NOTES



NOTES GASTROINTESTINAL CANCERS

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Tumors arising from cells in gastrointestinal (GI) tract
- Multifactorial etiology; generally result from aberrant cellular signaling, unregulated cellular growth
 - Genetic alterations (e.g. point mutations, amplifications, rearrangements, deletions)
 - Epigenetic influence (e.g. DNA methylation, chromatin remodeling)
 - Environmental factors (e.g. exposure to carcinogens, chronic inflammation)
- Risk increases with age

SIGNS & SYMPTOMS

- Highly variable clinical presentation; see individual disorders
- Fatigue, anorexia, weight loss

DIAGNOSIS

DIAGNOSTIC IMAGING

Imaging studies

Localization, staging

- Grading, TNM staging for treatment
 - T: characteristic of primary Tumor (e.g. invasion of nearby tissue)
 - N: involvement of regional lymph Nodes
 - M: Metastasis; spread from primary tumor to other body parts

LAB RESULTS

Biopsy

Histopathological diagnosis

TREATMENT

MEDICATIONS

Chemotherapy

SURGERY

See individual disorders

OTHER INTERVENTIONS

Radiation therapy

CARCINOID TUMOR

osms.it/carcinoid-tumor

PATHOLOGY & CAUSES

- Uncommon, well-differentiated, slowgrowing neuroendocrine tumor; originates in tubular digestive tract; also found in bronchopulmonary system, genitourinary tract
- Benign/malignant; tendency for liver metastasis
- Carcinoid: tumors of different morphology, less aggressive than GI tract adenocarcinomas; low grade (proliferative activity); low mitotic rate

TYPES

• Embryonic origin of Gl tract (e.g. foregut, midgut, hindgut)

Foregut tumors (e.g. stomach)

- Type I
 - Most common
 - Originates from enterochromaffin-like (ECL) cells
 - In association with high gastrin levels secondary to chronic atrophic gastritis
 - Small, usually benign
- Type II
 - Originates from ECL cells
 - In association with high gastrin levels induced by gastrinomas (e.g. Zollinger– Ellison syndrome) in conjunction with multiple endocrine neoplasia type 1 (MEN1)
 - Often large, indolent; low-grade malignancy
- Type III
 - Not associated with high gastrin levels
 - Large, aggressive; local lymphatic/ hepatic metastases; produce serotonin (5-HT)

Midgut tumors

- Small bowel (most common)
 - Originates from intraepithelial endocrine

cells

- Commonly located in ileum; may arise from Meckel's diverticulum
- Potential for lymph node/hepatic metastasis
- Appendix
 - Originates from subepithelial endocrine cells
 - Relatively low potential for metastasis

Hindgut tumors

• Rectum, colon, cecum (most common)

COMPLICATIONS

- Depend on tumor's location, size, local biochemical attributes
 - Local/distant metastasis
 - Pain: obstruction, intussusception, bowel ischemia, mechanical pressure from tumor
 - Desmoplasia: intense, local reaction characterized by overproduction of extracellular matrix proteins + myofibroblast cell proliferation → fibrosis, obstruction
 - Carcinoid syndrome: tumor-related humoral factors (e.g. serotonin, histamine, etc.) → cutaneous flushing, pruritic rash; excessive lacrimation; wheezing; diaphoresis

SIGNS & SYMPTOMS

- Often asymptomatic, discovered incidentally (e.g. imaging, surgery, endoscopy)
- Vary according origin site
 - Nonspecific, vague abdominal pain
 - Loss of appetite, vomiting, diarrhea, constipation
- Desmoplasia (with CT scan)

DIAGNOSIS

DIAGNOSITC IMAGING

CT scan, MRI, labeled somatostatin receptor-based diagnostic imaging

- Localization, TNM staging
- Presence of hepatic lesions

Endoscopy with biopsy

- Tumor visualization
- Histopathological analysis, grading

LAB RESULTS

5-hydroxyindoleacetic acid, chromogranin



Figure 32.1 Histological appearance of a carcinoid tumor of the lung.



