
بسم الله الرحمن الرحيم

Upper Gastrointestinal Bleeding
(Incidence, causes)

By

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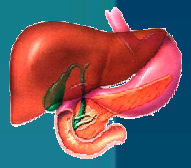
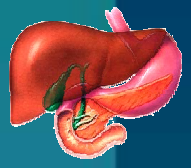


- **Acute upper gastrointestinal bleeding, represents the most common gastrointestinal emergency, accounting for:**
  - 50 – 150 admissions to hospital per 100.000/year of the population in U.K (2004).
  - 60 – 160 admissions to hospital per 100.000/year of the population in U.S (2005).
  - 26% of gastrointestinal emergency admission in Egypt (Kaser – ElAny 2001).



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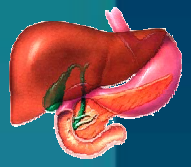
- In Mansoura in Medical gastroenterology unit, ***(El-Sayed Salem et al.,1996)*** conducted a study in 2442 patients presented by acute upper GIT bleeding where he found that:
  - 86% of bleeding due to rupture esophageal varices.
  - 6.3% of bleeding due to peptic ulcer (G + D).
  - 7.7% of bleeding due to others eg.



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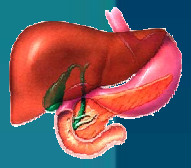
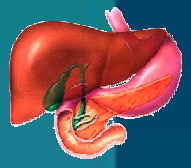
- **In U.S:**
  - 41% of bleeding due to peptic ulcer (G + D).
  - 10% of bleeding due to esophageal varices rupture.
  - 13% of bleeding due to erosive gastritis
  - 35.6% of bleeding due to others.
  - 0.4% of source of bleeding remains unidentified.



Clinical outcome

- 80% of cases of acute upper GIT bleeding stop with conservative therapy.
- 20% leads to hemodynamic instability or shock.
- 10% mortality was associated with massive upper GIT bleeding.
- Mortality due to peptic ulcer bleeding has been markedly decreased down to 5%.



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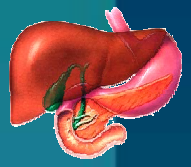
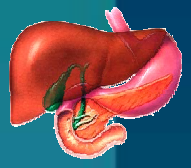
- Recurrent bleeding occurs in up to 50% of cases due to esophageal varices and 25% of cases due to duodenal and gastric ulcer.
- Without treatment after the first episode of esophageal varices bleeding, mortality is about 30% and after the 2nd attack of bleeding it raises up to 50%.



Age and comorbid disease:

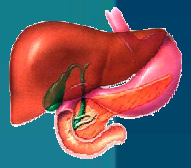
- Age above 60 years, doubles the mortality rate.
- ASGE survey assessed comorbid disease cateogories:
  - In absence of comorbid diseases, mortality risk was 2.6%
  - When three associated diseases, mortality rose to 14.6%.
  - While six associated diseases mortality reached 66.7%.



Pathogenesis

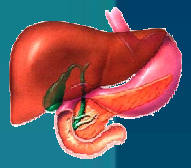
Portal hypertension

- It is the increase in portal venous pressure:
  - Normal portal pressure: 5 – 10 mmHg
  - Portal hypertension: > 12mmHg
  - Normal portal blood flow: 1- 1.5 L/min.
  - Elevated portal pressure increase the gradient between portal pressure and inferior vena cava pressure above the normal range 2 -6 mmHg.
  - Increased resistance to portal blood flow leads to formation of portosystemic collaterals that divert portal blood flow to the systemic circulation, effectively by passing the liver.



Pathophysiology of portal hypertension:

- Portal hypertension results from structural and functional alternation in the hepatic microvasculature.
- The principal site of these alternation are hepatic sinusoids although portal venules, hepatic arterioles and central and hepatic venules also may be involved.



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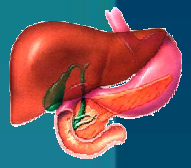
- The structural alternation in sinusoids include narrowing and increased trortuosity due to compression resulting from the swelling of adjacent hepatic parenchymal cells due to injury or the accentuation of lipid droplets, the accumulation of connective tissue in the space of disse and capillerization of the sinusoidal endothelium.



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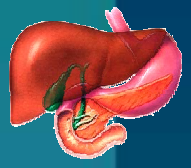
- Also the sinusoids become engorged with blood due to toxic injury leading to the formation of gaps through which red cells penetrate into space of Disse, leading to sinusoidal collapse or distintegarte reducing blood flow and increasing portal pressure.



