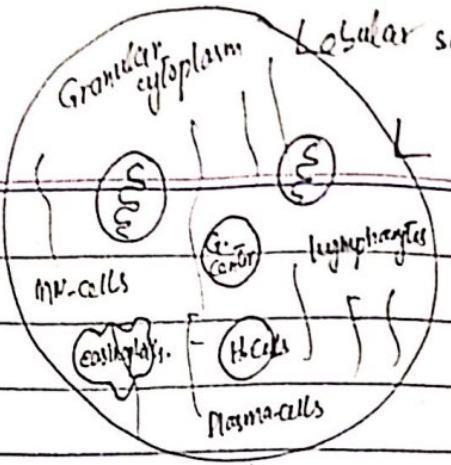
# ENDO

Q. A lady of middle age presents with painless in front of her neck for the last few months. On examination her thyroid gland is diffused enlarge. The provisional diagnosis of thyroiditis is made

Diagnosis ↓ Diffuse enlarge thyroid gland ↓ Thyroiditis

① Name 03 Type of Thyroiditis :-

- Hashimoto Thyroiditis
- Sub-acute Granulomatous de Quervain Thyroiditis
- Sub-acute lymphocytic Thyroiditis




Microscopic :-

Lobular Septa

Thyroid follicles with eosinophills, granular cytoplasm

Hurthle cell seen

Prominent mitochondria

Germinal center

Multiple Cells – Mono-nuclear

Lymphocytes

Plasma cells

Mose Fibrosis

A 35 years old female presented with lethargy, weakness, fatigue, Nausea, constipation, Fracture of bones, Renal stones.

* Diagnosis :-
  Primary hyper-parathyroidism.
* lab-findings:-
  PTH level increase
  $Ca^{+2}$ increase
  Phosphate level decrease (↓)

* Types:-
  Primary hyper-parathyroidism
  Secondary hyper-parathyroidism
  Tertiary hyper-parathyroidism

* Sheehan Syndrome:-
  1. Deficiency of ant. Pituitary hormones.
  2. Ischemic necrosis of ant. pituitary gland which results in arteriolar
  3. Spasm, severe hemorrhage or shock
  4. Usually Postpartum.
  5.

| | |
|---|---|
| ↓ FSH | FSH |
| ↓ LH | LH |
| ↓ TSH | ACTH |
| ↓ ACTH | TSH |
| ↓ GH. | GH |

papillary carcinoma of thyroid

* Classification :-

Adenoma :-

Follicular adenoma
Hurthle-cell adenoma

Carcinoma :-

Papillary Carcinoma
Follicular Carcinoma
Anaplastic Carcinoma
Medullary Carcinoma

Anaplastic

* Morphology of papillary carcinoma of Thyroid :-

① => Gross :-

May be solitary or multifocal
Well-circumscribed or encapsulated

② => Microscopic :-

Papillae formation ē cuboidal cell
Have fibro-vascular Coat/core
Abundant cytoplasm
Nucleus empty, Round glass orphan annie eye.
Nucleus ē line -> Nucleus groove
Nucleus over-lapping
Psammoma bodies -> calcium deposits

Ca++ calcium



Q. What will be the typical laboratory findings in myx-edema, Graves disease, Hashimotos Thyroiditis, multi-nodular goiter and Diffuse Non-toxic goiter.

Q. A lady of her-rec her Thyroid diagnosis

Q. 68 years old female having diabetes developed vomiting and become drowsy. She was taken to emergency and revealed blood-pressure of 95/60 mmHg with pulse-rate of 110/min and cold-extremities. She has acetone-like breath.

* Diagnosis:-

Diabetic ketoacidosis Coma.