MNEMONIC: CARCinoid Carcinoid syndrome components Cutaneous flushing Asthmatic wheezing Picht-sided valuular heart

Right-sided valvular heart lesions Cramping and diarrhea

TREATMENT

MEDICATIONS

• Somatostatin analogues suppress tumor proliferation, decrease symptoms

SURGERY

Surgical removal of tumor



Figure 32.2 Gross pathology of carcinoid tumor of the terminal ileum.

CHOLANGIOCARCINOMA

osms.it/cholangiocarcinoma

PATHOLOGY & CAUSES

- Rare bile duct cancers; arise from epithelial cells of intrahepatic, extrahepatic bile ducts (not including gallbladder, ampulla of Vater)
- High fatality due to late diagnosis; highly proliferative
- Mostly adenocarcinomas; minority squamous cell carcinomas

TYPES

- Determined by location (Bismuth-Corlette)

Type I

• Located below confluence of left, right hepatic ducts

Type II

Located at confluence

Type IIIa

Occludes common hepatic duct

Type IIIb

• Occludes right/left hepatic duct

Type IV

Multicentric

RISK FACTORS

- Primary
 - Existing liver, gallbladder disease: primary sclerosing cholangitis (PSC); chronic liver disease (e.g. viral hepatitis, cirrhosis)
- Congenital abnormalities of biliary tree
- Genetic disorders
 - Lynch syndrome; multiple biliary papillomatosis
- Obesity
- Liver fluke infection (undercooked fish)
- Intrahepatic cholangiocarcinomas
 - Associated with mutations in gene encoding isocitrate dehydrogenase 1

(IDH1)

- Risk increases with age
- Slightly more common in individuals who are biologically male

COMPLICATIONS

- Metastasis
 - Liver, lymph nodes, peritoneum, bone, etc.
- Bowel perforation, bleeding

SIGNS & SYMPTOMS

- Often asymptomatic initially; malaise, weight loss, abdominal pain
- Extrahepatic disease (when bile drainage obstructed)
 - Right upper quadrant pain, jaundice, pruritus, dark urine, clay-colored stools, weight loss
- Intrahepatic disease
 - Dull right upper quadrant pain, malaise, weight loss
- Other findings
 - Hepatomegaly, palpated mass



Figure 32.3 Histological appearance of a cholangiocarcinoma. There are normal hepatocytes in the top left of the image, with the tumour occupying the bottom right of the image.

DIAGNOSIS

- History, physical examination
 - Consistent with hepatobiliary disease

DIAGNOSTIC IMAGING

MRI, CT scan, PET, etc.

Detailed evaluation of lesion TNM staging

Transabdominal/endoscopic ultrasound (EUS) with biopsy

- Biliary obstruction, dilation of intrahepatic ducts
- Histolopathological analysis, grading

LAB RESULTS

- Tumor markers
 - Carbohydrate antigen (CA) 19-9; carcinoembryonic antigen (CEA)
- Liver function tests
 - Consistent with biliary obstruction, cholestasis
 - Elevated transaminases, gammaglutamyl transpeptidase, alkaline phosphatase
 - Prolonged prothrombin time/elevated INR
 - Elevated bilirubin



Figure 32.4 Histological appearance of a cholangiocarcinoma. This image shows the tumor edge, with normal hepatocytes on the right and tumor on the left. The tumor cells form tubular structures and are surrounded by fibrosis.

