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GNT pathology cases file Don't forget to check it frequently <u>Click</u>







Bassam Alasmari Rania Almutiri اللهم لا سهل الا ماجعلته سهلا و انت تجعل الحزن إذا شئت سهلا





1	Know the classification of intestinal tumors (small intestine and colon)									
2	Know the definition of a polyp.									
3	Compare adenomatous/neoplastic polyps and non neoplastic polyps (hyperplastic polyps, inflammatory polyp and hamartomatous polyp) with respect to pathology (gross and microscopic features).									
4	Know the three subtypes of adenomatous polyps, eg, tubular adenoma, villous adenoma, tubulovillous adenoma.									
5	Describe the adenomatous polyp-cancer sequence and the features associated with risk of malignancy, eg, polyp size, histologic architecture, and severity of epithelial dysplasia.									
6	Describe the classification of the hereditary syndromes involving the Gl tract and the syndromes associated with an increased risk of cancer (Peutz-Jeghers syndrome, familial adenomatous polyposis, and hereditary nonpolyposis colorectal carcinoma)									
Overview										
	Tumors of the small and large intestines									
1	Polyps Carcinoma Carcinoid Lymphoma									
Sigmoid colon Most common site Gl polyps, diverticula (outpouching/herniation of mucous into the bowel wall) and cancer										

Polyps

		Non-neoplastic	polyps (90%)			Neoplastic polyps (10%)	
	Hyperplastic polyps	Hamartomatous polyps	Inflammatory polyps	Lymphoid polyps		Adenoma	
Non-neoplastic polyps							
Hyperplastic polyps (Increased number of cells)							
- Asymptoma	itic, more than 5	0% are located in t	he rectosigmoid	- Most o	common type in	adults	
- Sawtooth (surfa (أسنان المنشار	ace & Star shaped	crypts				
- Composed	of well-formed g	lands and crypts lii	ned by differentia	ted goblet or al	bsorptive cells.		
- <u>No</u> malignant potential or polyposis syndromes, and No dysplasia.							
Hamartomatous polyps (Normal tissue but in <u>disorganized</u> matter)							

No tendency to cancer p	Juvenile polyps progression, only pain & sometimes bleeding	2) Peutz-Jeghers syndrome		
Juvenile Polyps (retention polyp)	Juvenile polyposis	- Rare, autosomal dominant		
 Developmental malformations affecting the glands and lamina propria Commonly occur in children under 5 years old in the rectum. In adult called <u>retention</u> <u>polyp</u>. 	Autosomal dominant:- TGF-β signaling pathway abnormalities- Juvenile polyps; risk of gastric, small intestinal, colonic, and pancreatic adenocarcinomaCowden syndrome:- Abnormality in PTEN- Hamartomatous polyps, lipomas,	 hamartomatous polyps accompanied by mucosa and cutaneous pigmentation around the lips, ora mucosa, face and genitalia, present with red blood in stool. Polyps tend to be large and pedunculated (bleeding). Increased risk of developing carcinoma of the pancreas, breast, lung, ovary and uterus. Mean age at presentation (10-15 years old) 		
 No malignant potential Smooth eroded surface with numerous mucus retention 	ganglioneuromas, inflammatory polyps; increased risk for colon cancer and cancer of thyroid and breast	 Gl lesions: Arborizing polyps (Small intestine > Colon > Stomach; Colonic adenocarcinoma) Selected extragastrointestinal manifestations 		
cysts, typical of sporadic juvenile polyps.	Cronkhite-Canada syndrome: - Non-Hereditary polyposis syndrome - Polyps plus ectodermal abnormalities (Nail atrophy, hair loss, abnormal skin pigmentation) cachexia (weight loss), and anemia	Mucocutaneous pigmentation; increased risk for thyroid, breast, lung, pancreas, gonadal, and bladder cancers. - LKB1/STK11 encodes a tumor suppressive protein kinase that regulates cellular metabolism		
Infl	ammatory polyps	Lymphoid polyps		
 longstanding IBD, especially in <u>chronic ulcerative colitis</u>. Represent an exuberant reparative response to longstanding mucosal injury called pseudopolyps. 		Collection of lymphoid tissue in the lamina propria . Common in children . It can be physiological & forms lymphoid-follicle like masses in the lamina propria .		

