Complications of gastric and duodenal ulcers.

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- Complications of gastric and duodenal ulcers.
- Perforated ulcer of the stomach and of the duodenum. Clinical picture. Diagnosis. Treatment.
- Gastro intestinal ulcerous hemorrhages. Clinical picture. Diagnosis. Treatment.
- Ulcerous stenosis. Diagnosis. Treatment.
- Penetration of the gastric and duodenal ulcer. Clinical picture. Treatment.

The most common complications include

- bleeding,
- perforation
- malignization
- **penetration** of the stomach or duodenal walls
- **obstruction** of the digestive tract.

PUD complications incidence

Every year peptic ulcer disease (PUD) affects 4 milion people around the world Complications are encountered in 10%-20% of these patients

Lau JY, Sung J, Hill C, Henderson C, Howden CW, Metz DC: Systematic review of the epidemiology of complicated peptic ulcer disease: incidence, recurrence, risk factors and mortality. Digestion. 2011, 84: 102-113. PMID: 21494041

Perforated ulcer of the stomach and of the duodenum.

This is when the ulcer completely erodes (perforates) the stomach or duodenal wall.
Perforation means that the ulcer has eaten through all stomach lining. It occurs in an <u>estimated 2-</u> <u>10%</u> of all cases. This causes the leaking of acidic gastric contents into the abdominal cavity and results in acute peritonitis (infection of the abdominal cavity).

Perforated Gastric Ulcer



incidence

- 2%-14% of the ulcers will perforate
- In the third world the clinical picture is different with a high male : female ratio (approximately 8 = 1), younger age, and a strong link with cigarette smoking. In addition in the third world there is a high incidence of patients who present late and this may partly account for the high mortality (20%) reported in some studies.

Bertleff MJ, Lange JF: Perforated peptic ulcer disease: areview of history and treatment. Dig Surg. 2010, 27: 161-169. [PMID: 20571260 doi:10.1159/000264653]

Perforated peptic ulcer (PPU) is relatively rare, but lifethreatening with the mortality varying from 10% to 40%

Svanes C: Trends in perforated peptic ulcer: incidence, etiology, treatment, and prognosis. World J Surg. 2000, 24: 277-283. [PMID: 10658061 doi:10.1007/s002689910045]

ethiology

- Helicobacter pylori is implicated in 70–92% of all perforated duodenal ulcers
- The second most common cause of perforated duodenal ulcer is the ingestion of Non-Steroidal Antiinflammatory drugs, in developed countries such that 40–50% of perforated duodenal ulcers are caused by them.
- The least common cause is pathologic hypersecretory states, such as Zollinger-Ellison syndrome.

Perforated ulcer of the stomach and of the duodenum.

• The first report of a series of patients presenting with perforation of a duodenal ulcer was made in 1817 by *Travers*. The earliest operative description was made by *Mikulicz* in 1884 but the first successful operation for a perforated duodenal ulcer was not until 1894.

Savieliev Clasification, 1976

• 1. <u>By origine</u>

- ulcer perforation; - hormonal perforation.

2. By localization:

- gastric ulcer perforation (smoll curve, big curve, anterior wall, posterior wall);
- duodenal ulcer perforation (anterior wall, posterior wall).
 Anatomy of the stomach



 lig. hepatogastricum; 2 — lien; 3 — ventriculus; 4 — lig. gastrocolicum; 5 — duodenum; 6 —lig. hepatorenale; 7 foramen epiploicum (Winslovi); 8 — lig. hepatoduodenale; 9 vesica fellea; 10 — hepar; 11 — lig. teres hepatis..

3. By clinical evolution:

- - perforation in the free peritoneal cavity
- Covered perforation
- Atipical perforation
- closed perforation in the vecinecy organs – pancreas,liver, gallblader, etc.);

Covered perforation

- the place of perforation can be temporary obturated by omentum or organ that is situation near.
- The **Ratner Rikker sign** persistent pain in the upper right area of the abdominal wall.