TREATMENT

MEDICATIONS

- Fluoropyrimidine-based chemoradiotherapy
- Chemotherapy

SURGERY

Resection

OTHER INTERVENTIONS

Radiation

COLORECTAL CANCER

osms.it/colorectal-cancer

PATHOLOGY & CAUSES

- Common malignancy of large bowel/rectum
- Third most common cancer worldwide
- Often arises from colonic epithelial tissue
 → adenomatous polyp formation →
 adenocarcinoma
- High metastatic potential after penetrating muscularis mucosa

RISK FACTORS

- Hereditary
 - Familial adenomatous polyposis;
 Lynch syndrome, MUTYH-associated polyposis
- Inflammatory bowel disease
- Lifestyle
 - Smoking, physical inactivity
- Dietary
 - High alcohol consumption; processed red meat; low consumption of fruits, vegetables
- Obesity
- Diabetes mellitus, insulin resistance
- Low socioeconomic status
- History of abdominal radiation
- Lack of screening colonoscopy



Figure 32.5 Gross pathology of an exophytic colorectal carcinoma.

- Black people of African descent
 Highest rates in United States
- More common in individuals who are biologically male
- Risk increases with age
- Protective factors
 - Physical activity; regular use of aspirin, other nonsteroidal anti-inflammatory drugs (NSAIDs)

COMPLICATIONS

- Iron-deficiency anemia (due to bleeding)
- Local, distant metastasis
- Bowel obstruction
- Cachexia
- Bowel perforation \rightarrow peritonitis

SIGNS & SYMPTOMS

- May be asymptomatic initially
- Vague constitutional symptoms
 Fatigue, anorexia, weight loss
- Change in bowel habits
 - Narrowing of stool, constipation, diarrhea
- Rectal bleeding
 Frank/occult
- Rectal pain, tenesmus (feeling of incomplete defecation)
- Nausea, vomiting
 - Bowel obstruction from advanced malignancy

DIAGNOSIS

DIAGNOSTIC IMAGING

Colonoscopy/flexible sigmoidoscopy; biopsy, CT colonography

 Tumor visualization, histopathological analysis, grading, TNM staging, potential for resection



Figure 32.6 Histological appearance of adenocarcinoma of the colon. The tumor is composed of malignant cells which continue to form glandular structure. The left side of the image displays normal colonic mucosa.

LAB RESULTS

- Tumor marker: CEA
- Stool guaiac testing
 - Positive for occult blood

OTHER DIAGNOSTICS

Digital rectal exam

Palpable mass if distal rectal mass



Figure 32.7 A CT scan in the axial plane demonstrating a tumor in the cecum.

TREATMENT

MEDICATIONS

Chemotherapy

SURGERY

- Polypectomy with clear margins
- Surgical resection
- Sessile polyps: colectomy

OTHER INTERVENTIONS

Chemoradiation therapy



Figure 32.8 Positron emission tomography with high levels of tracer accumulation in the pelvis (rectal tumor) as well as the liver and kidneys (metastases).

ESOPHAGEAL CANCER

osms.it/esophageal-cancer

PATHOLOGY & CAUSES

- Rare malignancy of esophageal epithelium
- Squamous cell carcinoma (most common)/ adenocarcinoma
- Commonly diagnosed when disease advanced
- Tendency for rapid metastasis

CAUSES

Chronic exposure to irritants → metaplasia
 → dysplasia → malignant transformation

RISK FACTORS

- Smoking
- Alcohol (esp. combined with smoking)
- Gastroesophageal reflux disease (GERD); reflux esophagitis, Barrett esophagus
- Hiatal hernia
- More common in individuals who are biologically male
- Risk increases with age



MNEMONIC: ABCDEF

Esophageal cancer risk factors

Achalasia Barret's esophagus Corrosive esophagitis Diverticulitis Esophageal web Familial

COMPLICATIONS

Esophageal obstruction; regurgitation
 → aspiration → aspiration pneumonia;
 metastasis

SIGNS & SYMPTOMS

- Asymptomatic initially; dysphagia; pyrosis; retrosternal pain; weight loss
- Late symptoms
 - Coughing, chest discomfort when swallowing; hiccups if spread to diaphragm

DIAGNOSIS

DIAGNOSTIC IMAGING

EUS guided biopsy, CT scan, PET, integrated fluorodeoxyglucose (FDG)

 Tumor visualization, histopathological analysis, grading, TNM staging, potential for resection

Bronchoscopy

 In carina identifies potential lung involvement

OTHER DIAGNOSTICS

Palpable supraclavicular lymphadenopathy



Figure 32.9 Endoscopic appearance of an esophageal tumor. The tumor sits at the gastroesophageal junction and is viewed from above.