Polyps Non-neoplastic polyps (90%) Neoplastic polyps (10%) Hyperplastic Hamartomatous Inflammatory Lymphoid Adenoma polyps polyps polyps polyps **Neoplastic polyps** If the polyp is single Adenomatous Polyp (adenoma) - Occur mainly in large bowel - Sporadic (Acquired, few) and familial (>100 polyps) - Vary from small pedunculated (مثل العنق) to large sessile. - Epithelium proliferation and dysplasia - Divided into three types (Depending on surface/villous architecture) :

1)Tubular adenoma	2) Villous adenoma	3) Tubulovillous adenoma
 Most common less than 25% villous architecture (smooth surface) Represents 75% of all neoplastic polyps. 75 % occur in the distal colon and rectum. Sigmoid colon most common site. Lining epithelium is dysplastic. 	 villous architecture over 50% The least common, largest and most ominous of epithelial polyps (most likely to undergo malignant transformation). Age: 60 to 65 years, 75% located in rectosigmoid area Present with rectal bleeding or anemia, large ones may secrete copious amounts of mucoid material rich in protein and potassium which will result in : Large tumors can produce hypoalbuminemia and hypokalemia (causes secretory diarrhea). 	-villous architecture between 25 and 50%. - 20%–30% of polyps - Intermediate in size, degree of dysplasia and malignant potential between tubular and villous adenomas.

Multiple neoplastic polyps Familial Polyposis Syndrome (FAP)							
Patients have genetic tendencies to develop neoplastic polyps. ★Mutated Gene: APC ★Gl lesion: Multiple Adenomas. - Divided into three types:							
□Familial polyposis coli (FPC)	Gardener's syndrome	□Turcot syndrome					
 Genetic defect of Adenomatous polyposis coli (APC). APC gene located on the long arm of chromosome 5 (5q21). APC gene is a tumor suppressor gene Innumerable neoplastic polyps in the colon (500 to 2500) Polyps are also found elsewhere in alimentary tract The risk of colorectal cancer is 100% by midlife Treatment : colectomy "due to risk of colorectal cancer developing " Classic FAP : 10-15 years old (congenital RPE hypertrophy) Attenuated FAP : 40-50 years old . 	 Mean age at presentation: 10-15 years old Selected extragastrointestinal manifestations: Polyposis coli, Multiple osteomas, Desmoids (tumor like fibroma in the soft tissues), Skin (Epidermal) cysts, Fibromatosis. 	 Mean age at presentation: 10-15 years old Selected extragastrointestinal manifestations: Polyposis coli, CNS tumors, Medulloblastoma, Glioma, Fibromatosis. 					

Relationship of Neoplastic Polyps to Carcinoma

Adenoma to carcinoma sequence is documented by several genetic alterations and morphological appearance.