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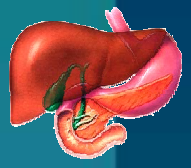
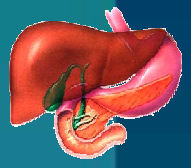
- Functional alternation in sinusoide include narrowing and increased tortusity due to contraction of activated stellate cells and possibly sinusoidal endothelial cells (SEC) stimulated by increased levels of endothelin-1, endothelin receptor expression and decreased levels of NO and perhaps CO.



The response of hepatic microvasculature are of to basic types

- An inflammatory response involving paracrine activation of the sinusoidal endothelial cells by mediators (endotoxins, cytokines, chemokins, free radicals, nitric oxide, endothelin, carbon monoxide, leukocytes and platelets). The mediators are released from adjacent kupffer cells and adjacent hepatic parenchymal cells, these lead to up regulation of adhesion molecules and the subsequent adhesion of leukocytes to the SEC all restrict the sinusoidal blood flow.



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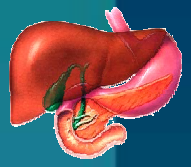
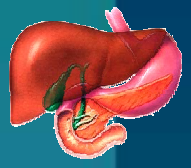
- Direct injury to SEC resulting in loss of fenestrate formation of gaps penetration of the sinusoidal lining by blood cells, destruction of SEC and obstruction of the sinusoid by SEC debris.



Nitric oxide (No):

- is a gaseous molecular implicated in development of portal hypertension.
- In the intrahepatic circulation, NO bioavailability is diminished due to defects in the post translational regulation of endothelial NO synthase, with ↑ intrahepatic resistance.



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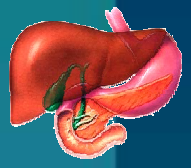
- in construct in the systemic and splanchnic circulation NO bioavailability is increased because of up regulation of Nos protein expression and post translational activation of e-NOs, leading splanchnic vasodilatation and ↑ portal venous flow.



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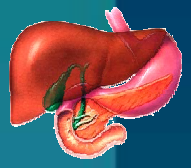
- Specific syndromes will developed due to ↑ NO in systemic circulation:
  - Hepatorenal syndrome.
  - Hepatopulmonary syndrome.
  - Hepatic encephalopathy.
  - Cirrhotic cardiomyopathy.
  - Esophageal varcies.



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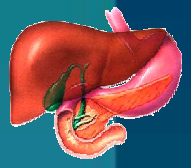
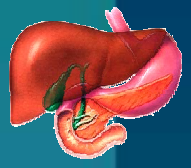
- So therapeutic implication to inhibit NO production in systemic circulation, still under investigation, new goal is to use a combination of vasoconstrictors to ↓ splanchenic blood flow and vasodilators to ↓ intrahepatic collateral resistance. Also activated factor VII and polytera-flourethylene covered Tips stent.



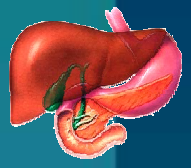
Causes:

- **Hematemesis:** is the vomiting of blood from the mouth due to a cause below the pharngeo esophageal junction.
- **Fale hematemesis:** vomiting of blood due to a cause above the pharngeo esophageal junction e.g. epistasix, hemoptysis, lesion in gum, tongue, throat, tonsils, hysteria or malingering.
- **Melena:** tarrary stool, more than 60 cc of blood.



Classication causes

- **Esophageal causes:**
  - Esophgeal varices.
  - Esophagitis
  - Foreign body.
  - Ulcers due to hiatus hernia.
  - Mallory-Weiss syndrome.
  - Ruptured aortic aneurysm.
  - Tumor.



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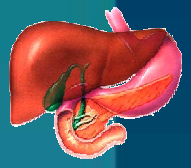
- **Gastric causes:**
  - Gastric ulcer
  - Gastric varcies.
  - Gastritis.
  - Erosive gastritis due to drugs.
  - Corrosives.
  - Anastomotic ulcer.