Atipical perforation:

- PERFORATION HAEMORAGIC SINDROM, or Malletgui sign.
- Perforation in bursa omentalis most cases present with extraluminal air, pancreatitis, or lesser sac abscess.
- Perforation in the retroperitoneal space .**The Vigiara sign** paraumbilical emphizema the sign of retroduodenal perforation.
- Perforation into mediastinum associated with peumopericardium, subcutaneous emphysema.
- **Podlah sign** supraclavicular emfizama show the possible perforation in thre cardiac area of stomach.
 - Perforation into pleural cavity associated with tension pneumothorax gastropleural fistula, gastrobronchial fistula,
- Reperforation

The clinical pictures usualy have 3

periods:

- The first step or Septic Shock period last usually 6 hours. The Ps, T/A are normal. The skin cover with cold swet and become pale, vomiting can be, constipation.
- **The second period** false improve, beetwen 6 and 12 hours the pain and rigidity lessen, he feels better and a "silent interval" begins. Vomiting disappares, the paim become slight, but the abdomen examination present generalized tenderness, rebound tenderness, guarding, and rigidity and peritoneal irritation sign.
- The third period peritonitis

Pain

• A perforated peptic ulcer usually present with a sudden onset of severe, sharp, intense and steady pain. The perforation allows the contents of the stomach or small intestine to leak into the abdominal cavity. As more contents enter the abdominal cavity, the pain intensifies and spreads throughout the abdomen, radiating to include pain in one or both shoulders. The pain caused by a perforated peptic ulcer often worsens when changing positions or breathing deeply. Most patients describe generalized pain; few present with severe epigastric pain. As even slight movement can tremendously worsen their pain, these patients assume a fetal position.

Pain

- **Dieulafoy sign** the epigastrical pain that begining like knife heat.
- Eleken sign the pain irradiated toward clavicula.
- Mondore triade ulcer history, the epigastrical pain that beginning like knife heat, muscle tenderness.
- Mandel sign the increasing of the abdominal pain during perecution in the projection of the perforation.
- **Blumberg sign** the sign of the peritoneal <u>irritation.</u>

Abdominal inspection

- 1. Patients with peritonitis exhibit abdominal swelling. The swollen abdomen feels tender when touched and worsens when pressed firmly and quickly released. Abdominal examination findings are usually consistent with generalized tenderness, rebound tenderness, guarding, and rigidity. However, the degree of peritoneal findings is strongly influenced by a number of factors, including the size of perforation, amount of bacterial and gastric contents contaminating the abdominal cavity, time between perforation and presentation, and spontaneous sealing of perforation.
 - 2. The Klark sign the "absens, of the liver tisuie during percution
- 3. Shlange sign sign of abdominal silent, the absens of the intestinal peristaltic
- 4. Kulenkamf sign the presens of painfull pelvic swelling during rectal examination.
- 5. Blumberg sign the sign of the peritoneal irritation

Diagnostic Procedures

- X-RAYS. Look for a thin linear gas shadow between his diaphragm and his liver or stomach.
- Free air under the diaphragm found on an upright chest X-ray is indicative of hollow organ perforation
- On an erect Chest X Ray free air can be seen in about 80% of cases.
- In doubtful cases a water-soluble gastroduodenogram will show the leak from the duodenum or its sealing.



X-RAYS

- CAUTION !
- 1. An ulcer can perforate almost silently in the very old, or in the course of another disease.
- (2) The absence of gas does not exclude the presence of a perforated ulcer.
- (3) Gas can also come from a ruptured diverticulum or an appendix (uncommon).
- Plain x-rays of the abdomen with the patient in the upright position have been used in diagnosing perforated ulcer. However, several case series have shown that in 30% to 50% of patients, the x-ray may be negative for free air, particularly in the elderly.
- Similarly, use of water-soluble contrast medium with an upper gastrointestinal tract series or computed tomography scan may increase the diagnostic yield.

CT scans

The increased use of CT scans has greatly improved our ability to detect perforation. Suspicious findings on CT scan include unexplained intraperitoneal fluid, pneumoperitoneum, bowel wall thickening, mesenteric fat streaking, mesenteric hematoma and extravasation of contrast. We suggest Erect CXR as initial routine diagnostic assessment in case of acute abdomen from suspected free perforation of PU.

In case of negative AXR and/or erect CXR, we suggest CT scan as second level diagnostic tool since its higher sensitivity in detecting intra-abdominal free air.

In case of negative findings of free intra-abdominal air and persistent suspicion of PPU, we suggest adding oral water soluble contrast or via NGT.

Blood analisis

 The leukocyte count varied from 15,000 to 25,000 cells per cubic millimeter in 60 percent of the patients;

USG examination

 show the prezens of free fluid and air in the free peritoneal cavity.