* Pathogenesis:-

Glucose level ↓

Insulin ↓ — Glucagon ↑

I ↓ — ↓

L ↑ — Lipophysis ↑ — ↓

F ↑ — FFA-production ↑ — ↓

β-oxidation ↑ ← CPT-1 ↑ — ↓ — ACC ↓ ↑ GAC

↓

Ketone-body production

↓

DKA

* 04 Metabolic features:-

Metabolic acidosis

Hyper-glycemia

Hyper-ketonemia

Poly-dipsia

Poly-Uria

* Clinical

Dehydration

✓ Acetone-like breath

✓ Cold-extremities

✓ Coma

✓ Tachycardia

✓ Tachypnea

Lethargy

Cerebral edema

Google

4 top tests for diabetes

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**Tests for Type 1 Diabetes, Type 2 Diabetes, and Prediabetes**


- A1C Test. The A1C test measures your average blood sugar level over the past 2 or 3 months. ...
- Fasting Blood Sugar Test. ...
- Glucose Tolerance Test. ...
- Random Blood Sugar Test. ...
- Glucose Screening Test. ...
- Glucose Tolerance Test.

CDC https://www.cdc.gov › getting-tested

Diabetes Tests | CDC

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# LIVER

Program

Liver

Q-1

A) What 03 features are needed To establish a diagnosis of cirrhosis?

① → Fibrous Septa
② → Parenchymal Nodules
③ → Vascular Shunting

B) Enumerate Liver-function Tests :-

*- Hepatocyte Integrity :-
- AST
- ALT
- LDH

① Hepatocyte integrity
② Hepatocyte synthetic function
③ Biliary excretory function

*- Biliary Excretory Function :-
- Serum Billirubin (SBR)
- Urine Billirubin (UBR)
- Serum bile-acids (SBA)
- Serum alkaline phasphatase (SAP)
- Serum Glutamyl transpeptidase (SGTP)

*- Hepatocyte Synthetic function :-
- Serum Albumin (SA)
- Coagulation factor (I, V, VII, IX, X)
- Hepatocyte Metabolism
- Serum Ammonia (SA)
- Aminopyrine breath Test



c) Precursor of hepatocellular carcinoma?

*- Biliary Excretory Function :-

- Serum Billirubin (SBR)
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- Hepatocyte Metabolism
- Serum Ammonia (SA)
- Aminopyrine breath Test



c) Precursor of hepatocellular carcinoma?

Lesion

*→ Hepato-cellular Adenoma
*→ Small-cell change
*→ Large-cell change
*→ Low-grade dysplastic nodule
*→ High-grade dysplastic nodule

Q.2

Q. Describe the pathogenesis & Lab-findings of hepatocellular carcinoma?

* Pathogensis :-

Normal hepatocyte
↓
Alchohal
Hepatitis B-virus
Hepatitis C-Virus
↓
Acute hepatitis
↓
Chronic hepatitis
↓
Cirrohis
↓
Hepatocellular Carcinoma.

* Lab-Findings of HCC :-

① → ↑ Alpha-feto protein
② → ALT
③ → AST
④ → Radio-imaging
⑤ → Liver-biopsy



Q.3

Q.No.06

Q. A 58 years old chronic alchohlic man presented with Nodular Liver. Liver biopsy revealed fibrous septa dividing liver paranchyma in different nodules.

* Diagnosis :-

Cirrohsis

* Different Causes :-

- Alchohal Abuse
- Biliary Disease
- ~~Viral hepatitis~~
- Iron Loaded
- Metabolic Disease

} Diseases

- NASH
- Hepatitis B
- Hepatitis C

} Hepatitis

* Morphology of different stages of alchohlic hepatitis.

① → Hepatic steatosis :-

Lipid chop accumulation in hepatocyte

Hepatocytes nucleus moves towards periphery of cells due to lipid accumulation

Nucleus.

Fatty change

* Hepatitis C

* Morphology of different stages of alcoholic hepatitis.

① → Hepatic steatosis :-

Lipid drop accumulation in hepatocyte

Hepatocytes nucleus moves towards periphery of cells due to lipid accumulation

Nucleus.

Fatty change



Alcoholic (Steato) hepatitis :-

- Mallory bodies
- Neutrophilic Reaction.

Alchoholic Steatofibrosis :-

Fibrosis

Micro-nodules → Hobnail appearance

Macro-nodular pattern

Chicken-wire fence pattern 

Laennec cirrhosis.

Q.No.07

Q. A 35 year male patient presents with malaise, fever, Jaundice. His serological Tests are positive for HBsAg.

* Enlist Serum markers of Hep.B.Virus.