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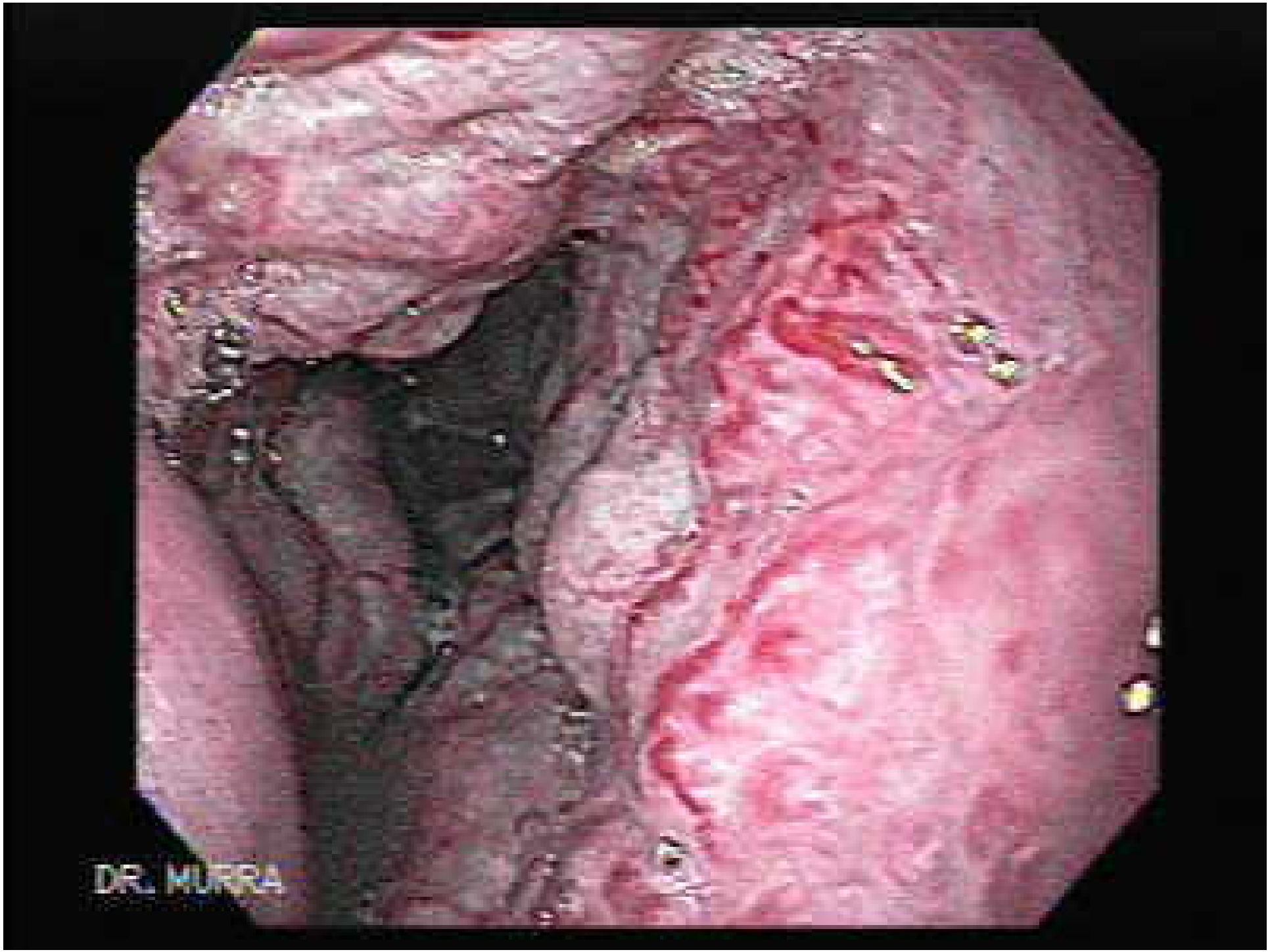
- **Gastric causes:**
  - Carcinoma
  - Other benign tumors as leiomyoma.
  - Foreign body.