Treatment of the perforated ulcer Non-operative treatment

- In 1935 *Wangensteen* noted that ulcers are able to self seal and reported on seven cases treated without surgery.
- In 1946 this observation was confirmed by *Taylor* and he treated 28 cases without surgery with good success.
- This was in the context of the high mortality and morbidity associated with surgical management at the time.

NON-OPERATIVE TREATMENT FOR A PERFORATED PEPTIC ULCER

- INDICATIONS.
- (1) A perforation which appears to have sealed itself already, as shown by diminished pain and improved abdominal signs.
- (2) Heart or lung disease, which increases the surgical and anaesthetic risks.
- (3) The patient who is admitted after a day or two and is almost moribund with diffuse peritonitis.

Surgical treatment Laparoscopic Surgery

- The traditional management of a perforated duodenal ulcer has been a Graham Omental Patch and a thorough abdominal lavage.
- More recently this has been shown to be able to performed using a laparoscope. The only proven advantage of the laparoscopic technique appears to be decreased postoperative pain.
- Operating times are longer compared to open techniques and hospital time appears to be similar to conventional treatment.
- This technique has not been subjected to any large prospective trials and at present must not be considered as standard management.

Surgical treatment simple closure and lavage

- There is good evidence that, in the emergency situation, highly selective vagotomy (proximal gastric, or parietal cell vagotomy) combined with simple omental patch closure of the perforation, in patients without the risk factors mentioned above, is just as effective as that performed in the elective setting (Grade C).
- This is associated with a less than 1% mortality rate and a 4–11% ulcer recurrence rate. The success of this operation is surgeon-dependent.
 - Truncal vagotomy with drainage has its advocates as an expedient operation familiar to most surgeons.

Operative treatment of perforation of ulcer patch of omentum





Surgical tactic in the pacient with perforated ulcer

- **Type 1 ulcer by Jonson classification, stable** – necessary to be performe distal gastrectomy Bilrot 1 method.
- Type 2 ulcer by Jonson classification, unstable – biopsy,patch the perforated ulcer and give treatment for H. pilori eradication.
- Type 2 or 3 ulcer by Jonson classification
 patch , closure with treatment of H. pilory

• It must be recalled, however, that in the developed world the surgeon's major role in the management of PPU will continue to be the performance of lifesaving operations in elderly unfit patients

Indication for vagotomy with ulcer excizion or stomach rezection

- Young person
- Not more than 6 hours after perforation



heineke-mikulicz pyloroplasty


FINNEY PYLOROPLASTY



JABOULAY PYLOROPLASTY



Haemorrhage (bleeding)

 This complications of a peptic ulcer occurring in between 15-30 % of cases.

Symptoms and Signs

- Hematemesis vomiting of blood
- Melena tarry and black stool
- Hematochezia red blood stool
- Ocult bleeding

Pathogenesis Histologic Features

 Histologic examination of a surgically resected gastric ulcer associated with bleeding reveals an artery eroded by the crater of the ulcer. In most cases the diameter of the bleeding artery is small (mean, 0.7 mm; range, 0.1 to 1.8)4. A larger arterial size is probably associated with increased morbidity and mortality, as well as a decreased likelihood of success with endoscopic therapy. A retrospective review noted that the arterial diameter ranged from 1.5 to 3.4 mm in approximately a quarter of patients with fatal bleeding ulcers

Forrest endoscopic classification

• Acute hemorrhage

- Forrest I a (Spurting hemorrhage)
- Forrest I b (Oozing hemorrhage)
- Forrest I c (capillary hemorrhage)
- Signs of recent hemorrhage
- Forrest II a (Visible vessel)
- Forrest II b (Adherent clot)
- Forrest II c (Hematin on ulcer base)
- **Lesions without active bleeding**
- Forrest III (Lesions without signs of recent hemorrhage)

Forrest's classification for PU bleeding

I-a (arterial jet)



II-b (adherent clot)



I-b (oozing)



II-c (black spot)



II-a (visible vessel)



III (clean base)



BLEEDING. Classification.

 Compensative hemorrhage: loose of blood to 20% VOL (to 1000 ml), pulse, AP, Hb more be normal.

- II. Subcompensative hemorrhage: loose of blood to 30% VOL (to 1500 ml), pulse – tahicardia to100, AP decrease to 100, Hb decrease 100 g/l, olygurine.
- III. Decompensative hemorrhage: loose of blood more than 30% VOL (more than 1500 ml), pulse – tahicardia to120 and more, AP decrease to 80-70, decrease Hb, olygounurine.

