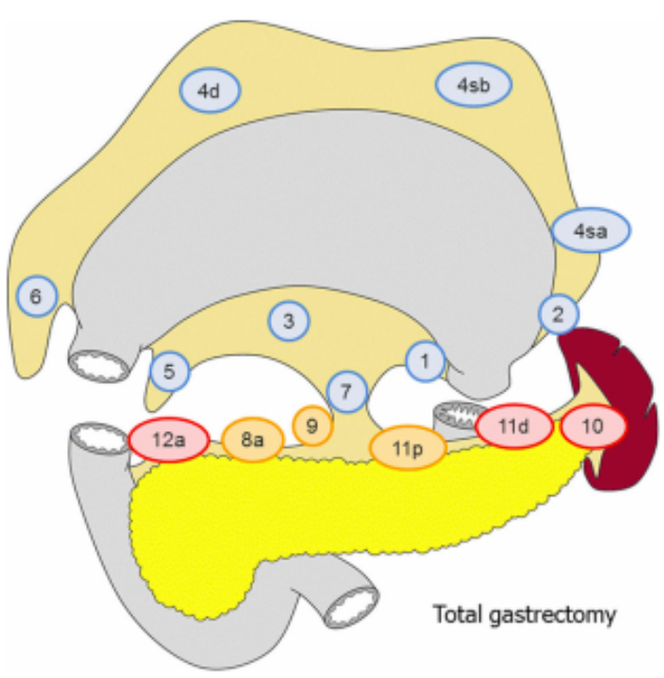
With A Cap-fitted Panendoscope

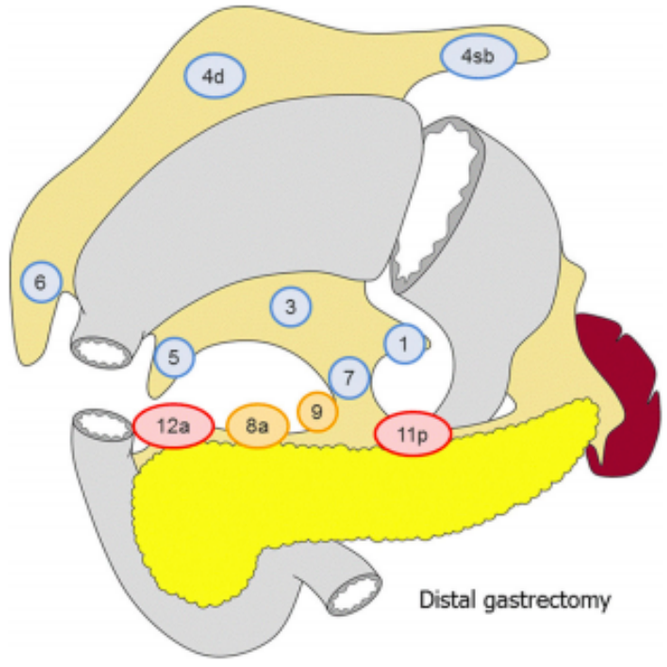
EMR-L; Akiyama et al., *Gastrointest Endosc*, 1997