  - Hypertensive gastropathy
  - CHF
  - Hereditary telangiectasia.
  - Dieulafoy lesion.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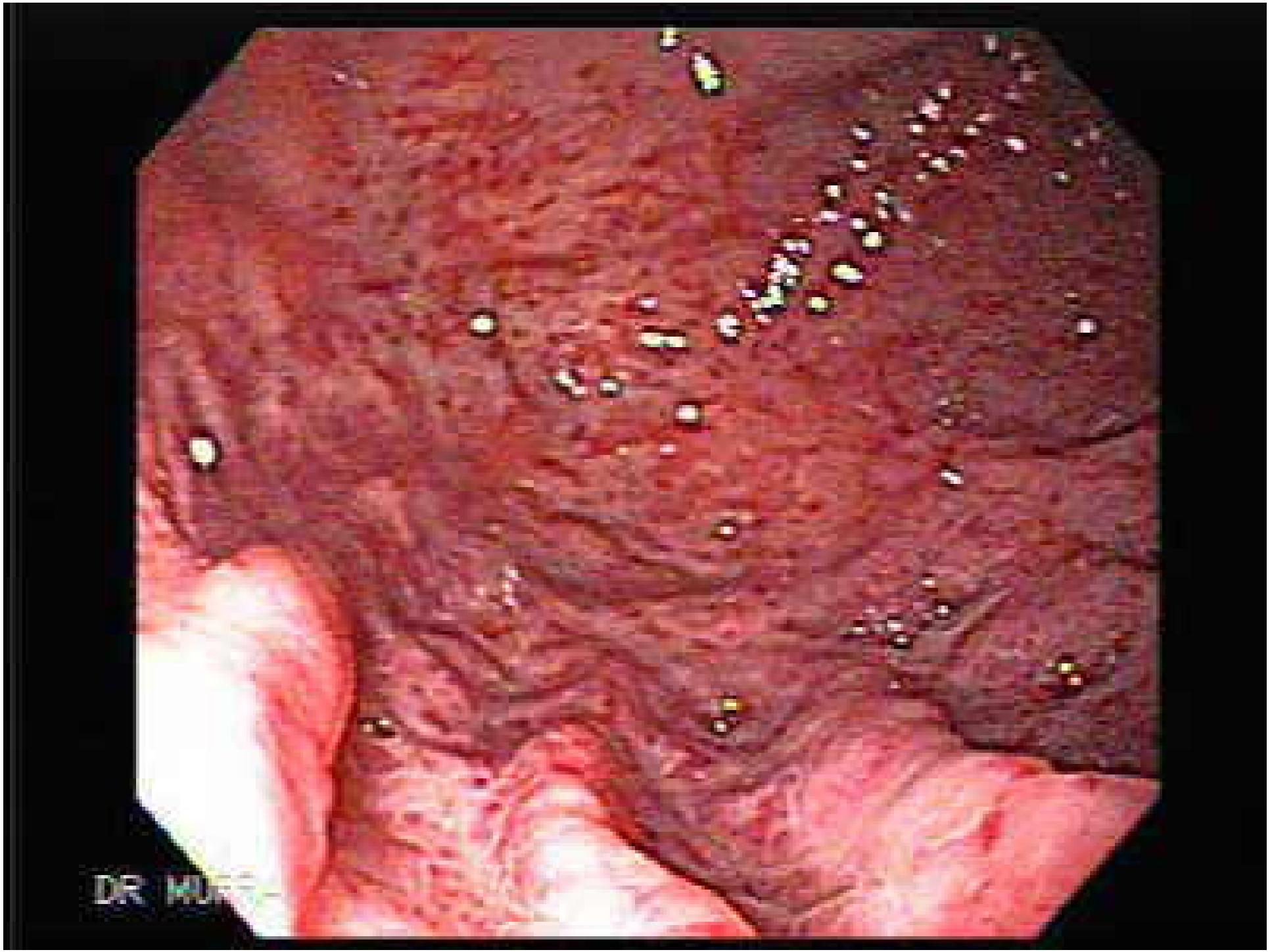