Figure 32.10 A barium swallow demonstrating a tumor distorting the normal outline of the esophagus.

TREATMENT

MEDICATIONS

Chemotherapy

SURGERY

Resection of primary tumor, associated nodes

OTHER INTERVENTIONS

Radiation

Esophageal stenting

• Therapeutically enlarges esophageal lumen, reduces dysphagia

GALLBLADDER CANCER

osms.it/gallbladder-cancer

PATHOLOGY & CAUSES

- Uncommon malignancy; most frequently diagnosed cancer of biliary tract
- High fatality rate due to typically late diagnosis
- Most gallbladder cancers arise within fundus
- May obstruct bile flow at common bile duct/ duodenum

RISK FACTORS

Chronic gallbladder inflammation

- Cholelithiasis (gallstones), primary sclerosing cholangitis, porcelain gallbladder, gallbladder polyps, biliary cysts; chronic infection (e.g. Salmonella typhi, Helicobacter bilis)
- More common in individuals who are biologically female
- Obesity
- Cigarette smoking
- Occupational exposure to carcinogens: textile, oil, paper, chemical industries, radon (mining)
- Genetic predisposition

COMPLICATIONS

- Biliary fistula
- Local/nodal/distant metastases

SIGNS & SYMPTOMS

- Often asymptomatic in early stages; malignancy discovered incidentally after symptoms mimic benign gallbladder disease
- Non-specific symptoms
 - Malaise, pain, anorexia, nausea, vomiting, weight loss
- Clinical manifestations (when bile drainage obstructed)
 - Jaundice, dark urine
- Palpable gallbladder

DIAGNOSIS

DIAGNOSTIC IMAGING

EUS guided/percutaneous biopsy, CT scan, MRI, PET, MRCP

• Tumor visualization, histopathological analysis, grading, TNM staging, potential for resection

LAB RESULTS

- Tumor markers: CA 19-9; CEA
- Liver function tests
 - Consistent with biliary obstruction, cholestasis
 - Elevated transaminases, gammaglutamyl transpeptidase, alkaline phosphatase
 - Elevated bilirubin



Figure 32.11 Histological appearance of gallbladder adenocarcinoma. The tumor cells show increased nuclear size, prominent nucleoli and are forming tubular structures.

TREATMENT

MEDICATIONS

Chemotherapy

SURGERY

Simple/radical cholecystectomy

OTHER INTERVENTIONS

Radiation

HEPATOBLASTOMA

osms.it/hepatoblastoma

PATHOLOGY & CAUSES

- Common primary childhood hepatic malignancy; arises from primitive hepatic cells
- Usually occurs in right lobe of liver
- Morphologically diverse tumor: composed of many cell types including embryonal hepatocytes, tissues (e.g. bone, striated muscle)
- Extramedullary hematopoiesis may occur in sinusoids
- Usually present during first two years of life

RISK FACTORS

- Beckwith Wiedemann syndrome
- Trisomies 18, 21
- Familial adenomatous polyposis
- Type la glycogen storage disease
- Li–Fraumeni syndrome
- More common in individuals who are biologically male

COMPLICATIONS

- Ectopic gonadotropin → precocious puberty (uncommon)
- Fatal hepatic hemorrhage, rupture
- Metastasis: commonly lungs

SIGNS & SYMPTOMS

- Children
 - Abdominal mass; discomfort
- Anorexia, weight loss, precocious puberty

DIAGNOSIS

DIAGNOSTIC IMAGING

Ultrasound, percutaneous biopsy, CT scan with/without contrast, MRI

 Diagnostic workup for tumor visualization, histopathological analysis, grading, pretreatment staging system (PRETEXT); potential for resection

LAB RESULTS

- Elevated alpha-fetoprotein (AFP)
- Genetic testing



Figure 32.12 Histological appearance of a hepatoblastoma, a tumor of immature hepatocytes.