Initial Management.

Hemodynamic assessment (blood pressure, pulse, and postural changes) and, if necessary, institution of resuscitative measures are the first steps in the management of upper gastrointestinal bleeding.

• Clinical prognostic features and the initial response to resuscitation are used to decide whether a patient should be hospitalized and, if so, what level of care should be provided. Patients with clinical characteristics that indicate a high risk of further bleeding or death should be admitted to an intensive care unit

HDS moderate/severe

- 2 INTRAVENOSIS WAYS
- Sol. Na Cl 0,9% / sol Ringer
- oxigen,
- Nasogastric tube
- Transfuzion if Ht: > 30% in adult persons, > 20-25% young;
- clinical monitoring

Endoscopic Therapy



Endoscopic Therapy

Injection therapy

absolute ethanol, epinephrine (at a dilution of 1:10,000), polidocanol (a sclerosing agent usually injected immediately after the injection of epinephrine), fibrin, trombine, adrenalin and even normal saline. **laser therapy** Monopolar electrocoagulation bipolar electrocoagulation

heater-probe therapy

Argon plasma

metalic clipse

Eficiency > than 95 % of cases

Angiographic therapy

• Angiographic therapy is rarely used to treat patients with bleeding ulcers and should be considered only for severe, persistent bleeding if surgery poses an extremely high risk and endoscopic therapy has been unsuccessful or is unavailable. Ulcers may stop bleeding with an intraarterial infusion of vasopressin in up to 50 percent of cases. Uncontrolled studies suggest that arterial embolization with an absorbable gelatin sponge (Gelfoam), an autologous clot, tissue adhesives, or mechanical occlusion devices may control bleeding identified angiographically in approximately 75 to 80 percent of cases, although recurrent bleeding may occur in over half these cases

ANGIOEMBOLIZATION

Angiographic obliteration

 of the bleeding vessel is considered in patients with poor prognoses



Surgical treatment may be:

- - *urgent* Forest one bleeding
- - *early* repeted bleeding after 2-3 days
- *planic* in the cases of defenitive hemostasis.

Surgical Therapy - indications

- Patients who have recurrences of bleeding from ulcers despite medical therapy are candidates for surgical therapy;
- Indications for operation for bleeding duodenal ulcers are massive hemorrhage that is unresponsive to endoscopic control, and transfusion requirement of more than 4 to 6 units of blood, despite attempts at endoscopic control.
- Lack of availability of a therapeutic endoscopist
- Recurrent hemorrhage after one or more attempts at endoscopic control
- Lack of availability of blood for transfusion
- Repeat hospitalization for bleeding duodenal ulcer
- Concurrent indications for surgery such as perforation or obstruction, also are indications for surgery.

PU TREATMENT

Surgical intervention

- Only 10% of patients
- Indications
 - Failure of endoscopy
 - Significant rebleeding after 1st endoscopy
 - Ongoing transfusion requirement
 - Need for >6 units over 24 hours
 - Earlier for elderly, multiple co-morbidities

Bleeding Gastric Ulcer Surgical treatment

- Distal gastric resection to include the bleeding ulcer is the procedure of choice for bleeding gastric ulcer.
- Second best is V+D with oversewing and biopsy of the ulcer.

Operative treatment. Method of resection of stomach.





resection of stomach by Bilroth-I

resection of stomach by Bilroth-II

PU Surgical intervention

Gastric ulcer

- 10% are maliganant
- 30% will rebleed
 with simple ligation

Resection

- Distal gastrectomy
 Bilroth I or II
- Subtotal gastrector



Surgical treatment

- If the ulcer is situated on the posterior wall of the duodenum is indicated GASTRIC REZECTION.
- If the ulcer is situated on the anterior wall of the duodenum is indicated ULCER EXCIZION AND VAGOTOMY.
- The two operations most commonly used FOR BLEEDING DUODENAL ULCER ARE V+D (VAGOTOMY AND DRAINAGE PROCEDURES).
- Combined with oversewing of the ulcer, or vagotomy and antrectomy(V+A).
- IN SEVERITY CONDITION OF THE PACIENT IS POSIBLE TO DO THE SUTURE OF THE BLEEDING VASEL OF ULCER





The mortality rate:

• The mortality rate for surgery for bleeding duodenal ulcer is 10 to 20%. Early operation should be considered in patients over 60 years of age, those presenting in shock, those requiring more than 4 units of blood in 24 hours or 8 units of blood in 48 hours, those with rebleeding, and those with ulcers greater than 2 cm in diameter or strategically located as described above.