HBsAg
HBcAg
HBsAb
HBcAb
HBeAb
HBeAg

* How to detect in Window period :-

By looking at HBcAb, because it's always +ve during window period but HBsAg and HBsAb are disappeared and not detectable respectively.

* Interpretation :

HBsAg -ve
Anti HBcAg -ve → Vaccine is is given against Hep.B
Anti HBsAg -+ve



Q.No.08

Q. A 50 years old patient with history of drinking

Primary billiary cirrhosis.



Q.

| | Primary biliary Cirrhosis | | Primary Sclerosing cholangitis |
|---|---|---|---|
| Age ✓ | Middle age 50 years | ←①→ | Median age 30 years |
| Gender | 90% Female | ←②→ | 70% Male |
| Complication | Sjorgen Syndrome | ←③→ | IBD ✓ |
| | Sclerodema | | U. colitis |
| | Thyroid disease | | Pancreatitis |
| | Celiac disease | | Idiopathic fibrosing |
| Morphology | R. Arthritis | | Disease. |
| ★ | Florid duct lesion ✓ | ←④→ | Circumferential onion skin fibrosis |
| | Portal-Portal septal fibrosis | ⑦ | ★ Tombstone scar |
| ✓ | Mallory-Denk bodies | | - |
| ✓ | Nodular Regenerative hyperplasia | | - |
| ✓ | Hepatomegaly | | - |
| Antibodies | AMA | ←⑤→ | P-ANCA 65% |
| 95% | (Anti-mitochondrial antibody) | | |
| | ANCA (Anti-nuclear antibody) | | |
| Enzyme ↑ | Elevated Serum alkaline phosphate | ←⑥→ | ↑ Alkaline phosphate |
| Pigments | | | Bilirubin |
| | | | Hypergamaglobinemia |



Q.9

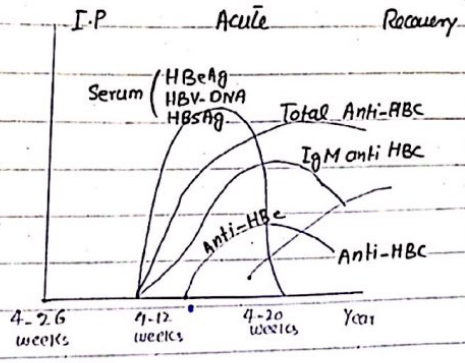
Q. English Metabolic Liver Disease:

Q. No:05 :-

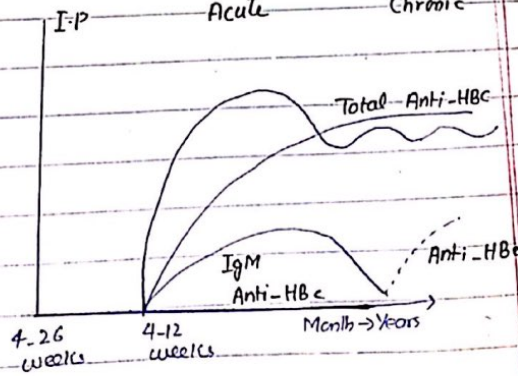
A 38-year old-male patient presents with malaise, fever, jaundice. His serological Test are +ve for HBsAg.

* Graphical Representation :-

Acute Infection :-




Chronic Infection :-



# GIT AND ORAL CAVITY

Diagnosis — Tumor markers.
Pathogenesis — Genetic mutation
Morphology — Gross, Microscopic — Prognostic factors + Dependance
Field of cancerization.

Q- A 62 old-male suffering from altered bowel habits, bleeding per-rectum and weight loss. His Colonoscopy reveals napkin ring constriction of colon. Chalky necrosis. Mass with irregular margins - Colonoscopy.

* Diagnosis :-
Colorectal Carcinoma.

* Altered bowel habits
* weight loss
* Colon mass
* colonoscopy
* Napkin ring constriction.
* Chalky necrosis.

* Pathogenesis :-
Normal
↓ APC-mutation ✓
Hyper-proliferation
↓
① - Early Adenoma
↓ KRAS mutation ✓
② - Inter-mediated adenoma
↓ Chromosome 18 loss ✓
③ - Late adenoma
↓ P-53 loss ✓
Cancer

☆ APC-Mutation
K-RAS
Ch-18 loss
P53 loss.