TREATMENT

MEDICATIONS

Chemotherapy

SURGERY

Resection

HEPATOCELLULAR CARCINOMA

osms.it/hepatocellular-carcinoma

PATHOLOGY & CAUSES

• Hepatic malignancy commonly diagnosed in presence of chronic liver disease

RISK FACTORS

- Hepatitis B/C infection, coinfection with hepatitis D
- Hereditary hemochromatosis
- Cirrhosis
- Smoking; frequent alcohol consumption
- Obesity
- Alpha-1 antitrypsin deficiency
- Gallstones
- Chronic exposure to aflatoxin (mycotoxin found in peanuts, soybeans, corn)
- More common in individuals who are biologically male

COMPLICATIONS

- Paraneoplastic syndrome: watery diarrhea, hypoglycemia, hypercalcemia, erythrocytosis; cutaneous lesions (e.g. pemphigus foliaceus)
- Extrahepatic metastasis: commonly lymph nodes, lungs, adrenal gland



MNEMONIC: ABC

Hepatocellular carcinoma etiology Aflatoxins Hep B

Cirrhosis

Hepatocellular carcinoma features

AFP increased: classic marker Bile-producing: DDx from cholangiocarcinoma

Most **C**ommon primary liver tumor

SIGNS & SYMPTOMS

- Often no symptoms aside from those of chronic liver disease
- Epigastric pain; appetite, weight loss
- Palpable abdominal mass; manifestations of decompensated cirrhosis (e.g. splenomegaly, ascites, jaundice); hepatic bruit



Figure 32.13 An abdominal CT scan in the axial plane demonstrating a massive hepatocellular carcinoma.

DIAGNOSIS

DIAGNOSTIC IMAGING

Ultrasound with biopsy, CT scan, MDCT, arteriography, portography, MRI

 Tumor visualization, histopathological analysis, grading, TNM staging, potential for resection

MRI angiography

• 3D characterization of lesion, hepatic circulation

LAB RESULTS

- Elevated aminotransferases, alkaline phosphatase, gamma-glutamyl transpeptidase; hyperbilirubinemia; hypoalbuminemia
- Elevated alpha-fetoprotein (most common serum marker)



Figure 32.15 Histological appearance of a hepatocellular carcinima. The cells show high nuclear variation, thickened nuclear envelopes and occasional prominent nucleoli. The cells also have abundant eosinophilic cytoplasm.



Figure 32.14 Gross pathology of hepatocellular carcinoma.

TREATMENT

MEDICATIONS

- Chemotherapy
- Systemic molecularly targeted therapy; sorafenib, nivolumab

SURGERY

- Partial hepatectomy
- Liver transplant

OTHER INTERVENTIONS

- Radiofrequency ablation
- Percutaneous ablation with ethanol/acetic acid
- Transarterial chemoembolization
- Cryoablation
- Radiation therapy; stereotactic body radiation therapy

ORAL CANCER

osms.it/oral-cancer

PATHOLOGY & CAUSES

- Oral cavity malignancy; arises from mucosal surfaces
 - Lips, buccal mucosa, anterior tongue, mouth floor, hard palate, gingiva, retromolar trigone
 - Most often: squamous cell carcinoma
- May arise from normal mucosa/ premalignant lesions (e.g. erythroplakia, leukoplakia); undergo malignant transformation