Ulcerous stenosis. (Obstruction)

- *Gastric outlet obstruction:* Obstruction may be caused by scarring, spasm, or inflammation from an ulcer. Repeated episodes of ulceration and healing can lead to pyloric scarring and a fixed stenosis with chronic gastric outlet obstruction. In cases of untreated duodenal ulceration, the lifetime risk of chronic pyloric stenosis approximates 10%.
 - Symptoms include recurrent, large-volume vomiting, occurring more frequently at the end of the day and often as late as 6 h after the last meal.

Pathogenesis/ pathophysiology Obstruction of the stomach

hypertrophy of the stomach

Dilatation

depressed acid secretion

Gastritis &

Complications / Effects

- Malnourishment weight loss
- Iron deficiency anaemia
- Vomiting of gastric content resulting in:
 - dehydration
 - shock
 - electrolyte imbalance(Na, Cl, 🕻) 🕴 🌡
 - metabolic alkalosis
 - -paradoxic aciduria
 - acute kidney injury

3 type of stenosis:

piloric stenosis
 duodenal bulb stenosis
 postbulbar stenosis

3 stage of stenosis

- : I stage (compensation)
- II stage (subcompensation)
- III stage (decompensation).

GASTRIC OUTLET OBSTRUCTION (stenos). Classification

- Compensation stenos: episode vomiting, wait barii in stomach to 6 hours, loose weight to 5 kg.
- II. Subcompensation stenos: everyday vomiting, wait barii in stomach to 12 hours, loose weight to 10 kg, disturbance of waiter-electrolyte balance.
- III. Decompensation stenos: no evacuation from stomach, vomiting after every food, wait bari in stomach more than 24 hours, loose weight more than 10 kg, severe disturbance of waiter-electrolyte balance, tetanus.

I stage (compensation)

• In the first stage the general condition of the pacient are relative satisfactory, nutrition condition are satisfactory also. The pacient fills epigastrical distension, the vomits have incidently characters. Loss of appetite with persistent bloating or fullness after eating also suggests gastric outlet obstruction.

II stage (subcompensation)

• In the II stage apare a havy senzation in the epigastrical area. Symptoms include recurrent, large-volume vomiting, occurring more frequently at the end of the day and often as late as 11-12 h after the last meal, but without putrefaction sign. The pacient have denutrition state.

III stage (decompensation).

- III stage documented by stasis and inceasing atony and gastric repletion. The general condition of the pacient will be worse, can br detected the sign of dehidratation.
- The fulness and distension in the epigastrical area provoke vomiting, that apears rare, but has enormous amount, Vomit with the food residue what was eated 1 till 2 days before. The food residue have fetid, very unpleasured smell. Prolonged vomiting may cause weight loss, dehydration, and alkalosis.

Endoscopic investigation

- FGDS reveal:
- st.I –skar ulcer deformation of the piloric canal (1-0,5 cm)
- st.II-III –stomac distension with total narrowing of the piloric canal .

x-rays may provide:

- evidence of retained gastric contents:
 6-12 ore in stage I,
- 12-24 ore in stage II,
- More than 24 ore in stage III.


investigations

- 1) Stabilise patient
- FBC (anaemia)
- SEUCR (hypochloraemia, hypokalaemia,hyponatraemia,elevated Hco3)
- BLOOD GASES(metabolic alkalosis)
- URINALYSIS (paradoxical aciduria)

Investigation

2)To confirm diagnosis

- Plain x-ray of abdomen:shows large gastric shadow and a large amount of gastric fluid.
- Gastric aspiration:a wide bore stomach tube is placed early in the morning and the stomach is aspirated of resting juice.if >400ml of juice is obtained a presumptive diagnosis of GOO can be made.

investigation

 Esophagogastroduodenoscopy + biopsy(histology and bacterioloical investigation).

Aim is to viualise the stomach mucosa and any ulcer.

Barium meal:

-markely dilated stomach with a lot of residue

-presence of an ulcer crater

-trifoil deformity of the duodenal cap.

Treatment

Endoscopic dilation of the gastric outlet is a reasonable course for treating this complication after medical therapy has failed. Balloon dilation can usually improve the acute problem. A lubricated balloon is passed through the endoscope and is inflated with water or air.