* Morphology :-
Gross →
→ Ulcer formation
→ Chalky Necrosis
Microscopic →
In Proximal Colon → Polypoid, Exophytic mass ✓
✓ Hyper-chromatic Nuclei
Necrosis ✓
✓ Signet-ring cells.
✓ Large Vacule, Expanded cytoplasm

Q. How will you differentiate diffuse type of gastric carcinoma from intestinal type.?

| Intestinal Carcinoma | Diffuse Carcinoma (Gastric) |
|---|---|
| Well differentiated | Poor differentiated |
| Mestastize in Liver | Metastize in ovary, peritonium |
| Better prognosis | Poor prognosis |

Gross

Morphology

Bulky Tumor
Heaped up border
Composed of columnar cells
Desmoplastic Stroma

Linitis Plastica type
Leather bottle stomach

Signet ring cells

crescent shape Nuclei

large Mucin vacule.

b) Define Early gastric carcinoma & Aduanced gastric carcinoma?

Early Gastric carcinoma:-
Lesion confined to mucosa and sub-mucosa

Advanced Gastric carcinoma:-
Extend below sub-mucosa into muscular wall and spread widely



what's Barret Oesphagus

Dia
Pal

- What's Barret Oesophagus
- Pathogenesis
- Morphology
- Stain

Oesophageus Tumor according To Location.

c) Barret Oesophagus :-

① Intestinal Metaplasia due to GERD

② Involve Lower end of oesophagus.

Pathogenesis :-

GERD (Chemic injury)
↓
Intestinal Metaplasia (Regurgitation)
↓
Squamous cell epithelium
↓
Columnar epithelium with Goblet cell.

Morphology :-

Gross :-

* Well-defined salmon pink area
* Velvety appearance

Microscopic :-

Dysplasia (High grade)
Squamous cell convert into columnar cell.
Intestinal metaplasia
Goblet-cell present

Stain :-

PAS stain
AB stain.

Esophagus Tumor
According: Location.
Sq. cell cell carcinoma : Upper and Middle part of esophagus
Adenocarcinoma. Distal 3rd of esophagus.

Q- A 62 old-male
bleeding per-re
reveals napic
Mass with
* Diagnosis :-
* Pathogenesis :
① ② ③
* Morphology
Gross →
Microsco
In Proxi

Diagnosis Tumor markers.
Pathogenesis Genetic mutation
Morphology - Gross

- What's Celiac Disease?
- Location
- Anti-bodies
- Morphology
- complication.

Q. Celiac Disease:-

Immune mediated enteropathy by ingestion of gluten containing feed barley w/s.

Location:- Duodenum
Proximal
Jejunum.

Anti-body:- *
IgA antibodies against tissue transglutaminase
IgA anti-endomysial antibody.

Morphology:-
(MARSH CRITERIA)
Villous atrophy
Crypt hyperplasia
Intra-epithelial lymphocytosis.
Loss of mucosa and Brush-borders.
↑ No of eosinophills, Plasma cells, mast cells.

Complication:-
Enteropathy assasiated T-cell lymphoma (EATL)
Small Intestine adenocarcinoma.

Q. Sub-mandible gland Tumor
Mixed-nature.

* Diagnosis:
Pleomorphic Adenoma

* Mutation:
PLAG-1

* Morphology:

Gross:
Well demarcated
Round
Benign Tumor.

Pleomorphic adenoma


Microscopic:
Myxoid stroma
Combination of epithelial cells and myoepithelial cells
Arranged Ducts
Acini
Tubules.