RISK FACTORS

- Tobacco (esp. with alcohol)
- Alcohol
- Human papillomavirus (HPV) infection: oropharynx
- Periodontal disease
- Chronic oral candidiasis
- Betel quid chewing
- Immunosuppression
- Hepatitis C infection
- Genetic polymorphisms: cytochrome P450 1A1 (CYPIA 1); glutathione S-transferase mu 1 (GSTM1); alcohol dehydrogenase 3 genotype → oropharyngeal cancers
- More common in individuals who are biologically male

MNEMONIC: PATH LAB

Oral cancer risks

Plummer-vinson syndrome Alcohol

Tobacco

Human papilloma virus

- Leukoplakia Asbestos
- Bad oral hygiene

COMPLICATIONS

- Surgical resection → airway, speech, mastication, cosmetic complications
- Metastasis

SIGNS & SYMPTOMS

- Asymptomatic initially
- Pain/burning sensation
- Lump/ulcer visualized, palpated
- Hard, fixed lymph nodes

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan/MRI

Local spread/location of additional primary tumors

LAB RESULTS

• Fine needle biopsy; histopathological diagnosis

OTHER DIAGNOSTICS

- Palpation and visualization
 - Of mucous membranes, oral cavity, lymph nodes
- Flexible laryngoscopy
 - Back of throat, vocal cords

TREATMENT

MEDICATIONS

Chemotherapy

SURGERY

Resection

OTHER INTERVENTIONS

Radiation

PANCREATIC CANCER

osms.it/pancreatic-carcinoma

PATHOLOGY & CAUSES

- Highly lethal malignancy of exocrine pancreas
- Usually unresectable at presentation

RISK FACTORS

- Chronic pancreatitis
- Malignant transformation of pancreatic intraductal papillary mucinous neoplasm (IPMN)
- Genetic mutations (e.g. BRCA-1, BRCA-2, ATM, PALB2, CDKN2A, MLH1)
- Smoking; obesity; sedentary lifestyle

COMPLICATIONS

- Hypercoagulability with possible venous/ arterial thromboembolism
- Paraneoplastic manifestations
 - Bullous pemphigoid; nodular fat necrosis (pancreatic panniculitis)
- Metastasis

SIGNS & SYMPTOMS

- Recent onset of diabetes mellitus
- Clinical presentation
 - Tumor location
- Pain
 - Epigastric, abdominal, may radiate to the back, may worsen after eating/when lying down; asthenia:
- Physical weakness, loss of strength; anorexia, nausea; weight loss; jaundice, dark urine
- Hepatomegaly; right upper quadrant mass; Courvoisier's sign (nontender, palpable gallbladder at right costal margin); cachexia; metastasis: left supraclavicular/periumbilical lymphadenopathy, ascites, abdominal mass

DIAGNOSIS

DIAGNOSTIC IMAGING

Transabdominal ultrasound

• Detects degree of biliary tract dilation, obstruction

ERCP

Increased visibility of pancreaticobiliary tree

MRCP

• Visualization of liver parenchyma, vascular structures

Laparoscopy

Determines resectability

Abdominal CT scan; contrast-enhanced CT scan, EUS guided/percutaneous biopsy

• Tumor visualization, histopathological analysis, grading, TNM staging

LAB RESULTS

- Tumor marker: CA 9-19
- Hyperbilirubinemia (mostly conjugated); elevated alkaline phosphatase

OTHER DIAGNOSITCS

Cardiac catheterization

• Measure pressure in right side of heart

TREATMENT

MEDICATIONS

 Chemotherapy with/without chemoradiotherapy

SURGERY

Resection (e.g. pancreaticoduodenectomy)
 Only curative treatment



Figure 32.16 Cytological preparation of a pancreatic fine needle aspirate which demonstrates pancreatic adenocarcinoma. The group on the left is the cancer, with large, pleomorphic nuclei, which overlap with one another. Contrast these with the smaller, regularly spaced pancreatic ductal epithelial cells on the right.



Figure 32.17 Histological appearance of pancreatic adenocarcinoma. The tumor cells form acini, small sack like spaces surrounded by malignant glandular cells.

