جَائِزَة ٱلْحُسَيْنُ بْن عَبْدِ ٱللَّهِ ٱلثَّانِي لِلْعَمَلِ ٱلتَّطَوُّعِي
AL HUSSEIN BIN ABDULLAH II AWARD FOR VOLUNTARY WORK

النادي الطبي
Medical Club

General Surgery

Subject: Malignant tumor of the liver and pancreas

Group no:

Done by: Sadeel Alfaqeer

Malignant tumours of the liver &pancreas

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Liver tumour

- Benign
- Malignant
  - primary
  - 2dry (mets) — most common

→ from liver itself

Primary

- **HCC** — MC primary liver Tumor
- **Intra hepatic cholangiocarcinoma** — from cholangiocyte in intrahepatic biliary tree
- **cystadenocarcinoma**
- **Other (EHE, Lymphoma, angiosarcoma)**

Secondary

→ ***(most common)*** origin from different site (not liver)

- **CRLM** — Colorectal liver metastasis
- **NET** — Neuroendocrine Tumor
- **Others (gastric , oesophagus , breast , melanoma , GIST , RCC , ovarian , testicular)** — Pancreatic & Others.

- What is the most common type of liver tumor?
  - secondary type
- what is the most common primary liver cancer
  - HCC

Primary

1- HCC(Hepatocellular carcinoma)

- The most common primary hepatic tumour
- The fourth Cancer related death worldwide
- Arising on background of the underlying liver cirrhosis in 80-90% as precursors for HCC
- 10-20% no underlying Cirrhosis but not a normal liver
- Hight risk for vascular invasion , multifocality and advance at presentation

risk of develop depend on type of cirr...

next slide

في كثير من الحالات يُكتشف HCC متأخر، لأن:

- الأعراض المبكرة غير واضحة
- المرض غالبًا يأتي على خلفية **cirrhosis**
- قد لا يكون منتظم screening

لذلك عند التشخيص غالبًا يكون:

- كبير tumor
- أو منتشر داخل الكبد
- أو مع vascular invasion

more than lesion of HCC with high tendency of vascular invasion & in Almost all cases advanced at time of presentation (multifocal & vas.inva)

لذلك بشكل عام نادر جدًا انه ال HCC to be originated from normal liver parenchyma, usually 10-20% abnormal liver without cirrhosis (injury or fibrosis maybe) و 80% - 90% with documented liver cirrhosis

Especially in area which contain inflammatory pathogen as sub sahara Africa (HBV, HCV)

- The geographic distribution of HCC varies widely according to ethnic groups and regions, with a clear association with exposure to hepatitis viruses and environmental pathogens- more in sub-Sahara Africa and south Asia
- The risk of tumour development varies with the type of cirrhosis; the highest risk is reported for chronic viral hepatitis, whereas lower risks are associated with other forms of cirrhosis such as primary biliary cirrhosis.

At high risk to have HCC

↳ low risk of HCC

Recurrent attacks or activation of the viral hepatitis → more hepatocellular injury → more mutations → risk of cancer develop-

primary biliary cirr... ل

* HCC might be arising from the liver parenchyma or non cirrhotic liver or cirrhosis .

- HCC in the absence of cirrhosis may be related to some of the same aetiologies as those responsible for HCC in cirrhotic livers such as HBV infection or alcohol abuse.
- Alternatively, HCC may occur as a result of conditions that infrequently lead to cirrhosis such as α1-antitrypsin deficiency, haemochromatosis, or in the setting of specific aetiologies that do not result in cirrhosis such as hormonal exposure or glycogenosis
- Male >female

because males are more prone to have viral hepatitis (بسبب طبيعة الشغل و المواد يلي بتعرضها)

هاي شغلات بتعمل liver damage بس ما بتوصل للـ Cirrho... يعني يكون precursor Abnormal tissue يعني هذا النسيج لحدوث الـ HCC .

Risk factor include any risk factor or cause of cirrhosis & other

- Chronic HBV infection: the most frequent risk
- HCV infection
- Alcohol
- Non-alcoholic fatty liver disease (NAFLD) recently this growing issue one of the commonest causes
- Obesity and diabetes also increase the risk of HCC
- Hereditary haemochromatosis
- Cirrhosis of other aetiologies (PBC = primary biliary cirrhosis, auto-immune hepatitis) lowest risk factor to have HCC among all aetiologies of liver cirrhosis
- Aflatoxin
- Metabolic liver diseases and HCC(α1- antitrypsin deficiency, porphyria cutanea tarda, tyrosinaemia and hypercitrullinaemia.)
- Adenoma, contraceptives and androgens

Clinical presentation

- Abdominal pain (MC) Due to { rupture of tumor / Tumor effect (stretching or tension)
- Weight loss
- Rupture 5-15%
- Symptoms and signs of advance liver disease
- Hepatomegaly
- Constitutional symptoms

• very unlikely for HCC to present with Jaundice (only 10 - 20 %)

Related to underlying liver dx يكون عادة

Tendency of HCC to invade blood vessel is more & more but very unlikely for HCC to invade biliary tree or have mass effect on biliary sys.

يعني عادة لما بيجوا HCC مع الـ Jaun يكون السبب advanced liver dx و ٩٠٪ من الـ HCC يكون عندهم liver dx بس ١٠-٢٠٪ يكون عندهم سبب

Mass effect of HCC on biliary tree or invasion.

Diagnosis

Keys : Hx & PE

Screening program in high risk cirrhotic pt or in pt evaluate for range LFT or cancerated symptoms

... Screening program: [illegible]

Either on screening program for high-risk Cirrhotic patient or in patient evaluated for deranged LFTs or cancer related symptoms

Usually if pt doesn't have any sign of decompensated liver dx may LFT within normal, but sometimes if the pt has advanced

Bloods –LFTs usually reflect the underlying liver disease , jaundice usually related to decompensated liver rather than tumour compression

Liver dx or decompensated liver may be abnormal LFT

US, Triple phase liver CT , Liver MRI

tumour marker : A-FB (AFP) (normal 0-20) High in 70-80% in 20% of HCC will be normal

Level >400 consider diagnostic

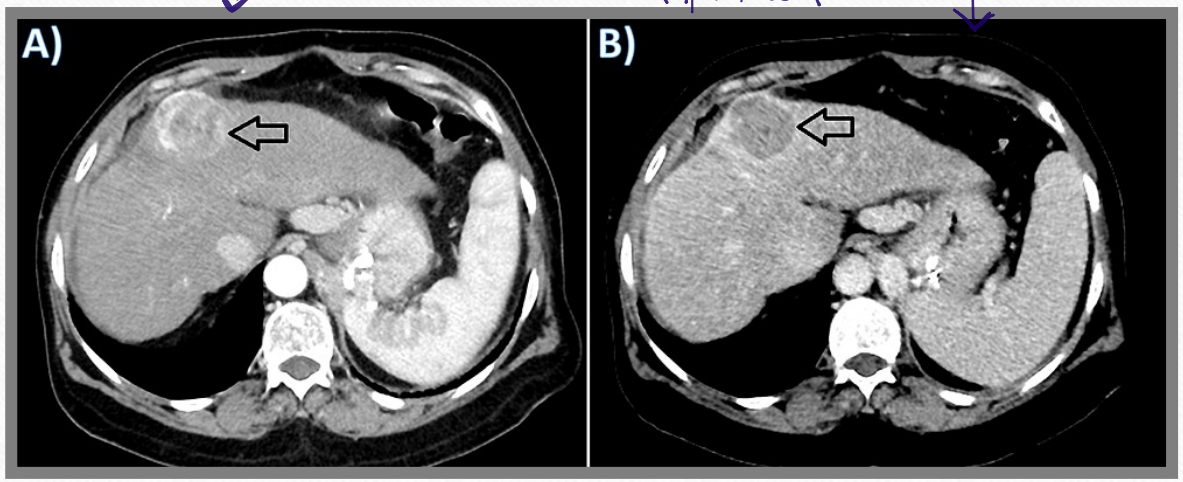
>10000 Associated with poor differentiation and vascular invasion

عادةً بكده نشخّص الـ HCC بالـ imaging بدون ما نحتاج لـ biopsy.

- CT feature of typical HCC
- **Typical feature of early arterial contrast enhancement and early washout out in the delayed phase on both CT and MRI**
- Always remember that the liver tumors either primary and secondary received their blood supple predominately from the hepatic artery not the Portal vein

Arterial phase: Aorta is already enhanced. HCC has ↑ vascularity

Delayed phase: porto-venous phase rapidly ↓ in enhancment due to rapid washout



Triphasic CT scan → Early arterial contrast enhancement (continuous rim of enhancement & on periphery) with rapid washout
MRI

Blood supply to liver come from { 75% portal vein / 25% Hepatic artery } ,This is in normal liver parenchyma, but in primary or 2dry liver tumor becomes from Hepatic artery predominantly

الـ portal ما بغذي الـ Tumor

لأنه التشخيص بكون متأخر وهو Advanced highly vascular invasion

- Remember: Only 15-20% of HCC is resectable, because rest of the tumors have • Multicentricity, Bilobar involvement Portal vein invasion, Lymphatic metastasis
- pre operative biopsy isn't mandatory for diagnosis in typical images feature

Rapid arterial ⟷ delayed phase

- Evaluation of the underlying liver disease and the stages of liver Cirrohis is mandatory using CPS or MELD score

Model for End-Stage Liver fDisease (MELD) score .

شوفيهم على النت و(حفظ)

↓ خارجي

Child-Pugh Classification of Cirrhosis

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وهو نظام لتقييم شدة تليّف الكبد (liver cirrhosis severity) وقدرة الكبد على التعويض (hepatic reserve). يُستخدم بشكل أساسي في مرضى HCC لكه
يحدد: إمكانية الجراحة (resection)، أمان TACE أو ablation، أهلية زرع الكبد (liver transplantation)، التوقعات (prognosis)

transarterial chemoemboli...

Factor	Units	1	2	3
Serum bilirubin	mol/L mg/dL	<34 <2.0	34-51 2.0-3.0	>51 >3.0
Serum albumin	g/L g/dL	>35 >3.5	30-35 3.0-3.5	<30 <3.0
Prothrombin time	seconds prolonged INR	0-4 <1.7	4-6 1.7-2.3	>6 >2.3
Ascites		None	Easily controlled	Poorly controlled
Hepatic encephalopathy		None	Minimal	Advanced

The Child-Pugh score is calculated by adding the scores of the five factors and can range from 5 to 15. Child-Pugh class can be A (a score of 5-6), B (7-9), or C (10 or above). Decompensation indicates cirrhosis with a Child-Pugh score of >7 (class B). This level has been the accepted criterion for listing liver transplantation.

Meld Score

MELD = 3.78 x $\log_e$ serum bilirubin (mg/dL) + 11.20 x $\log_e$ INR + 9.57 x $\log_e$ serum creatinine (mg/dL) + 6.43 (constant for liver disease etiology)

NOTES:
If the patient has been dialyzed twice within the last 7 days, then the value for serum creatinine used should be 4.0

Any value less than one is given a value of 1 (i.e. if bilirubin is 0.8, a value of 1.0 is used) to prevent the occurrence of scores below 0 (the natural logarithm of 1 is 0, and any value below 1 would yield a negative result)

Status	Meld Score	Patient Status
1		Includes patients with acute liver failure/disease with an estimated survival <7days (Highest priority for liver transplantation)
2a	>29	Patients with ESLD, severely ill and potentially hospitalized
2b	25-29	Patients with ESLD, severely ill, but not requiring hospitalization
3	<24	Patients with liver disease, too early for cadaveric transplantation, but may be candidate living donor transplantation

Table 2. Model for End-stage Liver Disease Score

Model for End-stage Liver Disease score = 6.43 + 3.78 Ln(serum total bilirubin [mg per dL]) + 11.2 Ln(International Normalized Ratio) + 9.57 Ln(serum creatinine [mg per dL])

Score	*90-day mortality (%)*
≥ 40	71.3
30 to 39	52.6
20 to 29	19.6
10 to 19	6.0
≤ 9	1.9

NOTE: *Although originally developed to predict three-month mortality in patients who had undergone transjugular intrahepatic portosystemic shunt procedure, the Model for End-stage Liver Disease score is now used to prioritize patients for liver transplant. Model for End-stage Liver Disease score calculators can be found at http://www.thedrugmonitor.com/meld.html and http://www.mdcalc.com/meld-score-model-for-end-stage-liver-disease-12-and-older.*

Information from reference 10.

3.78 x log(serum bilirubin) + 11.2 x log(INR) + 9.57 x log(serum creatinine) + 6.43 = MELD

The Child-Pugh Classification (CPC), also known as the Child-Turcotte-Pugh (CTP) score, is a system used to assess the prognosis of chronic liver disease, primarily cirrhosis. It evaluates liver function based on five clinical parameters and categorizes patients into three classes (A, B, or C) to guide treatment decisions and predict survival outcomes. Below is a detailed explanation of the scoring system and its clinical significans -

The total score (ranging from 5 to 15) determines the Child-Pugh class:

- **Class A (5–6 points)**: Mild liver disease; well-compensated liver function.
- **Class B (7–9 points)**: Moderate liver disease; significant functional compromise.
- **Class C (10–15 points)**: Severe liver disease; decompensated cirrhosis.

Interpretation and Prognosis

The Child-Pugh classification provides insights into survival rates and operative risks:

Points	Class	One-Year Survival	Two-Year Survival
5–6	A	~100%	~85%
7–9	B	~80%	~60%
10–15	C	~45%	~35%

- **Class A (5–6 points)**
  كبد متعوض (well compensated)
  → أفضل prognosis
  → ممكن resection أو علاجات أكثر شمولًا
- **Class B (7–9 points)**
  تعويض متوسط
  → خيارات علاج محدودة (غالبًا TACE أو supportive)
- **Class C (10–15 points)**
  فشل كبدي متقدم (decompensated)
  → غالبًا لا يتحمل أي تدخل كبير
  → قد يُفكّر فقط بـ transplant مناسب

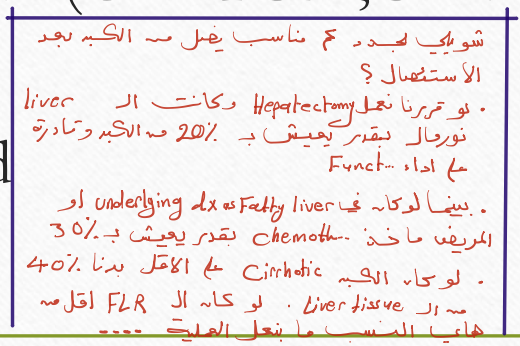
وهو نظام حسابي لتقدير شدة فشل الكبد (end-stage liver disease) واحتمال الوفاة خلال 3 أشهر، ويُستخدم بشكل أساسي لترتيب أولوية المرضى على قائمة liver transplantation.

الفكرة العامة
بعكس Child-Pugh (الذي فيه عناصر سريرية subjective)،
يعتمد على قيم مخبرية موضوعية بالكامل → أدق في التنبؤ بالوفيات MELD.

في أورام الكبد: مش حجم الورم لحاله اللي يحدد العلاج ، وظيفة الكبد أهم أحيانًا من الورم نفسه،، يعني مريض عنده tumor صغير لكن Child C → ممكن ما يتحمل أي

Treatment

- MDT decision not single surgeon decision (Hepatobiliary surgeons (2or3), oncologist, radiologist)
- CPS stage of Cirrhosis , performance status of the patient , number and the size of the tumour , extrahepatic metastasis and FLR ,all should be consider before making the decision of the treatment
- A lot of the staging system are used to stage the tumour (GRETCH ,CLIP CUPI, and TNM)
- BCLC guild line for management are most widely used



Cont . Treatment

- Curative : hepatectomy , liver transplant , RFA in absence of decompensated liver
- TACE and systemic therapy (Sorafenib) are the two palliative treatment modality that improve the survival in patient not candidate for curative treatment in absence of decompensated liver
- Best suppurative care and symptoms palliation in advance decompensated liver or not fit for any other treatment option (child C stage)
- No role for neo-adjuvant or adjuvant chemotherapy

الفكرة الأساسية من **TACE (Transarterial Chemoembolization)** في علاج **HCC (Hepatocellular carcinoma)** تعتمد على مبدأ فيزيولوجي مهم في الكبد:
أولاً، الكبد الطبيعي يستقبل التروية الدموية بشكل أساسي من **Portal vein**، بينما الورم الكبدي **(HCC)** يعتمد بشكل أكبر على **Hepatic artery** كمصدر رئيسي لـ **blood supply**. هذه النقطة هي الأساس الذي تُبنى عليه فكرة العلاج.
ثانياً، من الناحية الإجرائية **(Interventional Radiology procedure)**، يتم إدخال **catheter** عبر **Femoral artery**، ومن ثم يتم توجيهه تحت **fluoroscopic guidance** حتى الوصول إلى **Hepatic artery**، وبعدها يتم التقدم بشكل **super-selective** نحو الـ **feeding vessels** التي تغذي الورم مباشرة.
عند الوصول، يتم تطبيق خطوتين رئيسيتين:
الخطوة الأولى هي حقن **local chemotherapy** مباشرة داخل الشرايين المغذية للورم، مما يؤدي إلى تركيز عالي من الدواء داخل الورم نفسه **(high local concentration)** مع تقليل التأثيرات الجهازية.
الخطوة الثانية هي عمل **embolization** لهذه الشرايين باستخدام مواد خاصة **(embolic agents)**، مما يؤدي إلى إغلاق التروية الدموية عن الورم. النتيجة هي حدوث **ischemia** ومن ثم **tumor necrosis** بسبب انقطاع الـ **blood supply**.
بالتالي، التأثير العلاجي هنا مزدوج:
embolization نتيجة **Ischemic effect**
chemotherapy نتيجة الـ **Cytotoxic effect**
أما بالنسبة لـ **systemic chemotherapy (IV chemotherapy)**، سواء كان **neoadjuvant** أو **adjuvant**، فهو بشكل عام لا يملك دور فعّال في علاج **HCC**. السبب أن هذا النوع من الأورام يُظهر مقاومة عالية للـ **systemic chemotherapy**، ولذلك الاعتماد يكون على علاجات موضعية مثل **TACE** أو علاجات جهازية حديثة مثل **targeted therapy** و **immunotherapy** وليس الكيمو التقليدي.
باختصار:
بين الكبد الطبيعي والورم، ويوصل علاج مركز مباشرة للورم مع قطع ترويته، لتحقيق أقصى تأثير موضعي وأقل تأثير جهازي **blood supply** يستغل الفرق في **TACE**.

1. Neoadjuvant therapy

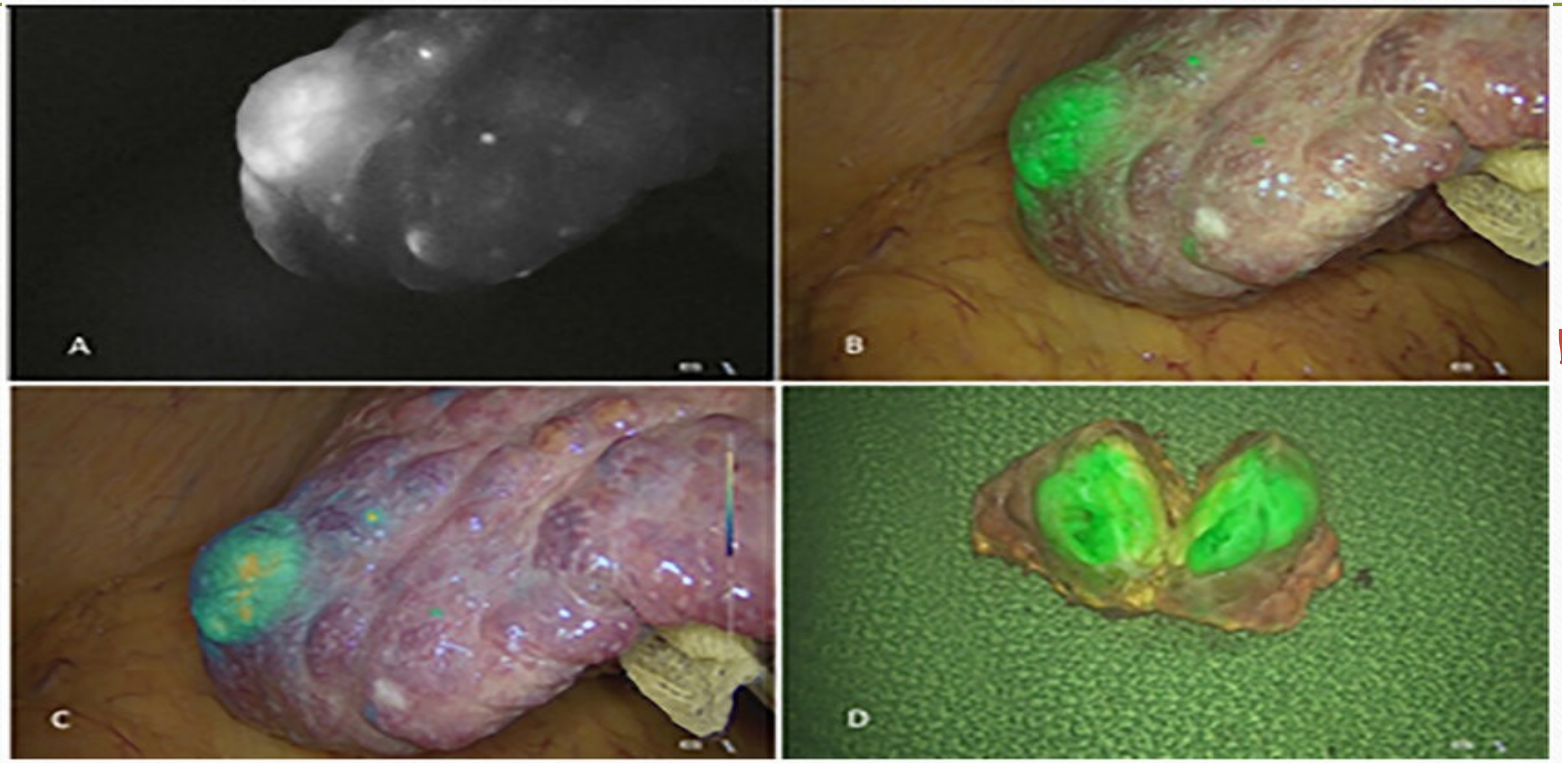
هو العلاج الذي يُعطى قبل الجراحة **(preoperative)**.
الهدف:
تصغير حجم الورم **(tumor downstaging)**
تحويل **tumor** غير قابل للجراحة → قابل للاستئصال
تقييم استجابة الورم للعلاج

2. Adjuvant therapy

هو العلاج الذي يُعطى بعد الجراحة **(postoperative)**.
الهدف:
القضاء على **micrometastases** غير المرئية
تقليل خطر **recurrence**
تحسين **overall survival**

(Intraoperatively photo)

Nodular liver (cirrhotic liver)



اللون الأخضر هاد
مادة بنعطيها بعليات
اسمها Laparoscopel
Indocyanin Green(ICG)
بتاخذها عند ال
بيريبا Hepatocyte
Malignant بال uptake
cells ، لذلك هاي
Nodules غالبا رح تكون
HCC

سبب الـrecurrance بـ HCC عم بـ ~90% underlying liver cirrhosis لأنه زي ما حكينا انه
بعد الاستئصال بضل الـ cirrhosis هو الـ risk factor لـ HCC وفي احتمالية recurrance.
وسبب آخر انه الـ Tumor high tendancy for vascular invasion.
او الـ Multifocality & Multicentricity بكون في very small nodule وما بينوا بالـ imaging فبتم استئصال الـ Nodules الكبار وهدول الصغار لاحقًا بيعملهم Tumor recurrance و growth

- Tumour recurrence is the major cause of death following resection of HCC in the cirrhotic patient. Its incidence is 40% within the first year
- 5- and survival rates of 43.9% ؟ survival على شو بعتمد الـ
- Independent predictors of survival were age, degree of liver damage, AFP level, tumour diameter, number of nodules, vascular invasion and surgical margins. → لو كانوا هاد +ve بعني انه incomplete resection.
- Best survival in case of small <2cm encapsulated tumour in patient with child A score

1 2 3 4 5. 6. 7.

single 5 cm — 3 nodules max diameter 3 cm → According to the Milan criteria, if these limits are exceeded, the patient is not considered a candidate for liver transplantation.

يعني موضوع الـ recurrance عليه نقاش هل الورم underlying cirrh-liver عم طلع de novo ولا True recurrance هاد هل هو
بس بالنهاية كله بنعتبره recurrance.

Variant of HCC

Fibrolamellar HCC

- Occurs in young adults without underlying cirrhosis
- Non-encapsulated but well, circumscribed, so high resectability rate than usual HCC
- Grows slowly and has better prognosis than usual HCC
- F>M عكس الـ usual
- AFP elevated in less than 10%
- Long-term survival can be expected in about 50-75% of patients after complete resection لأنه أغلب الحالات resectable.
- Recurrence is common and occurs in at least 80% of patients
- Presence of lymph node metastases predicts a worse outcome

→ HCC (mc type of primary liver Tumor)

Intrahepatic cholangiocarcinoma (ICCA)

- Is the second most frequent primary tumour of the liver after HCC.
- It arises from the peripheral intrahepatic biliary radicles 2nd & 3d order biliary tree
- In the Western world, the incidence of ICCA is 0.3–3 per 100000
- biliary intraepithelial neoplasia. and the intraductal papillary neoplasm of the bile duct are the two lesion usually precede invasive cholangiocarcinoma
- This tumour carries poor prognosis as it diagnosed at advance stage
- Equally between male and female , between the age of 55 and 75 years.

Sections & segm. لي بروحوا
Rt ant., Rt. pos., Lt. lat., Lt. med. & segmental from 1 to 8
ICCA هدول هم بكونوا
بكون الأورام
بيبتطلع Rt, Lt hepatic duct, confluence or CHD befor insertion of cystic duct → هدول hilar cholangiocarcinoma
واي Tumor بكون distal to cystic duct in CBD - Cholangiocarcinoma

Risk factors

(Usually no underlying risk factors) يعني بس

- in most patients with ICCA (more than 95%) none of these risk factors can be identified
- chronic biliary inflammation such as primary sclerosing cholangitis, chronic choledocholithiasis, hepatolithiasis, parasitic biliary infestation, Caroli's disease and choledochal cyst. (center process of carcinogenesis & mutation in recurrant inflammation)
- New risk factors are emerging, including chronic non-alcoholic liver disease, HBV infection, HCV infection, diabetes and the metabolic syndrome. → حسب اقل risk من ال HCC
- However, in contrast to HCC, most ICCAs develop without a background of liver disease.
- In surgical series, 75% of patients have normal livers, 16% have chronic hepatitis/liver fibrosis and 9% have cirrhosis

ال HCC ما بطلع من Normal liver بينما ICCA اغلب الحالات من Normal liver .

Clinical presentation and lab tests

Abd. pain, constitutional symptoms, maybe Jaundice

- abdominal pain, malaise, night sweats, asthenia, nausea and weight loss.
- jaundice may be present if the tumour compresses or invades the biliary confluence.
- LFTs non-specific even though an increase in liver enzymes (in particular GGT) may be the only initial finding in some patients
- Serum markers (Ca19.9, CEA) lack sensitivity and specificity.

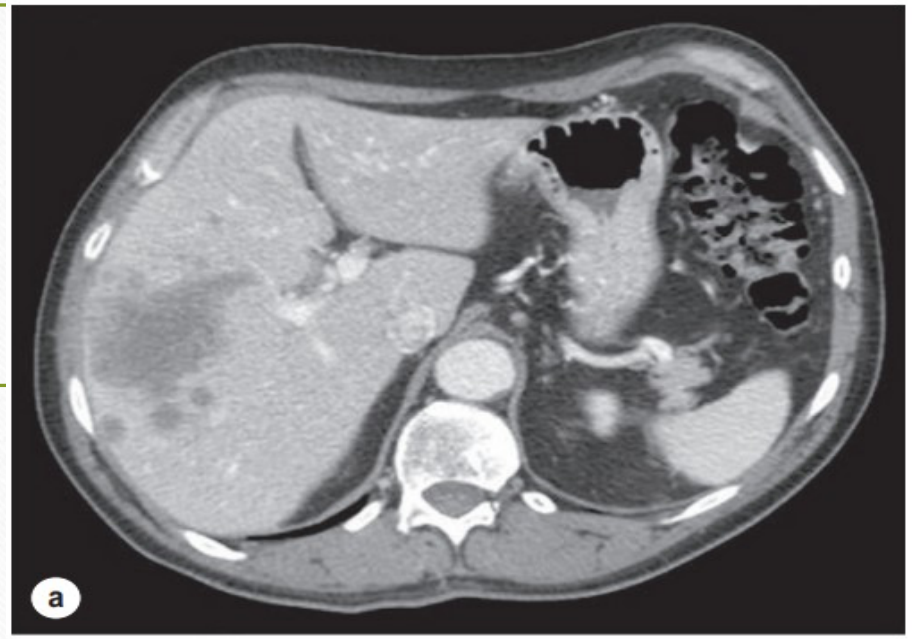
↳ not sensitive, not specific

Imaging

- Liver CT and liver MRI are used in diagnosis
- They are typically large, non-encapsulated, heterogeneous, associated with narrowing of adjacent portal veins and retraction of the liver capsule
- satellite nodules frequently develop in the vicinity of the tumour, and subsequently in the contralateral lob
- high propensity for lymph node invasion



بتعطينا فكرة عن الورم على الصورة



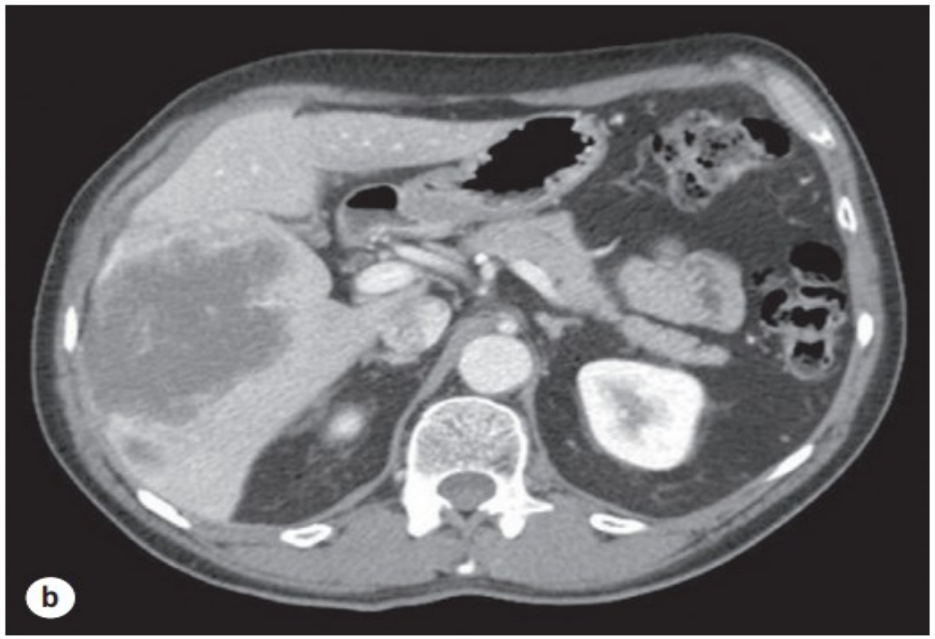




Figure 5.7 • CT of a patient with an intrahepatic/peripheral cholangiocarcinoma. Note the presence of typical satellite nodules at the periphery of the tumour **(a)**, the absence of vascular uptake **(b)** and the retraction of the capsula **(c)**.

No capsule, irregular edges, hypoenhancing
retraction of liver capsule

ما بيجوا بال Mini osce هيول الصور

Types

- Mass forming (MC)
- Intraductal-growth- rare but have a better long-term prognosis.
- Infiltrating type : ICCAs have a worse prognosis than the mass forming type due to spread along Glisson's capsule and high incidence of lymph node involvement
- Mixed type

MC → Mass forming.
beast prog → Intraductal
worse prog → infiltrating.

Treatment

unre ...

ICCA

Surgical resection is the only curative treatment.

if pt candidate for surgery

Unlike HCC, there is currently no place for liver transplantation except in very selective case

حتى لو عملت adequate preoperative imaging (مثل CT أو MRI) وبان لك إن الورم قابل للاستئصال، ما زال في risk تقريبا 20–30%إنك تكتشف أثناء العملية (at laparotomy) أشياء بتمنعك من عمل curative resection.

There is a significant risk (20–30%) that, despite adequate preoperative imaging, contraindications to a curative resection are identified at laparotomy

لذلك صار عندنا

Staging laparoscopy has been advocated

بندخل بالكاميرا و نتأكد ومع ذلك ممكن يبين عالـ staging انه resecta.. وبالعملية نكتشف

Systemic therapy in un resctable tumours

long term survival in unresectables بس

· Systemic the.. مع الـ very low بفضل

3. Biliary Cystadenocarcinoma

- Rare malignancy, typically intrahepatic in location. May arise from preexisting biliary cystadenomas. biliary cystadenomas have high tendency to become cystadenocarcinoma. Surgery بنعالجه ذلك
- Female to male ratio is 2 : 1
- CA 19-9 is raised
- The presence of an associated ovarian-like stroma in female patients appears to signify a favourable prognosis good prognosis
- Tends to be multilocular, fluid from the cyst can be blood stained, clear, or bile tinged.
- Preoperative cyst aspiration is not recommended because there is a risk for peritoneal tumour seeding (بنشخصه images على ال)

- The only potentially curative treatment is complete removal, by a major liver resection with clear margins.
- Survival rates for this disease have been reported in the range of 25% to 100% at 5 years if there is complete resection & pt fit enough to go for surgery

primary liver malignancy

#(Secondary) mc type in liver tumor

mets

4. Colo-rectal liver metastasis

- The liver is the most frequent site of metastases from CRC, with liver disease being detected either at the time of CRC diagnosis (synchronous; 20–25%) or subsequently (metachronous; 40%).
- The extent of liver disease is a key determinant of survival in patients with isolated colorectal liver metastases (CRLM).
- The diagnosis of CRLM is usually based on imaging during evaluation of patients with CRC , Rarely FNA biopsy

Unlikely Biopsy

Imaging

initial

- US is not a sensitive diagnostic test for CRLM and can fail to identify over 50% of metastatic lesions
- Triphasic liver multidetector CT
  triple-phase liver MDCT is the modality of choice for CRC staging and screening for liver metastases.
- CRLM typically appear as hypoattenuating lesions and are best identified in the portal venous phase of scanning but it can miss the lesion less than 1 cm
- MRI superior to CT in detecting and characterising indeterminate small lesions , more accurate in differentiating benign and malignant sub centimetre liver lesions, and more accurate in detecting and characterising CRLM if there is underlying liver parenchymal disease
- PET CT to identify metabolically active metastatic lesions. peritoneal nodule

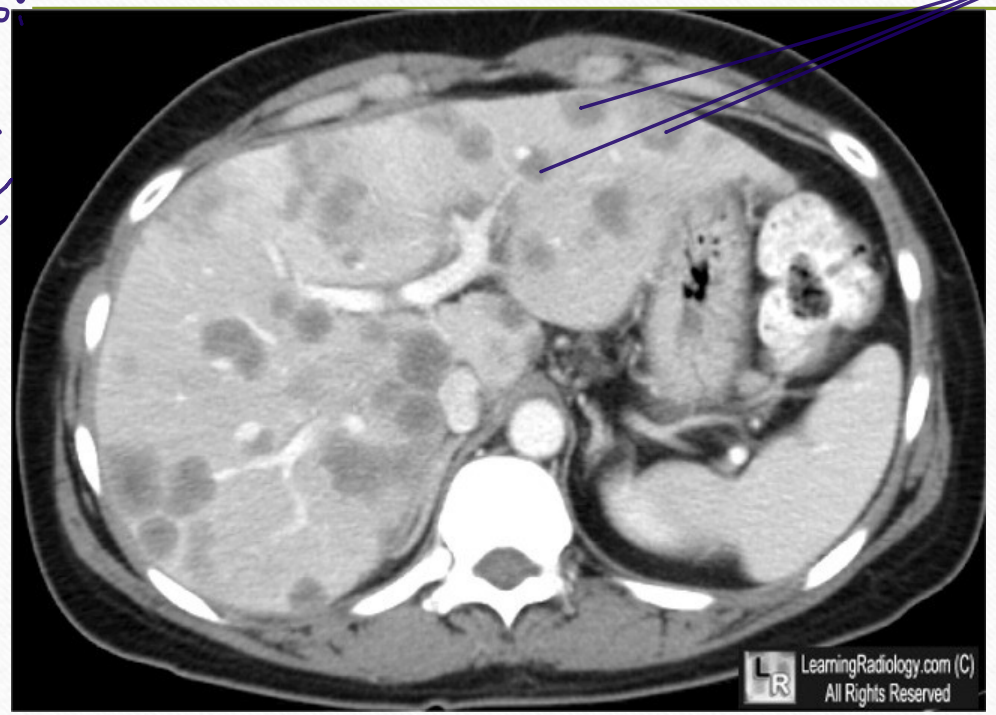
HCC ↓ عكس ال Arterial phase على ال enhance وبتكون ما

أحسن phase in CT نشوف فيها ال mets

لـ كيف نحلل الموضوع ؟

الكبد الطبيعي يعتمد في ترويته بشكل رئيسي على portal venous flow (حوالي 75%)، بينما يكون الاعتماد على hepatic arterial flow اقل في حالة primary liver tumor مثل HCC، يحدث tumor neoangiogenesis ويصبح الورم معتمداً بشكل أكبر على hepatic artery، لذلك يظهر arterial hyperenhancement في مرحلة الـ arterial phase، وهي المرحلة الأفضل لاكتشافه، ثم يحدث لاحقاً washout في الـ portal أو delayed phase. أمّا في colorectal liver metastases (CRLM)، فهي غالباً hypovascular lesions ولا تمتلك تروية شريانية قوية، وتكون ضمن كبد طبيعي يعتمد أساساً على الـ portal vein. لذلك في portal venous phase يحدث intense enhancement للكبد الطبيعي، بينما تبقى النقائل قليلة التعزز (hypodense)، فينشأ high lesion-to-liver contrast، وهذا ما يجعل الـ portal venous phase هي الأفضل لاكتشاف CRLM.

Late arterial, early portal venous phase



hypoattenuated or hypoenhancing lesions

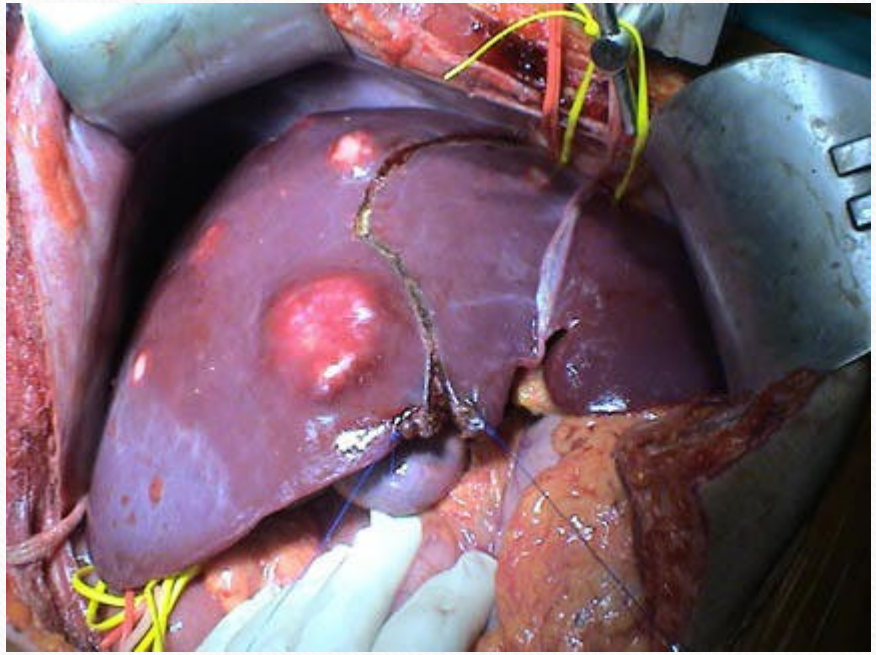


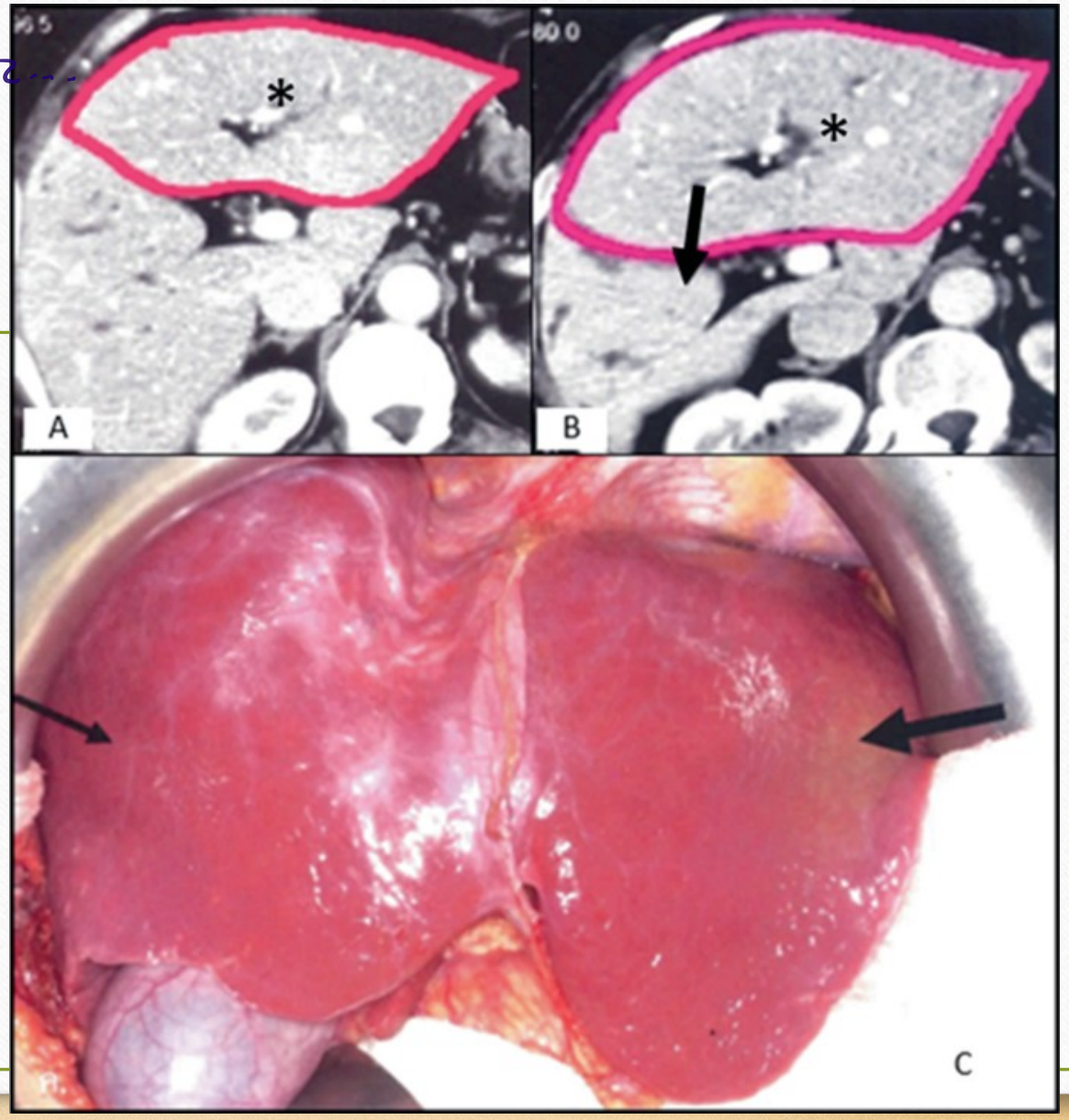
Treatment

- MDT decision should be applied not single. (Hepatobiliary & colorectal surgeons)
- Surgical resection with negative microscopic margins (R0 resection) offers patients with CRLM the best chance for long-term survival
- The decision depend on the extent of the liver mets and the presence of primary disease , extra hepatic mest , FLR (Future liver remenant) , prior neo adjuvant chemotherapy, vascular invasion and proximity to the major hepatic vein
- FLR is the future liver remnant post surgery (at least 20% in normal liver , 30% in chronic liver disease and 40 % in cirrhotic liver)
- Other options RFA , chemotherapy { Adjuvant / Neoadjuvant

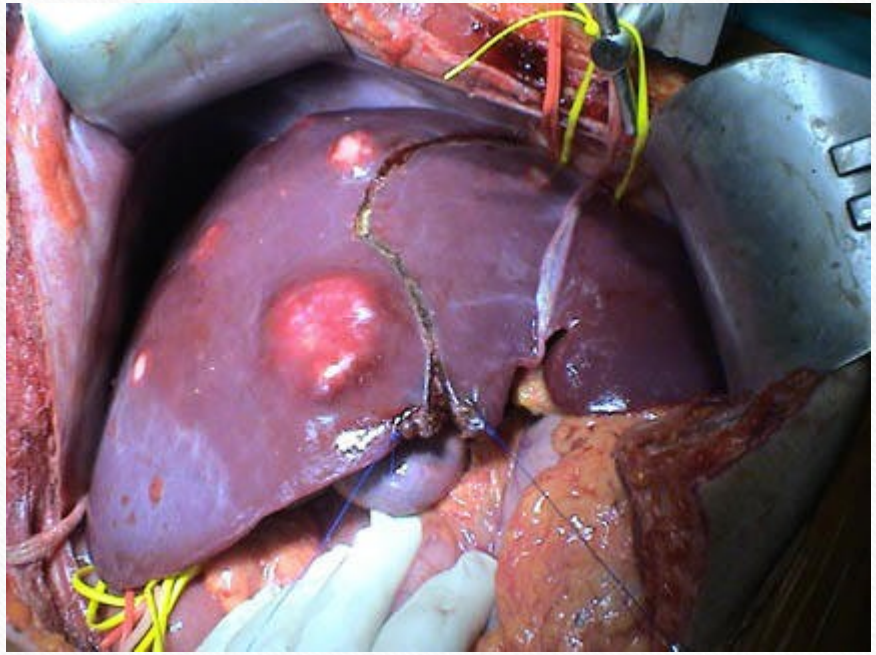
بدي أحكي عن Radiofrequency Ablation (RFA) كواحد من أهم أساليب العلاج الموضعي للأورام، خاصة في الكبد. هذا الإجراء يعتمد على إدخال إبرة خاصة داخل الورم تحت توجيه الأشعة، ثم تمرير تيار عالي التردد يولّد حرارة كافية لإحداث coagulative necrosis وتدمير الخلايا الورمية

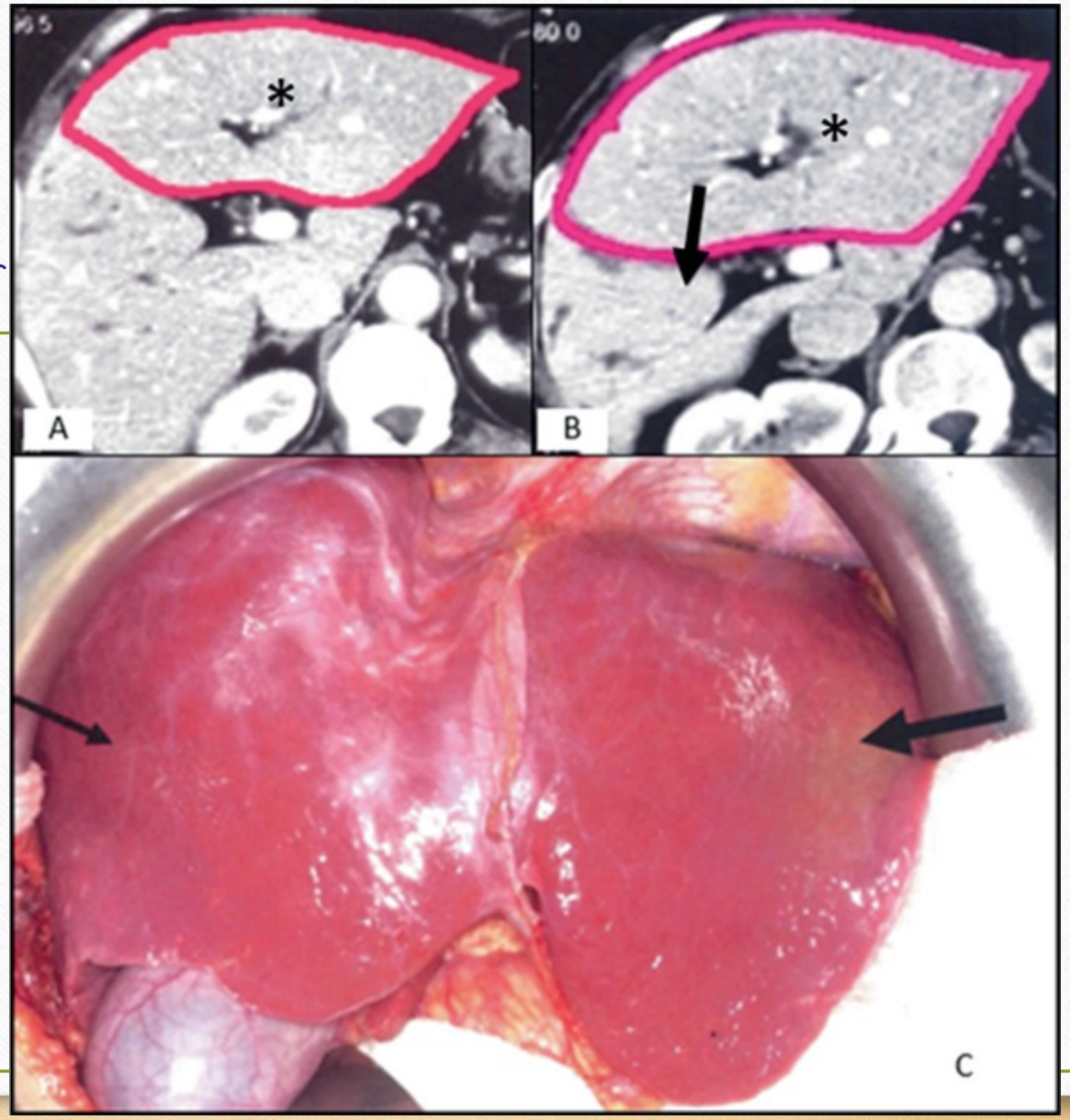
FLR & Portal vein emboliz...





لقينا الـ FLR مش كافي والحاتر بـ Right portal emb... بنعمل الـ Right lobe





- hepatic resection can be considered in patients with extrahepatic disease amenable to surgical resection or long-term oncologic control with adjuvant chemotherapy.

بس هم unresectable بتكون mets لما بيكون في Tumor حشكد عام ال
ميزات ال CRC انه حتى ال mets منه حيار نبقدر نعمل resectable
Liver resection to CRC بعض الحالات .

- In case of small FLR we can used Portal vein embolization of the lobe that have the mets before the surgery to induce atrophy of the affected liver lobe and hypertrophy on the non diseased lobe in effort to have adequate FLR and avoiding the post hepatectomy liver failure → because it can cause painful death to pt.

NET
Neuro-endocrine tumour mets

- Either from pancreatic or small bowel origin as ileum.
- Tend to be large , multifocal and bilobar → 80%
- Symptoms related either to the hormonally active disease or pressure symptoms and hepatomegaly
  - carcinoid syndrome: flushing, diarrhea, bronchospasm
  - بسبب كبر الكبد:
    hepatomegaly
    abdominal discomfort
    pressure symptoms
- less than 20% are candidates for surgery
- Liver resection may be performed with curative intent, symptom control or prolongation of survival in the palliative setting.
- Vascularity of the tumour is high → hyperenhancing or attenuated

CRLM ي

well differentiated, slow growing.

- Diagnosis : Liver triphasic CT , liver MRI
- Overall survival after surgery >80% at 5 years (more than the CRLM or other primary liver tumour)
- Treatment decision depend on the disease extent , symptoms , grade of the tumour
- Surgical treatment with curative intent is the best modality if the patient fit for the surgery and the extent of the disease permit doing safe hepatectomy otherwise cytoreductive surgery is the option for symptomatic control

تقليل كمية الخلايا السرطانية (tumor burden) داخل الجسم بدون استئصالها بالكامل

- Somatostatin analogue used for symptomatic treatment in hormonally active disease

Radio frequency Ablation

Peptide Receptor Radionuclide Therapy.

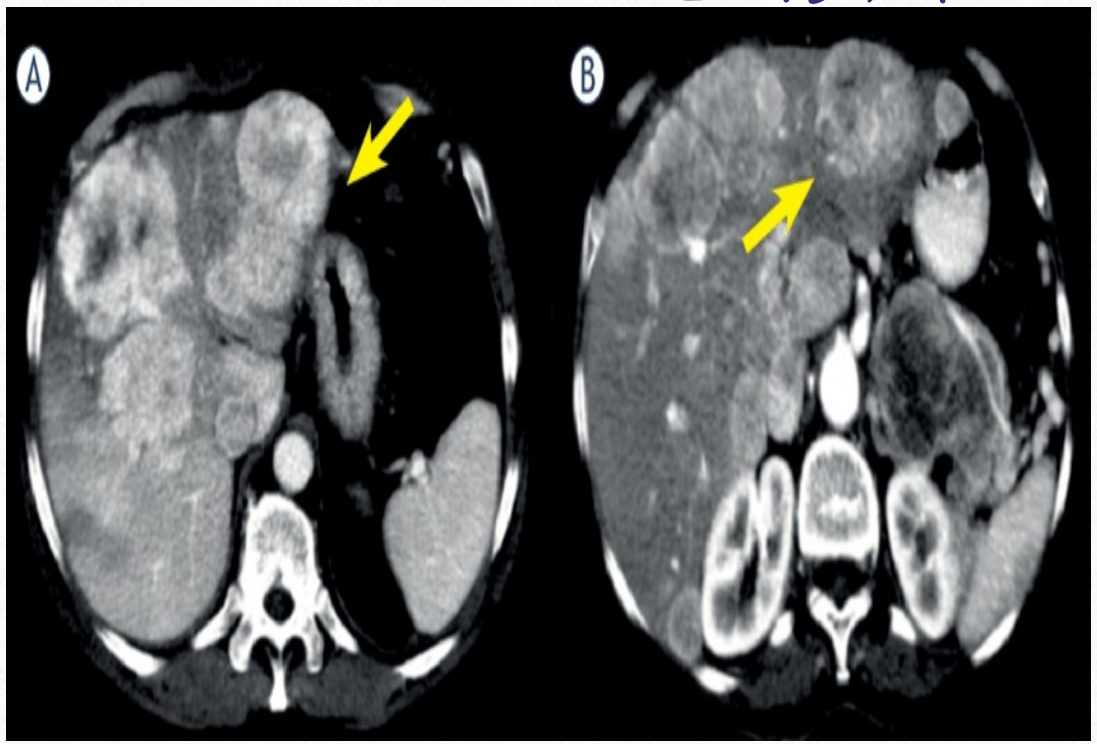
- Other option RFA , TACE , PRRT
- Limited role of systemic chemotherapy

No role

بنعطي peptide (زي somatostatin analogue)
مربوط بـ radioactive isotope (أشهره Lutetium-177)
→ هذا المركب يروح يرتبط بالـ receptors على الخلايا السرطانية
→ يدخل الخلية
→ يطلق إشعاع β-radiation
→ يؤدي إلى DNA damage وموت الخلية
يعني ببساطة: targeted internal radiotherapy موجه مباشرة للورم.
ثانيًا: متى نستخدم PRRT؟ (Indications)
حسب المراجع مثل Harrison's و ENETS guidelines:
Metastatic أو unresectable NETs
لازم يكون الورم somatostatin receptor positive (يتأكد عبر imaging مثل DOTATATE PET)
غالبًا في well-differentiated tumors
يُستخدم بعد أو مع somatostatin analogues إذا المرض مستمر أو symptomatic
ثالثًا: شو فائدته سريريًا؟
يقلل tumor progression
يحسن progression-free survival
ممكن يعطي symptom control (خصوصًا hormonal symptoms)
أحيانًا shrinkage للورم
مش عادة curative، لكنه قوي جدًا كـ disease control.

Hyperintense lesion that enhanced on CT with contrast during the arterial phase

CRLM





Pancreatic malignant tumour

- Exocrine (MC type)
- Endocrin

Exocrine :

the most common

Including PDAC , Mucinous , adenosquamous (The worse type+worse prognosis)

Pancreatic Ductal Adenocarcinoma

Endocrine : P-NET -5% of pancreatic tumour
(insulinoma , Glucagonoma , Gastrinoma , VIPoma)

Pancreatic malignant tumour

- PC is the seventh leading cause of cancer-related deaths worldwide
- The annual incidence rate worldwide for all histologic types of pancreatic cancer is approximately 4.9 new cases per 100,000 persons, ranking 12th among all cancers globally
- In Jordan 3.7 new case per 100,00 person

- Most common type of pancreatic tumor is Exocrine type. (PDAC)
- ~ ~ ~ ~ Exocrine ~ ~ ~ PDAC

PDAC

- Pancreatic ductal adenocarcinoma (PDAC) is most common pancreatic cancer and the most common exocrine pancreatic cancer
- MC site: Head (75%) > Body (15%) >Tail (10%) of pancreas
- More common in Men, African Americans, mean age at diagnosis is 72 years

Clinical presentation

- MC symptom for patients with PDACs in the periampullary region is jaundice which could be painless or painful
- Pain typically arising in epigastrium & radiating to the back.
- Weight loss affecting more than 50% of individuals.
- For tumours of body & tail of pancreas, pain & weight loss become more common at presentation.
- A palpable distended gallbladder in 1/3rd of patients with periampullary PDAC (Courvoisier sign)
- With widespread disease, a left supraclavicular node (Virchow's node)may be palpable. Periumbilical lymphadenopathy may be palpable (Sister Mary Joseph's node)

Cont.. Clinical presentation

- vomiting reflecting gastric outlet obstruction, in head.
- peripheral migratory venous thrombosis (Trousseau's sign) Thrombo تتنقل من مكان لمكان (ايد، رجل...) عند -phlebitis
- pancreatitis in the absence of other potential precipitants such as gallstones or regular alcohol use
- pancreatic insufficiency (either exocrine and/or endocrine) ie: Late-onset diabetes Signs of malabsorption without defined cause

(other Risk factor)

- **Association** between risk of **pancreatic cancer**, **H. pylori** colonization, and **ABO** blood groups
- **Established risk factors**: **Smoking (Tobacco)** and **Inherited susceptibility**Q

- **K-ras2** oncogene is **activated** (by point mutation) in **>95%** of pancreatic cancers (**MC gene mutation**)Q

↳ MC

Pt with genetic → go for screening program (Endoscopic US ☆, MRI ☆ for early detection

Risk factor

Box 16.1 • Risk factors for pancreatic cancer

- Age (above 60 years)
- Smoking
- Obesity
- High fat diet
- Alcohol abuse
- Pancreatitis
  - Chronic pancreatitis
  - Hereditary pancreatitis
- Diabetes mellitus
- Family history of pancreatic cancer
- Genetic predisposition
  - Peutz–Jeghers syndrome
  - Li–Fraumeni syndrome
  - Fanconi syndrome
  - Familial adenomatous polyposis
  - Lynch syndrome
  - Gardner syndrome
  - Multiple endocrine neoplasia
  - *BRCA1*
  - Von Hippel–Lindau syndrome

مطلوبين

Investigation

Blood test

largely unhelpful in diagnosis(normochromic anaemia, raised bilirubin and ALP , amylase and lipase may be elevated in those presenting with pancreatitis (5%)

TM –Ca19.9

biliary cystadeno-carcinoma

كانه يرتفع بال

CA 19-9 has suboptimal sensitivity (41–86%) and specificity (33–100%) for detecting pancreatic cancer

4–15% of the general population do not express the antigen and hence do not have detectable serum CA 19-9 levels

Ca19.19 Can be elevated in benign condition like cholecystitis , cholangitis , chronic pancreatitis

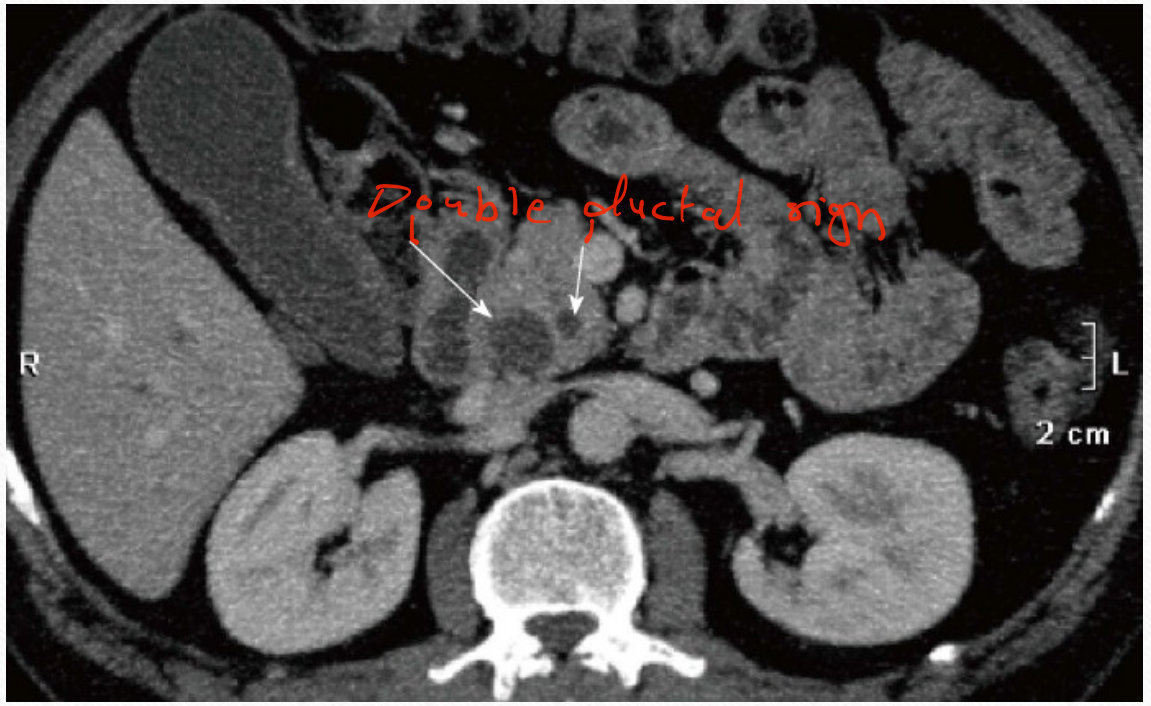
يبقى ما يرتفع كتير يغيب عكس
(normal = 37 هو ال بالاربعينات)
بنيما ال بكون Cancer اكثر من ١٠٠

Cont .. Investigation

- because of these limitations, CA 19-9 is mostly used as a prognostic marker to assess response to therapy in patients already diagnosed with pancreatic cancer
- US : Initial modality in jaundice patient looking for other cause (CBD stone), dilated CBD , ascites , liver mets
- CT- pancreas , more sensitive than the US , investigation of choice for the evaluation of lesions arising in the pancreas

SMA → Sup. mesentric Artery , & MV → sup. mesenbric Vein & (PV) → portal v

- CT shows the pancreatic mass site , any evidence of vascular invasion to SMA, SMV-PV , liver and extra-abdominal mets , assessment of the resectability
- Signs on CT : hypointense mass
- double ductal Sign (dilated CBD &PD) on CT scan is on the the sign of pancreatic cancer on the CT but in general this sign is seen more commonly with ampullary tumour


Double ductal sign
R
L
2 cm

Investigations

(Endoscopic US)

EUS: For identifying lesions that do not appear on CT and in case if we need to have a biopsy

عشان العملية

يعني لا biopsy مش ضروري للتشخيص ممكن اكتفي بال (images)؟ لا

Tissue diagnosis is not necessary prior to routine resection unless the patient planned to have the chemotherapy in this case biopsy is required

لا اذا العلاج كيموثيرابي

to confirmed dx

Magnetic resonance imaging (MRI) is mainly used as an adjunct to CT for planning treatment options

but not routinely used

PET-CT scanners are able to detect small (up to 7mm) pancreatic neoplasms and diagnose metastatic

Staging

Don’t memorize it

TNM CLASSIFICATION OF PANCREATIC CANCER

8th AJCC (2017) TNM Classification of Pancreatic Cancer	
Tis	Carcinoma in situ
T1	Tumor limited to pancreas **upto 2 cm** in greatest dimension
	T1a: Tumor **≤0.5 cm** in greatest dimension
	T1b: Tumor **>0.5 cm** but **≤1 cm** in greatest dimension
	T1c: Tumor **>1 cm** but **≤2 cm** in greatest dimension
T2	Tumor limited to pancreas **>2-4 cm** in greatest dimension
T3	Tumor **>4 cm** in greatest dimension
T4	Tumor involves **celiac axis**, **superior mesenteric artery** and/or **common hepatic artery**
N1	Metastasis in **1-3** regional LN
N2	Metastasis in **4 or more** regional LN
M1	Distant metastasis

Stage 0	Stage IA	Stage IB	Stage IIA	Stage IIB	Stage III	Stage IV
Tis N0 M0	**T1** N0 M0	**T2** N0 M0	**T3** N0 M0	**T1-T3 N1** M0	**T1-T3 N2** M0 **T4** AnyN M0	Any T AnyN **M1**

Treatment

- MDT
- Assessment if the tumour is resctable → Resectable → go for surgery
  ↓ Unresectable
- Palliative option ?
- Role of ERCP and stenting in jaundice patient: pre op , or palliative or if the patient planned for neoadjuvant chemotherapy
- Symptoms of GOO
- Only 20 % are surgically resctable at the time of diagnosis

1. pancreatic tumor with Jaundice → ERCP & stent قبل العملية حتى تخف الجاندس
2. ~ tumor + advanced symptoms & Jaundice, Tumor was unresectable → ERCP & stenting as palliative care.
3. pt will go for chemotherapy (neo adjuv)
   ↳ ERCP + stent when Jaundice ↓ , go for Chemotherapy

مش مطلوب

TABLE 62.2 National Comprehensive Cancer Network® (NCCN®) Criteria Defining Resectability Status

Resectability Status	Arterial	Venous
Resectable	No arterial tumor contact with CA, SMA, or CHA	No tumor contact with SMV or PV or ≤180-degree contact without vein contour irregularity
Borderline resectable	*Head/uncinate process* • Solid tumor contact with the SMA of ≤180 degree • Solid tumor contact with the CHA without extension to CA or bifurcation of the hepatic artery to allow safe and complete resection and reconstruction • Presence of variant arterial anatomy (ex: accessory right hepatic artery, replaced right hepatic artery, replaced CHA and the origin of replaced or accessory artery) and the presence and degree of tumor contact should be noted if present as it may affect surgical planning. *Body/tail* • Solid tumor contact with the CA ≤180 degrees • Solid tumor contact with the CA of >180 degrees without involvement of the aorta and with intact and uninvolved gastroduodenal artery	Solid tumor contact with the SMV or PV of >180 degrees, contact of ≤180 degrees with contour irregularity of the vein, or thrombosis of the vein but with suitable vessel proximal and distal to the site of involvement allowing safe and complete resection with reconstruction Solid tumor contact with the IVC
Unresectable	Distant metastasis (including non-regional lymph node metastasis) *Head/uncinate process* • Solid tumor contact with SMA >180 degrees • Solid tumor contact with CA >180 degrees • Solid tumor contact with first jejunal SMA branch *Body/tail* • Solid tumor contact of >180 degrees with the SMA or CA • Solid tumor contact with the CA and aortic involvement	*Head/uncinate process* • Unreconstructible SMV/PV due to tumor involvement or occlusion • Contact with the most proximal draining jejunal branch into SMV *Body/tail* • Unreconstructible SMV/PV due to tumor involvement or occlusion

CA, Celiac axis; *CHA*, common hepatic artery; *IVC*, inferior vena cava; *PHA*, proper hepatic artery; *PV*, portal vein; *RHA*, right hepatic artery; *SMA*, superior mesenteric artery; *SMV*, superior mesenteric vein.

Don't memorize this table –its beyond your level just note the highlight items to understand the management

- Criteria to assess if the tumour is resectable , borderline resecatble or unresectable

Resectable → Mets عامل الورم مش
لا لل Liver و peritoneum و
invasion عامل مش و lung لل
PV و SMV و SMA لا
Unresectable → Mets عامل
Borderline → blood vessels ال واحد
degree of ال على بتعتمد وهذا

invasion.

Resectable

- Surgical resection remains the only potentially curative treatment of pancreas cancer if the patient fit to go for it
- Tumours of head of the pancreas(Whipples –classical or PPPD) (Pylorus preserved pancreaticoduodenectomy) Antrum بنشيل ال وال pylorus
- Tumours of body & tail of the pancreas-Distal pancreatectomy and en-bloc splenectomy
- In addition, post operative adjuvant chemotherapy .

Borderline resectable

Chemoth... بقدر ابلش معه

- Neo adjuvant chemotherapy
- Then re assessment if become resecatble after the chemotherapy we will follow the same treatment plan of the resectable cancer (surgery)
- If not we will go for palliative treatment

Unresectable palliative

- Palliative chemotherapy
- Symptomatic management of pain , jaundice and GOO as shown in the table :

Gastric outlet obs.

Palliative Therapy for Pancreatic Cancer	
• Biliary obstruction	• **ERCP** with **metal stent placement** (**Best**)[Q] or • Roux-en-Y hepaticojejunostomy
• Gastric outlet obstruction	• **Endoscopic stenting** (**Preferred**)[Q] or • Double bypass (Roux-en-Y hepaticojejunostomy + gastrojejunostomy)
• Pain	• **NSAIDs** or **opiates**[Q] • **Celiac nerve block**[Q]

Prognosis

- Five-year survival after curative resection (pancreaticoduodenectomy) approaches 15-20 % very poor prognosis

Median Survival in Carcinoma Pancreas	
• **Resectable** disease (stage I and II)	• **15-20** months[a]
• **Locally advanced** disease (stage III)	• **6-10** months[a]
• **Metastatic** disease (stage IV)	• **3-6** months[a]

20% of cases resectable → وأعاريبي الله وبالكثير بعيشوا 15-20 month.

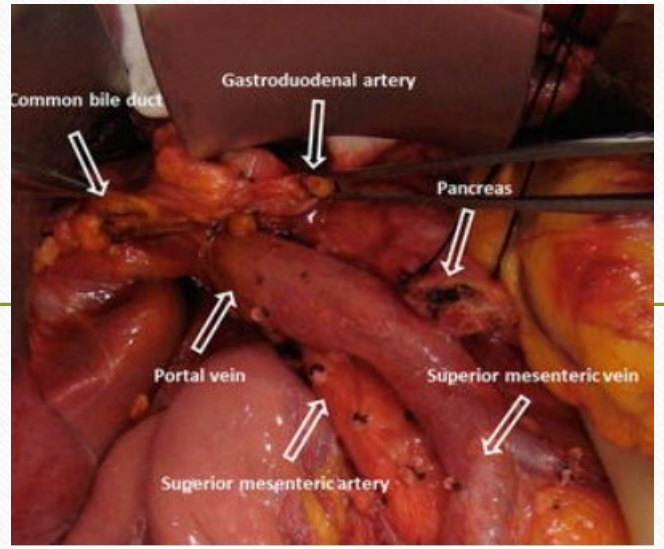
80 % unresectable → 10 months

one of the bigest surgery done for pt with periambullary or pancreatic head tumors or distal cholangiocarcinoma, we remove:

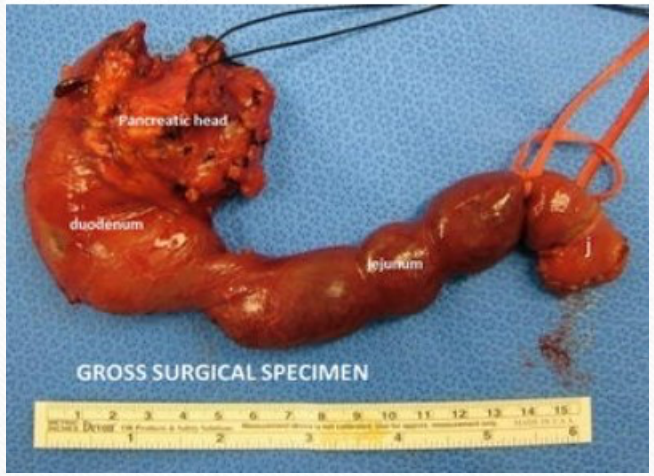
Whipple's Procedure Involves Resection of	
• Distal stomach[Q]	• Duodenum[Q]
• Gallbladder[Q]	• Proximal jejunum[Q]
• CBD[Q]	• Regional lymphatics
• Head of pancreas[Q]	

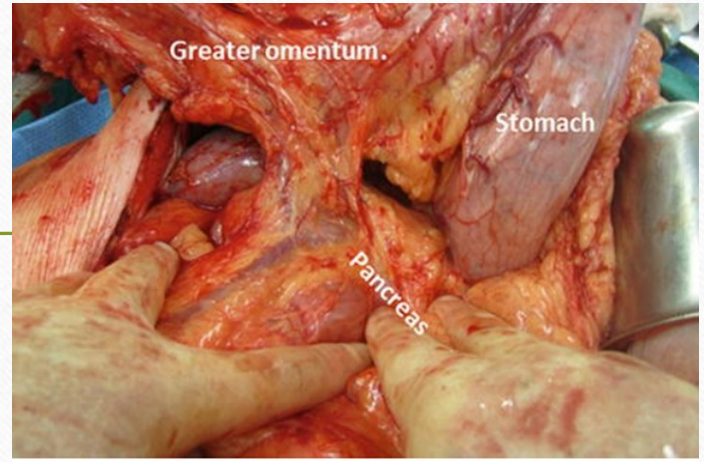
3 Type of Restoration بعملين جديدين of GI contin...

Restoration of GI Continuity Requires		
• Pancreaticojejunostomy[Q]	• Hepaticojejunostomy[Q]	• Gastrojejunostomy[Q]

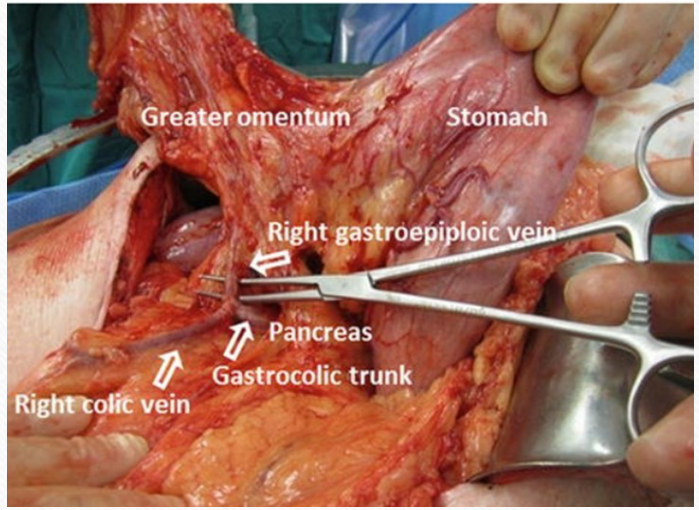


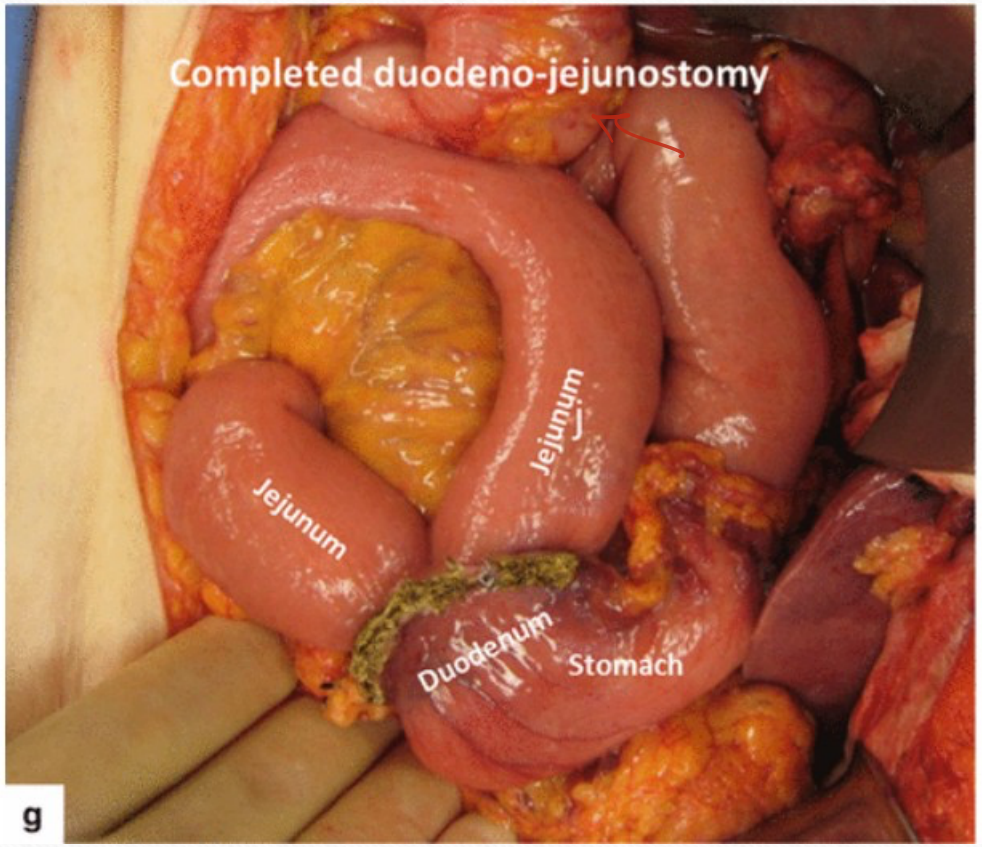
Figs. 19.16
Transected CBD

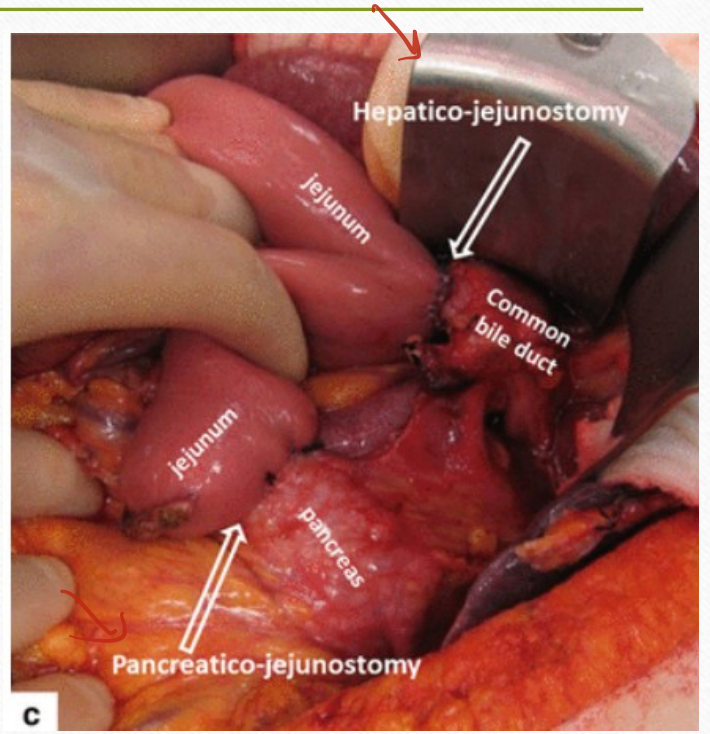




s. 19.4
tering the lesser sac




Completed duodeno-jejunostomy
Jejunum
Jejunum
Duodenum
Stomach
g


Hepatico-jejunostomy
jejunum
Common bile duct
jejunum
pancreas
Pancreatico-jejunostomy
c

P-NET

Pancreatic Neuroend-
Tumor.

Non functional (most common)

Functional

Insulinoma is the most common functional P-NET

Gastrinoma is the most common malignant functional P-NET

functional tumors are associated with better survival than nonfunctional PNETs because they are more often identified at an earlier stage pts come with symptoms.

Low- and intermediate-grade (G1 and G2, respectively) PNETs have significantly better 5-year OS (75% and 63%, respectively) than do G3 tumors (7%)

على شو بعتمد التصنيف G1,2,3 ؟

Differentiation , Mitotic rate & Ki-67 index (Tissue biopsy)

حفظا.

TABLE 65.1 Grading System for Pancreatic Neuroendocrine Tumors[a]

TERMINOLOGY	DIFFERENTIATION	GRADE	MITOTIC RATE (mitoses/2 mm^2)	Ki-67 INDEX (PERCENT)
NET, G1	Well differentiated	Low	<2	<3
NET, G2	Well differentiated	Intermediate	2 to 20	3 to 20
NET, G3	Well differentiated	High	>20	>20
NEC (small cell or non-small cell)	Poorly differentiated	High	>20	>20

Neuroendocrine Carcinoma

(Differentiat.. بنميز بينهم بال NEC , Grad 3)

Functional PNET

1. Insulinoma

- The average age at diagnosis is 45 years, Typically small
- 97% in pancreas (equal distribution in the head, body, and tail)
- 3% in duodenum, splenic hilum, or gastrocolic ligament
- Diagnostic hallmark is Whipple's triad: Fasting-induced neuroglyopenic symptoms of hypoglycemia (diaphoresis, shaking, mental confusion, obtundation, and seizures), low blood glucose levels (40 to 50 mg/dL), and relief of symptoms after the administration of glucose
- Significant weight gain: Patients eat frequently to prevent hypoglycemia.
- It is a painless condition
- Sporadic in majority of the cases not Genetic

- Gold standard test for the diagnosis of insulinoma is the 72-hour fasting test
- An insulin-to-glucose ratio > 0.4 is consistent with insulinoma
- CECT or MRI: Hyperattenuating → hypervascular
- Portal venous sampling for insulin with or without arterial stimulation with calcium is the best pre-operative method of localization
- Treatment
- 1- Diazoxide decreases beta cell release of insulin
- 2- surgery

insulin
glucose
pro.insulin

factitious hypogly... لـ عشان نتأكد انها مش الناتج عن جرعات زائدة من الأنولينا عشان نرفض الـ DM

invasive proc.. بنعمل 72-h-F.T في بعض الحالات لكن ما بزيد فيها Portal v. sampling هي الـ

is portal... not 72 h. F.T.

sympt. control to block B. cell to secrete insulin

identify site

Either inniculation (only remove lesion) or distal pancreatectomy (Tail or body) or wipples if it in head.

2. (Malignant)

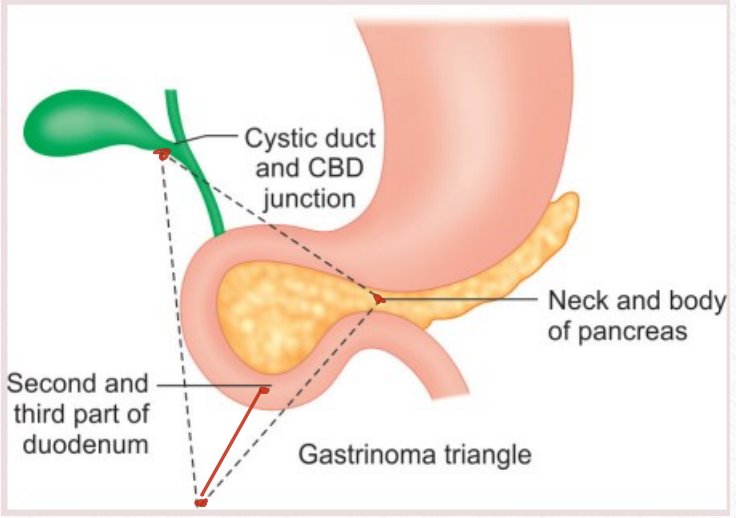
GASTRINOMA/ZOLLINGER-ELLISON SYNDROME

- Gastrinoma is MC functioning malignant pancreatic endocrine tumor. •
- More common in men, mean age 50 years
- • ZES occur in two forms: Sporadic (75%) & MEN-1 association (25%)
- • Those associated with MEN-1 are almost always multiple, early onset, more common in duodenum
- MC site is duodenum (50-70%) followed by Pancreas (20-40%)

- • About 70-90% of gastrinomas are located within the Passaro's triangle

Boundaries of Passaro's Triangle
• Junction of cystic duct and CBD[Q]
• Junction of 2nd & 3rd part of duodenum[Q]
• Junction of neck & body of pancreas

in duodenal side > pancreatic site



Clinical Features

- • Gastric acid hypersecretion causes peptic ulcer disease often refractory severe diarrhea
- MC presenting symptoms are abdominal pain(70-100%), diarrhea (50-70%) & GERD (30-35%)
- 100% patients will have a fasting serum gastrin level >100 pg/mL
- If the diagnosis is in doubt, the provocative tests are highly useful: • Secretin Provocation Test: An increase of >200 pg/mL in the gastrin value after administration of secretin is diagnostic

- Somatostatin receptor scintigraphy (SRS) is imaging test of choice for localizing both primary and metastatic gastrinomas
- Treatment : PPI +Surgery depending on the location
  - Tail / body → Distal...
  - Head & duoed... → Whipple.
- Consider genetic testing in those whom MEN1 is suspected with consideration to rule out parathyroid adenoma and pituitary adenoma

& to screen other family member.

Glucagonoma

- More common in females; 70% are malignant
- MC site: Body and tail of pancreas.
- Classic presentation of the 4Ds: Diabetes, dermatitis, DVT, and depression
- Fasting glucagon level >50 pmol/L is considered diagnostic + CT scan
- Necrolytic erythema migrans are MC manifestations of the disease, seen in 2/3rd of patients
- Resection is the treatment of choice distal pan -- + splenectomy

VIPomas (Verner-Morrison Syndrome)

- Also known as WDHA syndrome (watery diarrhea, hypokalemia, achlorhydria) or pancreatic cholera due to sever diarrhea.
- Usually solitary; MC site is tail of pancreas, Two-thirds are malignant
- Diagnostic triad: Secretary diarrhea + High levels of circulating VIP (vasoactive intestinal peptide.) + Pancreatic tumor
- Profuse, watery, iso-osmotic secretary diarrhea is MC presenting symptom
- Treatment :
- preoperative hydration and correction of electrolyte abnormalities & Octreotide
- Resection is the treatment of choice

Gastrinoma الـ حالات كمان نعطيها ↘
Symptomatic treat... insulinoma او

Precursor lesions

- These precursor lesions include:

1- pancreatic intraepithelial neoplasia (PanIN)(the commonest)

2- intraductal papillary mucinous neoplasm (IPMN)

3- mucinous cystic neoplasm (MCN).

We will speak only about 2& 3

IPMN
Intraductal Papillary Mucinous Neoplasm

- IPMN is also known as mucin-secreting carcinoma, villous adenoma of the duct of Wirsung
- Seen in 6th to 7th decade of life; Equal sex distribution
- • More common in head & uncinate process of the pancreas
- **Types of IPMN**
- – Side branch IPMN: Involves dilation of the pancreatic duct side branches
- – Main duct IPMN: Abnormal cystic dilation of the main pancreatic duct
- – Mixed-type IPMN: Side branch IPMN that has extended to involve the main pancreatic duct

Clinical Features

- abdominal pain or recurrent pancreatitis, (caused by obstruction of pancreatic duct by thick mucin
- 5-10% have steatorrhea, diabetes, & weight loss secondary to pancreatic insufficiency.
- • Careful histologic examination of the entire specimen (invasive component in (36%–100%)in Main duct type and 12-30% in branch type

Diagnosis

- Endoscopy: Mucus extruding through a large, fish-mouth like papillary orifice is virtually diagnostic of IPMN
- CT scans: Dilated main pancreatic duct, cysts of varying sizes, and possibly mural nodules
- Aspirated fluid: Mucinous content with elevated CEA & amylase level
- Pancreatic resection is the standard treatment for MCNs

دائما عالي

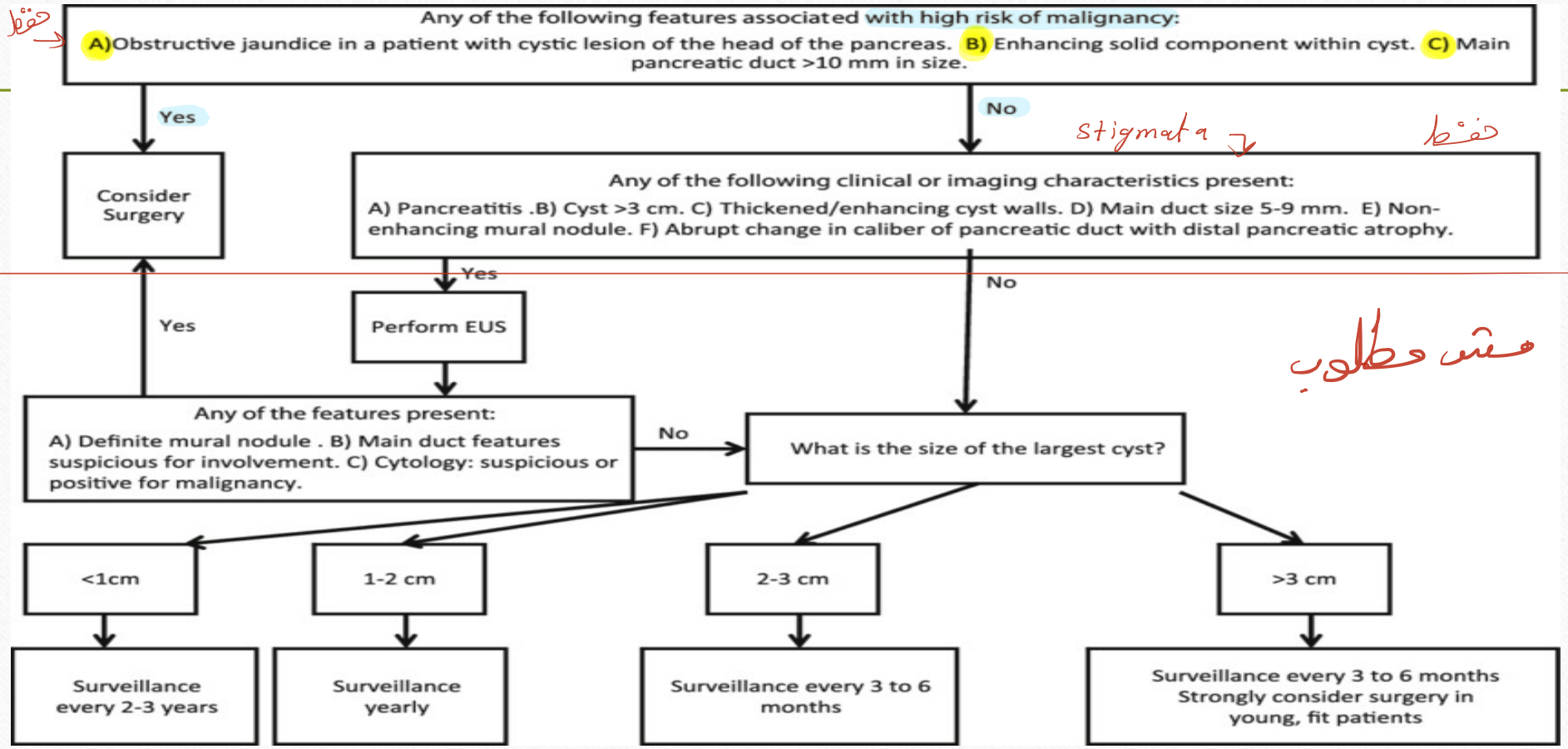
مش دائما عالي

communicate with pancre.. duct

Treat. Algorithim

W/ IPMN

Treatment


Any of the following features associated with high risk of malignancy:
A) Obstructive jaundice in a patient with cystic lesion of the head of the pancreas. B) Enhancing solid component within cyst. C) Main pancreatic duct >10 mm in size.
Yes
No
Consider Surgery
Any of the following clinical or imaging characteristics present:
A) Pancreatitis .B) Cyst >3 cm. C) Thickened/enhancing cyst walls. D) Main duct size 5-9 mm. E) Non-enhancing mural nodule. F) Abrupt change in caliber of pancreatic duct with distal pancreatic atrophy.
Yes
No
Yes
Perform EUS
Any of the features present:
A) Definite mural nodule . B) Main duct features suspicious for involvement. C) Cytology: suspicious or positive for malignancy.
No
What is the size of the largest cyst?
<1cm
1-2 cm
2-3 cm
>3 cm
Surveillance every 2-3 years
Surveillance yearly
Surveillance every 3 to 6 months
Surveillance every 3 to 6 months
Strongly consider surgery in young, fit patients

Mucinous Cystadenoma of Pancreas

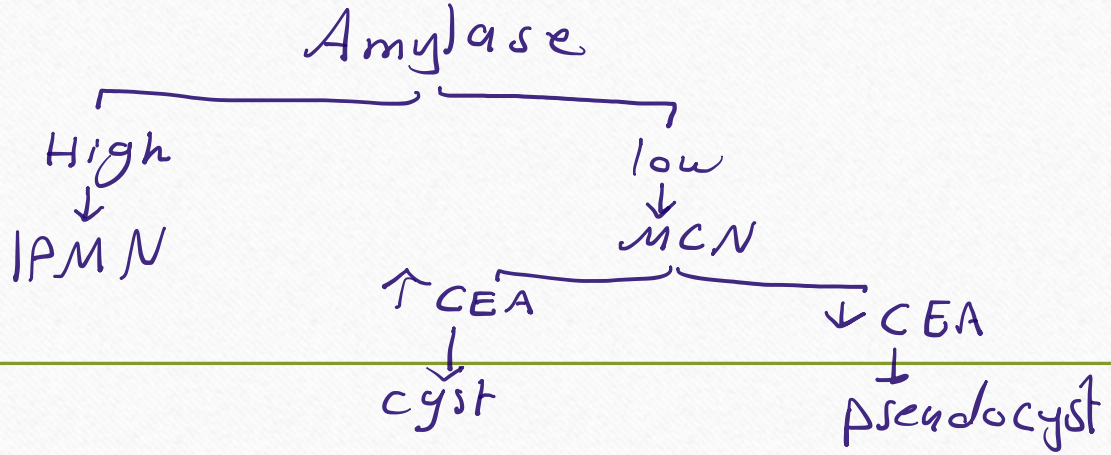
- Most Common cystic neoplasm of the pancreas
- Frequently seen in young women, mean age 5th decade
- More common in the body & tail of the pancreas
- almost exclusively found in females
- ovarian-like stroma surrounding the columnar epithelium is considered a pathognomonic finding
- Risk of invasive carcinoma 10-50 %

- CT scan: Presence of a solitary cyst with fine septations & rim of calcification
- Cyst fluid analyses: Mucin-rich aspirate, high CEA & low amylase levels

no communicat :- with main pancr-. duct

pseudocyst → ↓CEA & ↓ Amylas--

IPMN ترقیم بینہادیبیں



Thank you