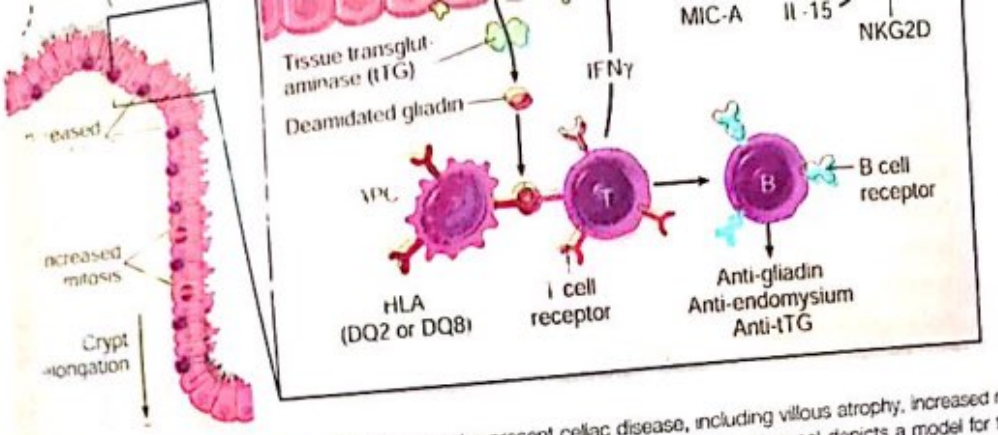



Figure 17-25 The left panel illustrates the morphologic alterations that may be present celiac disease, including villous atrophy, increased numbers of intac
epithelial lymphocytes (IELs) and epithelial proliferation with crypt elongation (compare to
celiac disease. Note that both innate (CD8+ intraepithelial T cells, activated by IL-15) and adaptive (CD4+ T cells, and B cells sensitization to gliadin) immu
components are involved in the tissue responses to gliadin.

- Antibodies to parietal cells that can be detected in serum and gastric secretions
- Reduced serum pepsinogen I concentration
- Endocrine cell hyperplasia
- Vitamin $B_{12}$ deficiency
- Defective gastric acid secretion (achlorhydria)

**Table 17-2** Characteristics of *Helicobacter pylori*–Associated and Autoimmune Gastritis

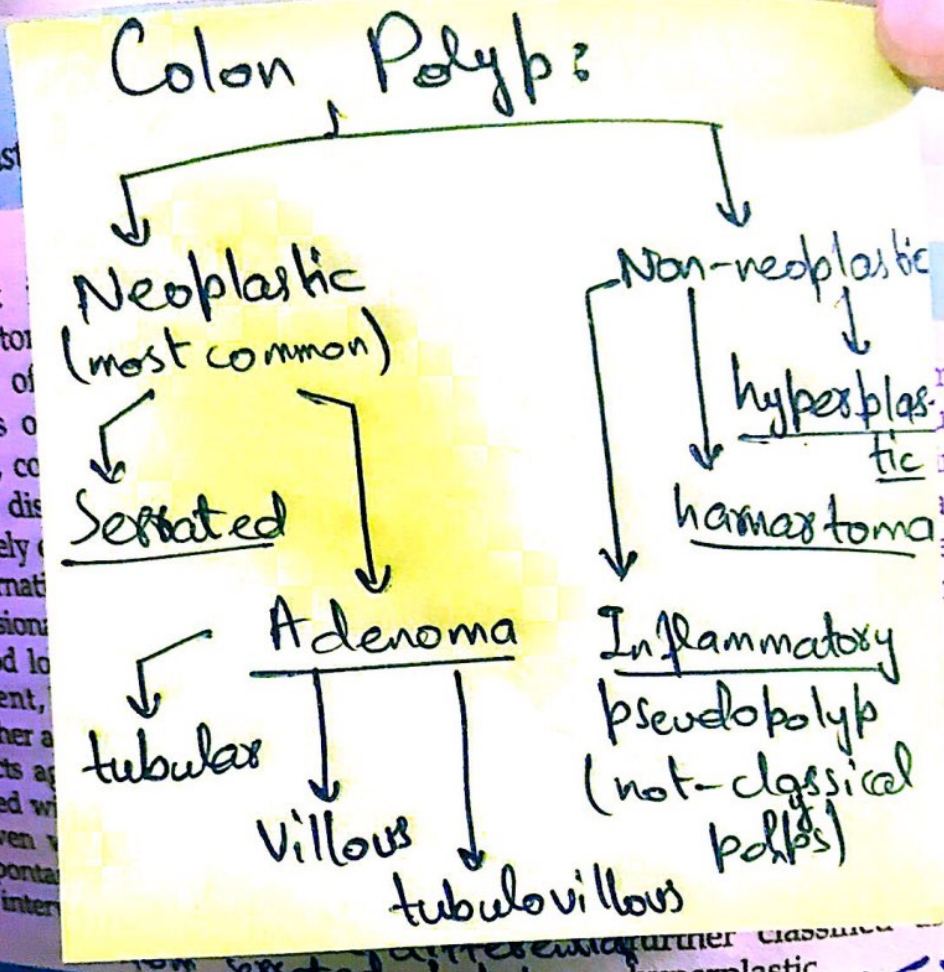
| | *H. pylori*–Associated | Autoimmune |
|---|---|---|
| Location | Antrum | Body |
| Inflammatory infiltrate | Neutrophils, subepithelial plasma cells | Lymphocytes, macrophages |
| Acid production | Increased to slightly decreased | Decreased |
| Gastrin | Normal to decreased | Increased |
| Other lesions | Hyperplastic/inflammatory polyps | Neuroendocrine hyperplasia |
| Serology | Antibodies to *H. pylori* | Antibodies to parietal cells ($H^+$,$K^+$-ATPase, intrinsic factor) |
| Sequelae | Peptic ulcer, adenocarcinoma, MALToma | Atrophy, pernicious anemia, adenocarcinoma, carcinoid tumor |
| Associations | Low socioeconomic status, poverty, residence in rural areas | Autoimmune disease; thyroiditis, diabetes mellitus, Graves disease |