		- Asymptomatic, rectosigmoid, No malignant potential or polyposis syndromes.
	Hyperplastic p	- Sawtooth surface & Star shaped crypts, composed of well-formed glands and crypts lined by differentiated goblet or absorptive cells.
		1/ Juvenile Polyps:
Non-neopla		- Affecting the glands and lamina propria, No malignant potential.
stic polyps		- In adult called <u>retention polyp</u> .
	Hamartomatous	- Smooth eroded surface with numerous mucus retention cysts, typical of sporadic juvenile polyps.
		2/ Juvenile polyposis
		3/ Peutz-Jehgers syndrome:
		- hamartomatous polyps accompanied by mucosal and cutaneous pigmentation around the lips, oral mucosa, face and genitalia, present with red blood in stool.
		- Polyps tend to be large and pedunculated.
		- Increased risk of developing carcinoma of the pancreas, breast, lung, ovary and uterus.
	Inflammatory p	- longstanding IBD (<u>chronic ulcerative colitis)</u> , have Pseudopolyps.
	Lymphoid p	_
		- In large bowel, small pedunculated to large sessile.
		- Epithelium proliferation and dysplasia
		1/ Tubular adenoma: less than 25% villous architecture in the distal colon and rectum.
	Adenomatous	2/ Villous adenoma: villous architecture over 50% in rectosigmoid area
		- Present with rectal bleeding or anemia, secrete copious amounts of mucoid material rich in protein and potassium (hypoalbuminemia and hypokalemia).
Neoplastic polyps		3/ Tubulovillous adenoma: villous architecture between 25 and 50%, Intermediate in size, degree of dysplasia and malignant potential between tubular and villous adenomas.
	Familial	1/ Familial polyposis coli: Genetic defect of Adenomatous polyposis coli (APC) on chromosome 5 (5q21).
	Polyposis	2/ Gardener's syndrome: Polyposis coli, Multiple osteomas, Desmoids, Skin (Epidermal) cysts, Fibromatosis.
	Syndrome (FAP)	3/ Turcot syndrome: Polyposis coli, CNS tumors, Medulloblastoma, Glioma, Fibromatosis.



Overview



Adenocarcinoma

- Adenocarcinoma of the colon is the most common malignancy of the Gl tract and is a major cause of morbidity and mortality worldwide.

- Constitutes 98% of all cancers in the large intestine.

 \star Incidence peaks at 60 to 70 years of age

	edisposing factors 1. IBD (ulcerative colitis) , adenomas (more villous) , polyposis syndrome. 2. Diet appears to play an important role in the risk for colon cancer: - Alcohol - Reduced intake of vit A, C & E. - Low fibre diet High fat content (Such as Fast food). why? * The reason: It is theorized that reduced fiber content leads to decreased stool bulk and altered composition of the intestinal microbiota. This change may increase synthesis of potentially toxic oxidative by-products of bacterial metabolism, which would be expected to remain in contact with the colonic mucosa for longer periods of time as a result of reduced stool bulk. High fat intake also enhances hepatic synthesis of cholesterol and bile acids, which can be converted into carcinogens by intestinal bacteria.								
Predisposing factors									
★ Prevention	Several epidemiologic studies suggest that aspirin or other NSAIDs have a protective effect. This is consistent with studies showing that some NSAIDs cause polyp regression in FAP patients in whom the rectum was left in place after colectomy.								
	70% are in the rectum, rectosigmoid and sigmoid colon.								
Morphology	Left-sided carcinomas tend to be annular, encircling lesions with early symptoms of obstruction	Right-sided carcinomas tend to grow as polypoid, fungating masses, obstruction is uncommon (bleeding & iron deficiency anaemia)							
	★ Mucinous adenocarcinoma secret abundant mucin that may dissect through cleavage planes in the wall.								
	If located closer to the anus: change in bowel habit, feeling of incomplete defecation, PR bleeding								
Signs and symptoms	A tumor that is large enough to fill the entire lumen of the bowel may cause bowel obstruction								
	★ Right-sided lesions are more likely to bleed while left-sided tumors are usually detected later and could present with bowel obstruction.								
colorectal carcinoma	★ Serum levels of carcinoembryonic antigen (CEA) - A to spread. They are helpful in monitoring for recurrence	umor marker , oncofetal protein - are related to tumor size and extent of of tumor after resection not used in diagnosis .							
Two	Car pathogenetically distinct pathways for the development of col	cinogenesis on cancer, both seem to result from accumulation of multiple mutations:							
	1)The APC/B-catenin pathway (85 %)	2) The DNA mismatch repair genes pathway							
- Chromosomal mutations in a s - Familial Aden the cause of fan individuals carry years.	 - Chromosomal instability that results in stepwise accumulation of mutations in a series of oncogenes and tumor suppressor genes. - Familial Adenomatous Polyposis: Hereditary mutation of the APC gene is the cause of familial adenomatous polyposis (FAP), where affected individuals carry an almost 100% risk of developing colon cancer by age 40 years. - These are referred to as MSI high, or MSI-H, tumors: 10%-15% of sporadic cases. - There is accumulation of mutations, Five DNA mismatch repair genes (MSH2, MSH6, MLH1, PMS1, AND PMS2) - give rise to the hereditary non polyposis colon carcinoma (HNPCC) syndrome. - This result in microsatellite instability and permit accumulation of mutations in numerous genes. 								
NORMAL COL	ON MUCOSA AT RISK ADENOMAS CARCINOMA	★ If these mutations affect genes involved in cell survival and proliferation, cancer may develop.							
Mucosa Submucosa Muscularis propria		 It progress from normal to sessile serrated adenomas to adenocarcinoma May produce abundant mucin that accumulates within the intestinal wall, and these carry a poor prognosis 							
Germline (inheri or somatic (ar mutations of o suppressor gr ("first hit")	ted) Methylation abnormalities accer suppressor genes ("second hit") Protooncogene mutation additional cancer suppressor genes ("second hit") Protooncogene mutation additional cancer suppressor genes ("second hit") Additional mutations ("second hit") Additional cancer suppressor genes ("second hit") Additional	NORMAL COLON Mucosa Submucosa Muscularis proprint Germline (inherited) or somatic (acquired) Materiation of second allele by LOH, mutation, or mutation, or Microsatellite instability/ mutation, or Microsatellite instability/ mutation, or Microsatellite							
Mismatch repair g e.g., MSH2 at 2	enes, β-catenin K- <i>ras</i> at 12p12 p53 at 17p13 Many genes D22 MSH2 LOH at 18q21	MLH1, MSH2 (MSH6, PMS1, PMS2) TGF\$RI, BAX, BRAF, TGF4, IGER, others							

In female slides only

<u>Girls' dr</u>: lt's just for your knowledge in the clinical practice ما أعتقد يجيبونها بالامتحان

6

Tumor-Node-Metastasis (TNM) classification of colorectal carcinoma

Tumor								
Tis carcinoma in situ, intramucosal carcinoma								
T1	tumor invades submu	tumor invades submucosa						
T2	tumor invades into b	ut not through muscula	aris propria					
Т3	tumor invades throug	h the muscularis propr	ia					
T4	tumor invades adjace	nt organs or visceral p	peritoneum					
	R	egional lymph node	es					
NX	regional lymph nodes	cannot be assessed						
NO	no regional lymph noo	le metastasis						
N1	metastasis in 1 - 3 reg	ional lymph nodes						
N2	metastasis in 4 or mo	re regional lymph node	es					
		Distant metastasis						
MX	MX Distant metastasis cannot be assessed							
M0	no distant metastasis							
M1	Distant metastasis or	seeding of abdominal o	organs					
	Colorect	al staging and	survival					
Stage	Tumor-No	de-Metastasis (TN	N) criteria	E Veer curring (%)				
Stage	Т	N	Μ	5-fear survivar (%)				
l	T1, T2	NO	M0	74				
	1	u						
llA	Т3	NO	M0	67				
llB	T4	NO	M0	59				
IIIA	T1, T2	N1	M0	73				
IIIB	T3, T4	N1	M0	46				
IIIC	Any T	N2	M0	28				

The most important prognostic factors are depth of invasion and the presence or absence of lymph node metastases and distant metastasis.

Any N

Any T

M1

١V

Carcinoid Tumors

- Neoplasms aris	sing from endocrine cells found along the length of GIT mucosa. 🚽 The peak incidence: sixth decade, but they may appear at any age.
★They compose	less than 2% of colorectal malignancies
- Almost half of	f small intestinal malignant tumors:
- 60 to 80% app	endix and terminal ileum - 10 to 20% rectum.
* Behavior	Aggressive behavior correlates with: 1. Site of origin: Appendiceal and rectal carcinoids infrequently metastasize, even though they may show extensive local spread 90% of ileal, gastric, and colonic carcinoids that have penetrated halfway through the muscle wall have spread to lymph nodes and distant sites at the time of diagnosis, especially those larger than 2 cm in diameter. 2. Depth of local penetration 3. Size of the tumor
Morphology	 ★Grossly : A solid, yellow-tan appearance ★Microscopically: The cells are monotonously similar, having a scant, pink granular cytoplasm and a round-to-oval stippled nucleus. Ultrastructural features (By electron microscope): neurosecretory electron dense bodies in the cytoplasm
Clinical features	- Asymptomatic - May cause obstruction, intussusception or bleeding. - May elaborate hormones (على حسب الهرمون اللي يُفرز): Zollinger-Ellison (secretes gastrin), Cushing's carcinoid or other syndromes.
	Carcinoid syndrome
- 1% of carcinoid	I tumor & in 20% of those of widespread metastasis
- Paroxysmal flu	ishing, episodes of asthma-like wheezing, right-sided heart failure, attacks of watery diarrhea, abdominal pain
- The principal o	chemical mediator is <mark>serotonin</mark>
- The syndrome secreted into po these symptoms	is classically associated with ileal carcinoids with hepatic metastases, Why ? In carcinoid tumor with normal liver: the abnormal serotonin is ortal circulation and then detoxified in the liver. But if there's liver metastasis, serotonin is secreted directly into the systemic circulation leading to 3,
	- Due to serotonin and other bioactive compounds (e.g., histamine, bradykinin)
	- Flushing of the skin (75%–90% of cases) Due to vasodilation; may be triggered by emotion, alcohol, other foods
Clinical findings	- Motility related Diarrhea (>70% of cases), due to increased bowel motility from serotonin
inteniõs	- Intermittent wheezing and dyspnea (25% of cases), due to bronchospasm
	- Facial telangiectasia
	- Tricuspid regurgitation and pulmonary stenosis, Because serotonin increases collagen production in the valves (right sided)
Serotonin and diarrhea	Patients with carcinoid syndrome often suffer from diarrhea, which has both a secretory and a motor component (secretory & motility related diarrhea). The secretory component of carcinoid diarrhea is attributable to excessive serotonergic stimulation of submucosal secretomotor neurons; the motor component includes faster small bowel and colon transit and an exaggerated tonic response of the colon to ingestion of a meal
	lymphoma
	Lymphoma

- Most often <mark>low-grade lymphomas</mark> arising in mucosal-associated lymphoid tissue (<mark>MALT</mark>) lymphoma or <mark>high-grade</mark> non-Hodgkin's <mark>lymphomas</mark> of B cell type.

- May occur in any part of the intestine;

- The ileocecal region is a favored site for Burkitt's lymphoma with right iliac fossa mass especially in children .

Tumors marker

 male's dr : tumor markers are not specific and can elevated in others diseases so if we have patients with high CEA we should do endoscopy

A tumor marker is a substance found in the blood , urine or body tissues that can be elevated in cancer, among other tissue types

Carcinoembryonic antigen (CEA)

Carbohydrate antigen (CA19-9)

Useful to assess disease recurrence (late stage)

male's dr : for your knowledge

CEA levels may also be raised in some non-neoplastic conditions like ulcerative colitis pancreatitis, cirrhosis COPD, Crohn's disease as well as in smokers

CA19-9 are raised in in patients with colon cancer and pancreatic cancer, esophageal cancer and hepatocellular carcinoma. Apart from cancer, elevated levels may also occur in pancreatitis, cirrhosis

Tissue inhibitor of metalloproteinases 1 (TIMP1)

Early as well as late stage disease

© Summary

		- Asymptomatic, rectosigmoid, No malignant potential or polyposis syndromes.
	Hyperplastic p	- Sawtooth surface & Star shaped crypts, composed of well-formed glands and crypts lined by differentiated goblet or absorptive cells.
		1/ Juvenile Polyps:
Non-		- Affecting the glands and lamina propria, No malignant potential.
neoplastic		- In adult called <u>retention polyp</u> .
polyps	Hamartomatous	- Smooth eroded surface with numerous mucus retention cysts, typical of sporadic juvenile polyps.
		2/ Juvenile polyposis
		3/ Peutz-Jehgers syndrome:
		- hamartomatous polyps accompanied by mucosal and cutaneous pigmentation around the lips, oral mucosa, face and genitalia, present with red blood in stool.
		- Polyps tend to be large and pedunculated.
		- Increased risk of developing carcinoma of the pancreas, breast, lung, ovary and uterus.
	Inflammatory p	- longstanding IBD (<u>chronic ulcerative colitis)</u> , have Pseudopolyps.
	Lymphoid p	-
		- In large bowel, small pedunculated to large sessile.
		- Epithelium proliferation and dysplasia
		1/ Tubular adenoma: less than 25% villous architecture in the distal colon and rectum.
	Adenomatous	2/ Villous adenoma: villous architecture over 50% in rectosigmoid area
		- Present with rectal bleeding or anemia, secrete copious amounts of mucoid material rich in protein and potassium (hypoalbuminemia and hypokalemia).
Neoplastic polyps		3/ Tubulovillous adenoma : villous architecture between 25 and 50%, Intermediate in size, degree of dysplasia and malignant potential between tubular and villous adenomas.
polypo	Familial	1/ Familial polyposis coli: Genetic defect of Adenomatous polyposis coli (APC) on chromosome 5 (5q21).
	Polyposis Syndrome (FAP)	2/ Gardener's syndrome: Polyposis coli, Multiple osteomas, Desmoids, Skin (Epidermal) cysts, Fibromatosis.
		3/ Turcot syndrome: Polyposis coli, CNS tumors, Medulloblastoma, Glioma, Fibromatosis.
		Predisposing factors:
		الBD، adenomas, polyposis syndrome، Alcohol, Reduced intake of vit A, C & E، Low fibre diet, High fat content.
		Morphology:
		- Left-sided carcinomas tend to be annular, encircling lesions with early symptoms of obstruction
Adenoca	arcinoma	- Right-sided carcinomas tend to grow as polypoid, fungating masses, obstruction is uncommon
		Symptomes:
		- closer to the anus: change in bowel habit, feeling of incomplete defecation, PR bleeding
		- A tumor that is large enough to fill the entire lumen of the bowel may cause bowel obstruction
		Carcinogenesis:
		 The APC/B-catenin pathway: mutation of the APC gene, K-ras Mutation , chromosome 18 and p53 The DNA mismatch repair genes pathway: Five DNA mismatch repair genes (MSH2, MSH6, MLH1, PMS1, AND PMS2), give rise to the <u>hereditary non polyposis colon carcinoma (HNPCC) syndrome</u>
		Morphology: Ultrastructural features: neurosecretory electron dense bodies in the cytoplasm
Carcinoi	d Tumors	Clinical features: obstruction, intussusception or bleeding, Zollinger-Ellison, Cushing's carcinoid or other syndromes, diarrhea due to excessive secretion of serotonin.
Lvmr	homa	- Most often <mark>low-grade lymphomas</mark> arising in mucosal-associated lymphoid tissue (<mark>MALT</mark>) lymphoma or <mark>high-grade</mark> non-Hodgkin's lymphomas of B cell type.
Lympnoma		- The ileocecal region is a favored site for Burkitt's lymphoma.

MCQs							
O1 A 55-year-old is identified in the r Microscopic examinarchitecture, but no	man unde ectum and nation reve signs of r	ergoes rou l resected. eals goblet nuclear aty	tine colonoscopy. A The surgical specim cells and absorptive pia. Which of the fol	small, raised, mucosa ien is shown in the in e cells with exaggerat llowing is the most lil	al nodule m nage. red crypt kely diagno	neasuring (osis?).4 cm in diameter
A) Adenocarcinom	าล	B) Hyper	rplastic Polyp	C) Inflammatory F	Polyp	D) Peutz	-Jeghers Polyps
02 A 65-year-old woman undergoes routine colonoscopy. During the procedure, a 2-cm mass is identified in the rectosigmoid region and resected. The surgical specimen is shown in the image . Microscopic examination shows irregular crypts lined by pseudostratified epithelium with hyperchromatic nuclei, without dysplastic features. Which of the following is the most likely diagnosis for this patient's colonic lesion?							
A) Adenocarcinom	าล	B) Carci	noid Tumor	C) Tubular Adenor	ma	D) Villou	is Adenoma
03 A 63-year-old woman complains of rectal bleeding of 1 week in duration. Laboratory studies show hypochromic, microcytic anemia (hemoglobin = 7.6g/dL and MCV = 70µm3). Colonoscopy reveals a large polypoid mass, which is removed (surgical specimen shown in the image). The arrow points to a malignant tumor. The patient asks about the relative risk of cancer arising in various types of gastrointestinal polyps. Which of the following types of colonic polyps is most likely to undergo malignant transformation?							
A) Tubular Adenoi	na	B) Villou	s Adenoma	C)Hyperplastic Po	olyp	D) Lymp	hoid Polyp
04 A 59-year-old man complains of progressive weakness. His friends have noticed that he has become pale, and he reports that his stools are tinged with blood. On abdominal palpation, there is fullness in the right lower quadrant. Laboratory studies show iron-deficiency anemia, with a hemoglobin level of 7.4g/dL. Stool specimens are positive for occult blood. Colonoscopy reveals an elevated and centrally ulcerated lesion of the sigmoid colon. The biopsy is shown in the image. Which of the following is the most likely diagnosis?							
A) Gastrointestinal tumor	stromal	B) Mucin	ous Cystadenoma	C) Lymphoma		D) Adenc	carcinoma
05 A portion of the large bowel was removed from a 34-year-old man with a familial disease that affects his gastrointestinal tract. The surgical specimen is shown in the image. This patient most likely carries a germline mutation in which of the following genes?							
A) APC		B) C-my	С	C) p53		D) Ras	
MOOF	C)1	00	07	0	Δ	ОГ

MCQs	01	02	03	04	05
Answer key	В	С	В	D	A

MCQs

06 Defect in which of the following genes can lead to Familial polyposis coli (FPC)?									
A) P53 located on the short arm of chromosome 5 (5p21)	B) P53 located on the long arm of chromosome 5 (5q21)	C) APC gene located on the long arm of chromosome 5 (5q21)	D) APC gene located on the short arm of chromosome 5 (5p21)						
07 Which of the following is true about Peutz-Jeghers syndrome ?									
A) Autosomal recessive	B) Increase risk of developing GlioblastomaC) Autosomal dominant		D) Autosomal dominant						
08 Right-sided Colorectal carcinoma usually present with ?									
A) lron deficiency anemia	3) obstruction C) Annular cricling lesions		D) Frank bleeding						
09 A 59-year-old man complains of progressive weakness. His friends have noticed that he has become pale, and he reports that his stools are tinged with blood. On abdominal palpation, there is fullness in the right lower quadrant. Laboratory studies show iron-deficiency anemia, Stool specimens are positive for occult blood. Colonoscopy reveals an elevated and centrally ulcerated lesion of the sigmoid colon. Which of the following is the most likely diagnosis?									
A) Gastrointestinal stromal tumor	B) Carcinoid tumor	C) Lymphoma	D) Adenocarcinoma						
10 Which one of the following is a neoplastic polyp ?									
A) Lymphoid polyps	B) Adenoma	C) Inflammatory polyps	D)Hyperplastic polyps						
11 Which one of the following abnormalities associated with adenocarcinoma of the colon ?									
A) Increase activity of TP53	B) Over stimulation of ACP gene	C) Increase activity of β-catenin.	D) None of the above						
12 Right-sided carcinoma (Colorectal carcinoma) associated with?									
A) Sickle cell anemia	B) Hypochromic microcytic anemia	C) lron deficiency anemia	D) Bleeding						

QUIZ!

MCQs Answer key	06	07	08	09	10	11	12
	С	D	А	D	В	С	С



This Lecture done by

- **Organizer**
- Member
- Note taker
- **Reviser**

Special thanks to **Renad alhomaidi** for her amazing work & efforts



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