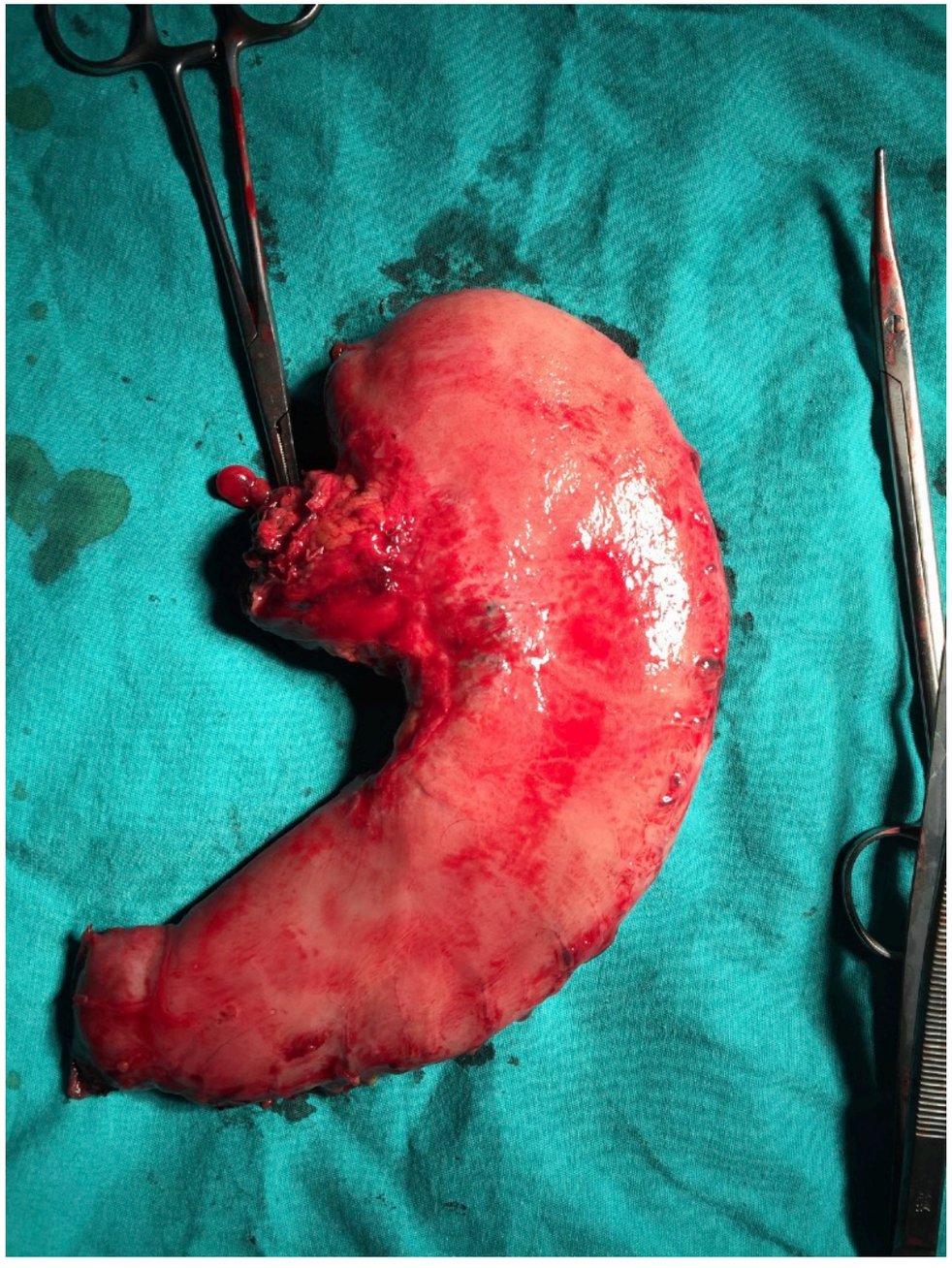
ESD

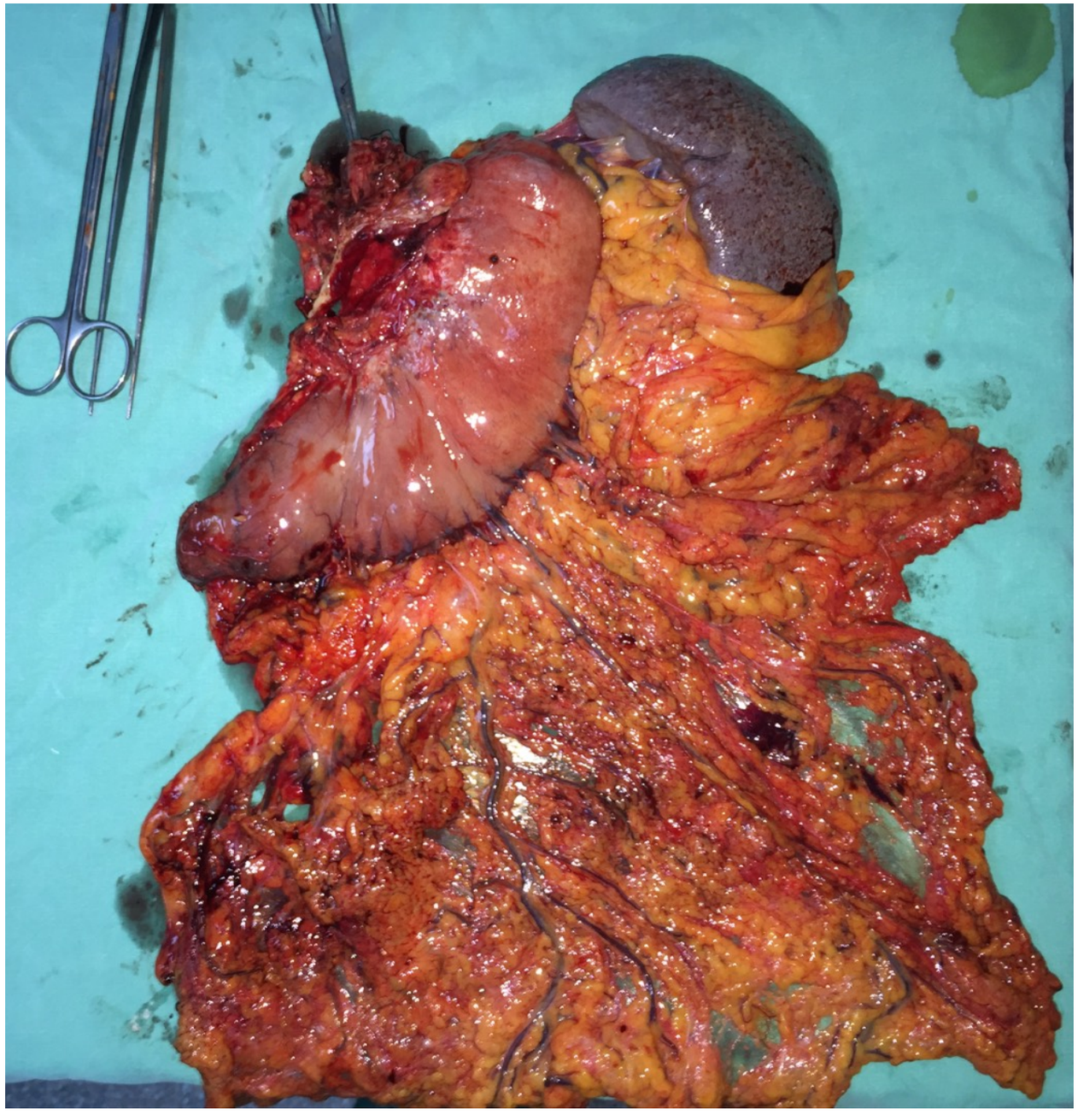



Stomach
Fundus
Cardia
Body
Pylorus
a
Proximal gastrectomy
b
Distal gastrectomy
c
Total gastrectomy
d


4d
4sb
4sa
6
3
2
5
1
7
12a
8a
9
11p
11d
10
Total gastrectomy


4d
4sb
6
3
1
5
7
12a
8a
9
11p
Distal gastrectomy





Adjuvan tedavi

- CT
- CRT

Anastomozlar

Billroth-I
Billroth-II
Roux-enY

Mide cerrahisi sonrası komplikasyonlar

- Anastomoz ilişkili komplikasyonlar
  - Kaçak
  - Duodenal kaçak
  - Striktür
  - Obstruksiyon
  - Jejunal intusseption
  - Internal herni
  - Marginal ülser
  - Afferent loop sendromu