ple cell types, and their precise roles in the pathogenesis of Crohn disease have yet to be defined. However, all

**Table 17-9** Features That Differ between Crohn Disease and Ulcerative Colitis

| Feature | Crohn Disease | Ulcerative Colitis |
|---|---|---|
| **Macroscopic** | | |
| Bowel region | Ileum ± colon | Colon only |
| Distribution | Skip lesions | Diffuse |
| Stricture | Yes | Rare |
| Wall appearance | Thick | Thin |
| **Microscopic** | | |
| Inflammation | Transmural | Limited to mucosa |
| Pseudopolyps | Moderate | Marked |
| Ulcers | Deep, knife-like | Superficial, broad-based |
| Lymphoid reaction | Marked | Moderate |
| Fibrosis | Marked | Mild to none |
| Serositis | Marked | Mild to none |
| Granulomas | Yes (~35%) | No |
| Fistulae/sinuses | Yes | No |
| **Clinical** | | |
| Perianal fistula | Yes (in colonic disease) | No |
| Fat/vitamin malabsorption | Yes | No |
| Malignant potential | With colonic involvement | Yes |
| Recurrence after surgery | Common | No |
| Toxic megacolon | No | Yes |

All features may not be present in a single case.




Scanned with CamScanner

**Table 16-4** Histologic Classification and Incidence of the Most Common Benign and Malignant Tumors of the Salivary Glands

| Benign | Malignant |
|---|---|
| Pleomorphic adenoma (50%) (mixed tumor) | Mucoepidermoid carcinoma (15%) |
| Warthin tumor (5%-10%) | Adenocarcinoma (NOS) (10%) |
| Oncocytoma (1%) | Acinic cell carcinoma (5%) |
| Other adenomas (5%-10%) | Adenoid cystic carcinoma (5%) |
| Basal cell adenoma | Malignant mixed tumor (3%-5%) |
| Canalicular adenoma | Squamous cell carcinoma (1%) |
| Ductal papillomas | Other carcinomas (2%) |

NOS, Not otherwise specified.
Data from Ellis GL, Auclair PL: Tumors of the Salivary Glands. Atlas of Tumor Pathology, Fourth Series. Washington, DC, Armed Forces Institute of Pathology, 2008.

benign and malignant tumors is listed in Table 16-4; not included are the rare benign and malignant mesenchymal neoplasms.

As indicated in Table 16-4, a small number of neoplasms makes up more than 90% of salivary gland tumors, and so our discussion is restricted to these. Overall, these neoplasms are relatively uncommon and represent less than 3% of all tumors in humans. About 65% to 80% arise within the parotid, 10% in the submandibular gland, and the remainder in the minor salivary glands, including the sublingual glands. Approximately 15% to 30% of tumors in the parotid glands are malignant. In contrast, approximately 50% of submandibular, 50% of minor salivary gland, and 70% to 80% of sublingual tumors are cancerous. Thus, [illegible] gland tumor being malignant