The goal of surgery for gastric outlet obstruction is twofold:

- 1) improvement of the obstruction
- 2) treatment of the predisposing ulcer with an acid-reducing procedure.
- Vagotomy, in which the vagus nerve is cut to reduce stomach acid, and antrectomy, the removal of the pyloric portion of the stomach, are recommended surgical procedures.

Surgical methods

- Surgical methods depended from general conditions of the pacient, stages of stenosis, dehydration level:
- Then general condition of the pacient permite the most comon operation are 2/3 rezection of the stomac by Bilrot II modification
- In the very old pacient, with asotiation pathology should to be performed combinated gastroenteroanastomosis with Braun enteronteroanastamosis



Penetration of the gastric and duodenal ulcer

• Ulcer penetration refers to penetration of the ulcer through the bowel wall without free perforation and leakage of luminal contents into the peritoneal cavity. Surgical series suggest that penetration occurs in 20 percent of ulcers, but only a small proportion of penetrating ulcers become clinically evident .Penetration occurs in descending order of frequency into the PANCREAS, omentum, Biliary Tract, LIVER, mesocolon, COLON, and vascular structures

Penetration ulcer. Clinical picture.

• Penetration often comes to attention because of a change in symptoms or involvement of adjacent structures. The change in symptom pattern may be gradual or sudden; it usually involves a loss of cyclicity of the pain with meals, and loss of food and antacid relief. The pain typically becomes more intense, of longer duration, and is frequently referred to the lower thoracic or upper lumbar region. The diagnosis of penetrating ulcer is suspected clinically when an ulcer in the proper region is found.

3 phase of penetration:

- <u>Phase I</u> Penetration of the ulcer through all levels of the gastric or duodenal wall.
- <u>Phase II Fibrosis conection,</u> adhision with vecinecy organ
- <u>Phase III</u>. Defenitive penetration in the vecinecy organ

- Mild hyperamylasemia can develop with posterior penetration of either gastric or duodenal ulcer, but clinical pancreatitis is uncommon .
- Penetration can be associated with a wide array of uncommon complications including perivisceral abscess (evident on CT or ultrasonography)],
- erosion into vascular structures leading to exsanguinating hemorrhage (aortoenteric fistula), or erosion into the cystic artery.
- Rare biliary tract complications include erosion into the biliary tree with choledochoduodenal fistula, extra hepatic obstruction, or hematobilia.
- Fistulization into the pancreatic duct has also been reported with penetrating duodenal ulcer fistulae are seen with greater curvature gastric ulcers, particularly marginal ulcers . Typical features of this complication include pain, weight loss, and diarrhea; feculent vomiting is an uncommon, but diagnostic symptom.
- A duodenocolic fistula can also occur .No rigorous studies are available to guide the management of penetrating ulcers. One can assume that management should follow the intensive measures outlined for refractory ulcers.
- In many cases, fistulas caused by ulcers can heal with treatment of the ulcer. Sometimes surgery may be needed to close the fistula.

Penetration ulcer treatment

• If after 1-2 course of hospital treatment the ulcer don't heal the surgical tratment will be performed

<u>Ulcer malignization.</u> 3 – 15%

• It is know that only gastric ulcer should be malignizated. In modern medical literature we don't know the cases of malign transformation of the duodenal ulcer, or theoreticly is posible. To be detected the time of ulcer malignization are clinically very dificult, usually disapear the periodicity of the pain durind the day and during spring – autum period of time

достаточно яркой и лаконичной форме принципы хирургической тактики при язве желудка сформулировал С.С. Юдин:

 «Чем больше язва, чем глубже ниша, чем старше больной, чем ниже кислотность, тем более опасность возникновения рака из язвы, а следовательно, тем скорее показана резекция желудка» (С.С. Юдин, 1965).

- Disapear appetite, increase weight loss
- The pain is often constant, nonradiating, and unrelieved by food ingestion but less stronger. In a surprising number of patients, pain can be relieved, at least temporarily, by antacids or gastric antisecretory drugs. ANOREXIA, NAUSEA, AND WEIGHT LOSS AND ANEMIA ARE PRESENT.
- Can be unwilingnees of meet food and microscopic bleeding (pozitiv Adler-Creghersen test)

• Fibroscopic investigation is the most definitive diagnostic method when gastric neoplasm is suspected. In the initial stages, gastric cancers can appear polypoid, as flat plaquelike lesions, or as shallow ulcers.