  - Efferent loop sendromu
- Motilite ilişkili komplikasyonlar
  - Dumping sendromu
  - Diare
  - Gastrik staz
  - Alkali gastrit
  - Roux staz sendromu
- Cancer

GIST

Interstitial cells of Cajal (ICC)

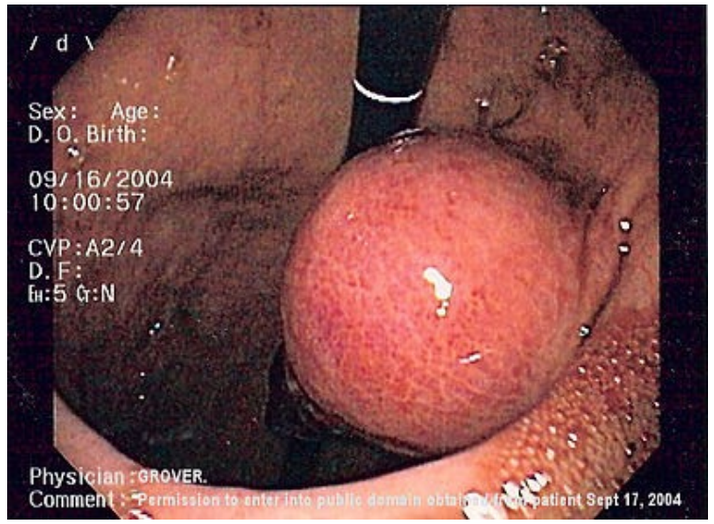
Spindle cell
Epiteloid type
Mixed type

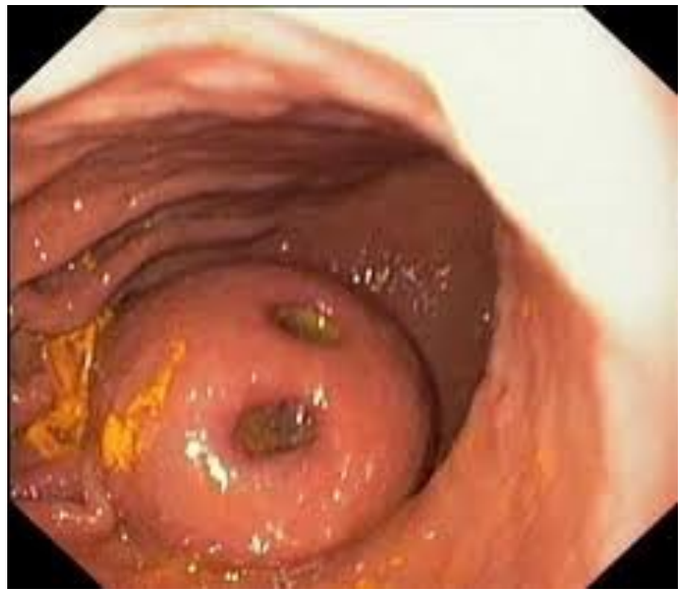
c-kit reseptörü (tirozin kinaz reseptörü)
CD-117
CD-34

PGFRa

Boyut-Mitoz sayısı-Orjin

rezeksiyon
Imatinib





	Size, mm	Mitotic index, per 50 HPF
Very low risk	<20	<5
Low risk	20–50	≤5
Intermediate risk	≤50	6–10
	50–100m	≤5
High risk	>50	>5
	>100	Any mitotic rate
	Any size	>10

HPF, High-power field.