# Cancerul Pancreatic – Note de curs

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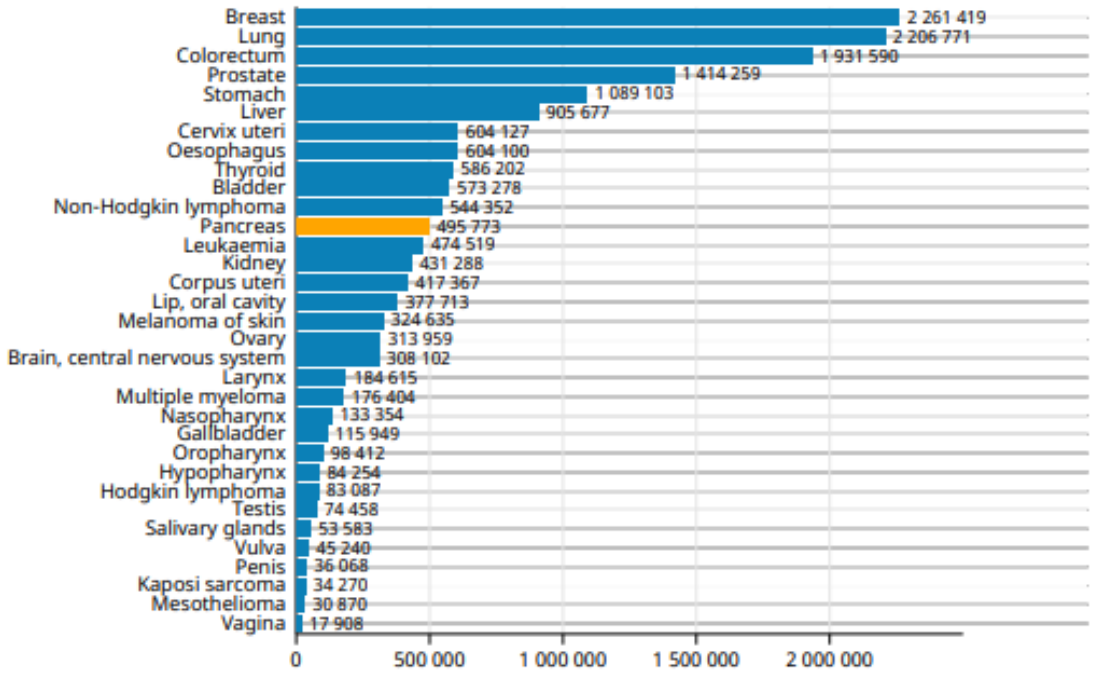
International Agency for Research on Cancer