STOMACH (GASTRIC) CANCER

osms.it/stomach-cancer

PATHOLOGY & CAUSES

 Aggressive adenocarcinoma arising from gastric mucosa

TYPES

Diffuse type (G-DIF): undifferentiated

- Impairment/lack of adhesion molecule E-cadherin
- Genetic mutation (germline, somatic, epigenetic methylation) of CDH1 gene
 → inactivation of CDH1 → nonfunctional
 E-cadherin → unregulated division
 (impaired tumor suppressor function);
 increased ability to spread, invade adjacent
 structures
 - Autosomal dominant inheritance pattern
 - More aggressive than G-INT

Intestinal type (G-INT): well-differentiated

Due to environmental factors; more

common in high-risk populations

 Intercellular adhesion molecules → adherence of tumor cells → arrangement in glandular formations

RISK FACTORS

- Primary cause (G-INT)
 H. pylori infection
- Family history of gastric cancer
- Autoimmune atrophic gastritis
- Lifestyle
 - Smoking, alcohol consumption
- Diet
 - Nitrates, nitrosamines, highly-salted foods; pickled/smoked foods
- Obesity
- Risk increases with age
- More common in individuals who are biologically male

- Protective factors
 - Intake of fruit, vegetables, fiber, folate

COMPLICATIONS

- Metastasis to liver, peritoneum, lymph nodes, etc.
- Paraneoplastic manifestations
 - Seborrheic keratoses, polyarteritis nodosa, Trousseau's syndrome (spontaneous, recurrent, migratory venous thrombosis)

Figure 32.18 Gross pathology of gastric carcinoma. The stomach has been pinned flat. The tumor is found in the antrum.

SIGNS & SYMPTOMS

- Asymptomatic initially
- Early symptoms
 - Vague constitutional symptoms (e.g. malaise, loss of appetite, dyspepsia)
- With disease progression
 - Epigastric pain, nausea, vomiting, dysphagia, weight loss
- If GI bleeding
 - Anemia, melena, coffee-ground hematemesis
- Pseudoachalasia syndrome (difficulty moving food, liquids from esophagus to stomach)
 - If tumor extends to Auerbach's plexus/obstruction occurs near gastroesophageal junction

DIAGNOSIS

DIAGNOSTIC IMAGING

Esophagogastroduodenoscopy with biopsy, barium studies, abdominopelvic CT scan

• Tumor visualization, histopathological analysis, grading, TNM staging, potential for resection

OTHER DIAGNOSTICS

Physical examination

- Enlarged supraclavicular, anterior axillary, periumbilical lymph nodes
- Palpable abdominal mass

Figure 32.19 The histological appearance of a well-differentiated gastric adenocarcinoma of intestinal type. The tumor is composed of disordered glands, the cells of which have large, hyperchromatic nuclei.

TREATMENT

MEDICATIONS

Chemotherapy

- G-INT, G-DIF differ in susceptibility to chemotherapeutic agents
- Eradication of H pylori infection

SURGERY

Resection

OTHER INTERVENTIONS

Chemoradiotherapy

WARTHIN'S TUMOR

osms.it/warthins-tumor

PATHOLOGY & CAUSES

- Uncommon benign tumor; arises from salivary gland
- AKA papillary cystadenoma lymphomatosum
- May involve submandibular/sublingual/ parotid gland (most common)
- Unilateral/bilateral, slow-growing

RISK FACTORS

- Smoking
- Risk increases with age
- More common in individuals who are biologically male

COMPLICATIONS

Malignant transformation (rare)

SIGNS & SYMPTOMS

• Development of painless nodular mass, usually near mandible angle

DIAGNOSIS

OTHER DIAGNOSTICS

Easily palpable tumor

Fine needle aspiration

Histopathological diagnosis

TREATMENT

SURGERY

Local resection/parotidectomy