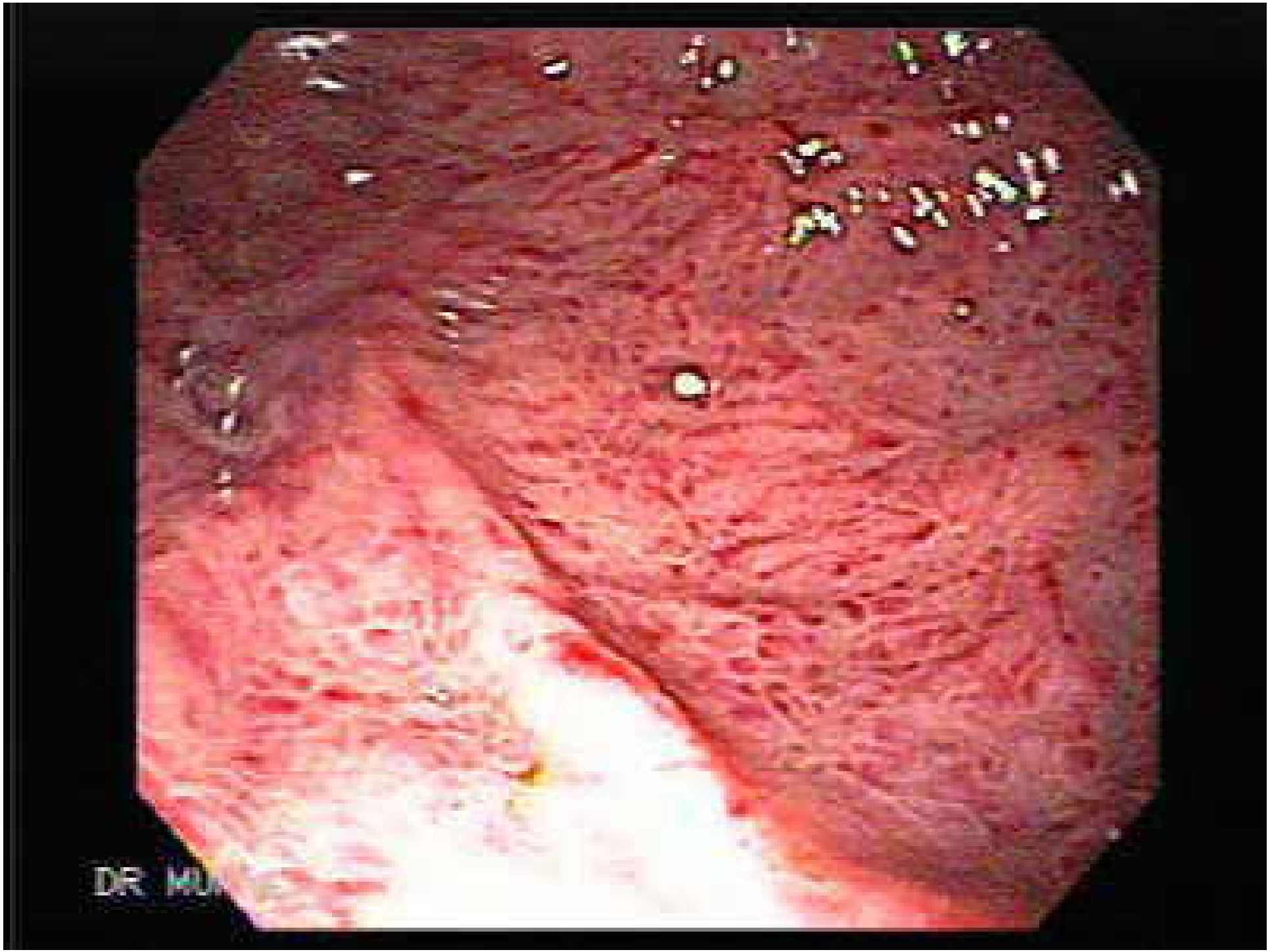

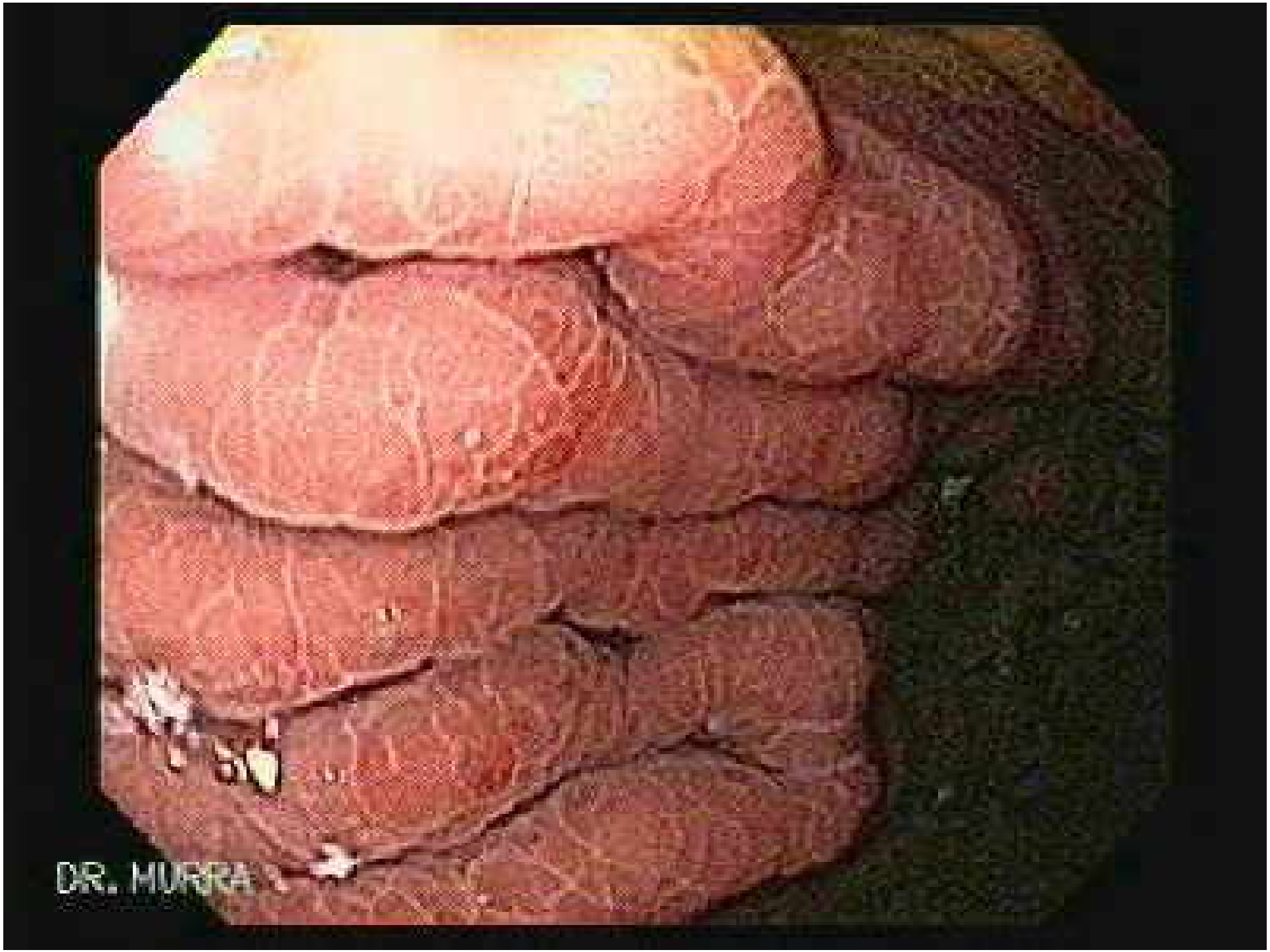