World Health Organization

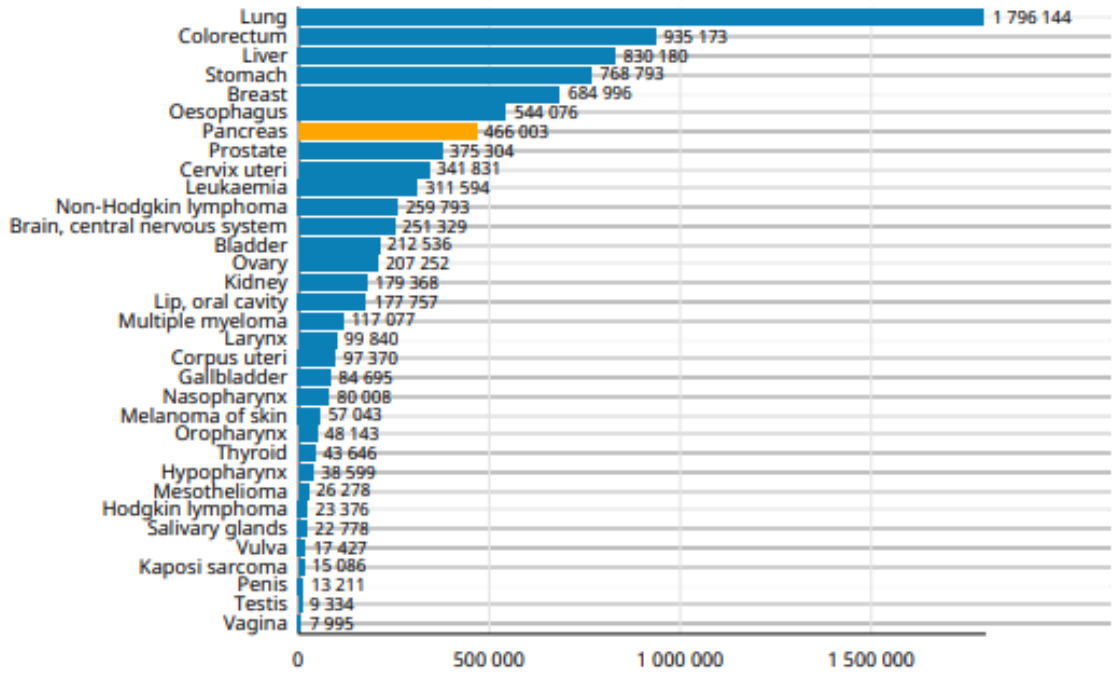
# Pancreas

Source: Globocan 2020

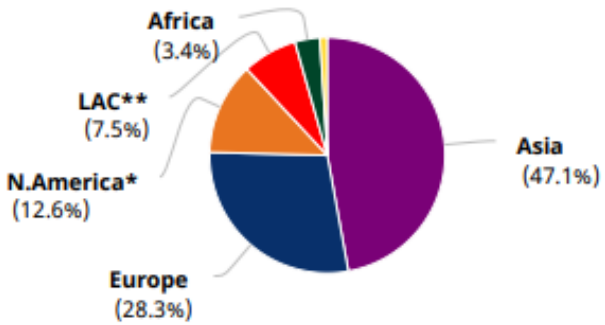
## Number of new cases in 2020, both sexes, all ages




## Number of deaths in 2020, both sexes, all ages

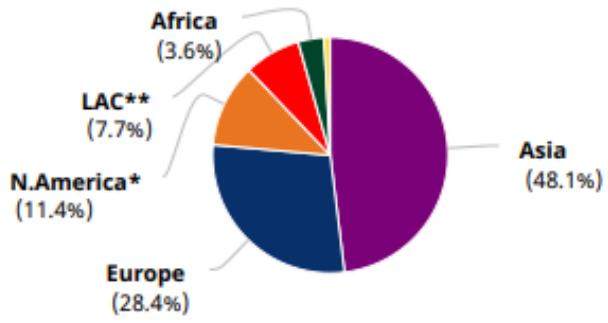


## Incidence, both sexes



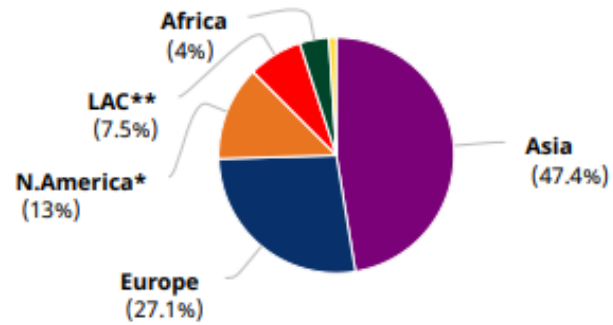

| Population | Number |
|---|---|
| Asia | 233 701 |
| Europe | 140 116 |
| *Northern America | 62 643 |
| **Latin America and the Caribbean | 37 352 |
| Africa | 17 070 |
| Oceania | 4 891 |
| Total | 495 773 |

## Mortality, both sexes




| Population | Number |
|---|---|
| Asia | 224 034 |
| Europe | 132 134 |
| *Northern America | 53 277 |
| **Latin America and the Caribbean | 36 030 |
| Africa | 16 549 |
| Oceania | 3 979 |
| Total | 466 003 |

## 5-year prevalence, both sexes

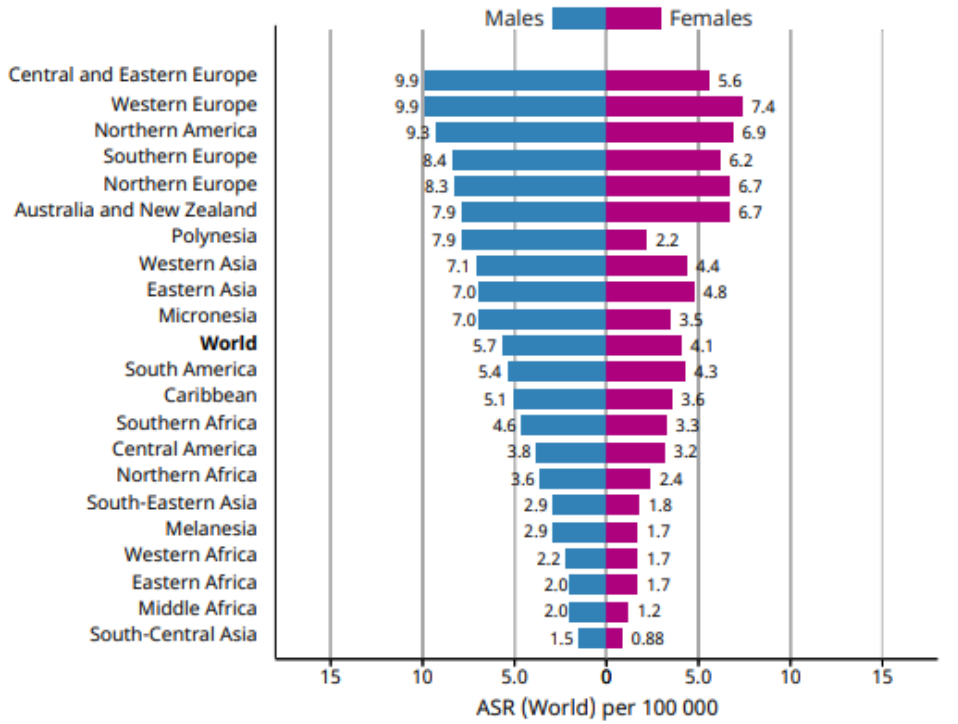



| Population | Number |
|---|---|
| Asia | 180 110 |
| Europe | 103 072 |
| *Northern America | 49 358 |
| **Latin America and the Caribbean | 28 356 |
| Africa | 15 280 |
| Oceania | 3 782 |
| Total | 379 958 |

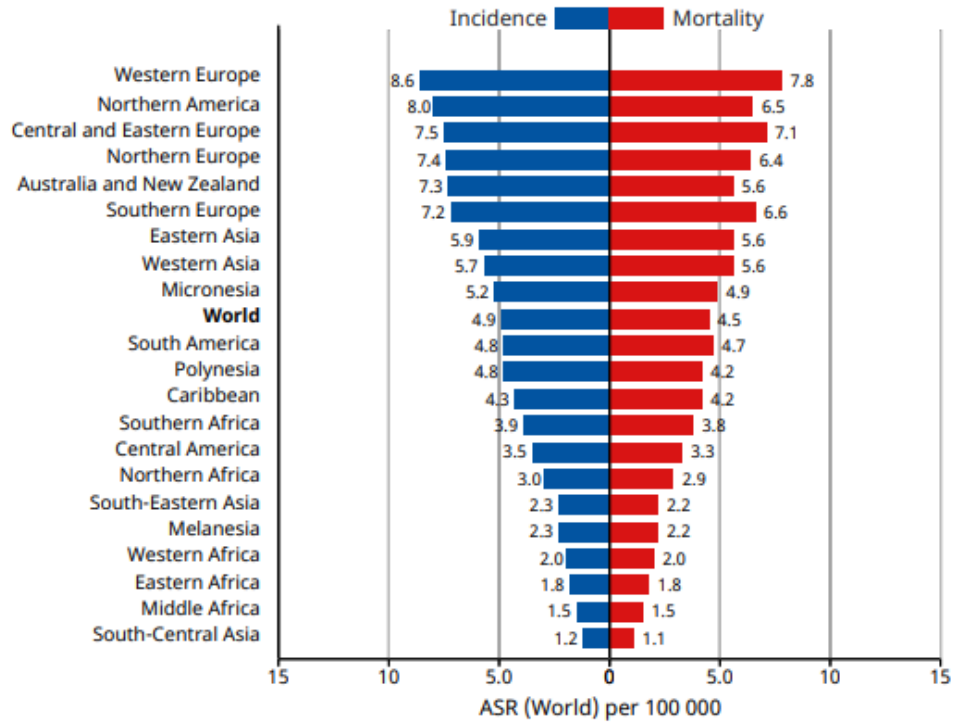
Data source: GLOBOCAN 2020
Graph production: IARC (https://gco.iarc.fr/today)
World Health Organization



## Age standardized (World) incidence rates, pancreas, by sex




## Age standardized (World) incidence and mortality rates, pancreas



# De retinut!

- **Incidența este în creștere.**
- 80% dintre pacienți aparțin decadelor a 6-a și a 7-a de vârstă.
- **Factori de risc**: fumatul, vârsta, dieta bogată în grăsimi, diabetul zaharat (DZ), alcoolismul și pancreatita cronică.
- Expunerea la naftalină și benzidină.
- **Factorul ereditar**: 1 din 20 de pacienți diagnosticați cu cancer pancreatic prezintă o anamneză eredo-colaterală agravată.

American Journal of Epidemiology

Society for Epidemiologic Research

Johns Hopkins Bloomberg School of Public Health



Volume 170, Issue 4
15 August 2009

**Article Contents**



# Cigarette Smoking and Pancreatic Cancer: A Pooled Analysis From the Pancreatic Cancer Cohort Consortium

Shannon M. Lynch, Alina Vrieling, Jay H. Lubin, Peter Kraft, Julie B. Mendelsohn, Patricia Hartge, Federico Canzian, Emily Steplowski, Alan A. Arslan, Myron Gross ... Show more

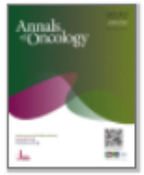
*American Journal of Epidemiology*, Volume 170, Issue 4, 15 August 2009, Pages 403–413,
https://doi.org/10.1093/aje/kwp134
**Published:** 26 June 2009 **Article history**



**Abstract**

Smoking is an established risk factor for pancreatic cancer; however, detailed examination of the association of smoking intensity, smoking duration, and cumulative smoking dose with pancreatic cancer is limited. The authors analyzed pooled data from the international Pancreatic Cancer Cohort Consortium nested case-control study (1,481 cases, 1,539 controls). Odds ratios and 95% confidence intervals were calculated by using unconditional logistic regression. Smoking intensity effects were examined with an excess odds ratio model that was linear in pack-years and exponential in cigarettes smoked per day and its square. When compared with never smokers, current smokers had a significantly elevated risk (odds ratio (OR) = 1.77, 95% confidence interval (CI): 1.38, 2.26). Risk increased significantly with greater intensity (≥30 cigarettes/day: OR = 1.75, 95% CI: 1.27, 2.42), duration (≥50 years: OR = 2.13, 95% CI: 1.25, 3.62), and cumulative smoking dose (≥40 pack-years: OR = 1.78, 95% CI: 1.35, 2.34). Risk more than 15 years after smoking cessation was similar to that for never smokers. Estimates of excess odds ratio per pack-year declined with increasing intensity, suggesting greater risk for total exposure delivered at lower intensity for longer duration than for higher intensity for shorter duration. This finding and the decline in risk after smoking cessation suggest that smoking has a late-stage effect on pancreatic carcinogenesis.

Annals of Oncology
Volume 23, Issue 7, July 2012, Pages 1880-1888



original articles
epidemiology

# Cigarette smoking and pancreatic cancer: an analysis from the International Pancreatic Cancer Case-Control Consortium (Panc4)

C. Bosetti [1, 2], E. Lucenteforte [1, 3, 4], D.T. Silverman [5], G. Petersen [6], P.M. Bracci [7], B.T. Ji [5], E. N Li [8], H.A. Risch [9], S.H. Olson [10], S. Gallinger [11], A.B. Miller [12], H.B. Bueno-de-Mesquita [13, 14], R. Ta J. Polesel [15], P. Ghadirian [16], P.A. Baghurst [17], W. Zatonski [18] ... C. La Vecchia [1, 3, 26]

Show more

## Results

Compared with never smokers, the OR was 1.2 (95% confidence interval [CI] 1.0–1.3) for former smokers and 2.2 (95% CI 1.7–2.8) for current cigarette smokers, with a significant increasing trend in risk with increasing number of cigarettes among current smokers (OR = 3.4 for ≥35 cigarettes per day, *P* for trend <0.0001). Risk increased in relation to duration of cigarette smoking up to 40 years of smoking (OR = 2.4). No trend in risk was observed for age at starting cigarette smoking, whereas risk decreased with increasing time since cigarette cessation, the OR being 0.98 after 20 years.

## Conclusions

This uniquely large pooled analysis confirms that current cigarette smoking is associated with a twofold increased risk of pancreatic cancer and that the risk increases with the number of cigarettes smoked and duration of smoking. Risk of pancreatic cancer reaches the level of never smokers ~20 years after

# Incidence of pancreatic cancer is dramatically increased by a high fat, high calorie diet in KrasG12D mice

Hui-Hua Chang, Aune Moro, Kazuki Takakura, Hsin-Yuan Su, Allen Mo, Masako Nakanishi, Richard T. Waldron, Samuel W. French, David W. Dawson, O. Joe Hines, Gang Li, Vay Liang W. Go, James Sinnett-Smith, [ ... ], Guido Eibl
[ view all ]

Published: September 8, 2017 • https://doi.org/10.1371/journal.pone.0184455





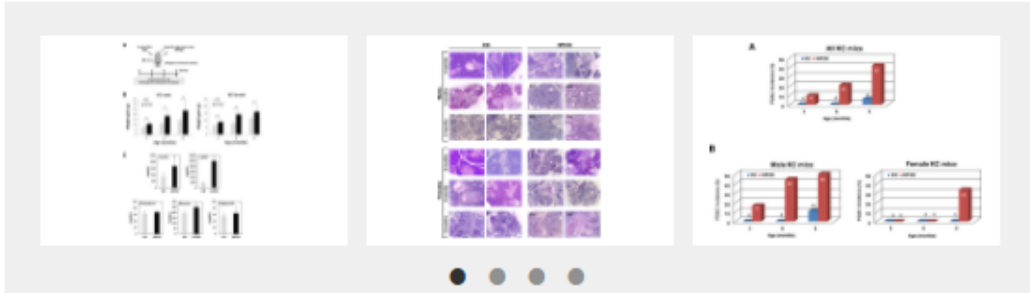
## Abstract

Epidemiologic data has linked obesity to a hi
mechanisms are poorly understood. To allow
model mimicking diet-induced obesity and pa
(HFCD) was given to *P48*$^{+/Cre}$;*LSL-KRAS*$^{G1}$
oncogenic Kras mutation. The mice were ran



Epidemiologic data has linked obesity to a higher risk of pancreatic cancer, but the underlying mechanisms are poorly understood. To allow for detailed mechanistic studies in a relevant model mimicking diet-induced obesity and pancreatic cancer, a high-fat, high-calorie diet (HFCD) was given to *P48*$^{+/Cre}$;*LSL-KRAS*$^{G12D}$ (KC) mice carrying a pancreas-specific oncogenic Kras mutation. The mice were randomly allocated to a HFCD or control diet (CD). Cohorts were sacrificed at 3, 6, and 9 months and tissues were harvested for further analysis. Compared to CD-fed mice, HFCD-fed animals gained significantly more weight. Importantly, the cancer incidence was remarkably increased in HFCD-fed KC mice, particularly in male KC mice. In addition, KC mice fed the HFCD showed more extensive inflammation and fibrosis, and more advanced PanIN lesions in the pancreas, compared to age-matched CD-fed animals. Interestingly, we found that the HFCD reduced autophagic flux in PanIN lesions in KC mice. Further, exome sequencing of isolated murine PanIN lesions identified numerous genetic variants unique to the HFCD. These data underscore the role of sustained inflammation and dysregulated autophagy in diet-induced pancreatic cancer development and suggest that diet-induced genetic alterations may contribute to this process. Our findings provide a better understanding of the mechanisms underlying the obesity-cancer link in males and females, and will facilitate the development of interventions targeting obesity-associated pancreatic cancer.

## Figures



Article

May 24, 1995

# Diabetes Mellitus as a Risk Factor for Pancreatic Cancer

## A Meta-analysis

James Everhart, MD, MPH; David Wright, PhD

Author Affiliations

*JAMA*. 1995;273(20):1605-1609. doi:10.1001/jar

Full Text

Full Text

## Abstract

**Objective.** —To evaluate diabetes mellitus as a risk factor for pancreatic cancer with the consideration that diabetes may also be a consequence of pancreatic cancer.

**Data Sources.** —Pertinent studies of diabetes mellitus and pancreatic cancer published between 1975 and 1994 were identified from a MEDLINE search and from citations in articles and books.

**Study Selection.** —Twenty of a total of 30 case-control and cohort studies met the two inclusion criteria: cases with a duration of diabetes of at least 1 year prior to either pancreatic cancer diagnosis or death and the ability to calculate an appropriate relative risk (RR) estimate and variance.

**Data Extraction and Synthesis.** —The pooled RR and 95% confidence interval (CI) of pancreatic cancer for diabetics relative to nondiabetics was 2.1 (1.6 to 2.8). There was a tendency for a higher RR for the nine cohort studies (RR, 2.6; 95% CI, 1.6 to 4.1) than for the 11 case-control studies (RR, 1.8; 95% CI, 1.1 to 2.7). Requiring diabetes duration of at least 5 years resulted in an RR of 2.0 (95% CI, 1.2 to 3.2).

**Conclusion.** —Pancreatic cancer occurs with increased frequency among persons with long-standing diabetes. (*JAMA*. 1995;273:1605-1609)

**Diabetes mellitus** and **pancreatic cancer** in a population-based case-control study in the San Francisco Bay Area, California

F Wang, S Gupta, EA Holly - **Cancer** Epidemiology and Prevention ..., 2006 - AACR

Background: **Diabetes** has been postulated to be both a risk factor and a consequence of **pancreatic cancer**, but the degree of risk and associated clinical factors remain unclear. Methods: We conducted a population-based case-control study of **pancreatic cancer** in the ...

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**Diabetes mellitus** as a risk factor for **pancreatic cancer**: a meta-analysis

J Everhart, D Wright - Jama, 1995 - jamanetwork.com

Objective.—To evaluate **diabetes mellitus** as a risk factor for **pancreatic cancer** with the consideration that **diabetes** may also be a consequence of **pancreatic cancer**. Data Sources.—Pertinent studies of **diabetes mellitus** and **pancreatic cancer** published between 1975 and ...

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**Diabetes mellitus** and **pancreaticcancer** mortality in a prospectivecohort of United States adults

EE Calle, TK Murphy, C Rodriguez, MJ Thun... - **Cancer** causes & ..., 1998 - Springer

Objectives: **Diabetes mellitus** and **pancreatic cancer** are known to be associated, but it is not known whether **diabetes** is a true risk factor, preceding development of the **cancer**, or if it is an early manifestation of the **cancer**. To address this uncertainty, we examined the ...



# Diabetes Mellitus and Pancreatic Cancer in a Population-Based Case-Control Study in the San Francisco Bay Area, California

**Furong Wang,[1] Samir Gupta,[2] and Elizabeth A. Holly[1,3]**

[1]Department of Epidemiology and Biostatistics, School of Medicine, and [2]Department of Medicine, Division of Gastroenterology, University of California San Francisco, San Francisco, California; and [3]Department of Health Research and Policy, School of Medicine, Stanford University, Stanford, California

## Abstract

Background: **Diabetes has been postulated to be both a risk factor and a consequence of pancreatic cancer, but the degree of risk and associated clinical factors remain unclear.**

Methods: **We conducted a population-based case-control study of pancreatic cancer in the San Francisco Bay Area between 1995 and 1999. Rapid case ascertainment through the Surveillance, Epidemiology and End Results registry for cases and random selection from the general population for controls were employed to identify study participants with no proxy interviews.**

Results: **Five hundred thirty-two cases and 1,701 controls were interviewed. Participants with pancreatic cancer were more likely to report a history of diabetes (13%) than were controls [9%; odds ratio (OR), 1.5; 95% confidence interval (95% CI), 1.1-2.1]. Compared with diabetics in the control group, diabetics in the case group had a shorter duration of diabetes ($P = 0.0003$) and a larger proportion of insulin users ($P = 0.002$). Risk for pancreatic cancer varied with duration of diabetes (OR, 2.4; 95% CI, 1.4-4.0 for 1-4 years; OR, 2.0; 95% CI, 1.2-3.4 for 5-9 years; and OR, 0.86; 95% CI, 0.52-1.4 for ≥10 years diabetes duration; $P_{trend} = 0.004$). Among diabetics, use of oral diabetes medication or insulin for ≥5 years was not associated with pancreatic cancer, but insulin use of <5 years was associated with a 6.8-fold risk for pancreatic cancer (95% CI, 3.7-12).**

Conclusion: **Recent-onset diabetes may be a complication or an early marker of pancreatic cancer. Diabetes of short duration with insulin use conferred a substantially elevated risk for pancreatic cancer and may reflect insulin resistance that is elicited by pancreatic cancer.** (Cancer Epidemiol Biomarkers Prev 2006;15(8):1458–63)

## Introduction

Pancreatic cancer is diagnosed in nearly 34,000 individuals per year in the United States, and the overall 5-year survival rate is <4% (1). Although 5-year survival of 15% has been shown in approximately twice the risk of developing pancreatic cancer compared with those without diabetes after censoring of pancreatic cancer diagnosed in the first year of follow-up

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doi: 10.1097/MPA.0b013e3182592c96

PMCID: PMC3896296
NIHMSID: NIHMS546833
PMID: 23000893

# Prevalence of diabetes mellitus in pancreatic cancer compared to common cancers

Gaurav Aggarwal, MD,[1] Pratima Kamada, MD,[2] and Suresh T. Chari, MD[1]

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## Objectives

In pancreatic cancer (PaC) the prevalence of diabetes mellitus (DM), especially new-onset DM (≤ 36 months of PaC diagnosis), is high. To determine if this observation is unique to PaC, we compared the prevalence and characteristics of DM in lung, breast, prostate and colorectal cancer with PaC and non-cancer controls.

## Methods

We retrospectively reviewed medical records of 500 consecutive cancer patients (100 each with lung, breast, prostate, colorectal and PaC) and 100 non-cancer controls.

## Results

Patients with PaC (mean age: 71.6±9.4 years; 53% male) had a significantly ($p<0.0001$) higher prevalence of DM (68%) compared to age-matched patients with lung (71.6±9.4 years, 59% male, 19.6% DM), breast (71.6±9.6 years, 100% female, 19.4% DM), prostate (71.3±9.4 years, 100% male, 14.8% DM), and colorectal cancer (71.6±9.5 years, 56% male, 20.7% DM), and non-cancer controls (70.7±9.2 years, 57% male, 23.5% DM). Among PaC patients, 40% developed DM in the 36 months preceding the diagnosis of PaC, as compared to 3.3-5.7% in the other groups ($p<0.0001$).

## Conclusions

While the prevalence of DM in PaC is very high, DM prevalence in other common cancers is no different from that in non-cancer controls. In particular, new-onset DM is a phenomenon that is unique to PaC.

**Keywords:** Pancreatic cancer, diabetes mellitus, screening

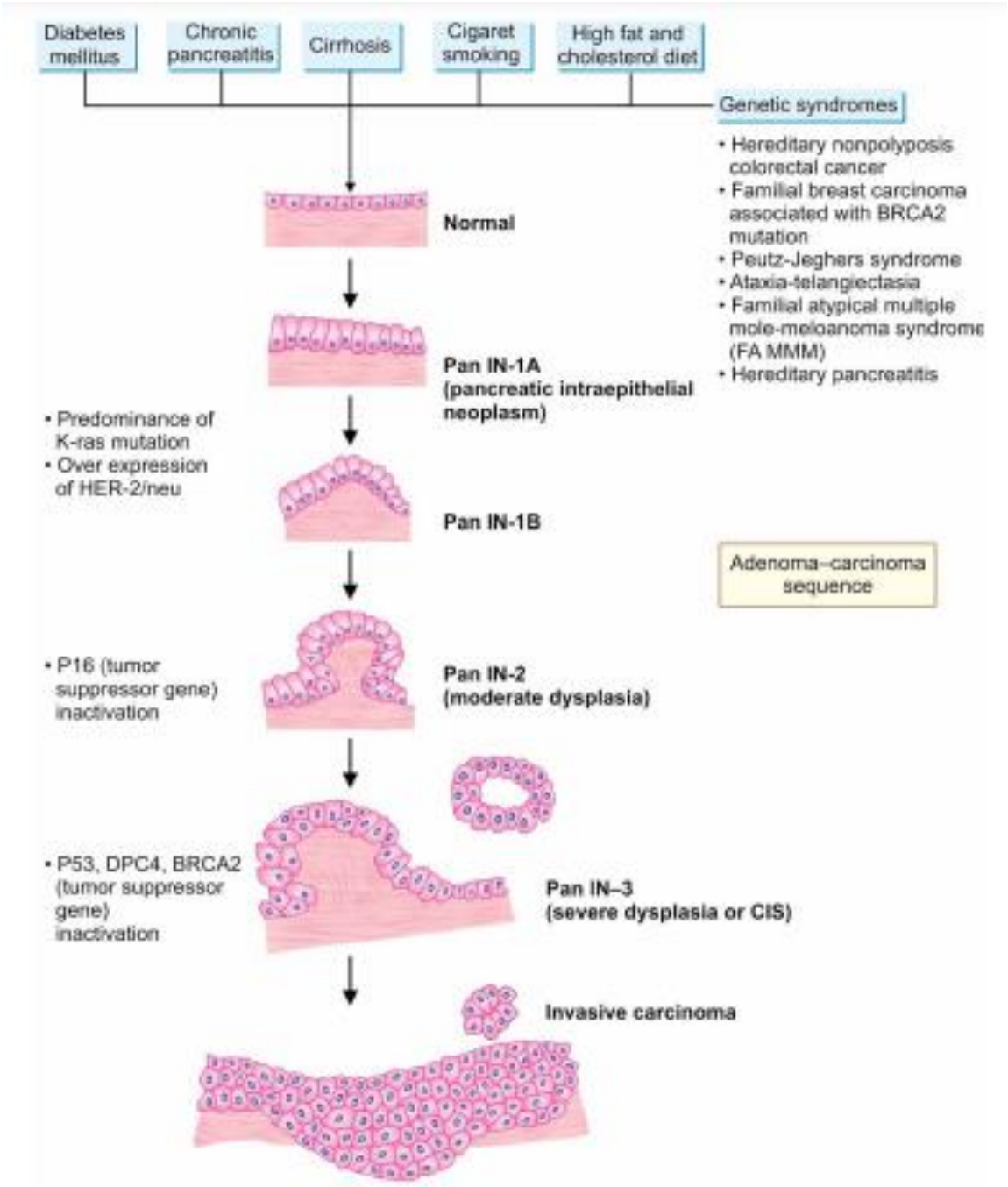
# Sindromul MEN 1 – ”WERMER”

- **Transmitere autozomal dominantă**, determinată de mutații care inactivează gena supresor tumorală MEN 1, situată pe cromozomul 11q3.
- 2-20 la 100000 populație, F=B.
- MEN 1 forma sporadică și MEN 2 forma familială.
- **Sindromul MEN 1** conține peste 20 de tumori endocrine și non-endocrine.

**Tumorile endocrine**:
•Tumori ale paratiroidelor 95-100% (hiperparatiroidismul primar)
•Tumori hipofizare 20-25%
•Tumori ale tractului gastro-intestinal 30-80% (gastrinomul/s. ZE, insulinom, glucagonom, VIPom)
•Tumori corticosuprarenaliene 20-40%
•Tumori carcinoide 10%

**Tumorile nonendocrine**:
•Angioame faciale
•Colagenoame
•Lipoame

# Etiologie:


Diabetes mellitus
Chronic pancreatitis
Cirrhosis
Cigaret smoking
High fat and cholesterol diet
Genetic syndromes
• Hereditary nonpolyposis colorectal cancer
• Familial breast carcinoma associated with BRCA2 mutation
• Peutz-Jeghers syndrome
• Ataxia-telangiectasia
• Familial atypical multiple mole-melanoma syndrome (FA MMM)
• Hereditary pancreatitis
Normal
Pan IN-1A (pancreatic intraepithelial neoplasm)
• Predominance of K-ras mutation
• Over expression of HER-2/neu
Pan IN-1B
Adenoma–carcinoma sequence
• P16 (tumor suppressor gene) inactivation
Pan IN-2 (moderate dysplasia)
• P53, DPC4, BRCA2 (tumor suppressor gene) inactivation
Pan IN–3 (severe dysplasia or CIS)
Invasive carcinoma

# Morfopatologie:

- **Adenocarcinomul ductal** este cea mai frecventă formă histologică (aprox. 85%) – se dezvoltă din celulele epiteliale ductale.
- **Carcinom din celule acinare**: tumori mari în dimensiuni, dar cu un prognostic mai bun.
- **Carcinom adenoscuamos**.
- **Carcinom nediferențiat cu celule gigante osteoclast-like**.
- **Carcinom coloid**.
- **Carcinom cu celule inel cu pecete**.

# Manifestări clinice:

- **Carcinom cefalopancreatic** (65%)
- **Icter mecanic** (90%) – datorat compresiei sau invaziei CBP. Semnul Courvoisier-Terrier (+).
- **Durere** (70%) în epigastru sau hipocondrul stâng, „vagă” cu iradiere în spate.
- **Hepatomegalie**, datorată metastazelor.
- **Anorexie**, greață, vărsături, fatigabilitate, dispepsie, prurit.
- **Semne de pancreatită acută** – primele semne clinice.
- **Thrombophlebitis migrans**

# FIZIOLOGIA BILEI

- Bilirubina se formează la degradarea hematiilor în splină.
- Este insolubilă și este transportată în ficat fiind atașată la albumină.
- Fiind preluată prin transport activ de către hepatocite, unde fiind conjugată, este excretată (hidrosolubilă) prin căile biliare în duoden.
- 10% din bilirubina conjugată este transformată în urobilinogen de către bacteriile intestinului subțire, reabsorbită în ileonul terminal și excretată în urină (circulația enterohepatică).
- 90% este transformată de către bacteriile colonice în stercobilinogen, care este excretată în fecale.

# ICTER

**Pre-hepatic (hemolitic)**

- Anomalii congenitale ale hematiilor (ex. sferocitoza ereditară, siclemia).
- Anemie hemolitică autoimună.
- Reacții adverse în timpul transfuziilor.
- Medicamentoasă.

**Hepatic (hepatocelular)**

- **Sindrom Gilbert** – Deficiență la preluarea bilirubinei.
- **Sindromul Crigler–Najjar** – Deficiența conjugării de către enzime.
- **Infecții**: virale (ex. hepatita A, B, C, EBV, CMV); bacteriale (ex. abcese hepatice, leptospiroză); parazitare (ex. amoebic).
- **Droguri**: ex. paracetamol, antipsihotice, antibiotice.
- **Hepatite ne-infecțioase**: ex. cronice, alcoolice.

**Post-hepatic (mecanic)**

- Coledoco-litiază
- Paraziți
- Neoplasm hepatic
- Neoplasm CBP
- Patologia Ampulei Water
- Neoplasm cefalopancreatic
- Atrezia CBP
- Colangită sclerozantă

# ICTER- Ce facem?

- **Istoric:**
- Anamneza eredo-colaterală.
- Plecări sau activități în alte zone geografice (expunerea la infecții).
- Medicație recentă.
- