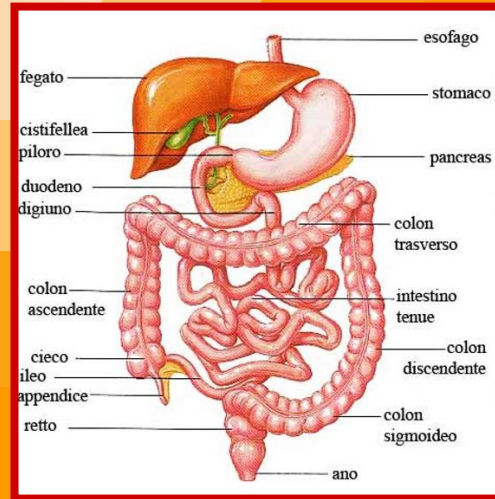


Corso di Anatomia Patologica



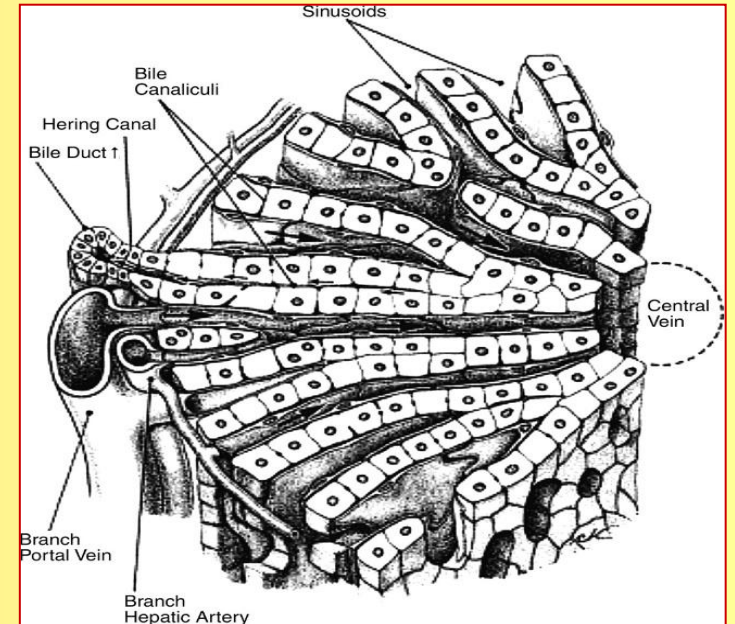
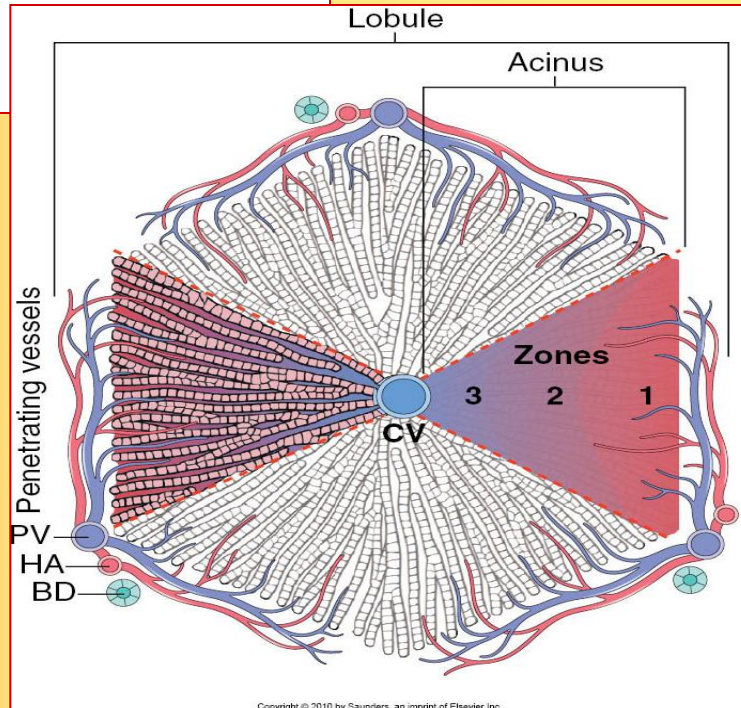
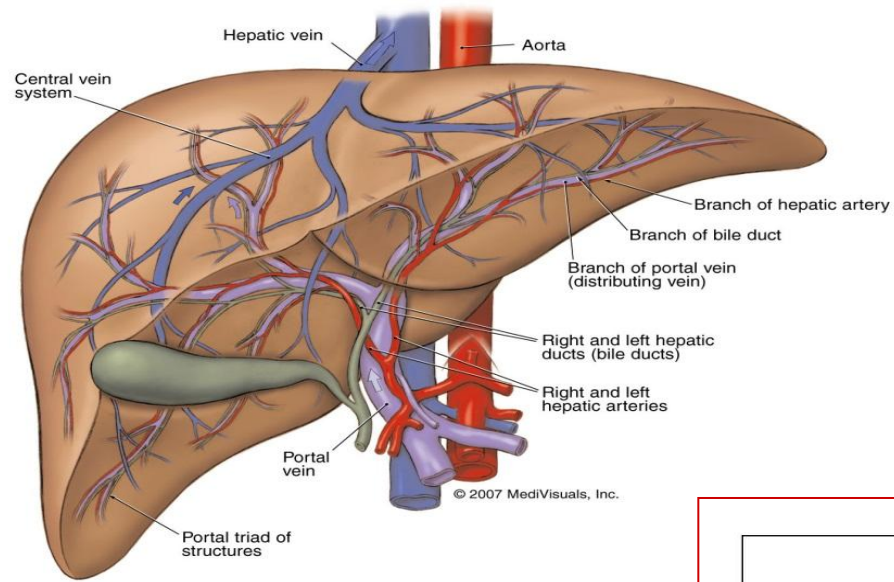
Dr. Stefano Ferretti
Dipartimento di Morfologia, Chirurgia e Medicina sperimentale
Università di Ferrara

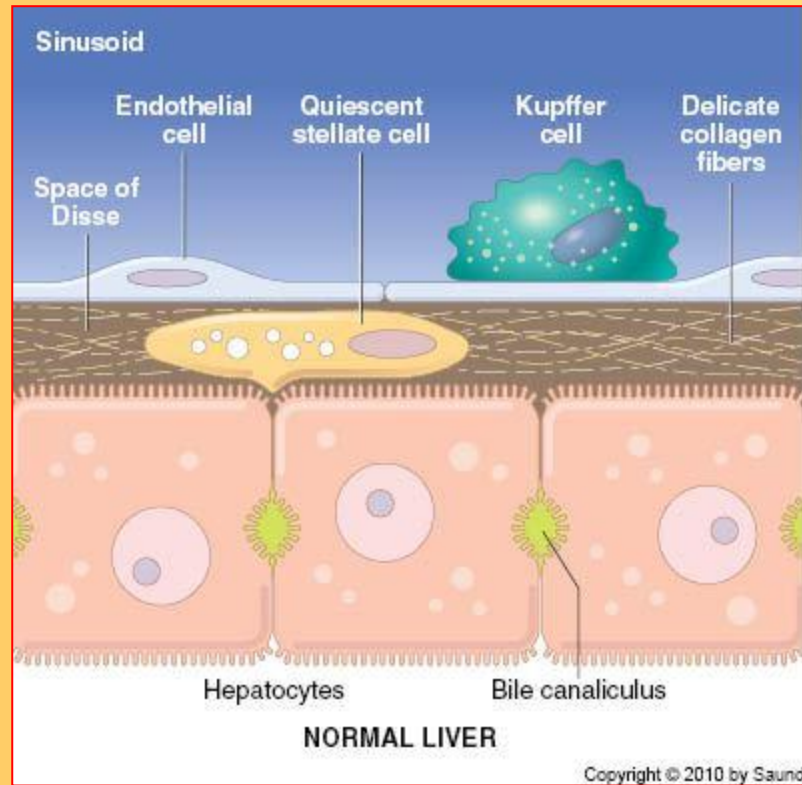
apparato gastroenterico
fegato, pancreas



Fegato

Internal Anatomy of Liver





Test Category	Serum Measurement
Hepatocyte integrity	Cytosolic hepatocellular enzymes [†] Serum aspartate aminotransferase (AST) Serum alanine aminotransferase (ALT) Serum lactate dehydrogenase (LDH)
Biliary excretory function	Substances normally secreted in bile [†] Serum bilirubin Total: unconjugated plus conjugated Direct: conjugated only Urine bilirubin Serum bile acids Plasma membrane enzymes (from damage to bile canaliculus) [†] Serum alkaline phosphatase Serum γ -glutamyl transpeptidase (GGT)
Hepatocyte synthetic function	Proteins secreted into the blood Serum albumin [†] Coagulation factors: Prothrombin (PT) and partial thromboplastin (PTT) times (fibrinogen, prothrombin, factors V, VII, IX, and X) Hepatocyte metabolism Serum ammonia [†] Aminopyrine breath test (hepatic demethylation) [†]

Funzioni principali del fegato

- regolazione metabolismo (proteine...)
- deposito
- escrezione
- detossificazione
- protezione
- emopoiesi / emocateresi
- produzione di bile
 - *digestione grassi*
 - *trasporto assorbimento prodotti digestione*
 - *rimozione bilirubina, colesterolo, xenobiotici*

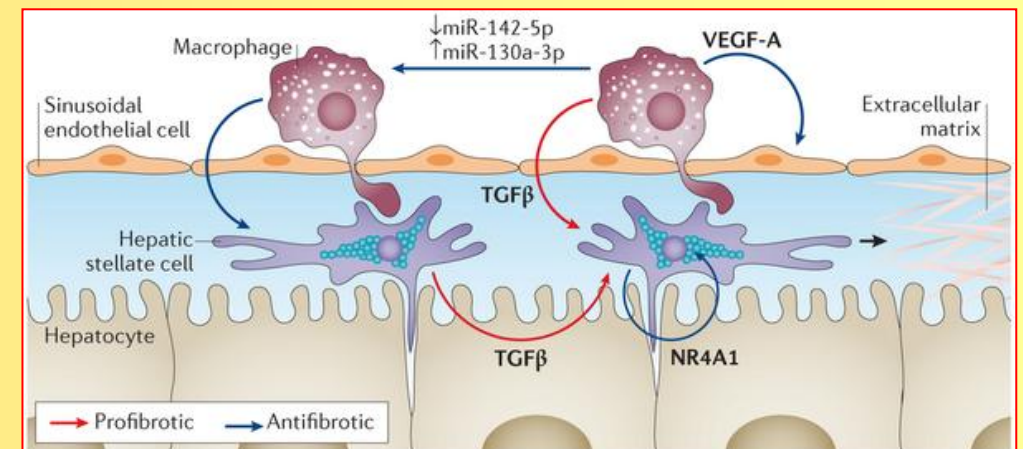
Danno epatico

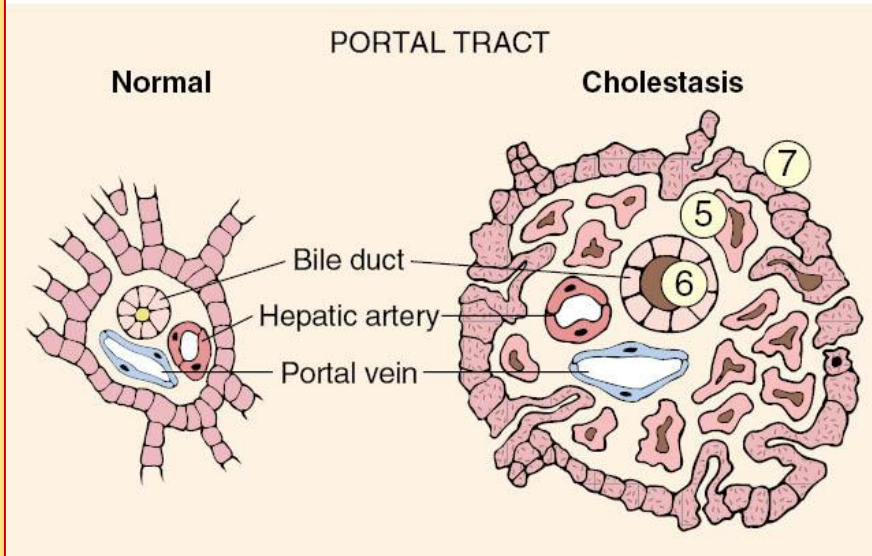
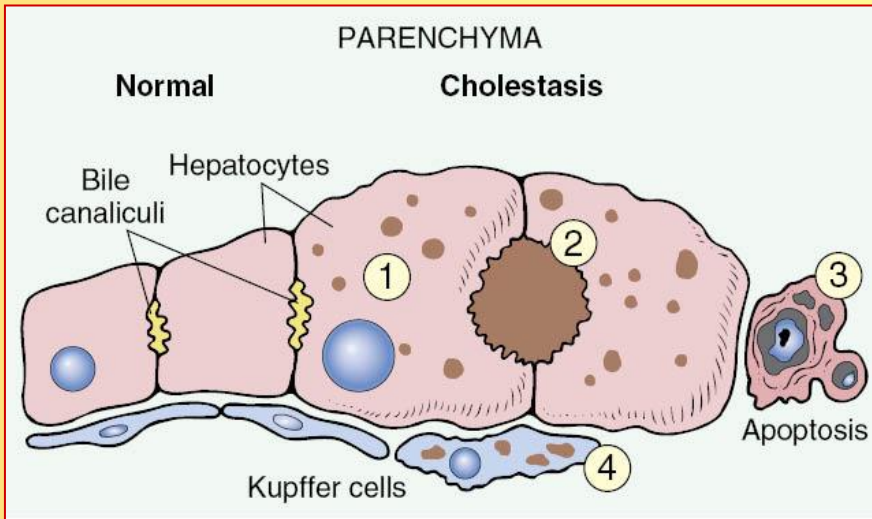
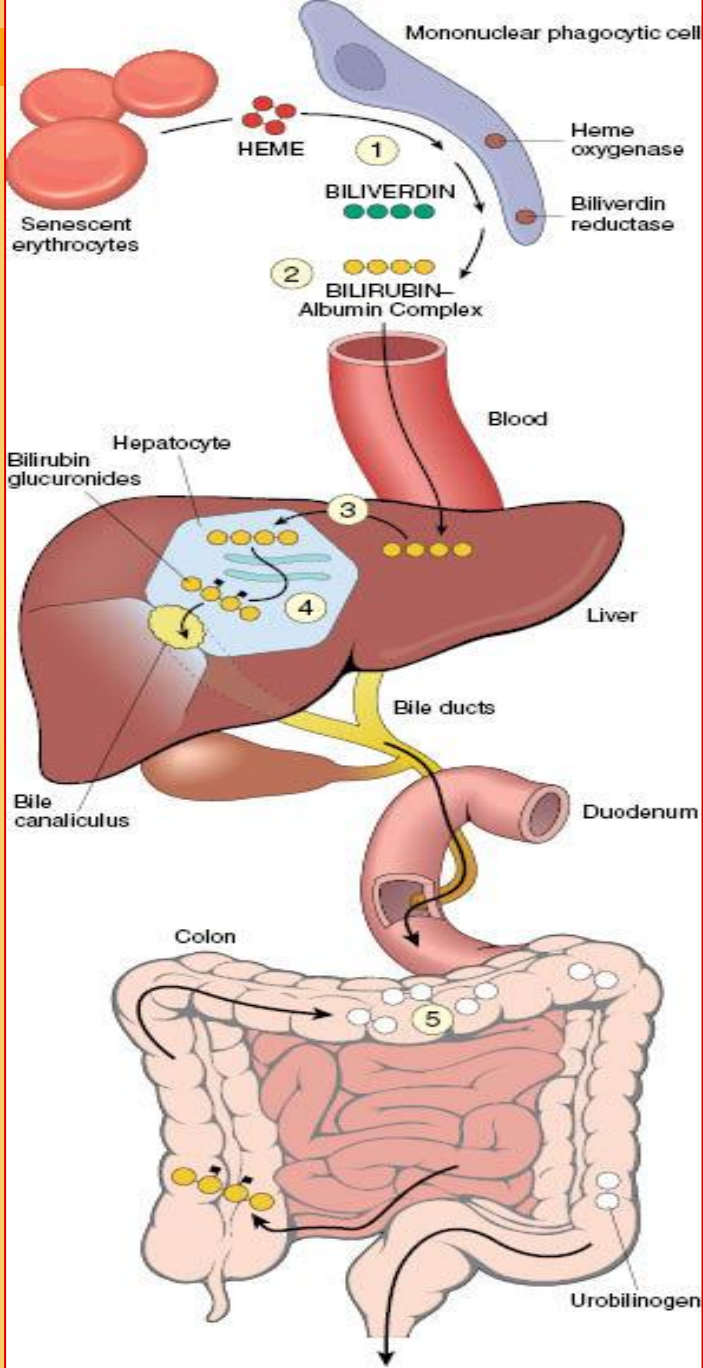
- tossiche (alcool, droghe, farmaci, ...)
- infettivo (epatiti)
- metabolico
- malattie sistemiche

Risposta

- necrosi, apoptosi
- rigenerazione
- fibrosi

Test Category	Serum Measurement
Hepatocyte integrity	Cytosolic hepatocellular enzymes [†] Serum aspartate aminotransferase (AST) Serum alanine aminotransferase (ALT) Serum lactate dehydrogenase (LDH)
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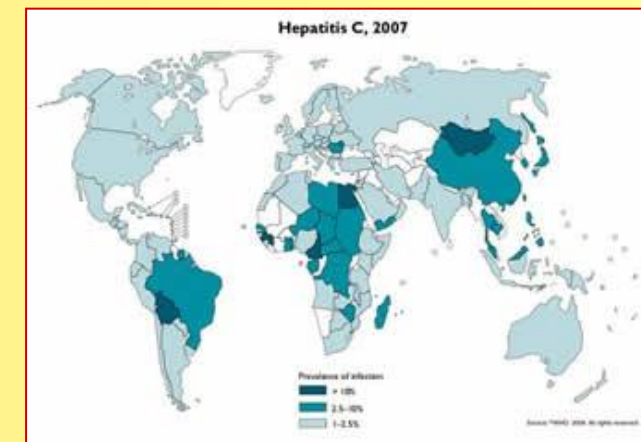
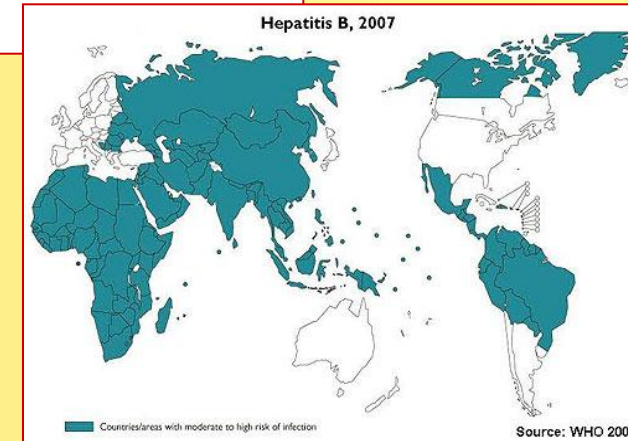
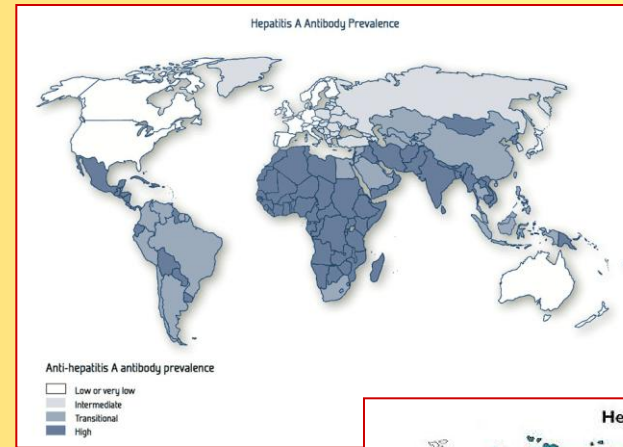


Etiologia:

- Virale
 - A,B,C,D,E
 - EBV
 - CMV (neonati, immunosoppr.)
 - Febbre gialla
 - Rosolia
 - Herpesvirus
 - Adenovirus
- Alcool
- Farmaci
- Tossine
- Deficit di α -antitripsina
- Autoimmunità
- Batteri
- Parassiti
- Elminti

Sindromi cliniche:

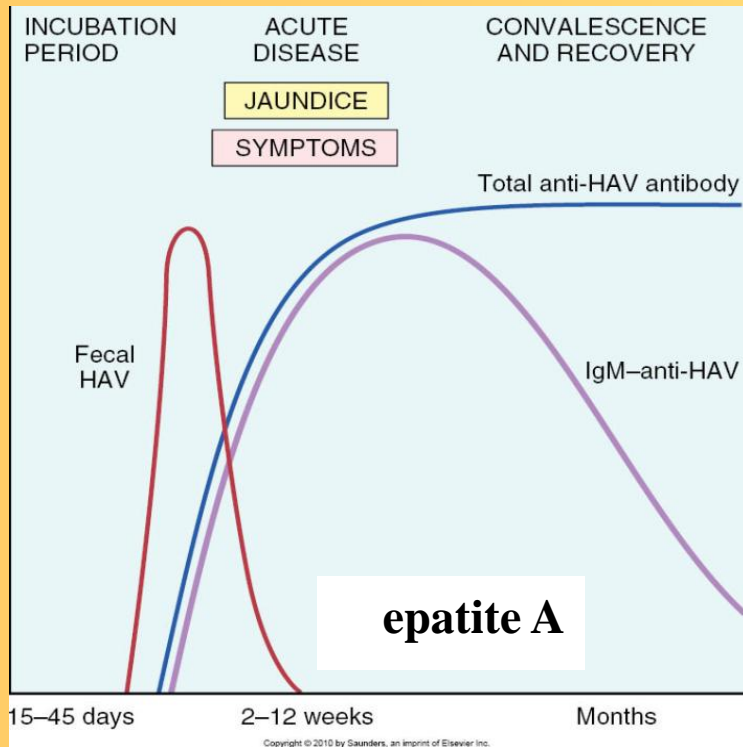
- Infezione acuta asintomatica con guarigione
- Infezione acuta sintomatica con guarigione
 - anitterica/itterica
- Epatite cronica
 - con/senza progressione a cirrosi
- Epatite fulminante con necrosi epatica massiva



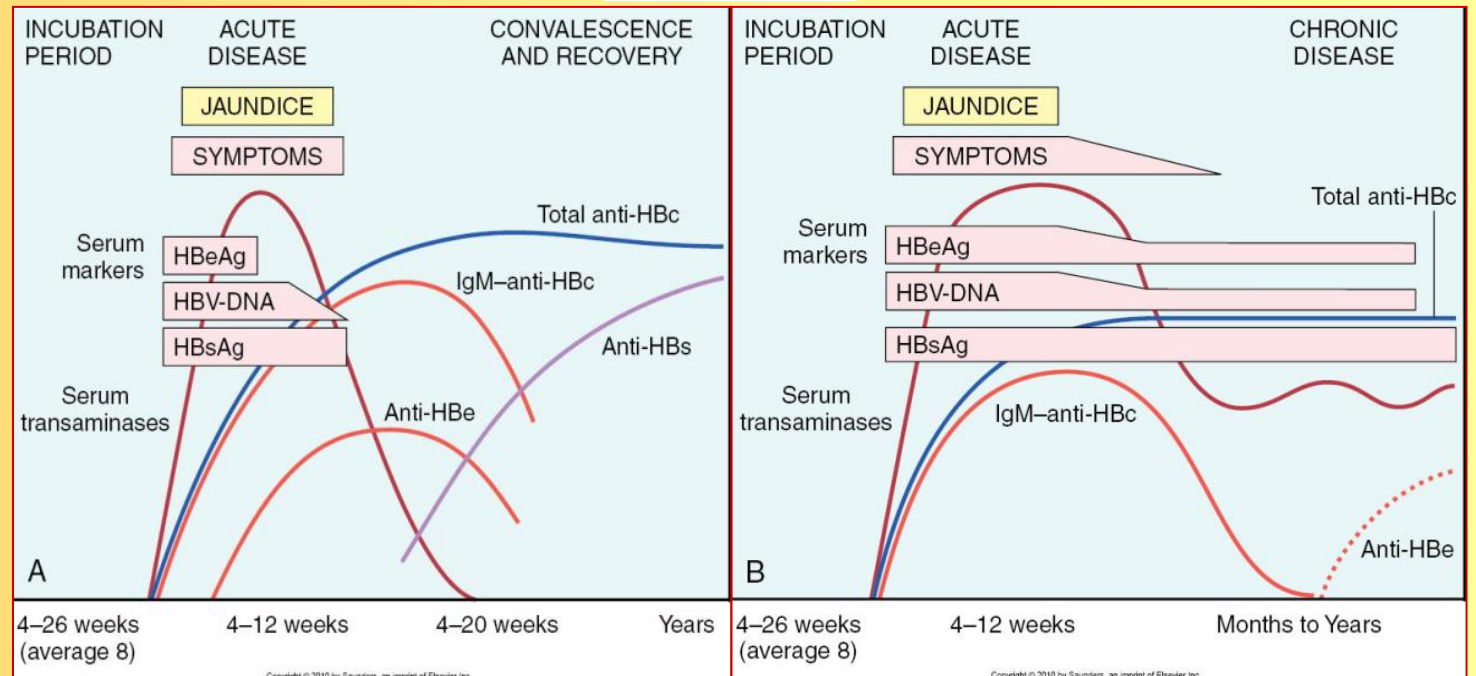
epatiti

Epatiti virali					
virus	A	B	C	D	E
Tipo	ssRNA	Parz. dsDNA	ssRNA	Circ. defect. ssRNA	ssRNA
Famiglia	Hepatovirus (picornavirus)	Hepadnavirus	Flaviridae	Partic. Subvirale (famiglia deltaviridae)	Calicivirus
Trasmisione	Oro-fecale	Parenterale	Parenterale	Parenterale	Oro-fecale
Incubaz. media	2-4 settim.	1-4 mesi	7-8 settim.	1-4 mesi	4-5 settim.
Evoluzione cronica	Mai	10%	≈80%	5%(coinfez.) >70%(superinf.)	Mai
Diagnosi	IgM ab	HBsAg ab HBcAg	HCV RNA	IgM, IgG HDV RNA (siero) HDAg (feg.)	PCR HEV IgM, IgG

epatite virale markers



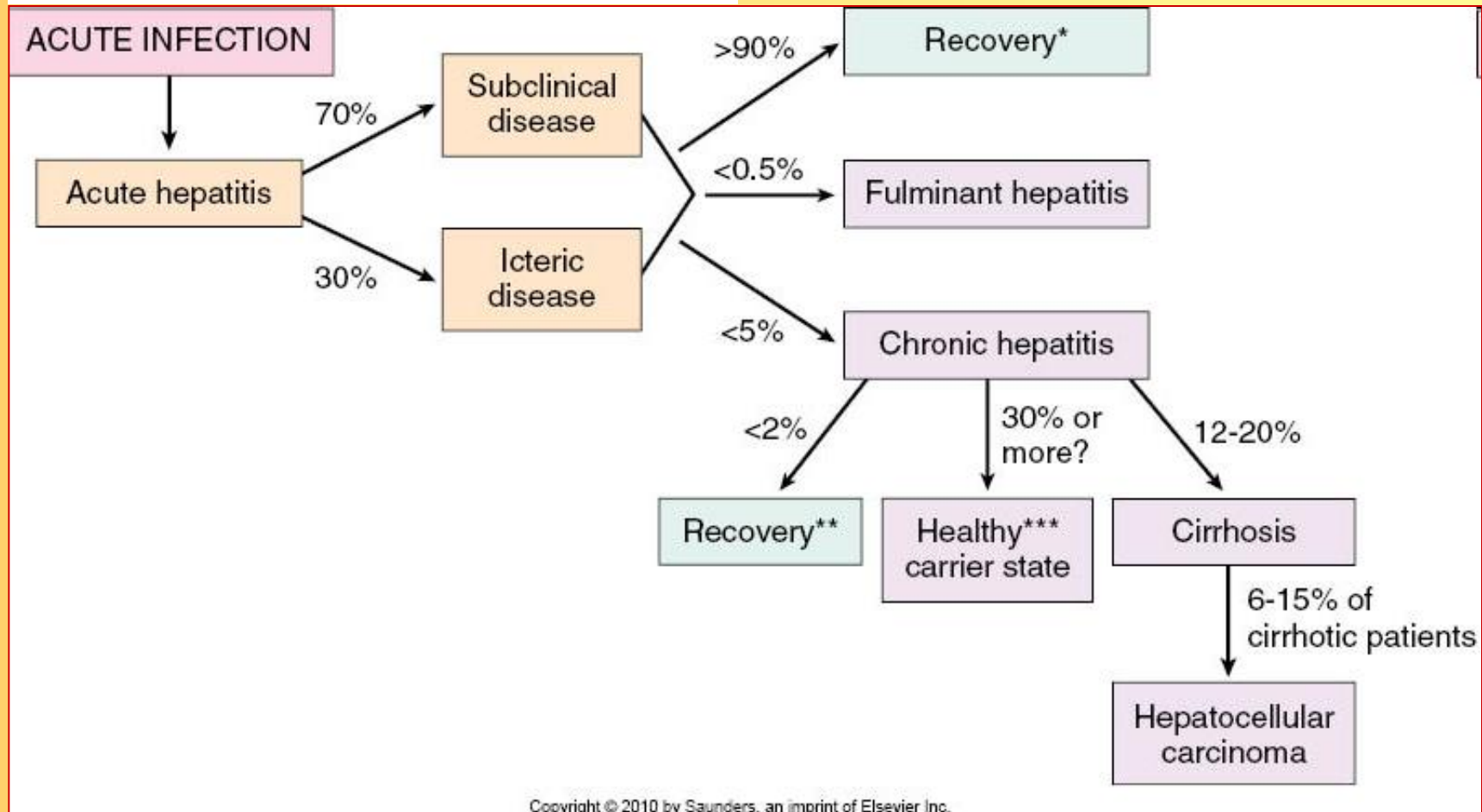
epatite B



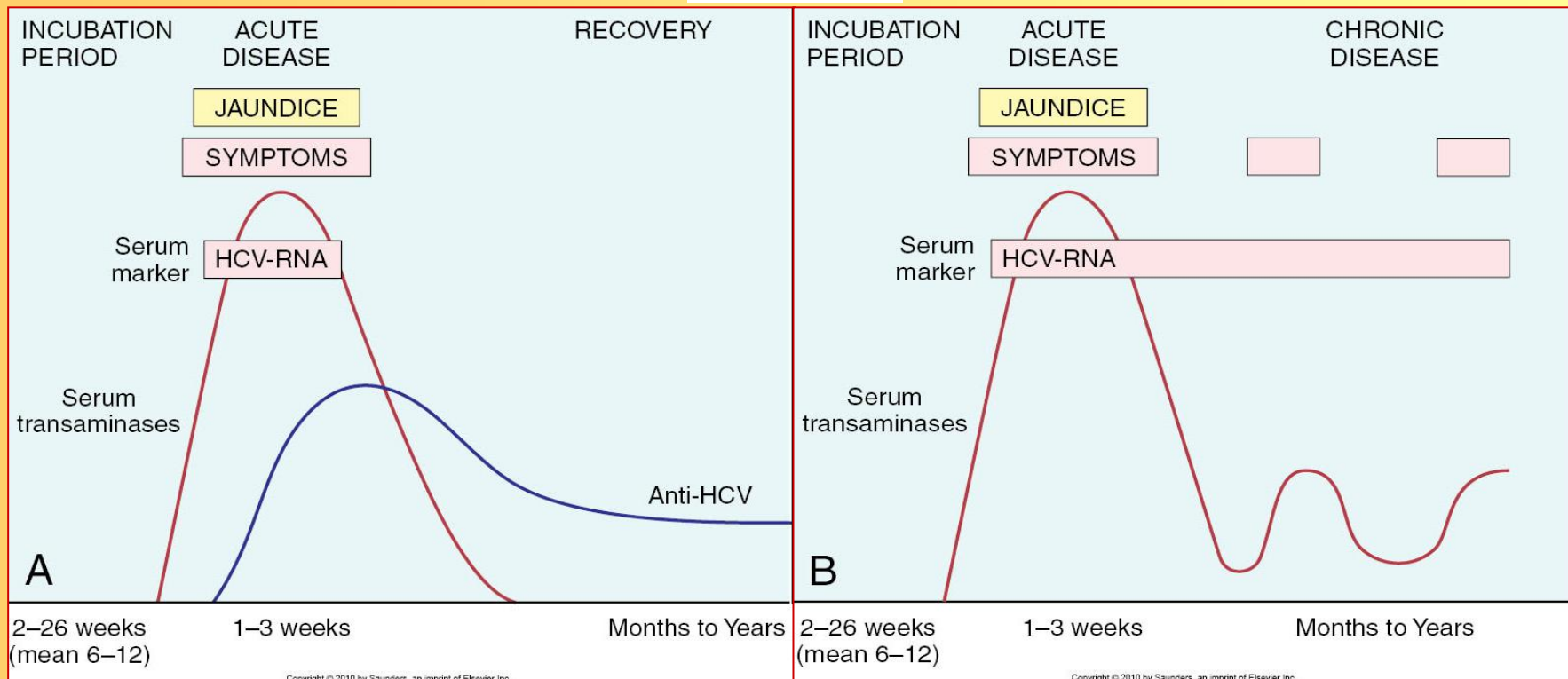
acuta-guarigione

cronicizzazione

Epatite B: evoluzione clinico-patologica



epatite C



acuta-guarigione

cronicizzazione

Quadro clinico:

Infezione acuta asintomatica con guarigione

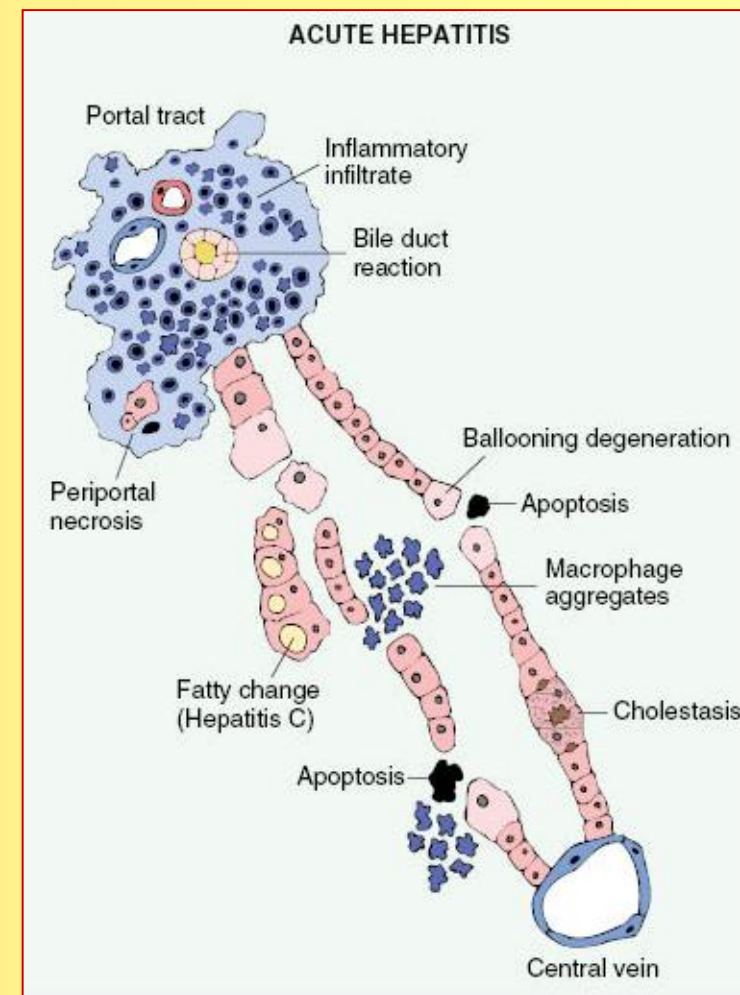
- Rialzo transaminasi, Ab
- HAV, HBV (neonati)

Infezione acuta sintomatica con guarigione

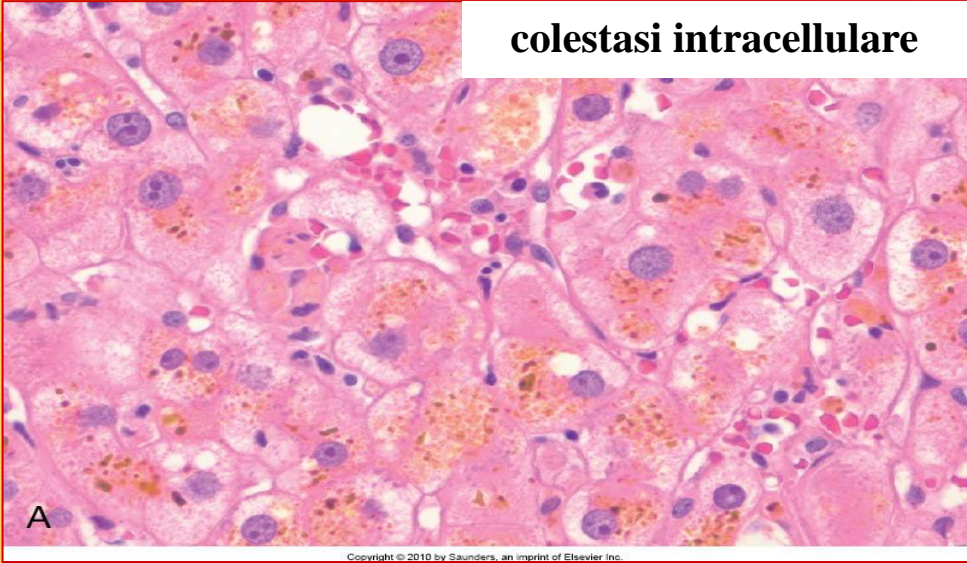
- Virus epatotropi
- Incubazione
- Fase preitterica
- Fase itterica
- Convalescenza

Anatomia Patologica:

- Granuli HBsAg (HBV*)
- Infiltrato linfocitario sp. portali (HCV+)
- Steatosi (forme tossiche)
- Deg. balloniforme
- Colestasi
- Infiammazione (portale)
- “Piece-meal necrosi”)
- Ipertrofia Kuppfer (fagocitosi)

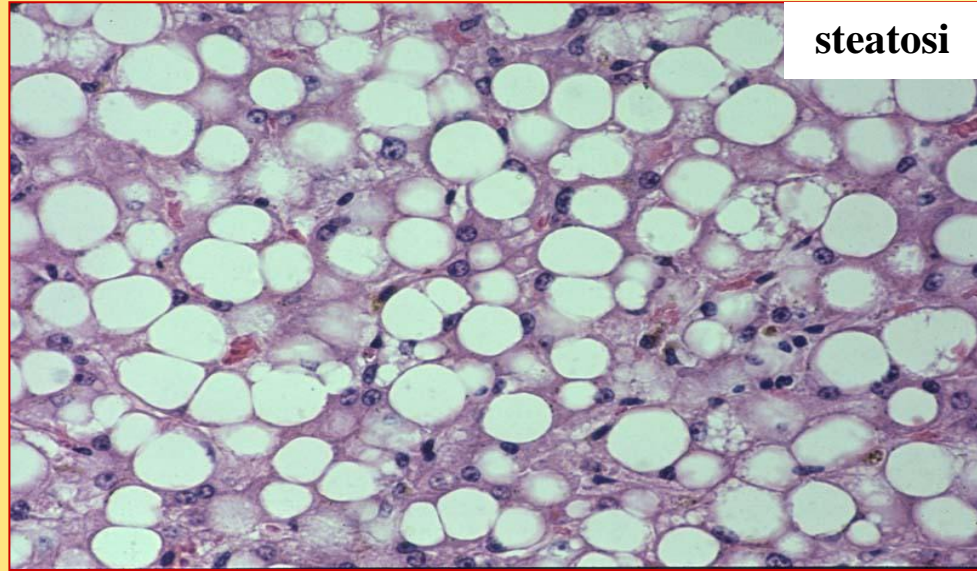


colestasi intracellulare



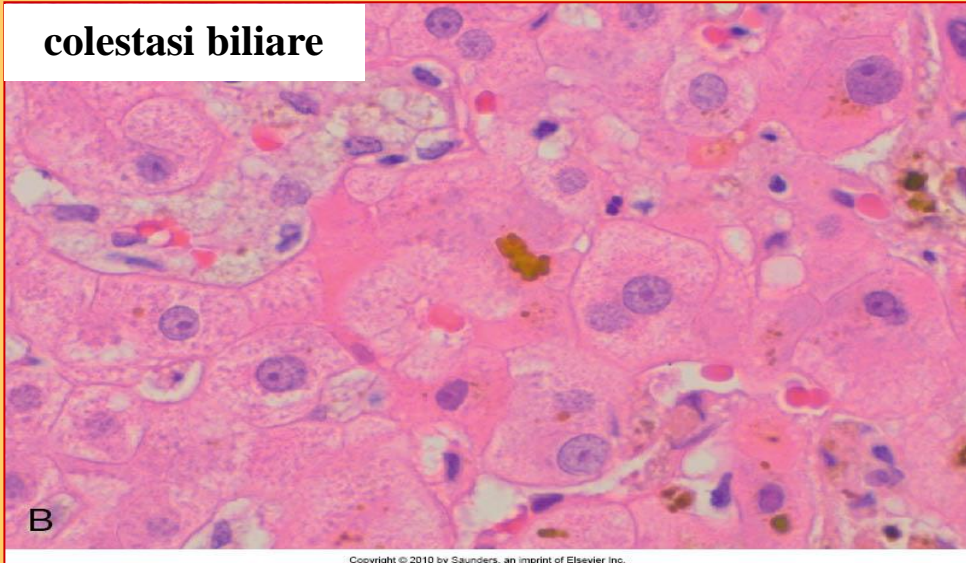
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steatosi



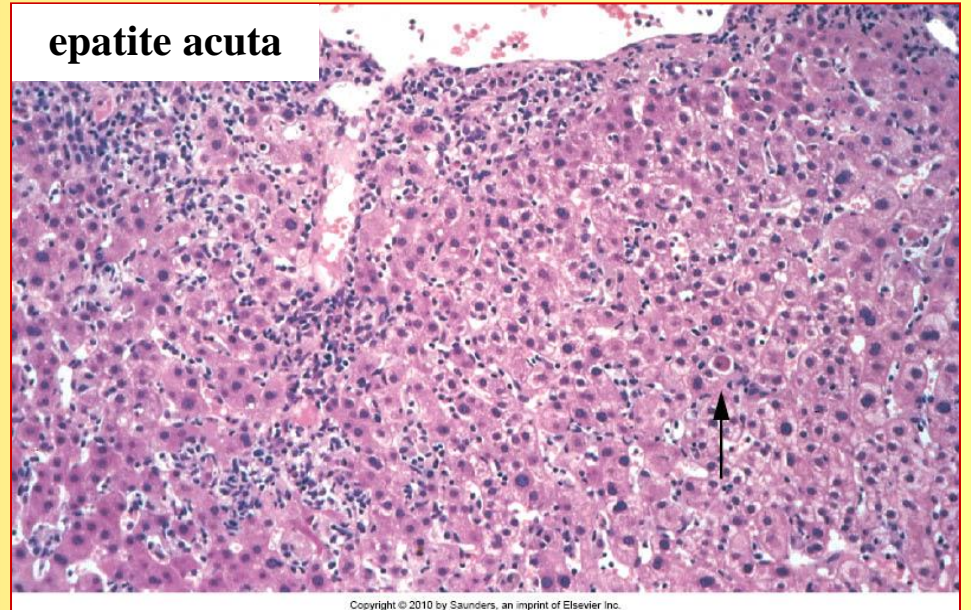
epatiti

colestasi biliare



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epatite acuta



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Quadro clinico:

Durata oltre i 6 mesi (siero, biochim., clinica)

Fatica, malessere, inappetenza, sub-ittero

Frequente etiologia HCV (+ raram. HBV)

Sintomi:

- Spider angioma, eritema palmare
- Lieve splenomegalia
- Aum. transaminasi, t. prothr., bilir., AF
- Aum. tempo protrombina
- (vasculiti da immunocompl.)
- Crioglobulinemia (35% epatite C)

Complicanze/evoluzione:

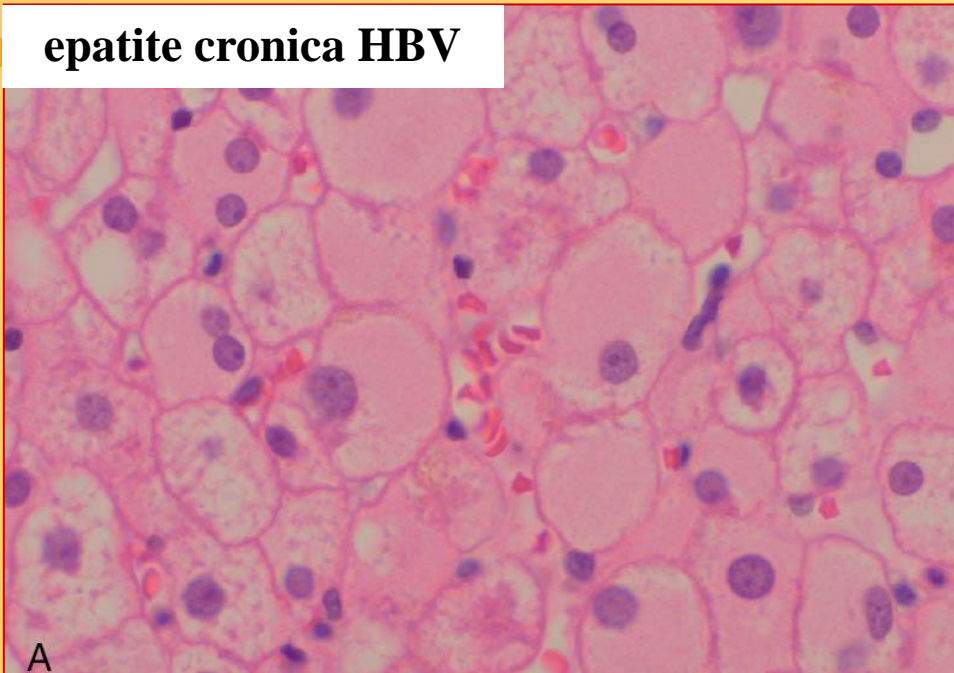
- In funzione dell'età
- Cirrosi
- Carcinoma

epatite cronica

Anatomia Patologica:

- Quadri a severità variabile
- Infiammazione portale
- Caratteri variabili (HBV, HCV,...)
- Steatosi
- Invasione lamina limitante
- Necrosi (a ponte)
- Fibrosi
 - Portale, p.-portale, p-centrale
- Evoluzione cirrotica

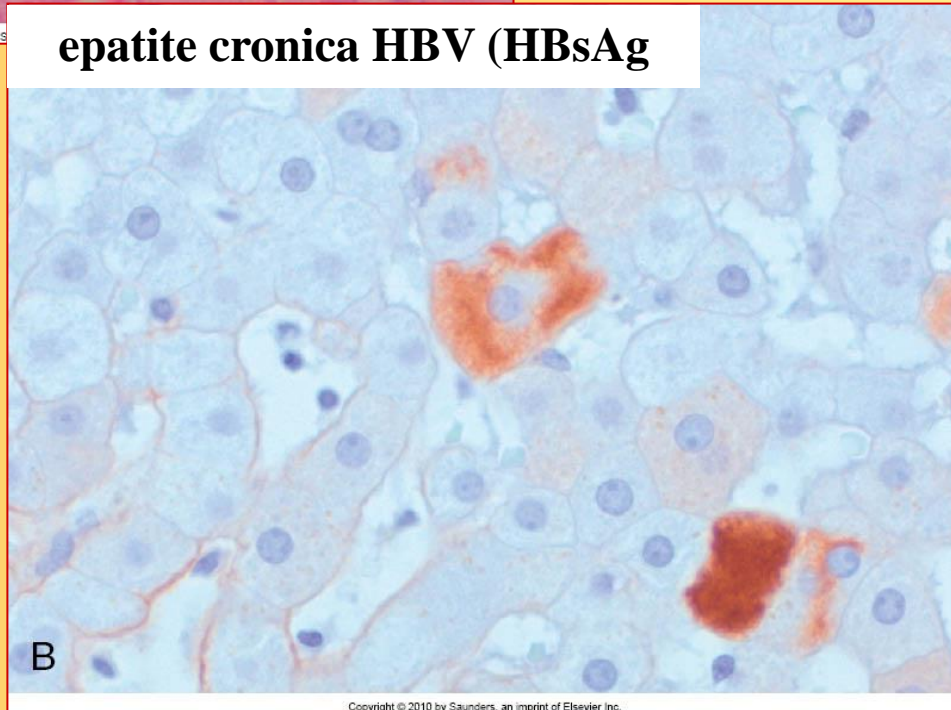
epatite cronica HBV



A

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epatite cronica HBV (HBsAg)

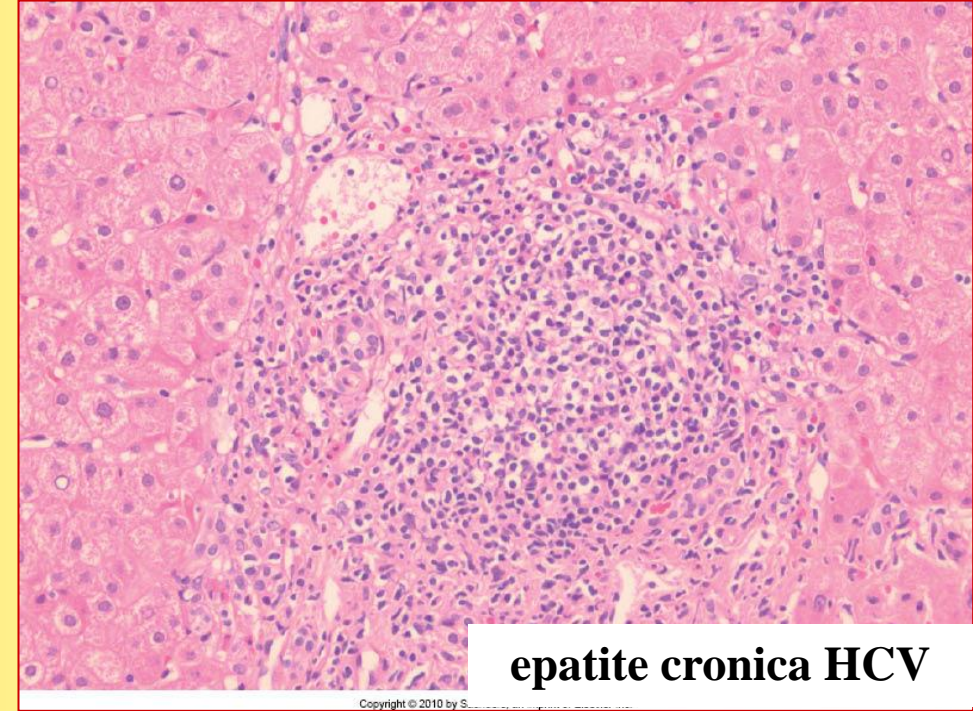


B

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epatite cronica

epatiti



epatite cronica HCV

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Quadro clinico:

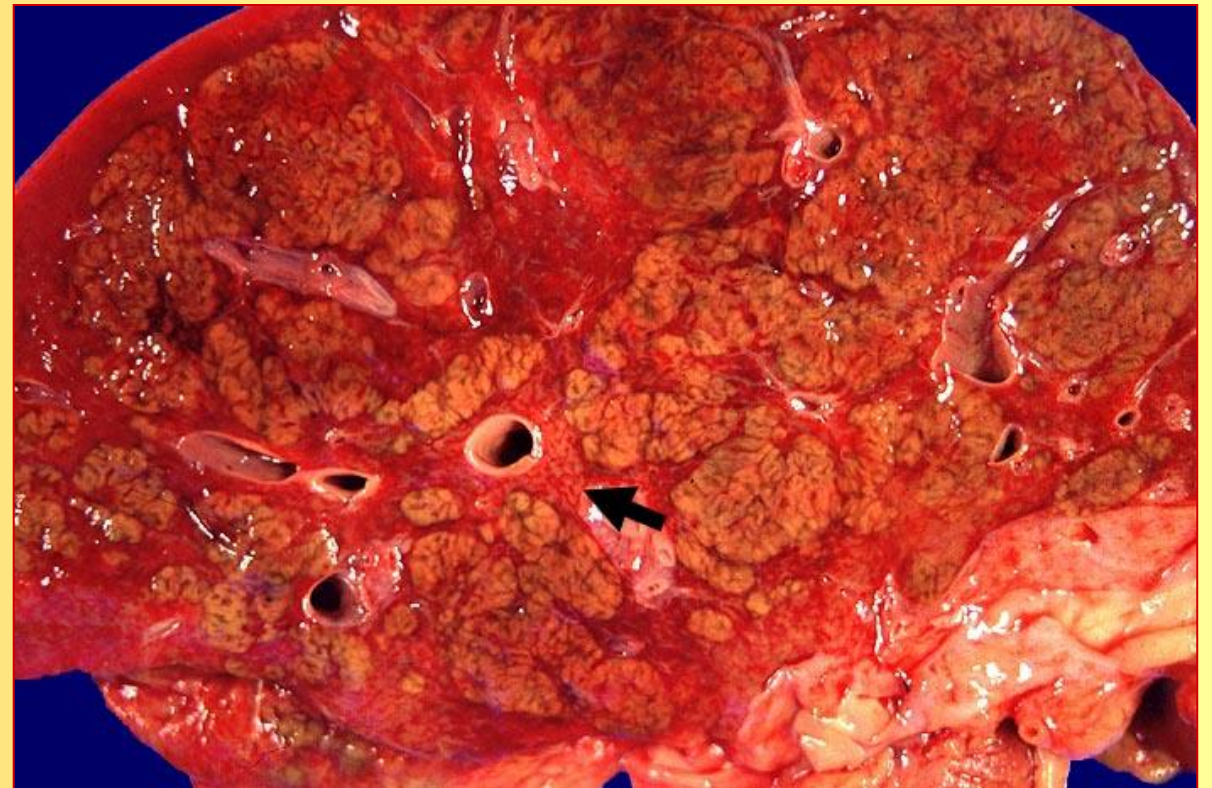
- Etiologia virale (HBV, HCV, HSV, Dengue)
- Etiologia tossica
- Etiologia “sconosciuta”
- Decorso rapido (2-3 settimane)
- Insufficienza epatica
- Encefalopatia
- Terapia di supporto
- Indicazione al trapianto

Anatomia Patologica:

- Necrosi massiva (emorragica)
- Scarsa flogosi
- Riduzione parenchima (500-700 g)
- Consistenza molle
- Raggrinzimento capsulare

epatite fulminante

epatiti

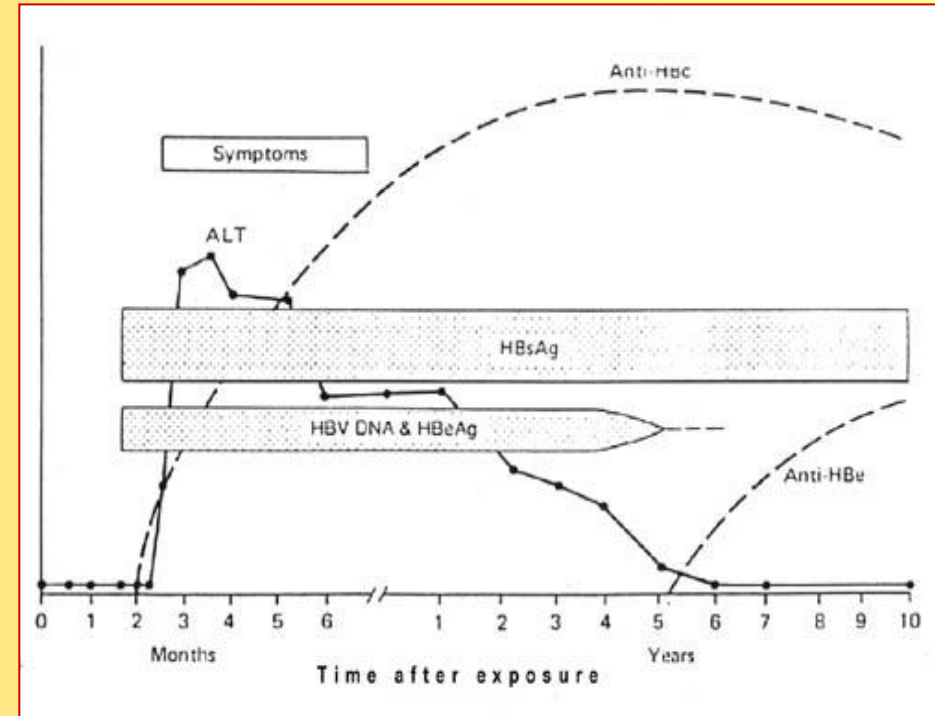
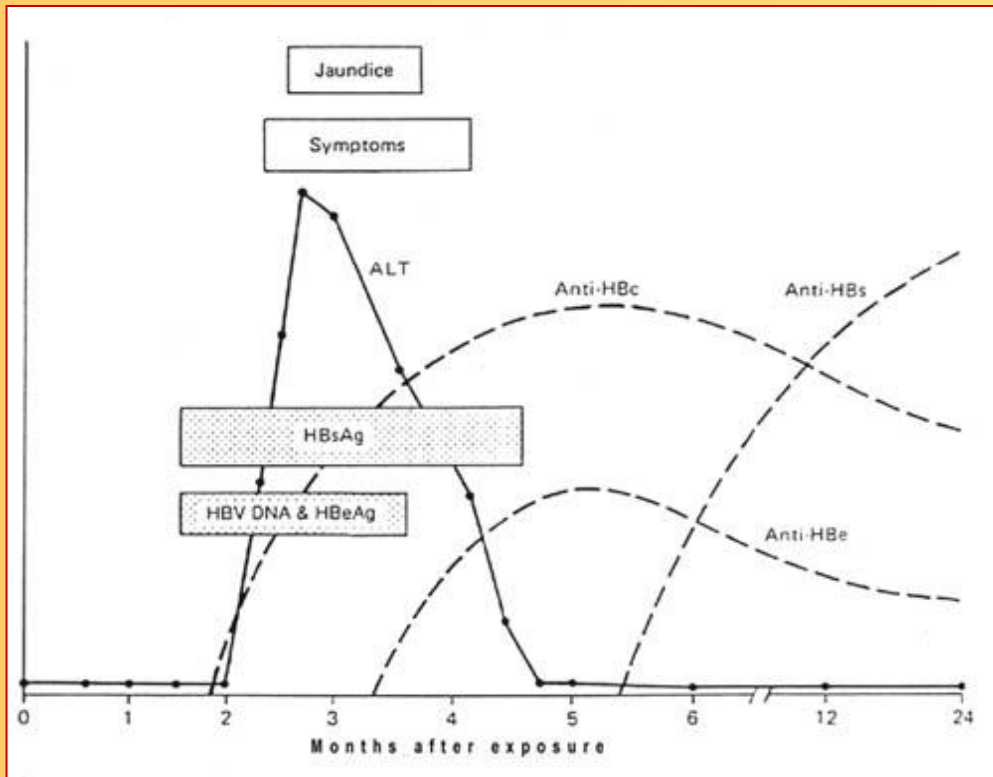


Quadro clinico:

Stato di “portatore”

- Assenza di sintomi
- Sieropositività
- Assenza di epatite (non progressione)
- HBV in 1% casi USA
- HBV in 90% casi aree endemiche

epatiti



Quadro clinico:

Coinfezione HIV

- 10% HBV+ e 30% HCV+ in HIV+
- Prognosi severa in epatite cronica HIV+
- Effetto esacerbante (non chiaro) HIV in HBV+ e HCV+

Quadro clinico:

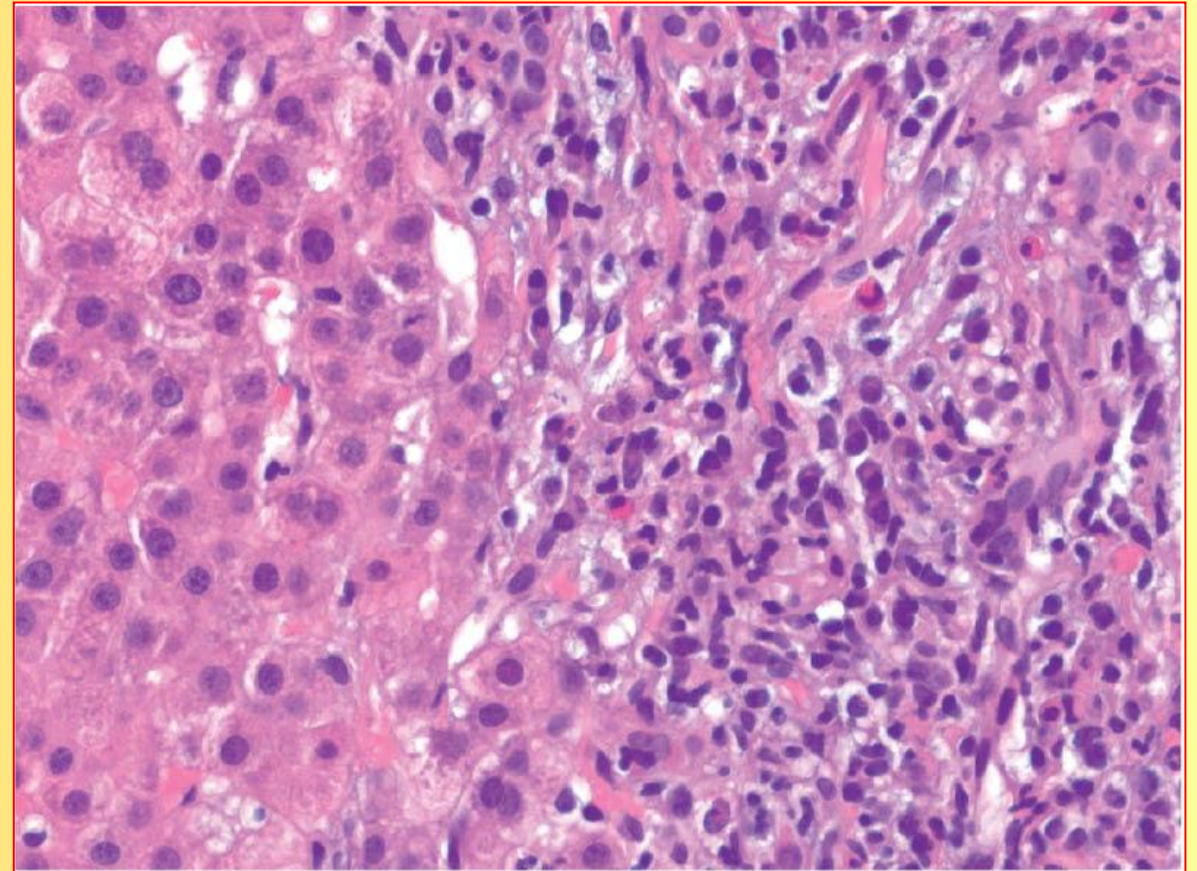
- Decorso variabile (lento-fulminante)
- Predominanza femminile (78%)
- Assenza di markers virali
- IgG e γ globuline elevate
- Tipo 1 (+ comune)
 - ANA, SMA, AAA, SLA/LP
- Tipo 2
 - ALKM-1, CYP2D6, ACL-1

Anatomia Patologica:

- Marcato infiltrato infiammatorio
- Clusters di pl.cellule porto-lobulari
- Necrosi
- Fibrosi
- Colangite autoimmune
- Prognosi severa (OS 60% a 4 mesi)
- Evoluzione cirrotica (40% survivors)
- Recidiva post-trapianto

epatite
autoimmune

epatiti



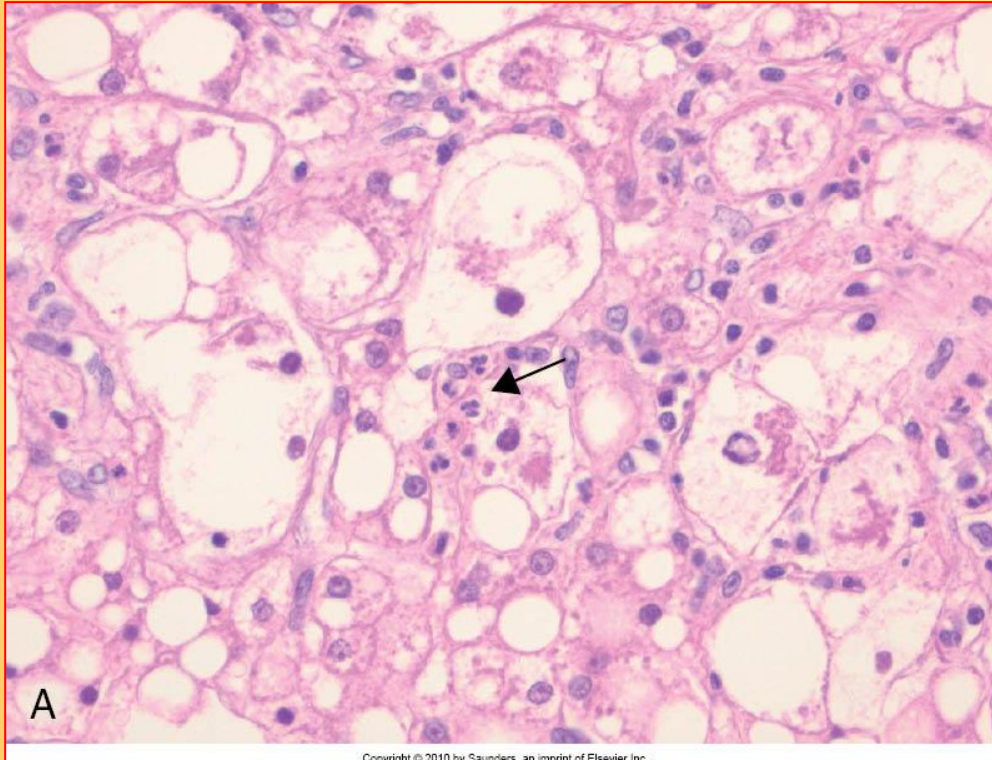
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Quadro clinico:

- Epatomegalia
- Aumento bilirubina e FA
- Sintomatologia variabile
- Anoressia
- Perdita di peso
- Remissione condizionata
- Evoluzione cirrotica
- Quadri fulminanti

epatite
alcolica

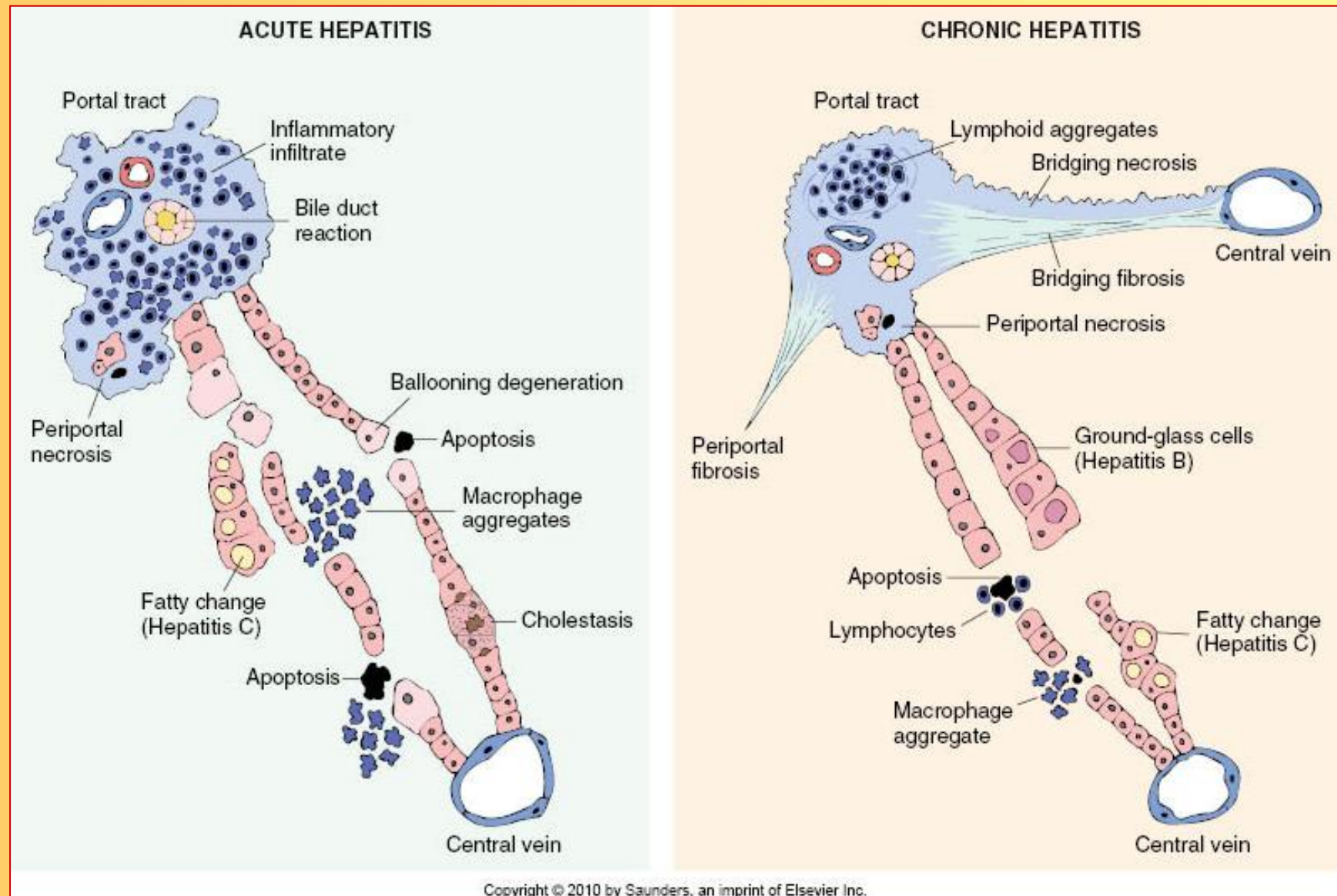
epatiti



Anatomia Patologica:

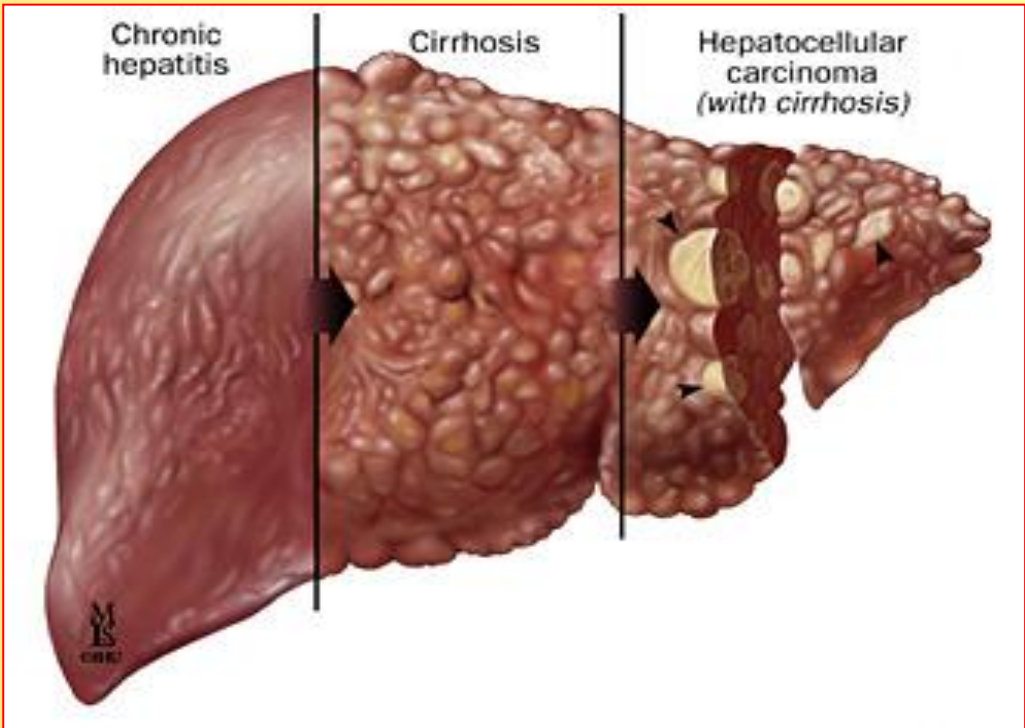
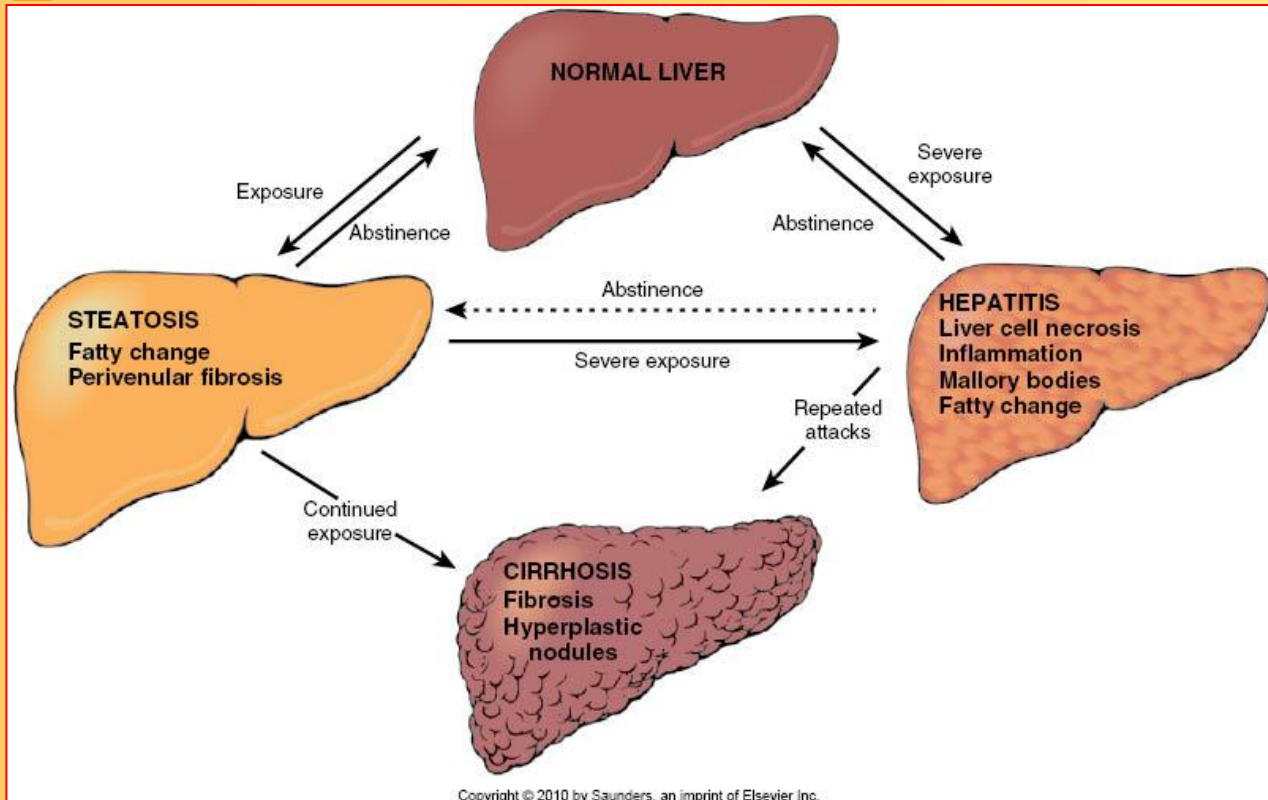
- Steatosi epatica
 - Micro/macrovescicole
 - Epatomegalia (4-6-kg)
 - Fibrosi assente/iniziale
 - Reversibilità
 - Epatite alcolica
 - Deg. balloniforme e necrosi epatocit.
 - Corpi di Mallory
 - Flogosi neutrofila
 - Fibrosi
 - Evoluzione cirrotica
 - Decorso lento e insidioso
 - Epatomegalia
 - Setti fibrosi p.-portali e p.-centrali
 - Disegno micronodulare
- Prognosi grave

Quadri di epatite tossica		
Quadro	Morfologia	Agenti associati
Colestasi	Colestasi senza infiammazione	Contraccettivi, steroidi, ormonot.sost.
Epatite colestatica	Colestasi con infiammaz., necrosi, Necrosi duttale bil.	Antibiotici, fenotiazine
Necrosi epatocellulare	Necrosi "spotty" Necrosi submass. zona 3 Necrosi massiva	Metildopa, fenitoina, acetaminofene, alotano, isoniazide
Steatosi	Macrovescicole	Etanolo, metotrexate, corticosteroidi, nutrizione parenterale
Steatoepatite	Microvescicole, C. di Mallory	Amiodarone, etanolo
Fibrosi, cirrosi	Fibrosi periportale e pericellulare	Metotrexate, isoniazide, inalapril
Granulomi	Epitelioidi, non caseosi	Sulfonamidi, altri
Lesioni vascolari	Occlusione venosa S. di Budd-Chiari Dilatazione sinusoidi Peliosi	Chemioterapia Contraccettivi orali Contraccettivi orali Steroidi, tamoxifen
Neoplasie	Adenoma epatico Carcinoma epatocellulare Colangiocarcinoma Angiosarcoma	Contraccettivi orali Thorotrast Thorotrast Thorotrast, CVM



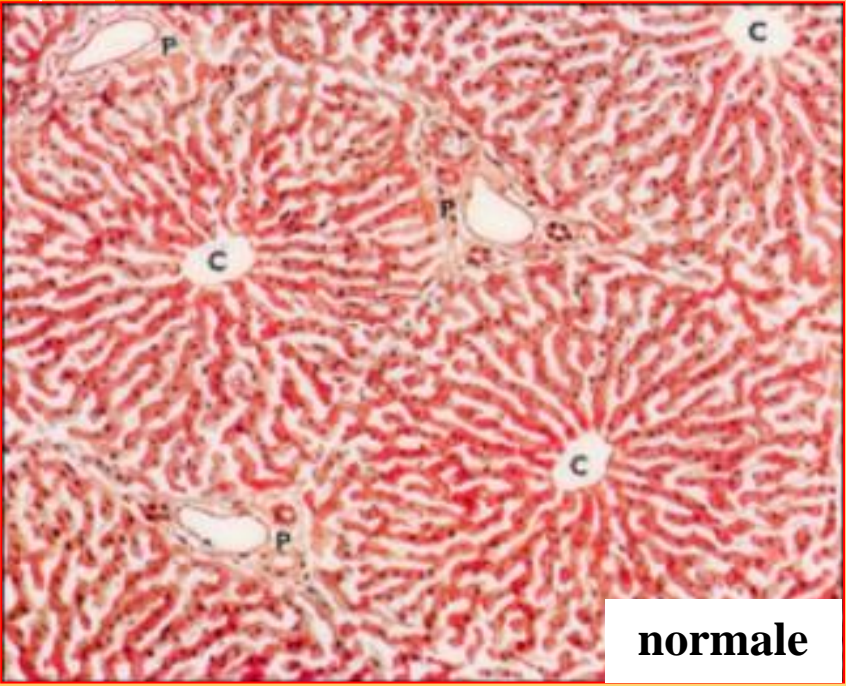
evoluzione

epatiti

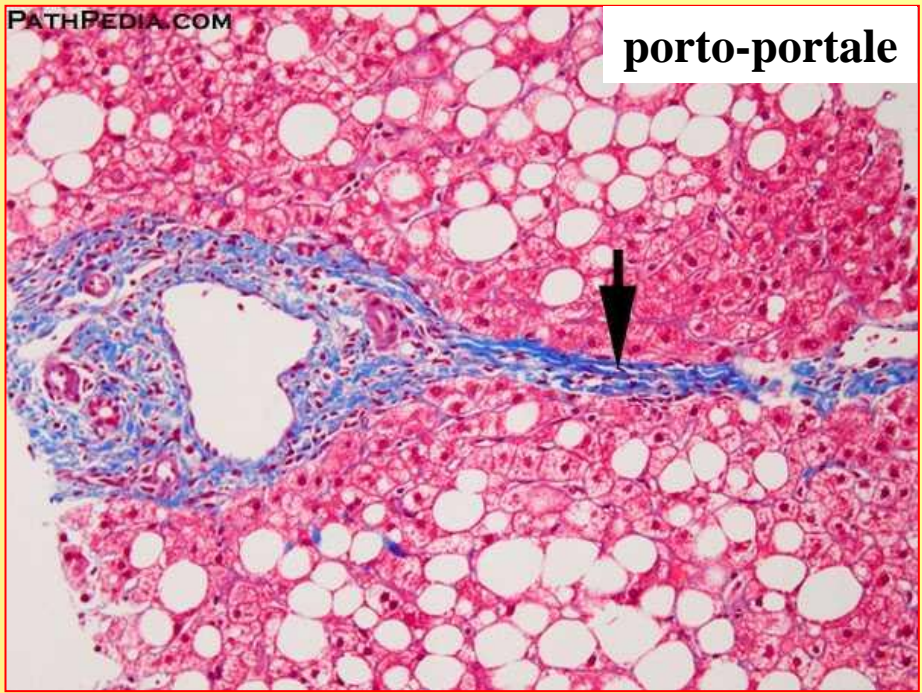


epatiti

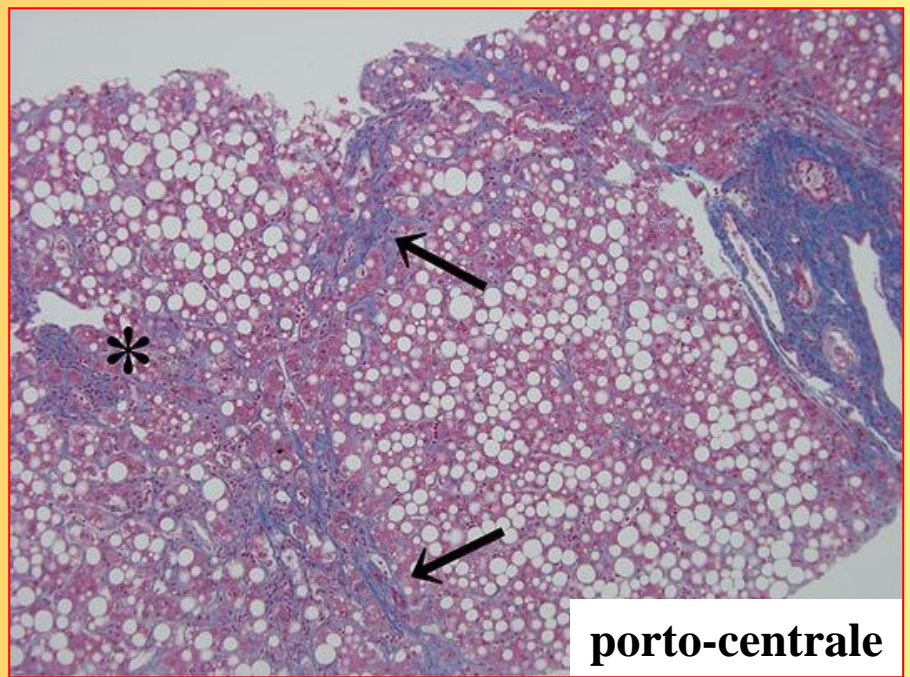
**evoluzione
fibrosi**



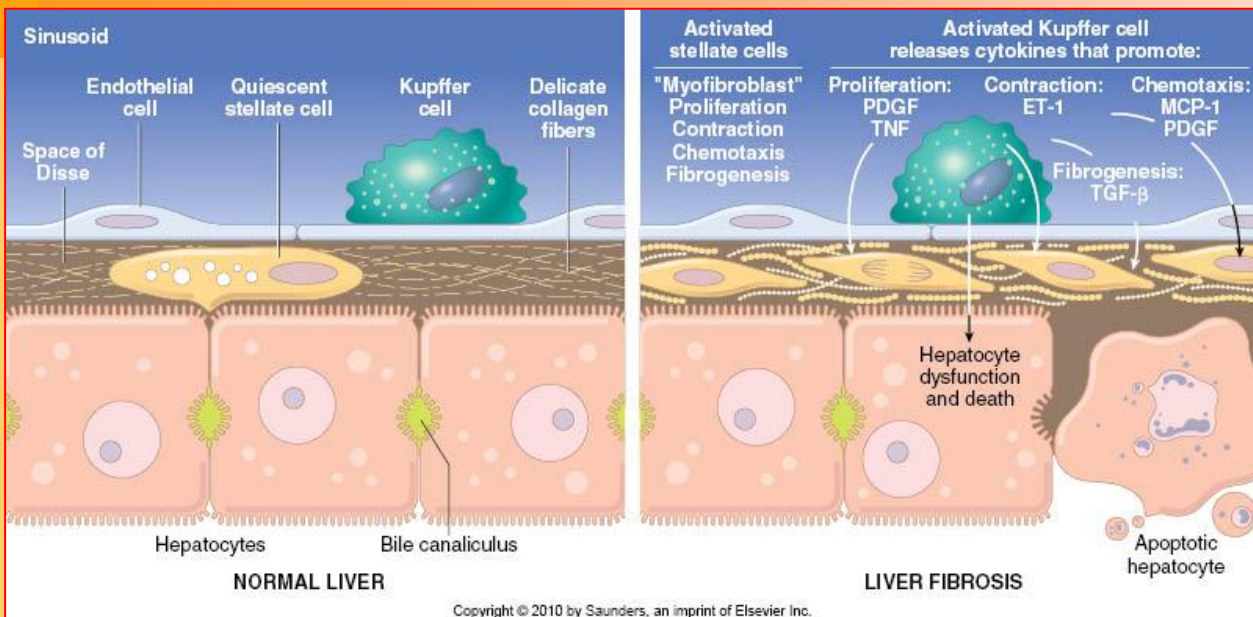
normale



porto-portale

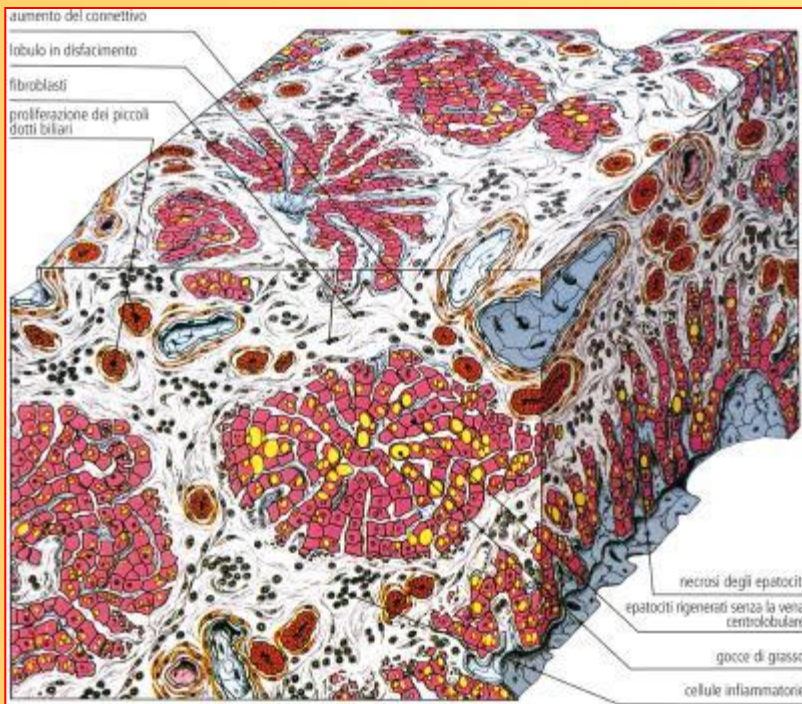


porto-centrale



cirrosi

patogenesi

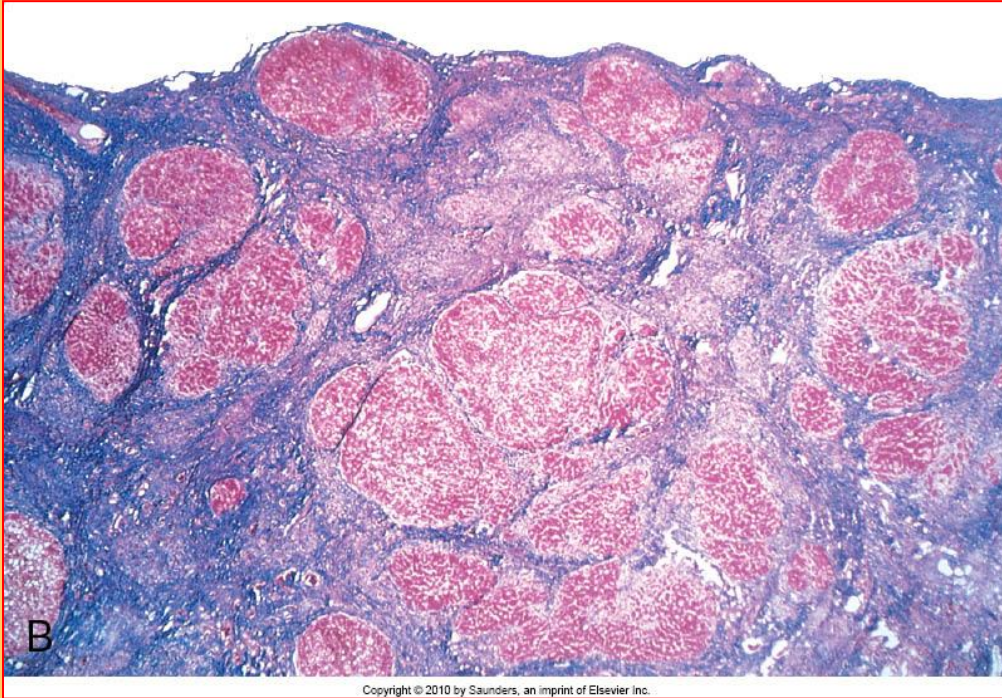


Anatomia Patologica:

- Setti fibrosi a ponte
- Noduli parenchimali
- Sovvertimento strutturale

morfologia

cirrosi



Quadro clinico:

- **Decorso lento e asintomatico**
- **Inizialmente: Anoressia, perdita peso, stanchezza**
- **Tardivamente: segni di insufficienza epatica**
- **Ipertensione portale**
- **Trasformazione maligna (epatocarcinoma)**

malattie delle vie biliari intraepatiche

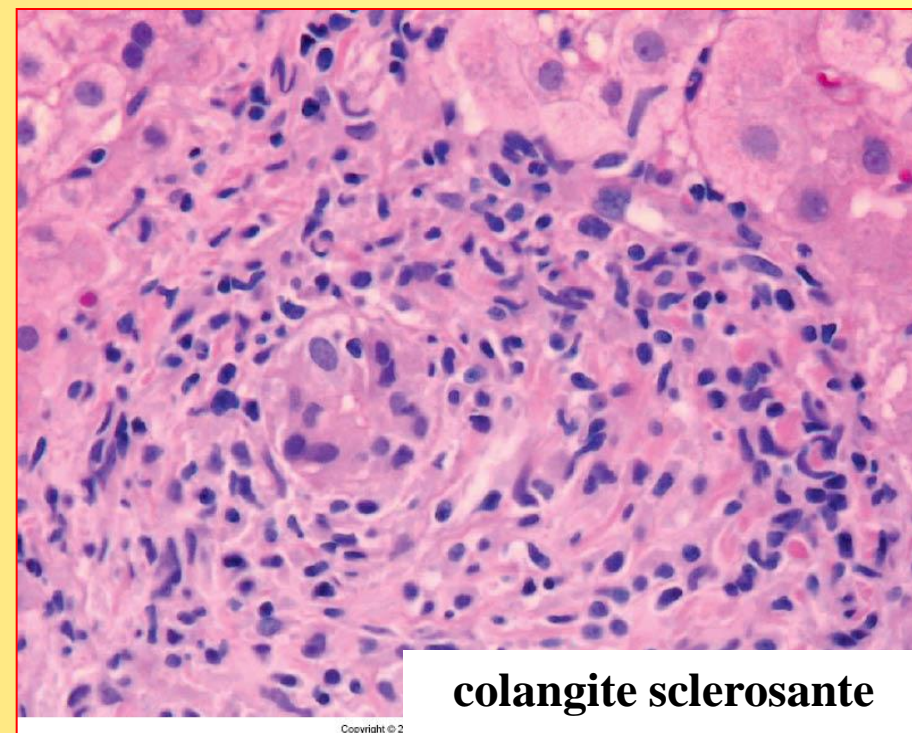
	Cirrosi biliare secondaria	Cirrosi biliare primitiva	Colangite sclerosante primaria
Eziologia	Ostruzione biliare extraepatica	Autoimmune	Sconosciuta (poss. autoimmune; 50-70% associata a IBD)
Genere	F = M	F:M = 6:1	F:M = 1:2
Semeiotica	Prurito, ittero malessere, urine brune, feci chiare, epatosplenomegalia	Idem CBS: insorgenza insidiosa	Idem CBS: insorgenza insidiosa
Laboratorio	Iperbilir. Diretta, aum. PA, ac.biliari, colesterolo	Idem CBS + IgM autoAb (M2, antimitoc.)	Idem CBS + IgM autoAb, iperyglob.
Fase precirrotica	Stasi biliare grave, iperpl. Duttulare, edema portale, neutrofili	Marcato infiltrato linfocitario; granulomi destruenti le vie biliari	Fibrosi periduttale portale, stenosi segmentaria dei dotti intra/extraepatici

cirrosi biliare primitiva



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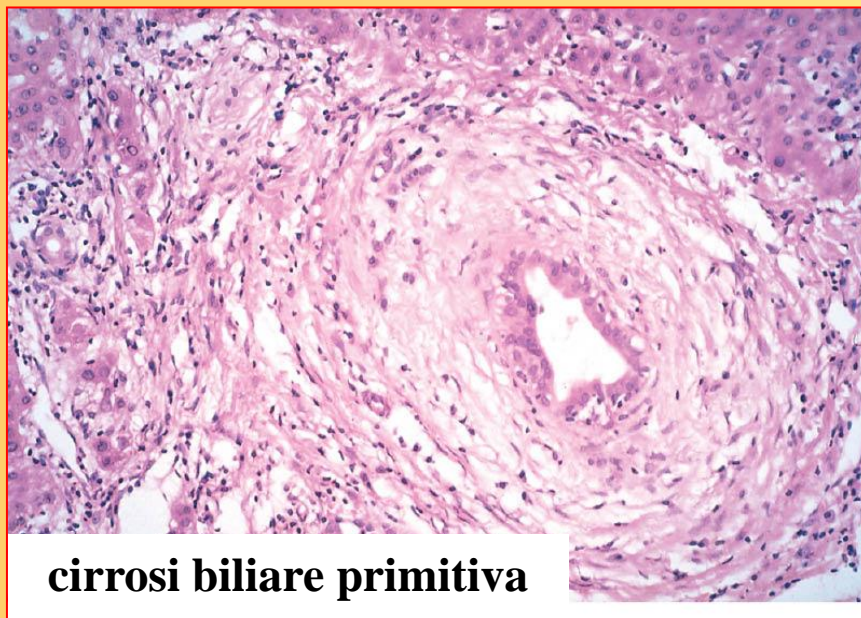
cirrosi biliare primitiva

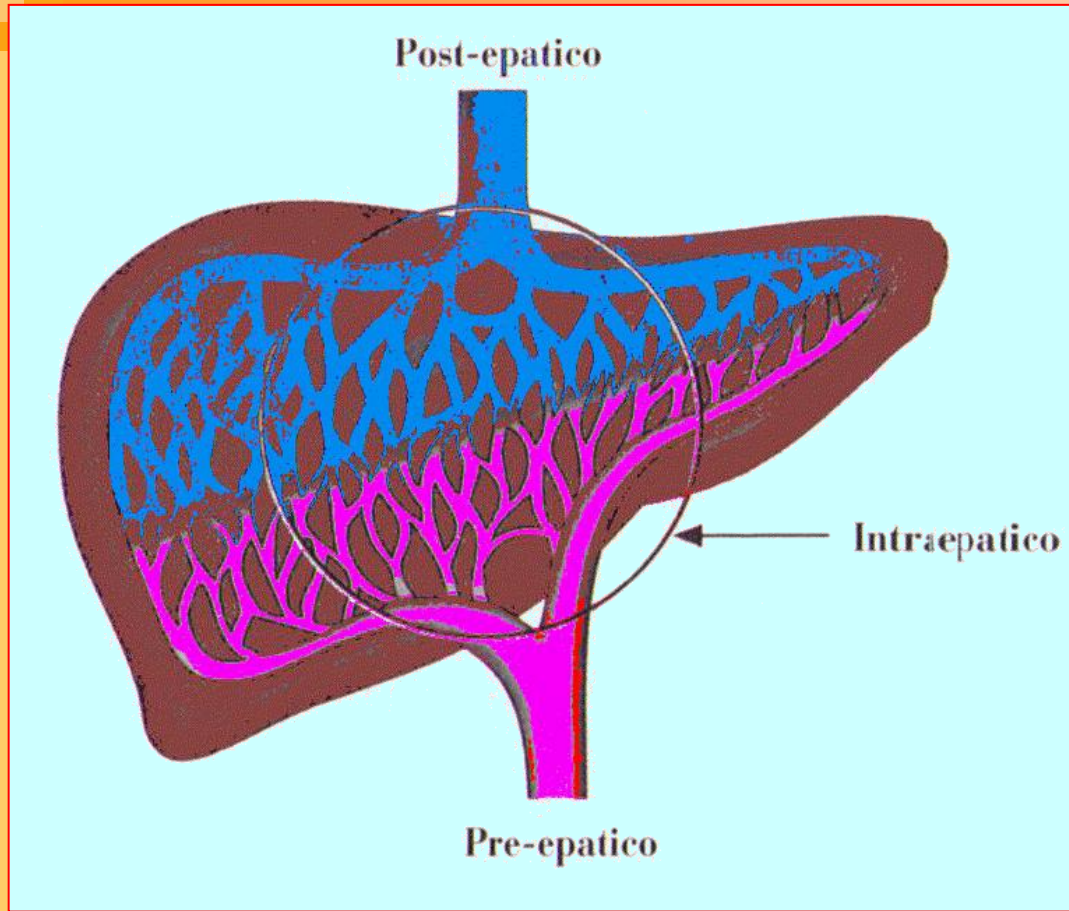


colangite sclerosante

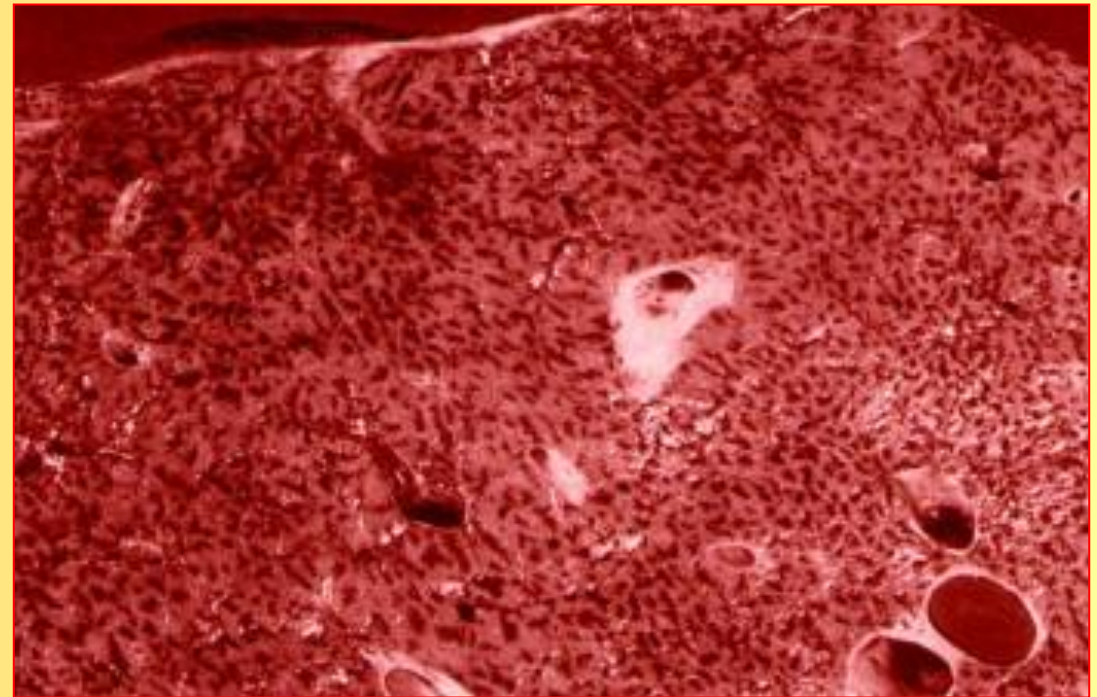
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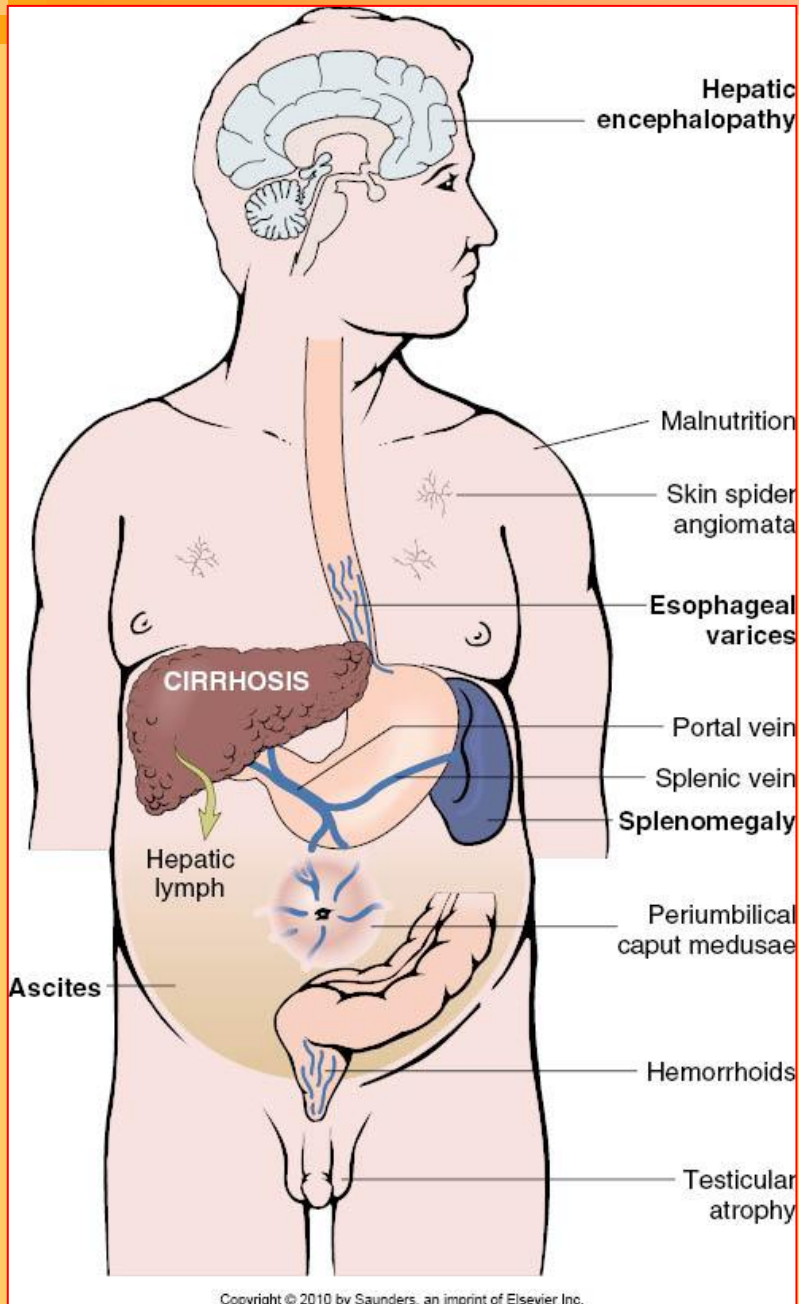
cirrosi biliare primitiva





ipertensione portale





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ipertensione portale

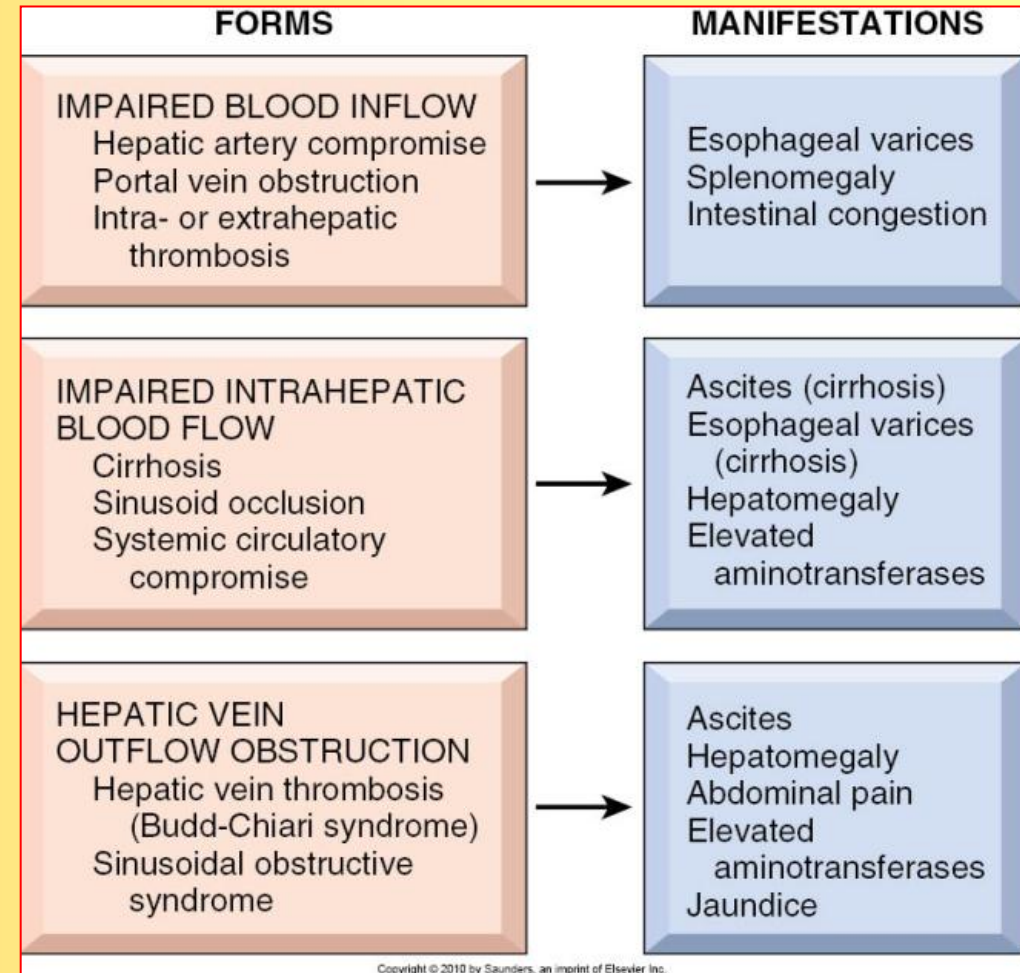
cirrosi



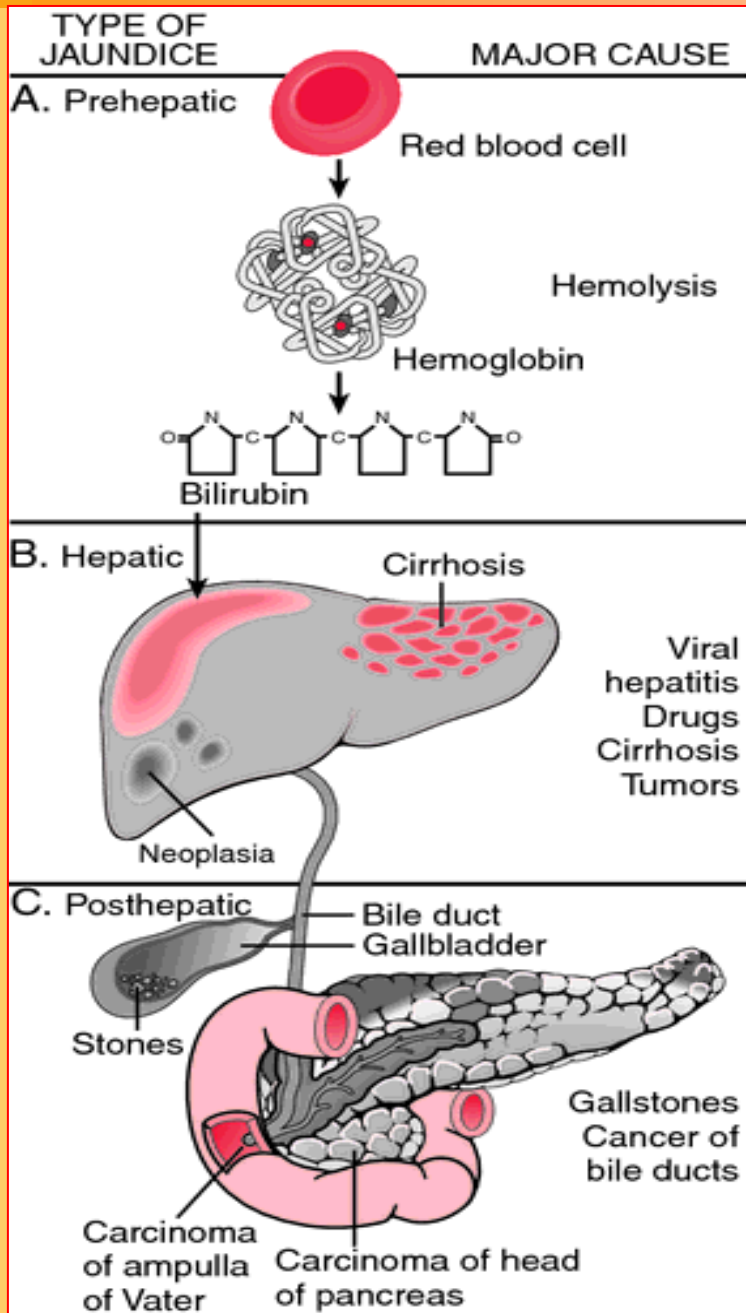
disordini vascolari



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**colestasi,
ittero**

«accumulo» sistemico di bilirubina
(non coniugata o coniugata) nei
tessuti

Cause di ittero

Da iperbilirubinemia non coniugata (i. rubinico)

- Eccesso di produz. bilirubina (emolisi, riassorbimento ematico)
- Ridotto uptake epatico
- Insufficiente coniugazione (neonati, dif.genetici, epatiti)

Da iperbilirubinemia coniugata (i. verdinico)

- Deficit di secrezione epatocitaria
- Stasi biliare

**colestasi,
ittero**

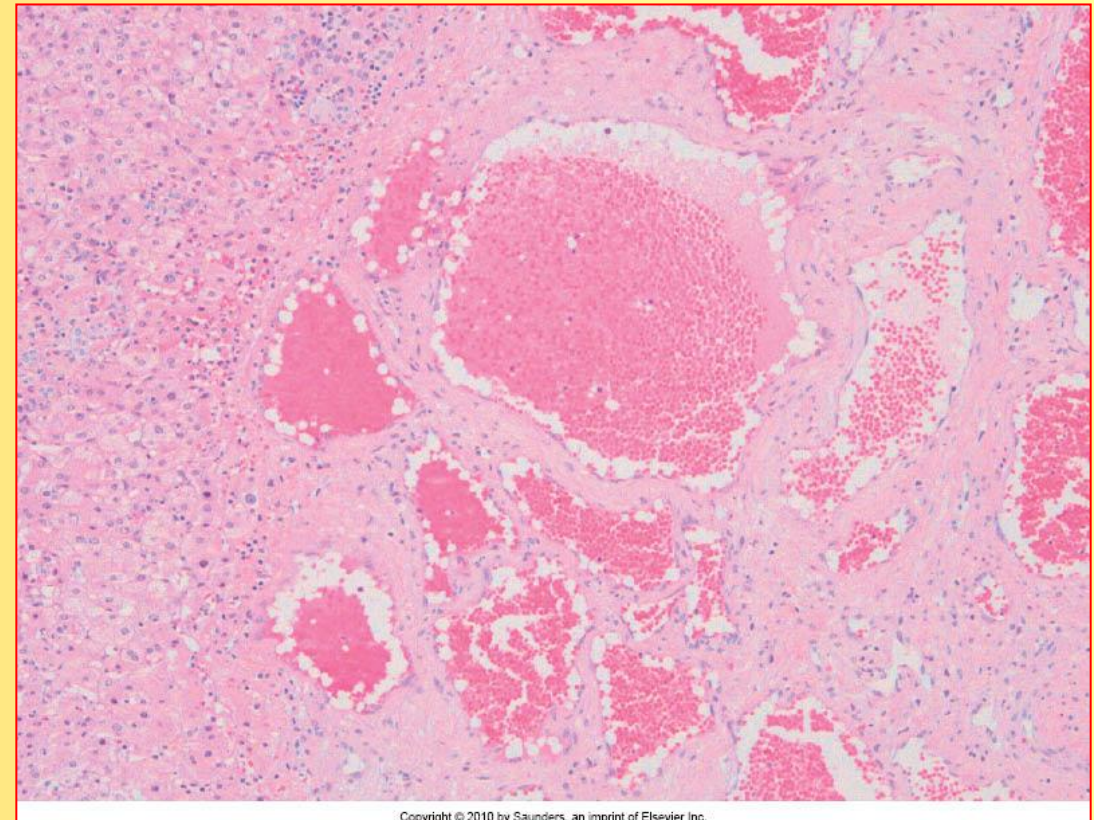


emangiomi

neoplasie
benigne

Clinica:

- Forme benigne più comuni
- DD metastasi
- Non indicata biopsia
- **Anatomia Patologica**
- Noduli rosso-bluastrò (1-2 cm)
- Localizz. spesso sottocapsulare
- Strutture vascolari, in contesto stromale
- Steatosi



Clinica:

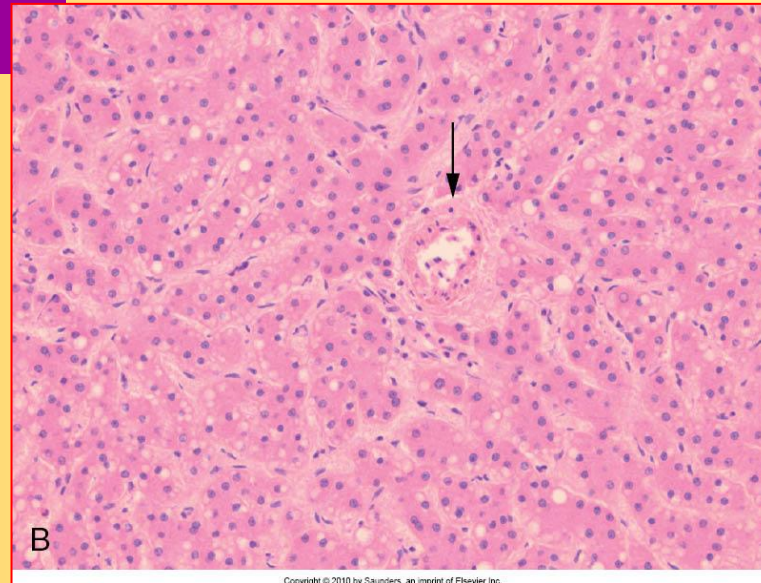
- Derivazione epatocitaria
- F > M
- Contraccettivi orali
- DD epatocarcinoma
- Tendenza alla rottura capsula (gravidanza)
- Rischio di insorgenza di carcinoma (raro)

Anatomia Patologica

- Nodulo giallastro (bile)
- Localizz. spesso sottocapsulare
- Tendenza ad aumento di volume
- Disegno a lamine e cordoni
- Strutture vascolari, assenza spazi portali
- Steatosi

adenoma epatocellulare

neoplasie
benigne



Fattori rischio:

- Infezione HBV, HCV
 - HBV endemico (RR=200)
- Etilismo
- Steatoepatite non alcolica
- Contaminanti alimentari (aflatox.)
- Altri fattori
 - Tirosinemia
 - Deficit α -antitripsina
 - Fatt. genetici
 - Fatt. ormonali

carcinoma epatocellulare

tumori maligni

Anatomia Patologica:

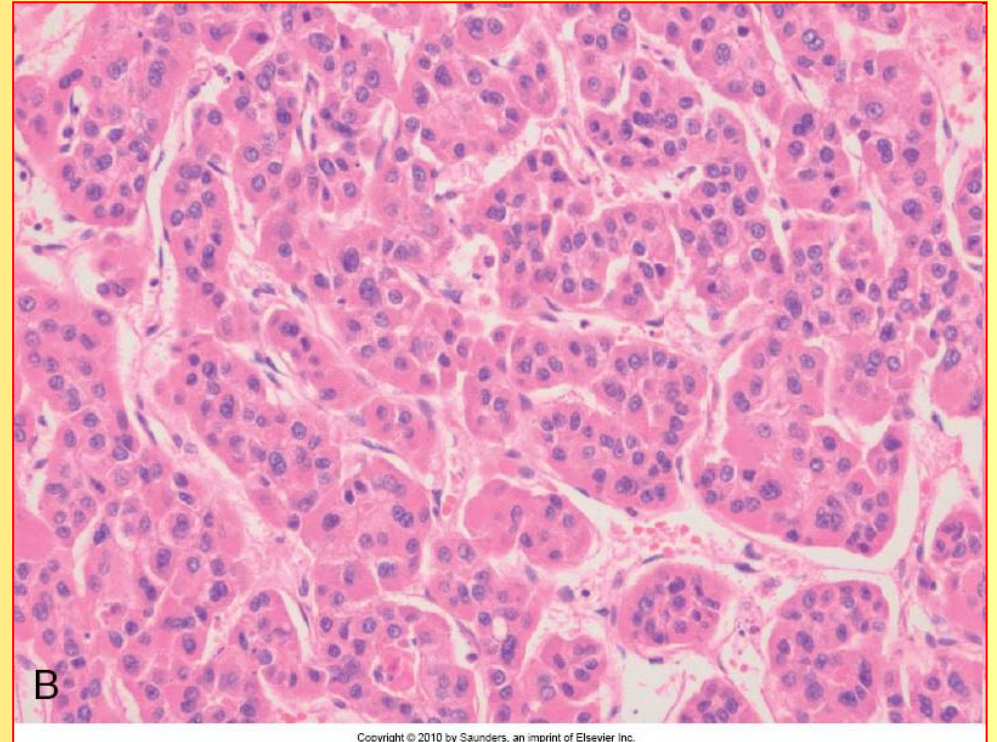
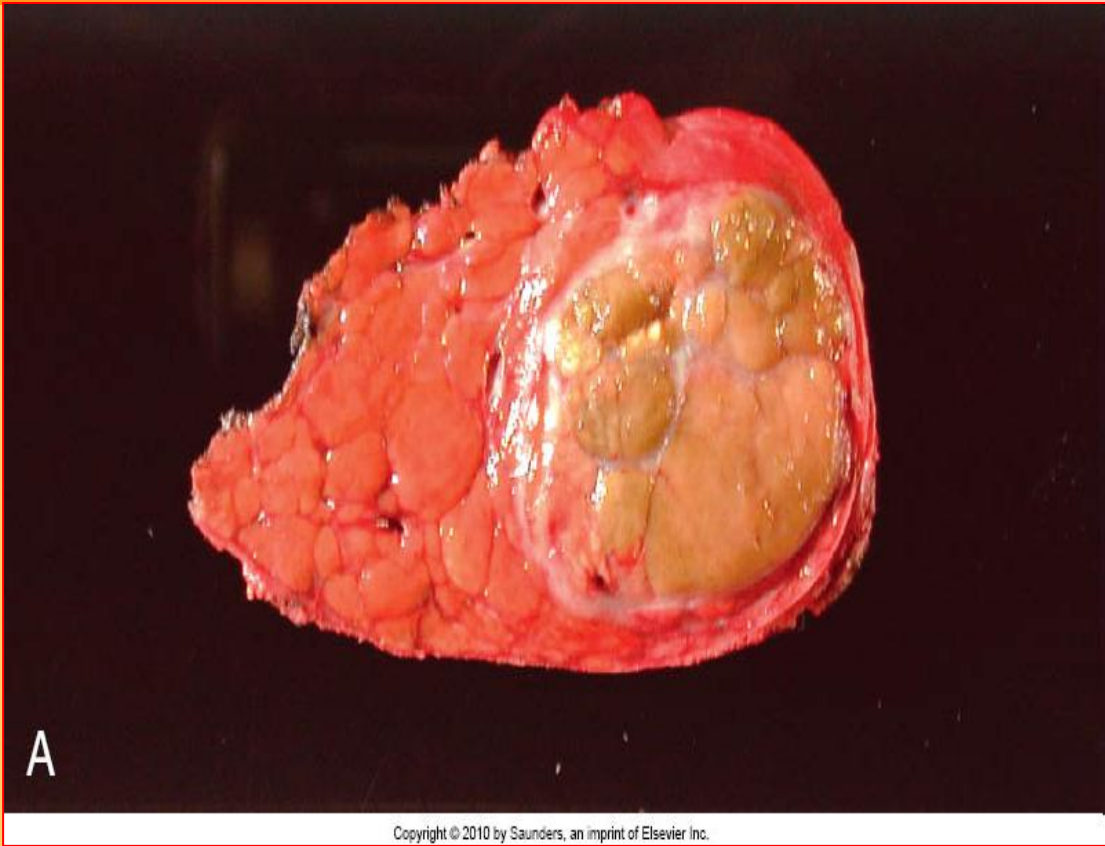
- Massa voluminosa, uni/multifocale
- Infiltrazione talora estesa (in cirrosi)
- Forme differenziate, secernenti
- Forme moder./scarsam. differenziate
- Invasione vascolare precoce
- Metastasi intraepatiche e sistemiche
- Variante fibrolamellare (5%) a miglior prognosi

Quadro clinico:

- Sintomi in relaz. a epatite/cirrosi
- Nessun sintomo (zone endemiche)
- Dolore, malessere, fatica
- Perdita peso, massa palpabile
- Ittero, febbre, varici
- Elevati livelli α -fetoproteina
- Evidenza eco/radiografica
- Progressione, metastasi
- Cachessia, varici, insuff.epat.
- Emorragie da rottura tumore
- Piletrombosi
- Prognosi infausta

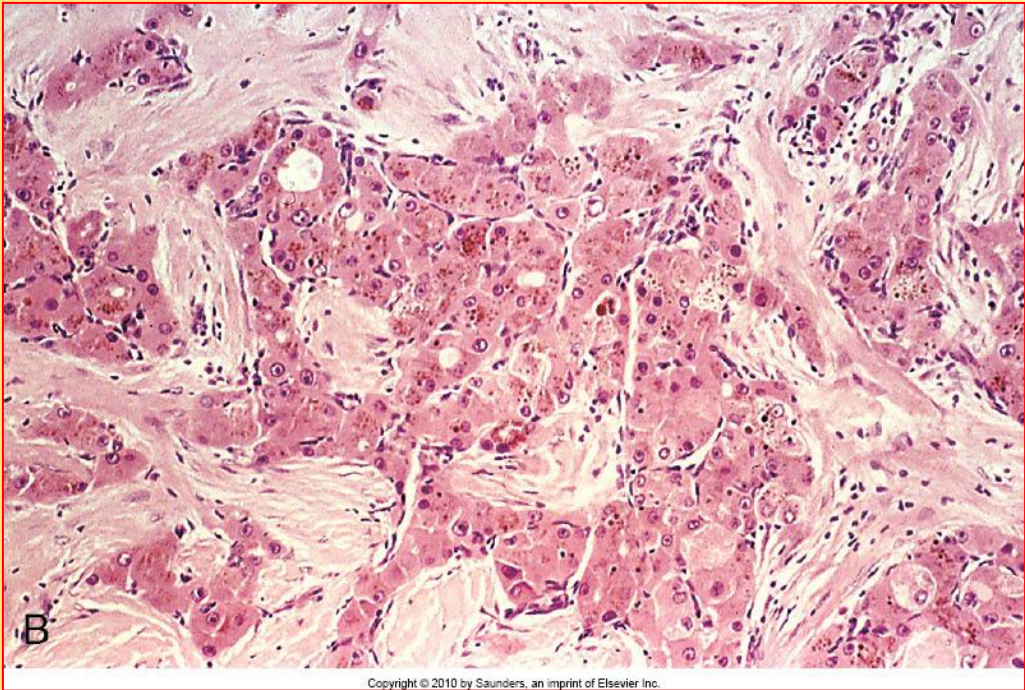
tumori maligni

carcinoma
epatocellulare



tumori maligni

carcinoma
epatocellulare
var. fibrolamellare



Fattori rischio:

- Colangite sclerosante
- Malattia policistica
- HCV
- Thorotrast

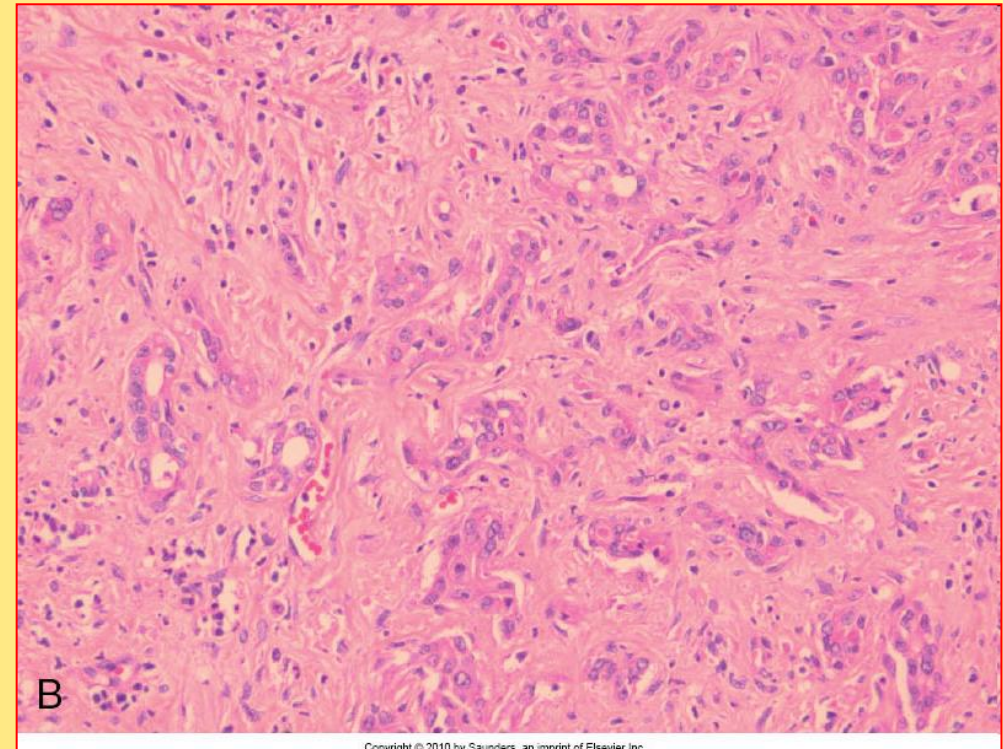


tumori maligni

colangiocarcinoma

Anatomia Patologica:

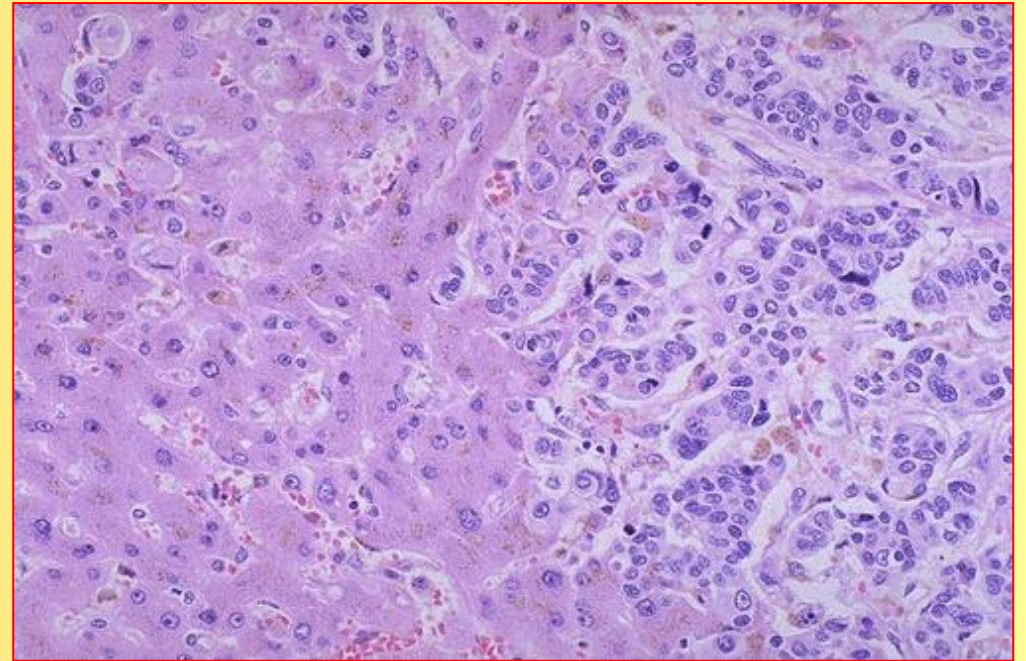
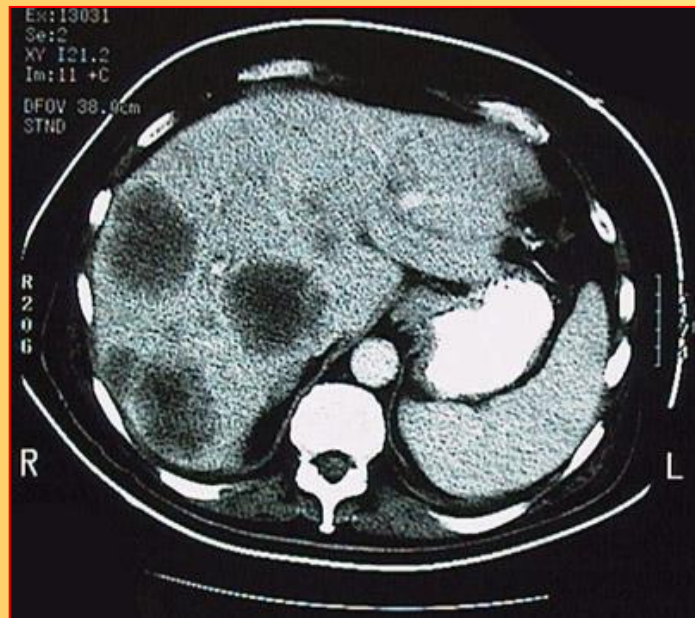
- Piccole lesioni alla diagnosi
- Noduli fissi, grigiastri
- Aspetto diff.infiltrativo/polipoide
- Talora secrezione mucinica
- Abbondante stroma fibroso
- Background non cirrotico
- Sviluppo lungo gli assi portali
- Metastasi a polmoni/ossa (vertebr.)
- Rare varianti miste HCC/CCA





tumori maligni

metastasi



**tumori maligni
epidemiologia**

**incidenza
mortalità**

Tumore del fegato

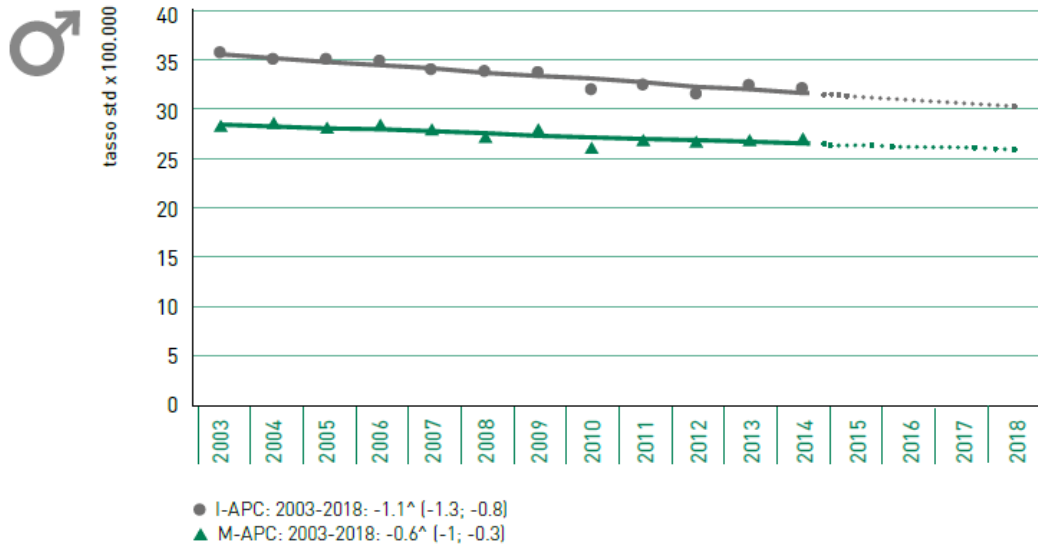


FIGURA 18A. Tumore del fegato, maschi. AIRTUM: stima dei trend tumorali di incidenza e mortalità 2003-2018. Tassi standardizzati nuova popolazione europea 2013

APC = Annual Percent Change (variazione percentuale media annua), I = incidenza, M = mortalità.

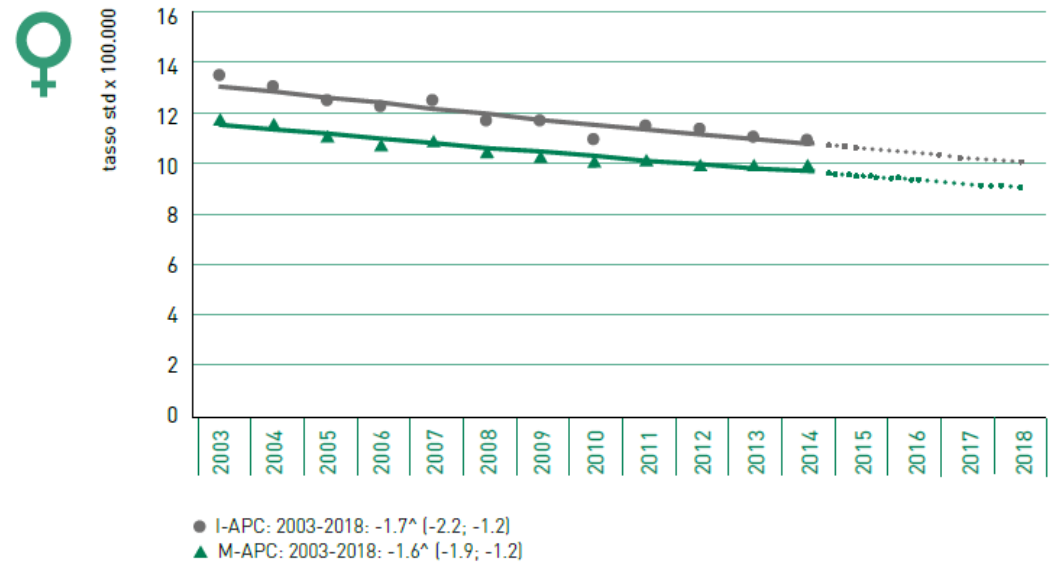


FIGURA 18B. Tumore del fegato, femmine. AIRTUM: stima dei trend tumorali di incidenza e mortalità 2003-2018. Tassi standardizzati nuova popolazione europea 2013

APC = Annual Percent Change (variazione percentuale media annua), I = incidenza, M = mortalità.

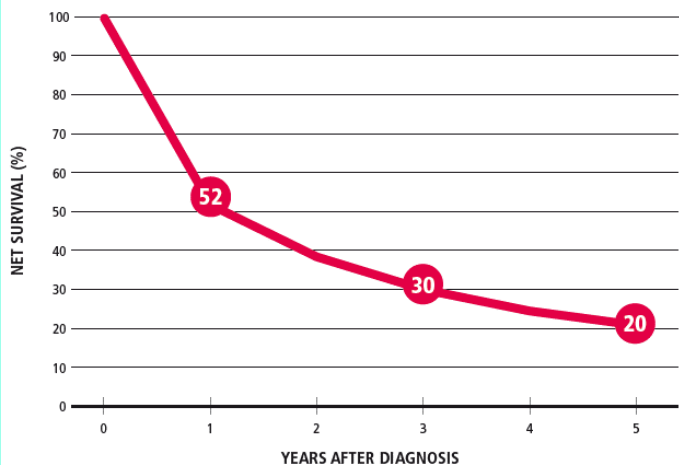


LIVER CANCER

MALES & FEMALES

C AGE-STANDARDIZED NET SURVIVAL (%), 2005-2009

POOL OF 42 CANCER REGISTRIES (29365 OBSERVED CASES)

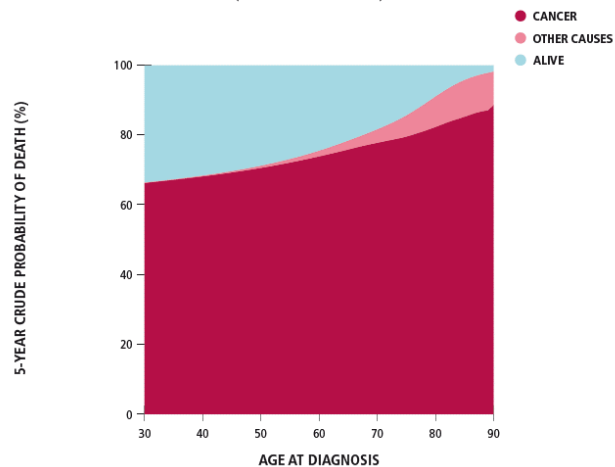


LIVER CANCER

MALES & FEMALES

D CRUDE PROBABILITY OF DEATH (%), 2005-2009

POOL OF 42 CANCER REGISTRIES (28886 OBSERVED CASES)



**tumori maligni
epidemiologia**

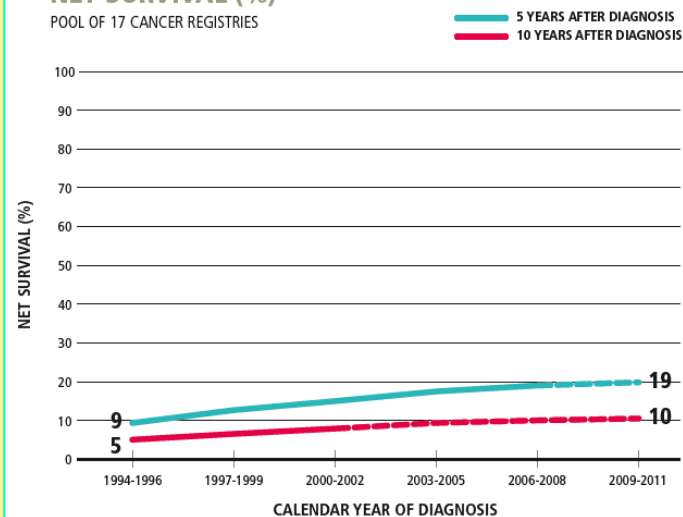
sopravvivenza

LIVER CANCER

MALES & FEMALES

H TIME TREND OF 5-, 10-YEAR AGE-STANDARDIZED NET SURVIVAL (%)

POOL OF 17 CANCER REGISTRIES

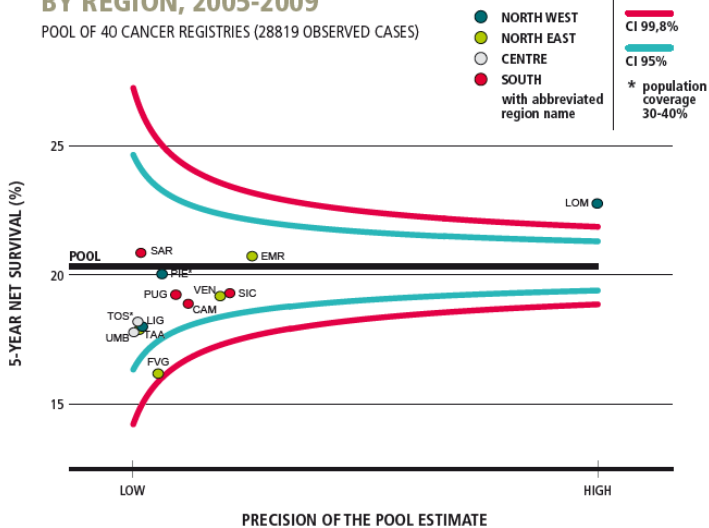


LIVER CANCER

MALES & FEMALES

E 5-YEAR AGE-STANDARDIZED NET SURVIVAL (%) BY REGION, 2005-2009

POOL OF 40 CANCER REGISTRIES (28819 OBSERVED CASES)



LIFE EXPECTANCY AT A GIVEN AGE OF DIAGNOSIS

● GENERAL POPULATION
● CANCER





Pool of Italian Cancer Registries - 1 January 2010

FEGATO

LIVER

(ICD-10 C22)

COMPLETE PREVALENCE BY YEARS SINCE DIAGNOSIS

YEARS →	≤ 2	(2 - 5]	(5 - 10]	(10 - 15]	(15 - 20]	> 20
No. →	10 693	7 245	3 841	1 263	421	208
% →	45%	31%	16%	5%	2%	1%
PROPORTION PER 100 000 →	19	13	7	2	1	0

MALE 70% **FEMALE 30%**

COMPLETE PREVALENCE BY SEX, MACRO-AREA, AND AGE

(PROPORTION PER 100 000)

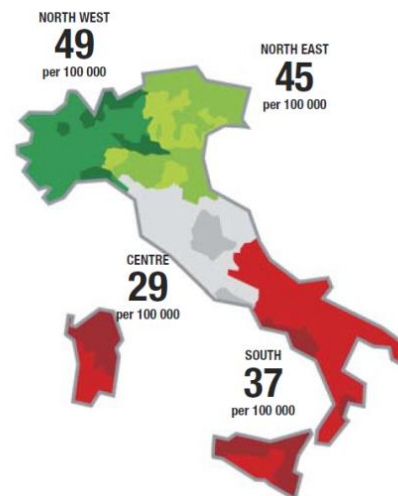
AGE CLASS →	0-44	45-59	60-74	75+	ALL AGES
MALE					
NORTH WEST	5	67	193	261	74
NORTH EAST	4	48	200	266	67
CENTRE	1	34	125	162	42
SOUTH	1	40	194	199	51
POOL	3	49	191	237	62
FEMALE					
NORTH WEST	2	12	49	100	27
NORTH EAST	2	10	43	101	24
CENTRE	3	7	33	68	17
SOUTH	3	9	84	82	24
POOL	2	10	56	94	24
BOTH SEXES					
NORTH WEST	4	39	116	158	49
NORTH EAST	3	29	118	161	45
CENTRE	2	20	77	105	29
SOUTH	2	24	136	128	37
POOL	3	29	119	147	42

2 587 347 CANCER SURVIVORS

23 671
LIVER
CANCER SURVIVORS

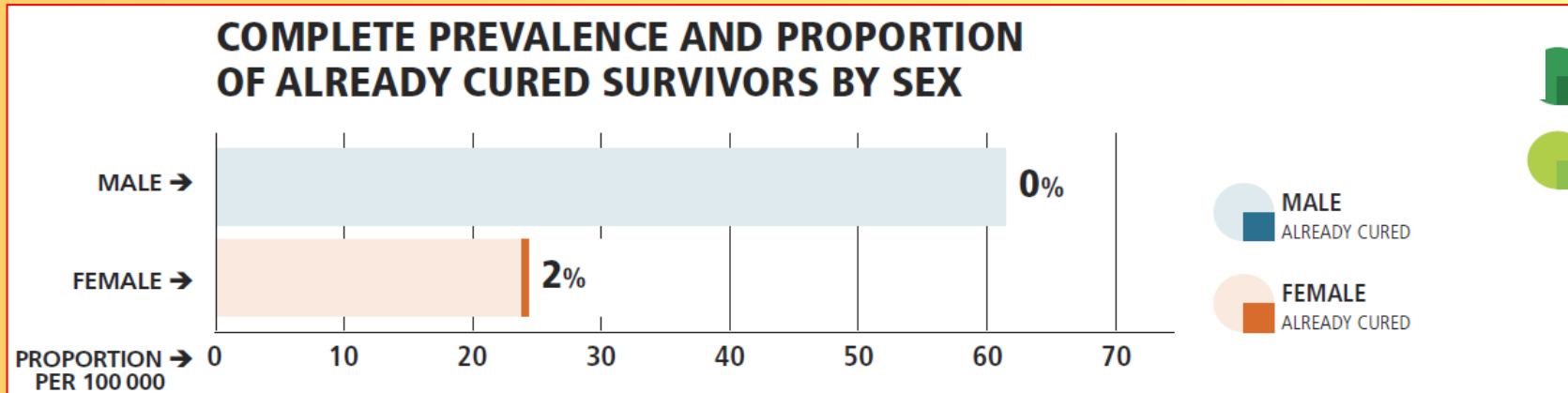
ALREADY CURED
at 1 JANUARY 2010

206 (0.9%)



tumori maligni
epidemiologia

prevalenza



CURE FRACTION AND TIME TO CURE BY AGE AT DIAGNOSIS AND SEX FOR CANCER PATIENTS DIAGNOSED IN ITALY IN 1985-2009

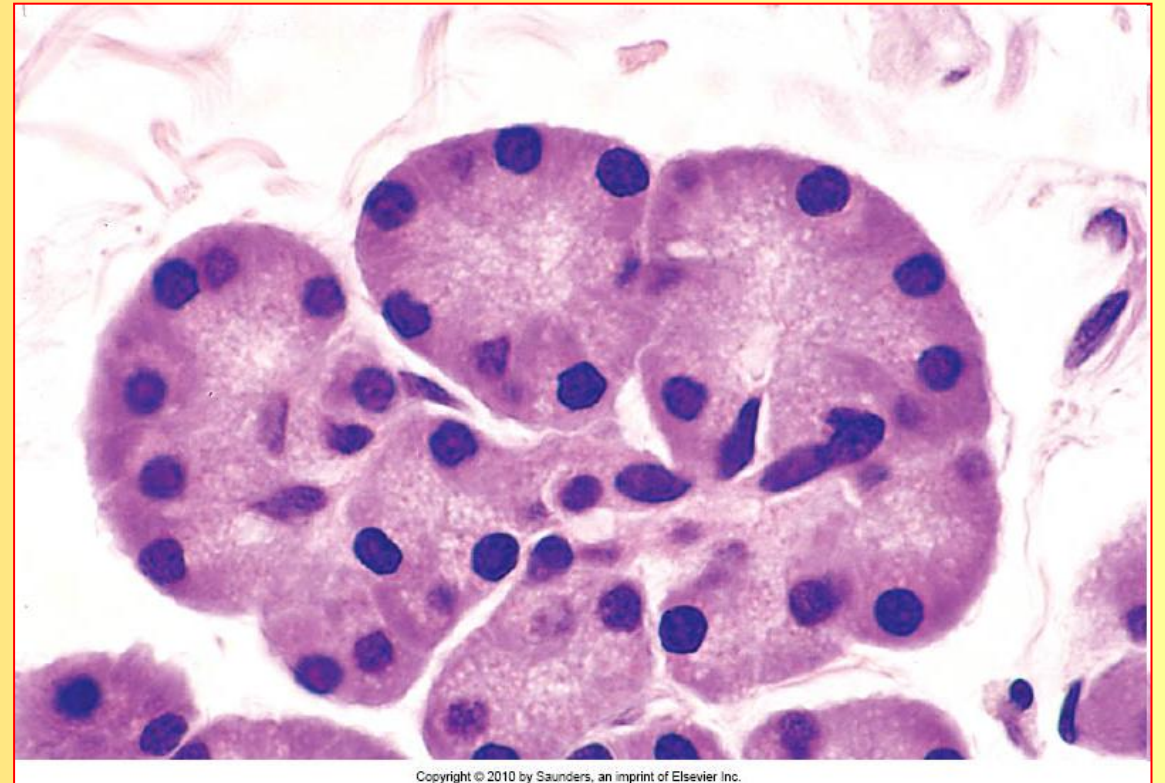
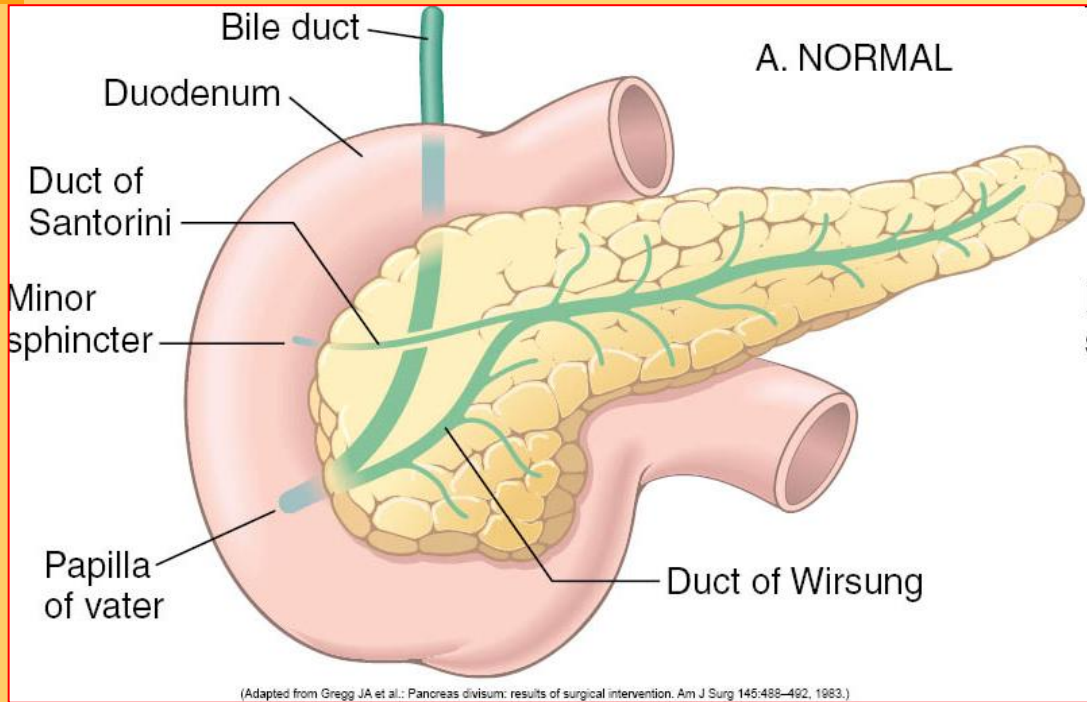
AGE AT DIAGNOSIS	CURE FRACTION %	
	MALE	FEMALE
0 - 44	6%	13%
45 - 59	3%	6%
60 - 74	1%	2%
75+	1%	1%

AGE AT DIAGNOSIS	TIME TO CURE YEARS	
	MALE	FEMALE
0 - 44	22	20
45 - 59	24	23
60 - 74	25	25
75+	>25	>25

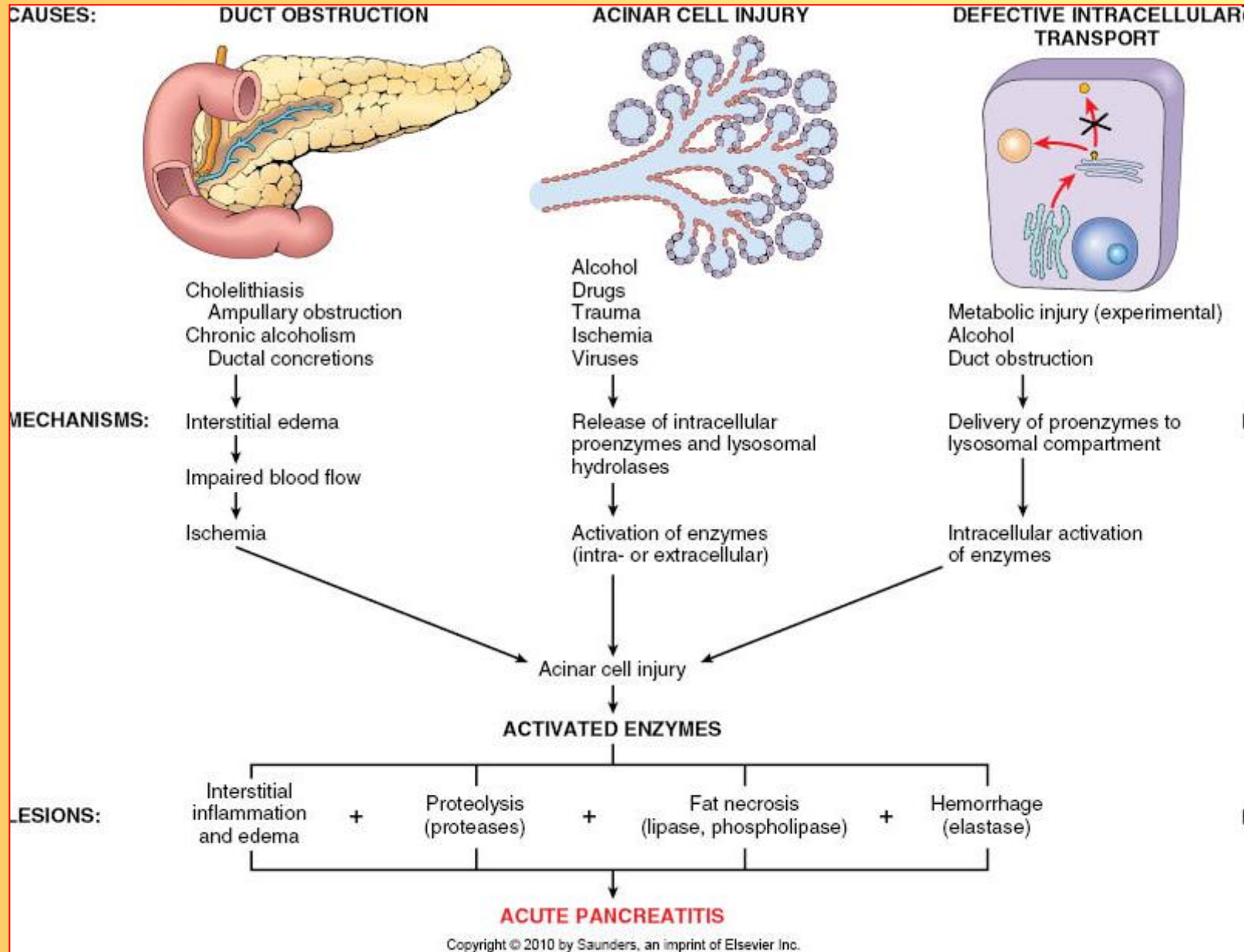


Pancreas

fisiopatologia



pancreatite acuta



Fattori etiopatogenetici:

METABOLICI

- Etilismo, iperlipoproteinemia, ipercalcemia,
- Farmaci

GENETICI

- Mutaz. Geni tripsinogeno cationico (PRSS1) e inibitore tripsina (SPINK1)
- Forme “ereditarie”

MECCANICI

- Litiasi, traumi (chir., endoscopia)

VASCOLARI

- Shock, ateroemboli, vasculiti

INFETTIVI

- Virus parotite

**pancreatite
acuta**

Clinica:

- Dolore addominale
- Anoressia, nausea, vomito
- Iperamilasemia
- “Dramma pancreatico”
 - Leucocitosi, emolisi
 - CID, ARDS
 - Necrosi adiposa
 - Necrosi tubulare acuta
 - Shock

Patogenesi attiv. enzimatica:

- Ostruzione duttale
- Danno cellule acinari
- Dif. trasporto intracell. proenzimi
- Inappr. attivazione tripsinogeno
- Attivazione altri enzimi
 - Fosfolipasi, elastasi, precallicr.
 - Fatt. Hageman XII

Anatomia Patologica:

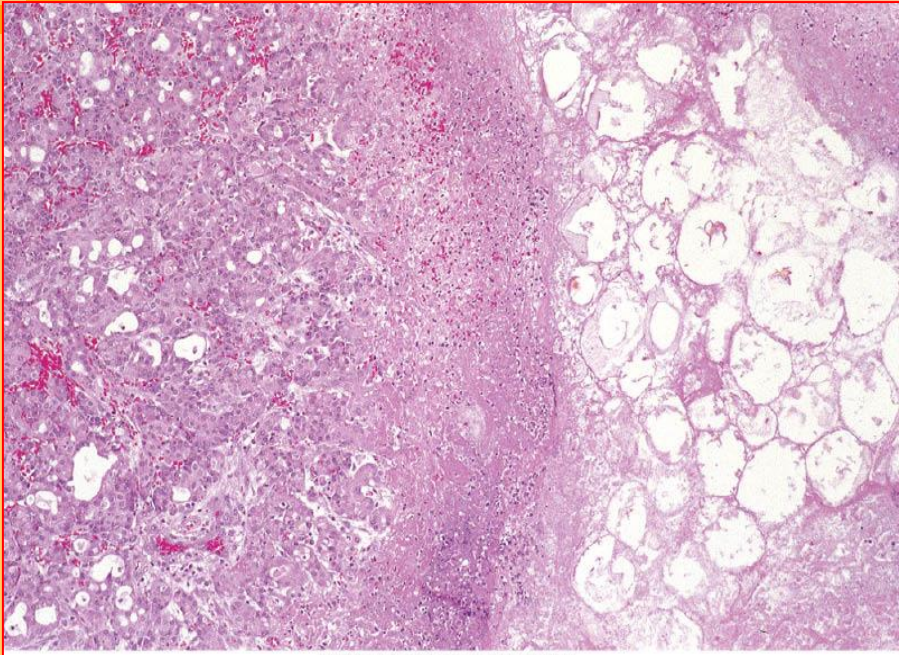
- Edema
- Necrosi lipidica
- Infiammazione acuta
- Distruzione parenchima e vasi
- Emorragia interstiziale
- Necrosi diffusa (saponificazione)



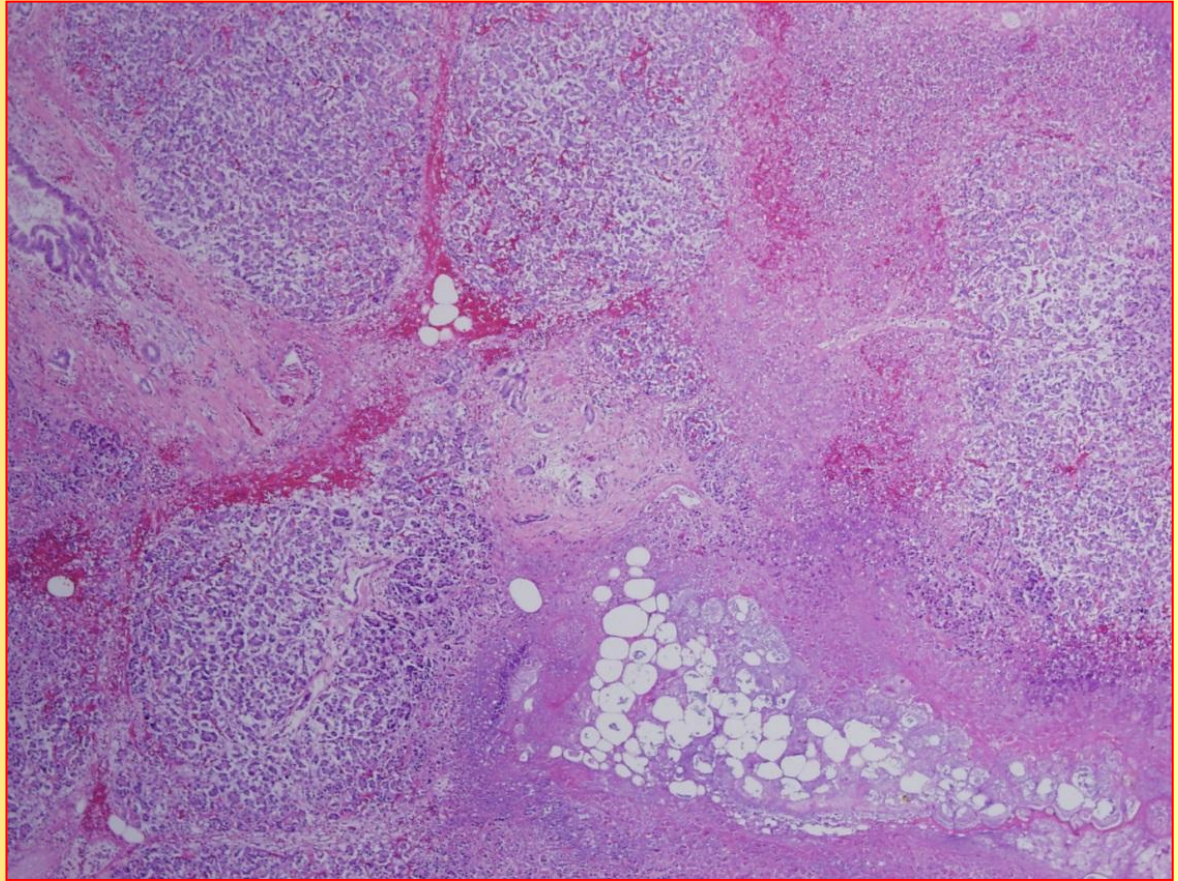
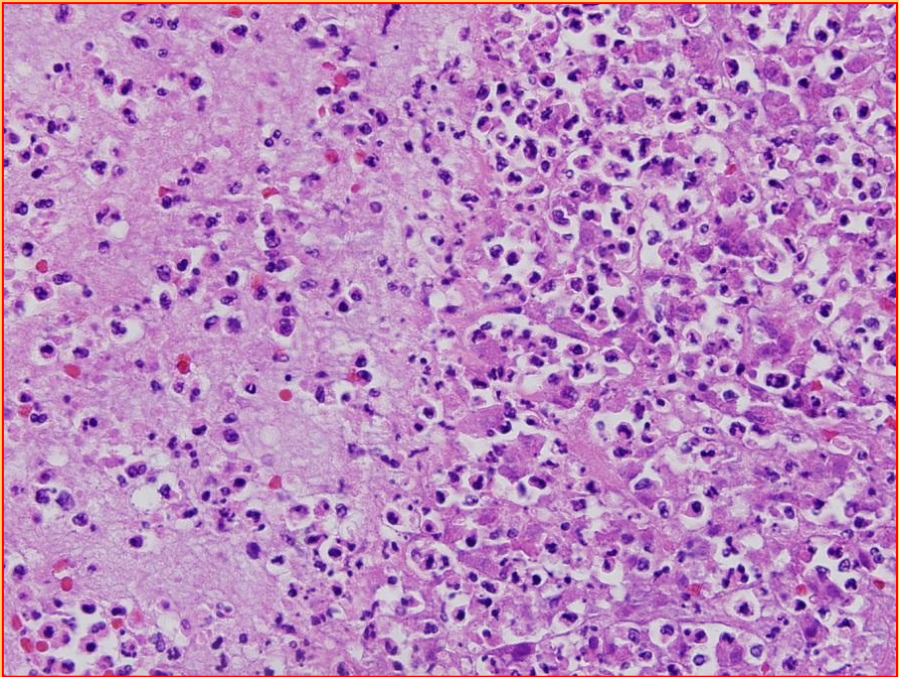
**pancreatite
acuta**



**pancreatitis
acuta**



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pancreatite cronica

Fattori di rischio

- Ostruzione cronica dotto pancreatico
- Danno autoimmune
- Predisposizione “ereditaria” (mut. PRSS1, SPINK1)
- Mutazione gene CFTR

Patogenesi:

- Ostruzione duttale
 - Precipit. proteici secreto
 - Calcoli
- Danno tossico
 - Tossine, alcool e metaboliti
- Stress ossidativo
 - Rad. liberi, chemochine (IL-8, TGF β)

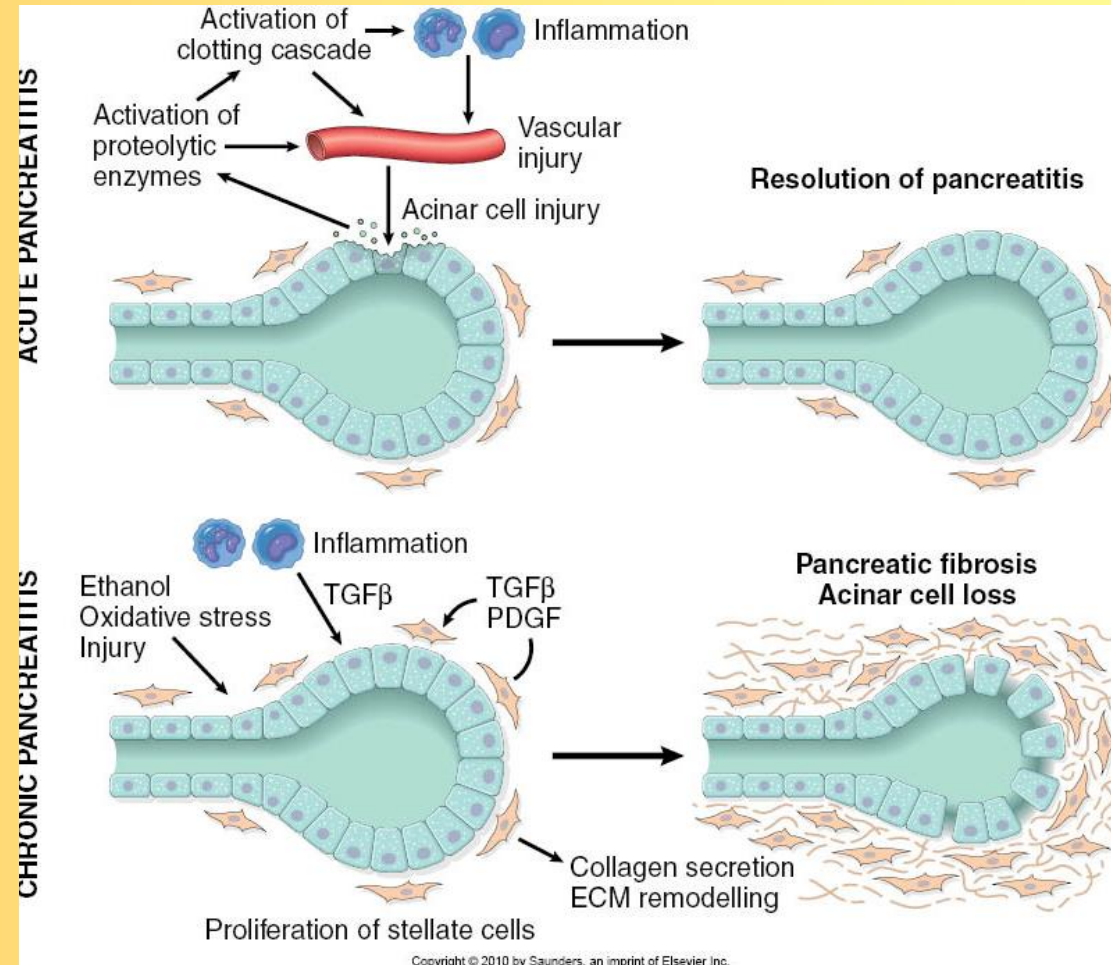
Clinica:

- Forme variabili
- Dolore moderato/severo
- Insuff. pancreatica
- Diabete mellito
- Ittero
- Febbre, iperamilasemia

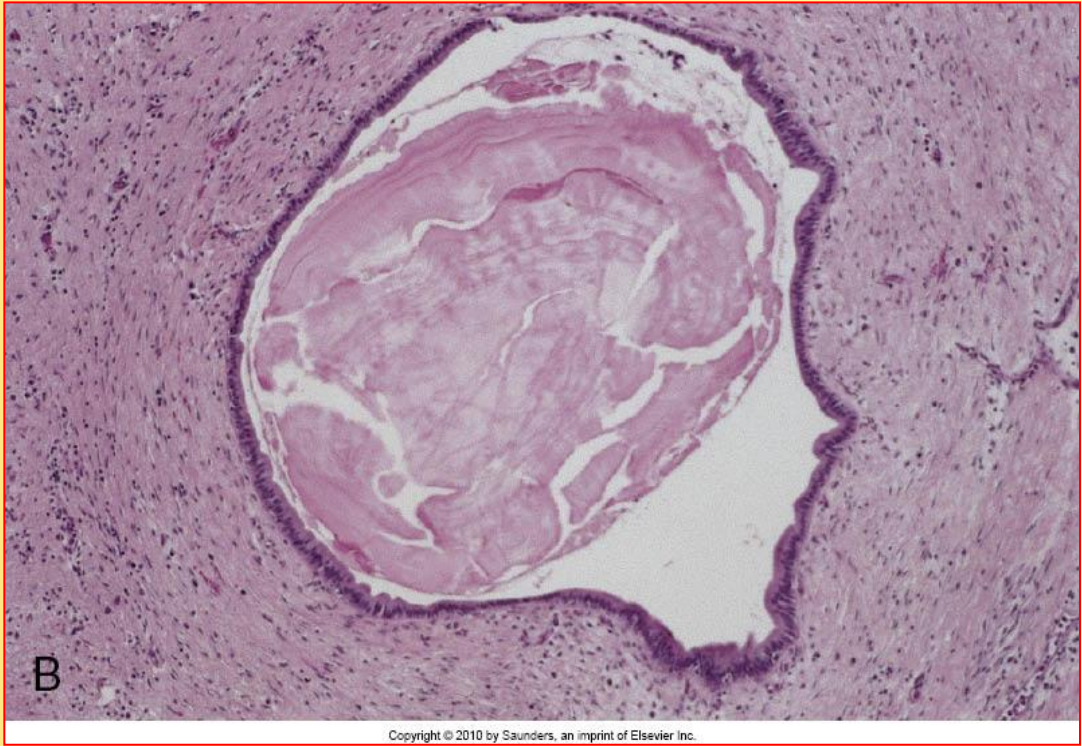
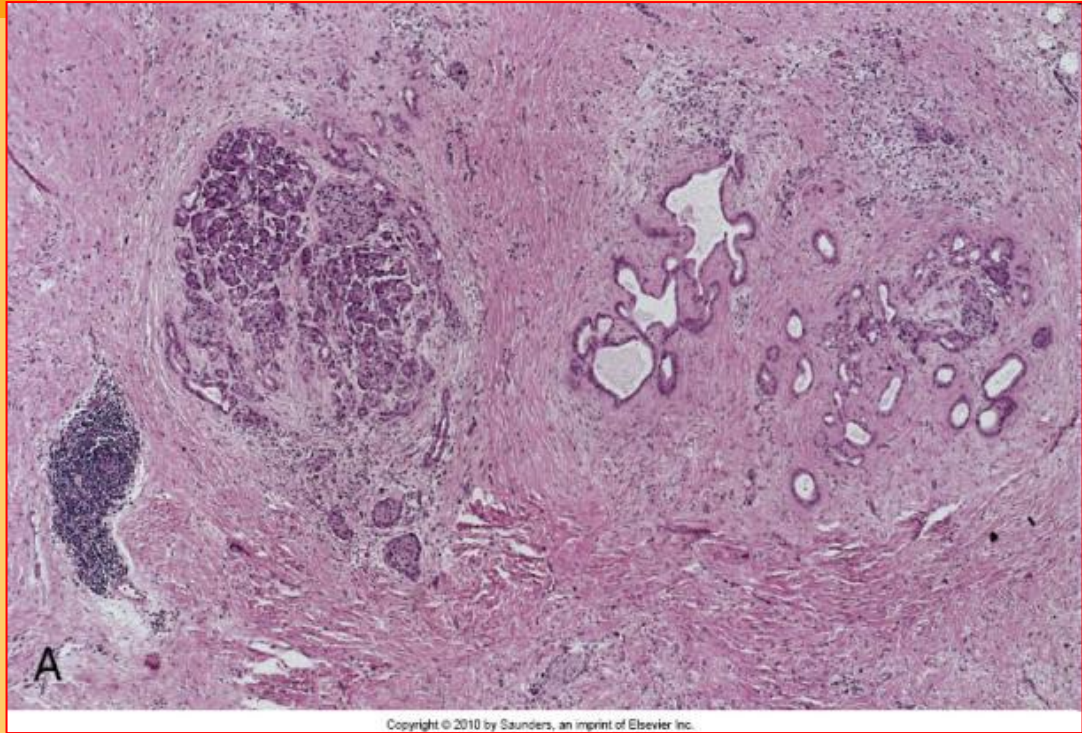
Anatomia Patologica:

- Fibrosi parenchimale
- Riduzione acinare
- Dilatazione duttale
- Is. Langerhans intatte
- Infiltrato inf. cronico
- Forme autoimmuni
- linfoplasmocitiche

pancreatite
cronica

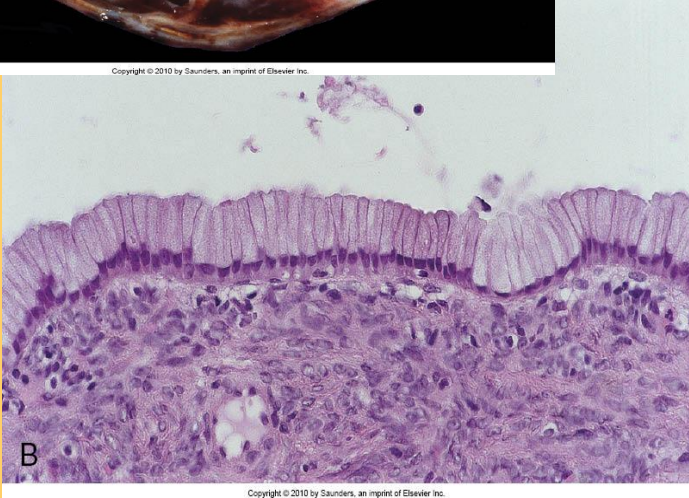
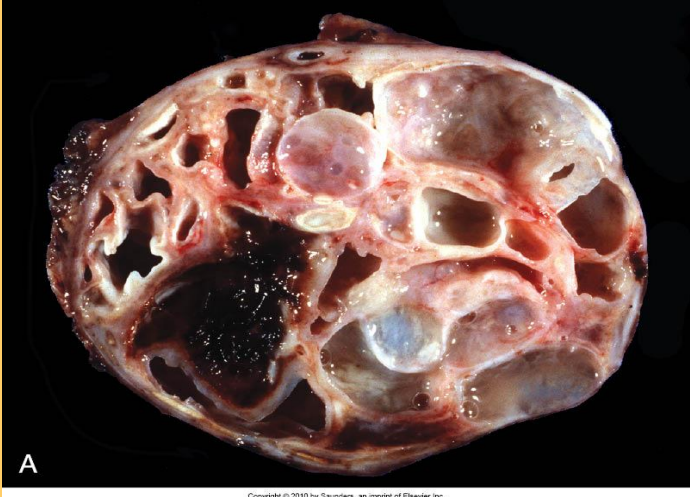


**pancreatite
cronica**

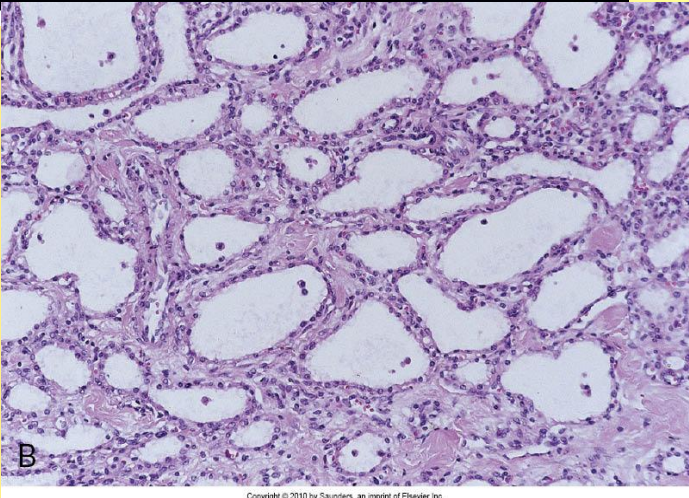
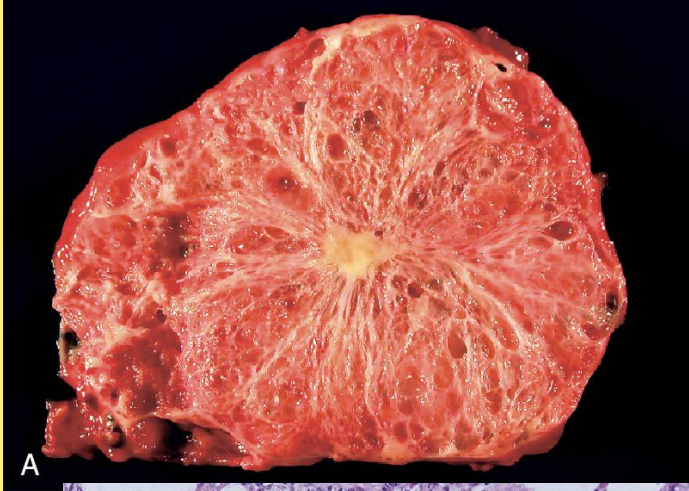


cistoadenoma

neoplasie
benigne

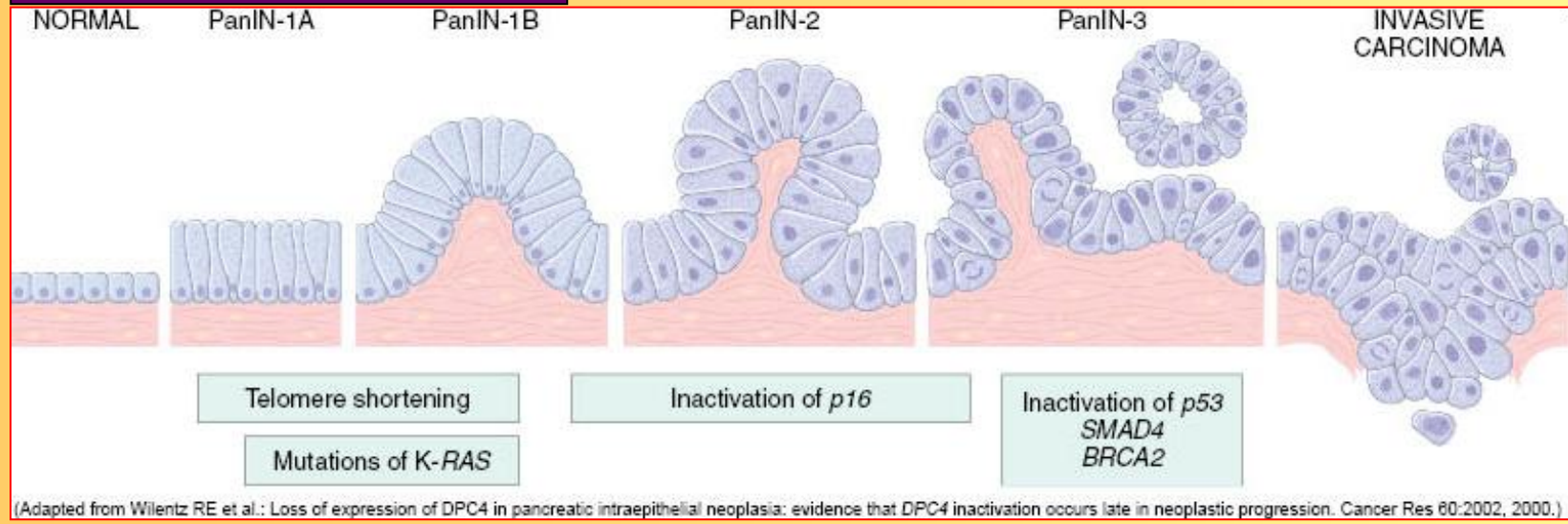


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Precursori e progressione:



- Carcinogenesi molecolare (geni):**
- KRAS
 - P16
 - SMAD4
 - P53
 - Altri geni
 - Anomalie di metilazione
 - Espressione genica

- Epidemiologia e fatt. rischio:**
- 80% casi inc. 60-80 aa.
 - Fattori razziali
 - Fumo di sigaretta (RR=2)
 - Dieta ricca di grassi
 - Pancreatite cronica

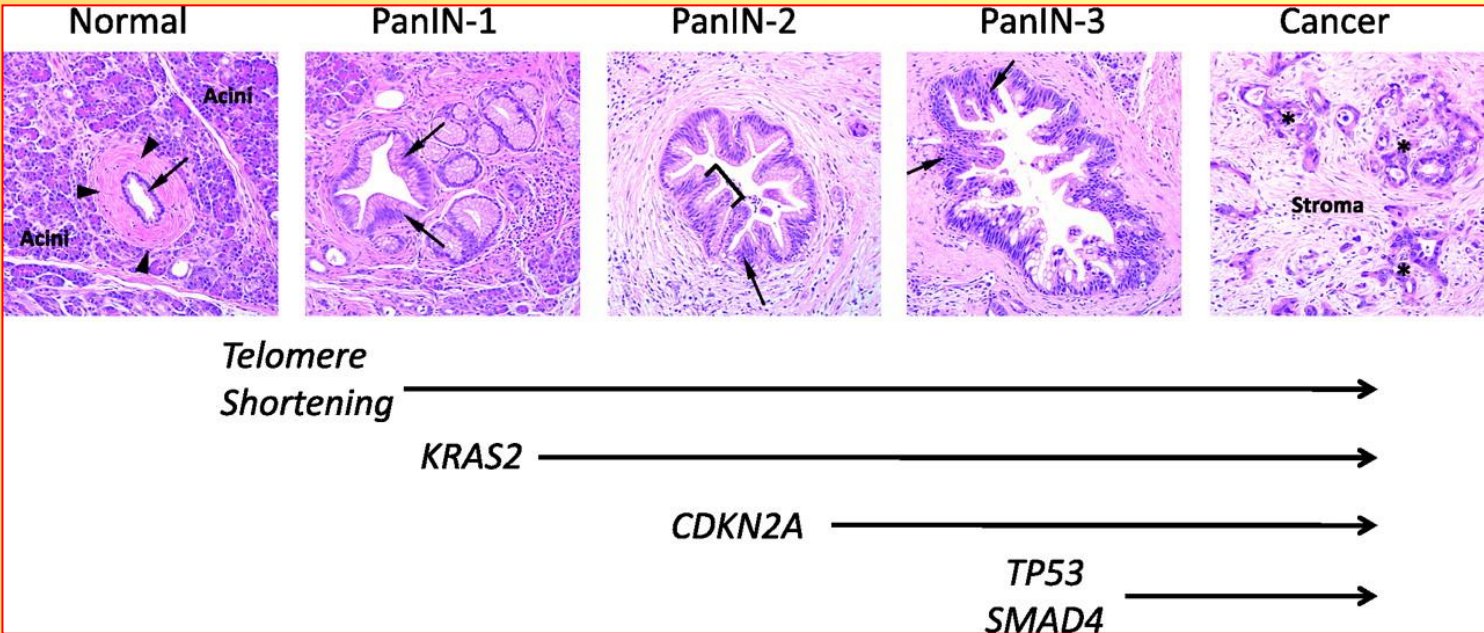
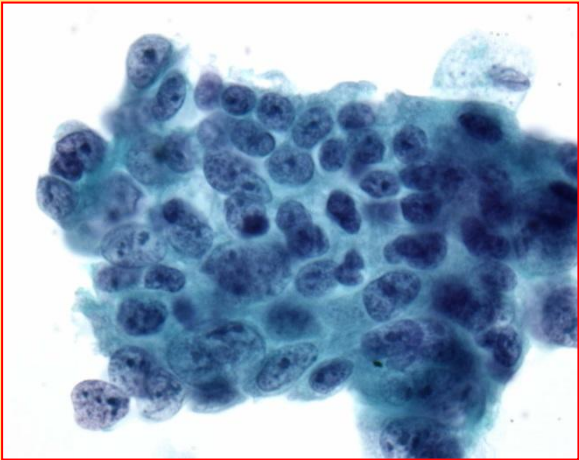
Anatomia patologica:

- **Localizzazione testa 60%**
 - Ittero, dilataz. duttale
- **Localizzazione corpo 15%**
- **Localizzazione coda 5%**
- **Forme diffuse 20%**
- **Consistenza dura**
- **Disegno stellato**
- **Colore grigio**
- **Istotipo prev. “duttale” (+ raro acinare)**
- **Formaz. Ghiandole e secrez. mucina**
- **Reazione desmoplastica marcata**
- **Carcinosi perineurale**
- **Massiva invasione circostante**
- **Differenziazione variabile**
- **Varianti poco frequenti**
 - Adenosquamose, colloidali
 - Epatoidi, midollari, indifferenziate

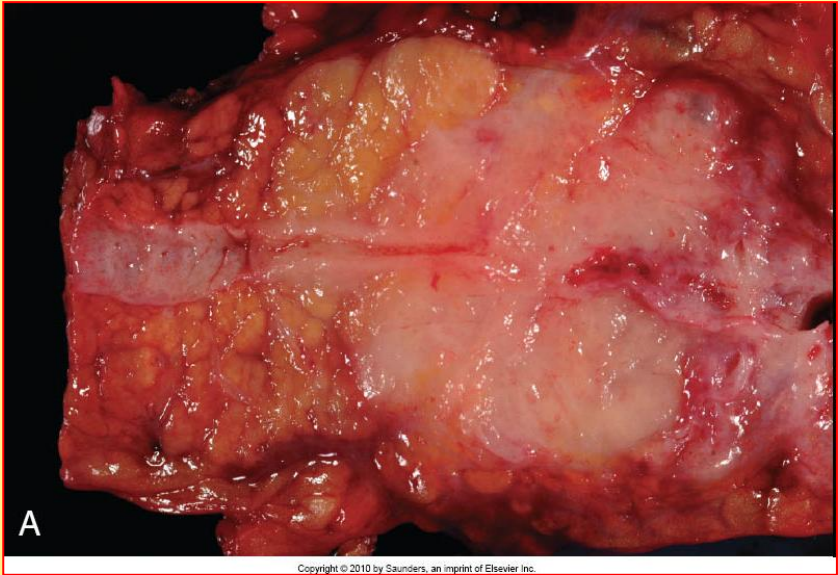
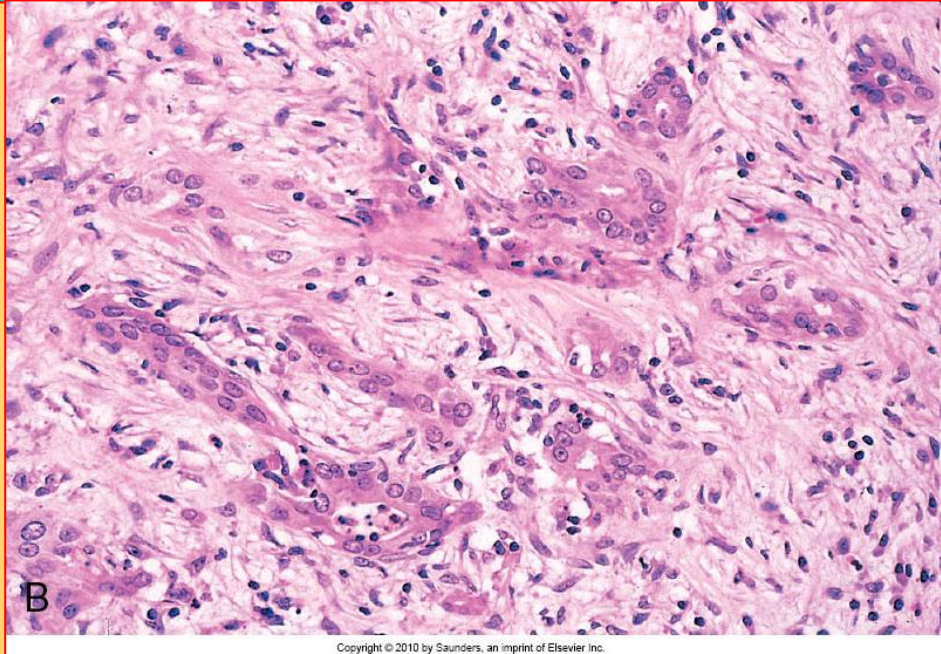
Clinica:

- **Lunga fase silente**
- **Sintomi legati all’invasione**
- **Dolore**
- **Ittero**
- **Tromboflebite migrante (10%)**
- **Markers (CA19.9)**
- **Comparsa di diabete mellito**
- **Decorso rapido e prognosi infausta**

carcinoma



carcinoma



carcinoma epidemiologia

incidenza
mortalità

Tumore del pancreas

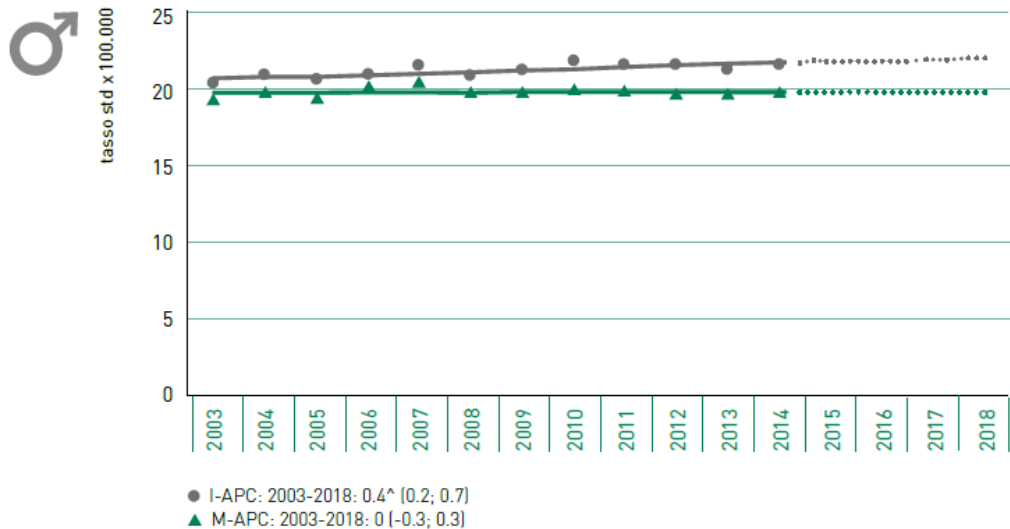


FIGURA 20A. Tumore del pancreas, maschi. AIRTUM: stima dei trend tumorali di incidenza e mortalità 2003-2018. Tassi standardizzati nuova popolazione europea 2013

APC = Annual Percent Change (variazione percentuale media annua), I = incidenza, M = mortalità.

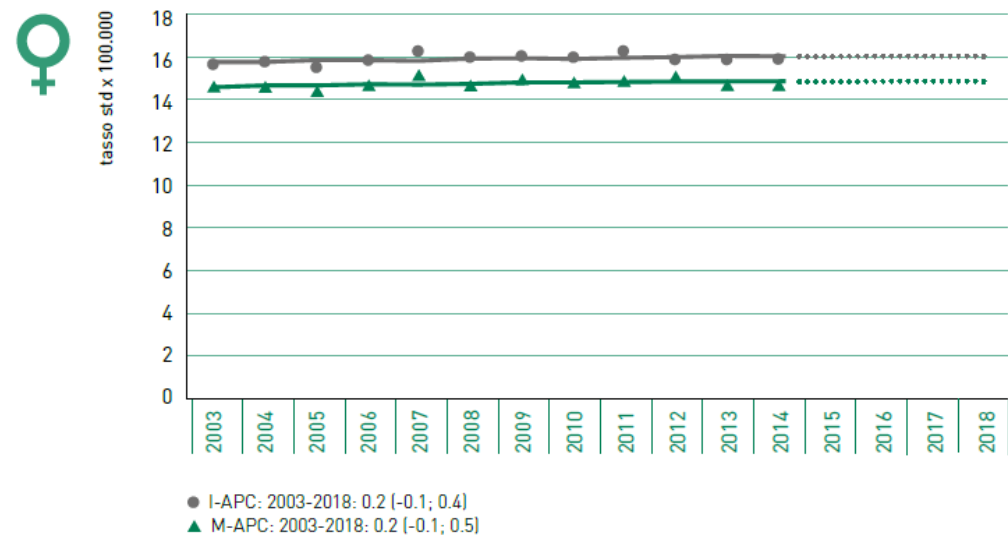


FIGURA 20B. Tumore del pancreas, femmine. AIRTUM: stima dei trend tumorali di incidenza e mortalità 2003-2018. Tassi standardizzati nuova popolazione europea 2013

APC = Annual Percent Change (variazione percentuale media annua), I = incidenza, M = mortalità.

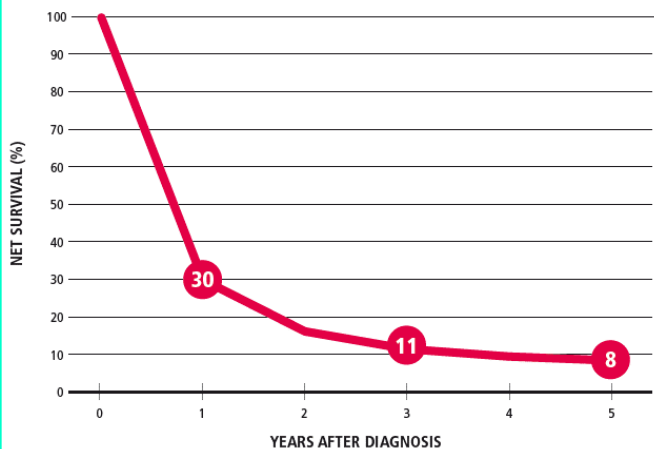


PANCREAS CANCER

MALES & FEMALES

C AGE-STANDARDIZED NET SURVIVAL (%), 2005-2009

POOL OF 42 CANCER REGISTRIES (26062 OBSERVED CASES)

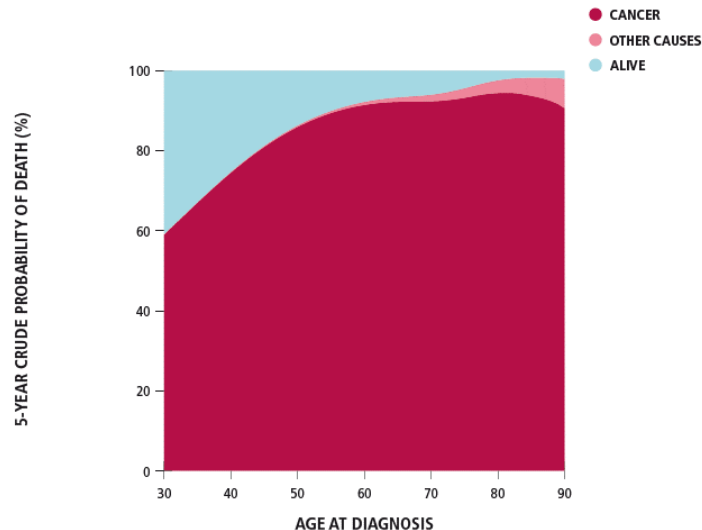


PANCREAS CANCER

MALES & FEMALES

D CRUDE PROBABILITY OF DEATH (%), 2005-2009

POOL OF 42 CANCER REGISTRIES (25127 OBSERVED CASES)



**carcinoma
epidemiologia**

sopravvivenza

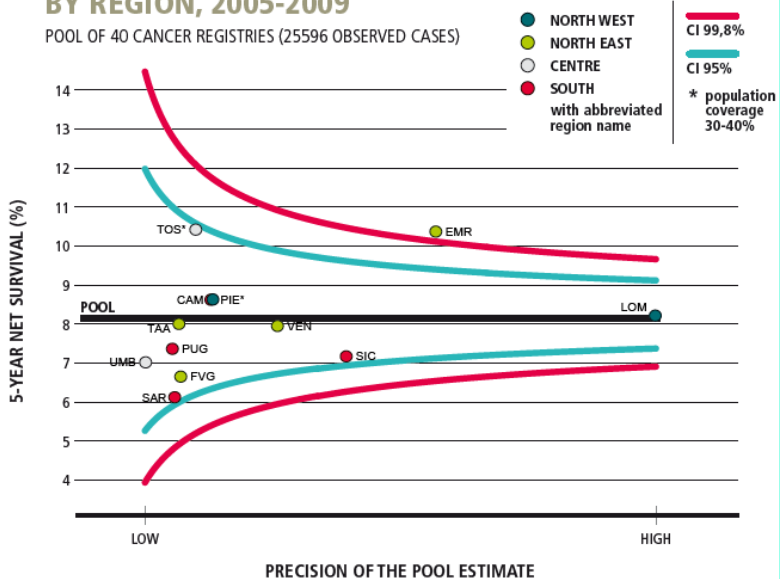


PANCREAS CANCER

MALES & FEMALES

E 5-YEAR AGE-STANDARDIZED NET SURVIVAL (%) BY REGION, 2005-2009

POOL OF 40 CANCER REGISTRIES (25596 OBSERVED CASES)

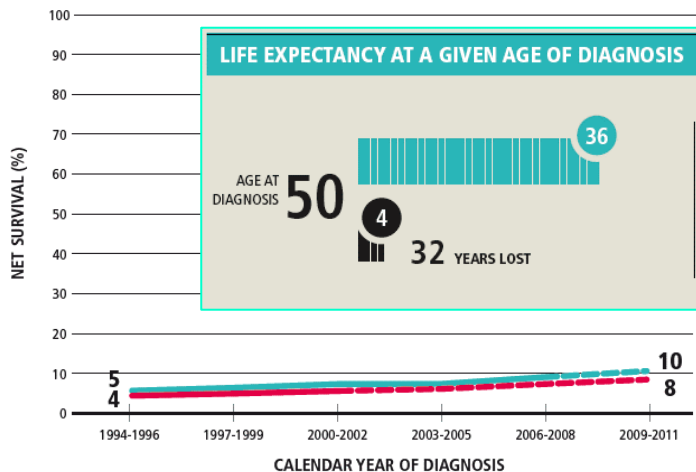


PANCREAS CANCER

MALES & FEMALES

H TIME TREND OF 5-, 10-YEAR AGE-STANDARDIZED NET SURVIVAL (%)

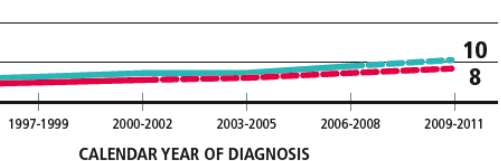
POOL OF 17 CANCER REGISTRIES



LIFE EXPECTANCY AT A GIVEN AGE OF DIAGNOSIS

Age at Diagnosis	General Population (Years Lost)	Cancer (Years Lost)
50	36	4
70	16	2

32 YEARS LOST (at age 50)
14 YEARS LOST (at age 70)





Pool of Italian Cancer Registries - 1 January 2010

PANCREAS

PANCREAS

(ICD-10 C25)

COMPLETE PREVALENCE BY YEARS SINCE DIAGNOSIS

YEARS →	≤ 2	(2 - 5]	(5 - 10]	(10 - 15]	(15 - 20]	> 20
No. →	6 730	1 792	1 671	772	368	261
% →	58%	15%	14%	7%	3%	2%
PROPORTION → PER 100 000	12	3	3	1	1	0
	MALE 45%			FEMALE 55%		

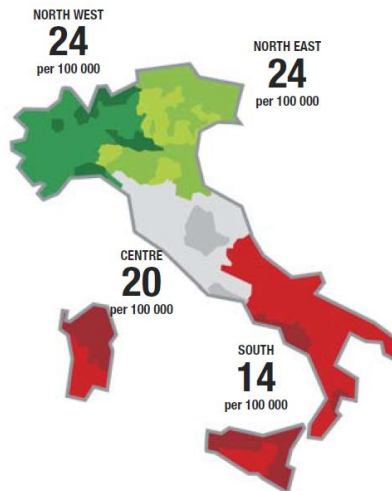
COMPLETE PREVALENCE BY SEX, MACRO-AREA, AND AGE (PROPORTION PER 100 000)

AGE CLASS →	0-44	45-59	60-74	75+	ALL AGES
MALE					
NORTH WEST	2	18	60	63	21
NORTH EAST	2	18	67	93	23
CENTRE	2	21	47	64	19
SOUTH	1	19	42	50	14
POOL	1	19	56	70	19
FEMALE					
NORTH WEST	3	15	57	81	26
NORTH EAST	3	19	51	80	25
CENTRE	2	15	46	67	20
SOUTH	2	19	39	40	15
POOL	3	17	49	69	22
BOTH SEXES					
NORTH WEST	2	16	58	75	24
NORTH EAST	2	19	59	85	24
CENTRE	2	18	47	66	20
SOUTH	1	19	40	44	14
POOL	2	18	52	69	21

2 587 347 CANCER SURVIVORS

11 594
PANCREAS
CANCER SURVIVORS

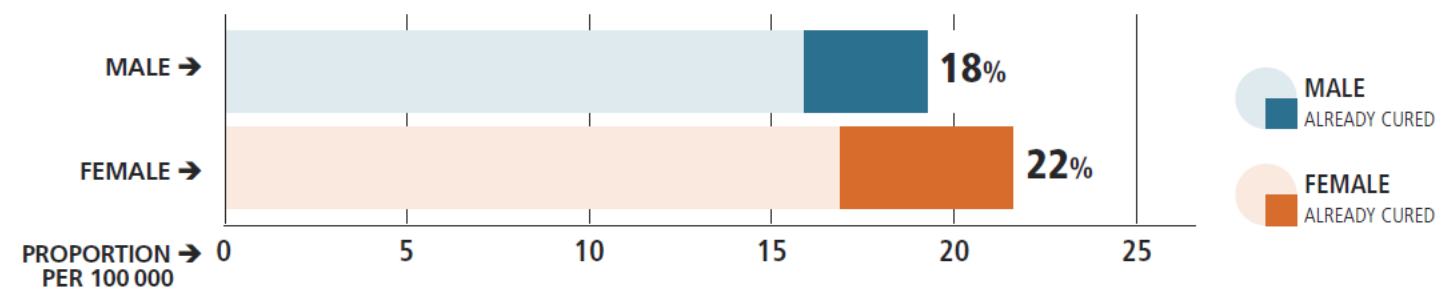
ALREADY CURED
at 1 JANUARY 2010
2 332 (20%)



**carcinoma
epidemiologia**

prevalenza

COMPLETE PREVALENCE AND PROPORTION OF ALREADY CURED SURVIVORS BY SEX



CURE FRACTION AND TIME TO CURE BY AGE AT DIAGNOSIS AND SEX FOR CANCER PATIENTS DIAGNOSED IN ITALY IN 1985-2009

AGE AT DIAGNOSIS	CURE FRACTION %		AGE AT DIAGNOSIS	TIME TO CURE YEARS	
	MALE	FEMALE		MALE	FEMALE
0 - 44	7%	14%	0 - 44	6	6
45 - 59	4%	7%	45 - 59	6	7
60 - 74	2%	3%	60 - 74	7	7
75+	1%	1%	75+	7	7



Continua...