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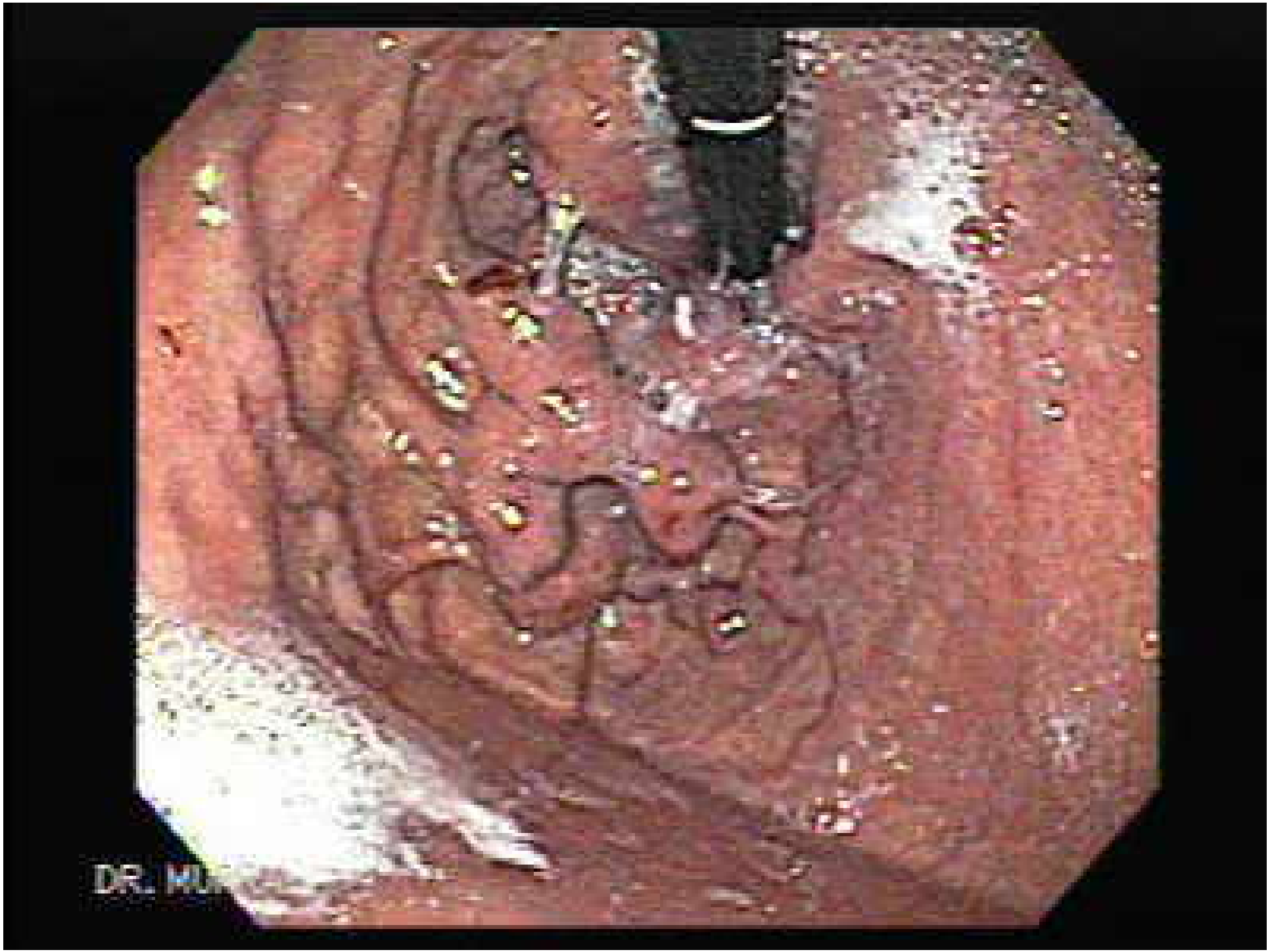

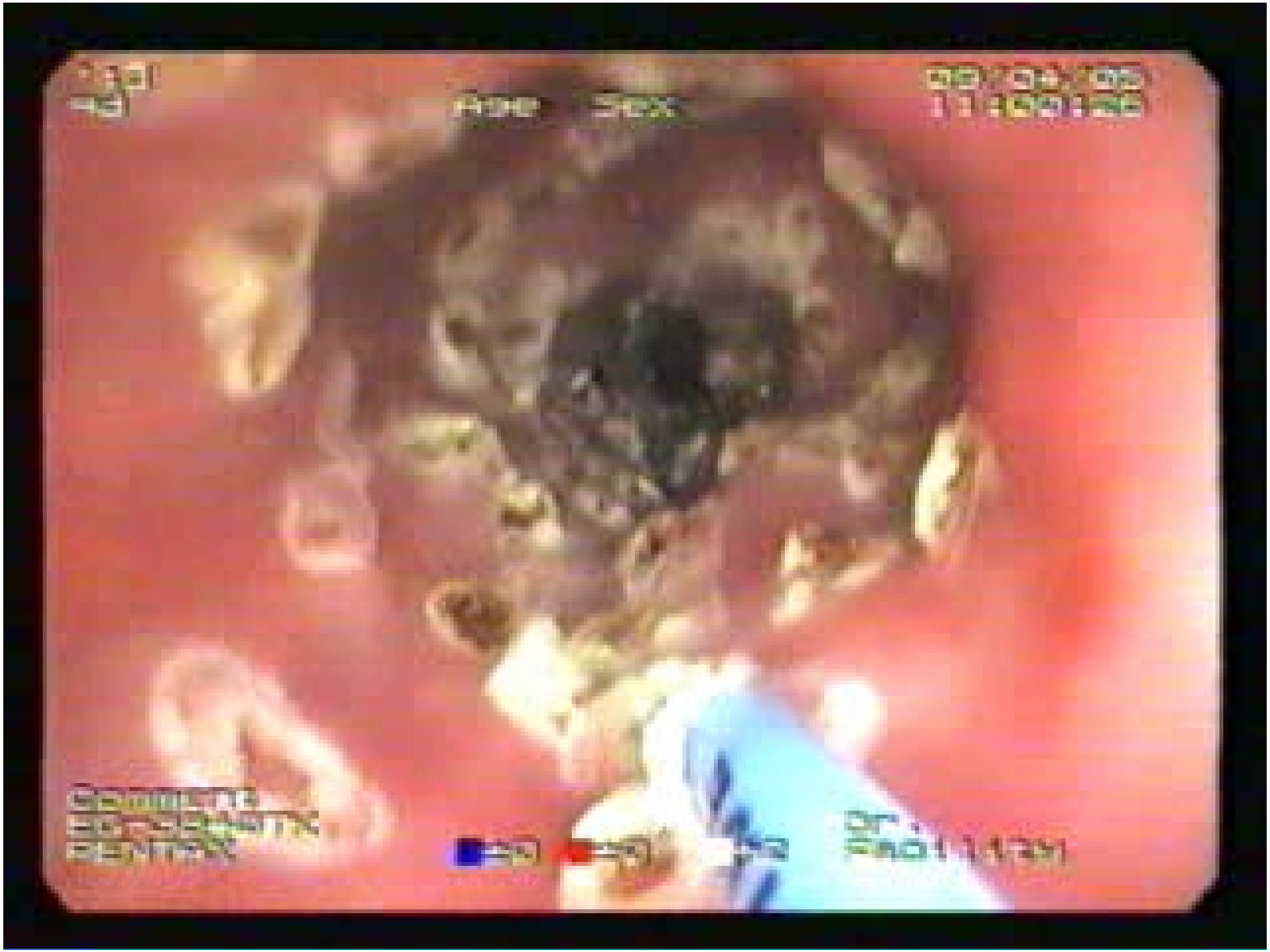

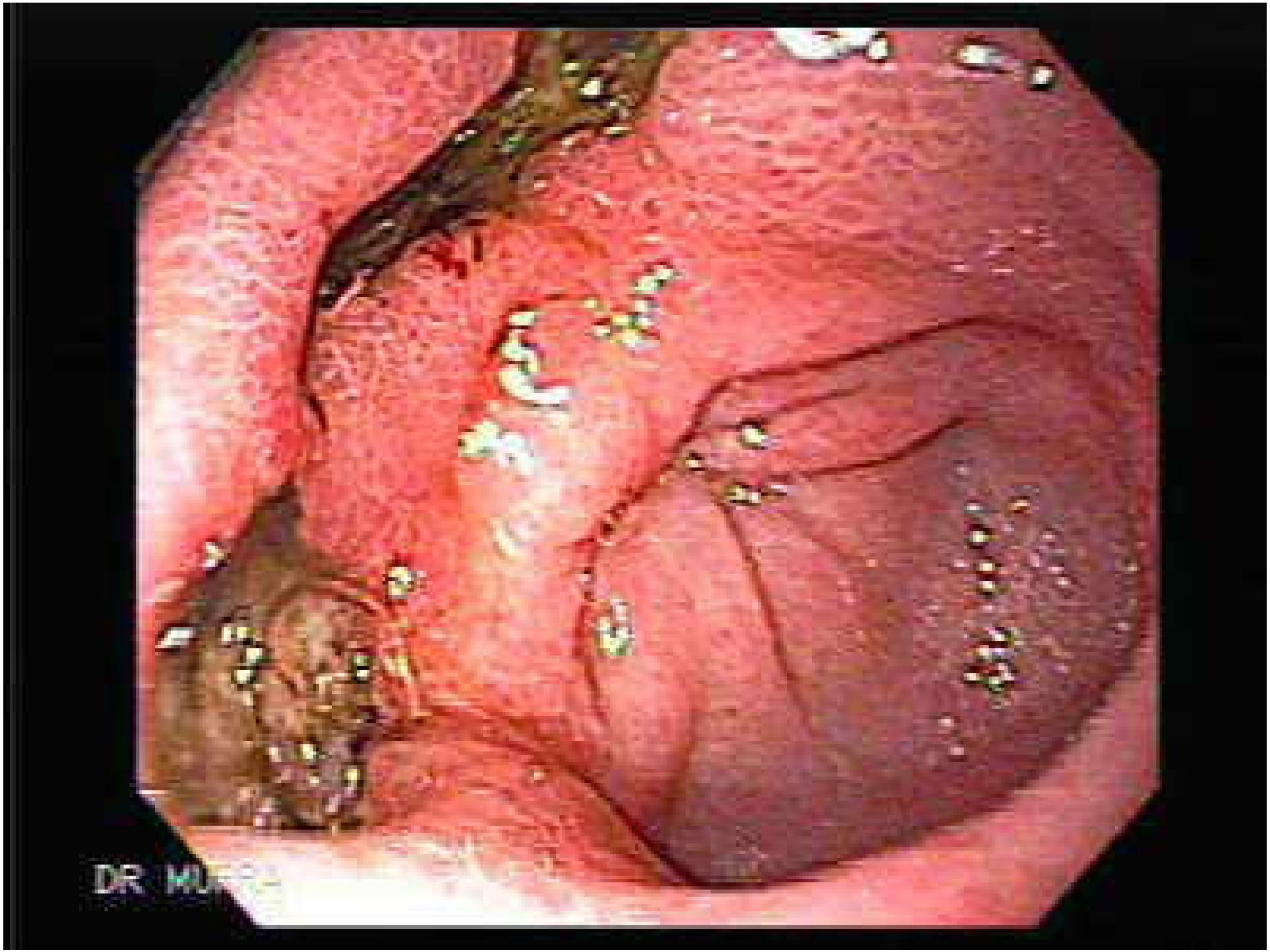

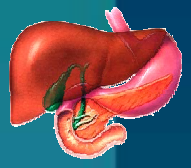

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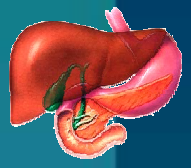
- **Duodenum:**
  - Duodenal ulcer.
  - Duodinitis.
  - Diverticulum.
  - Aortoenteric fistula.
  - Pancreatic pseudocyst.
  - Post sphincterotomy.
  - Cancer ampulla of vater.
  - Hemobilia.



General causes

- **Acute fever:**
  - Septicemia
- **Hemorrhogic tendencies:**
  - Pupura,
  - Hemophilia
  - Acute leukemia
  - Salicylate poisoning.
  - Anticoagulants.
  - Polycythemia vera.
  - Thrombosis of hepatic or portal veins.

Cont.

- Uremia
- Periartertitis nodosa.
- Pseudoxanthoma elasticum.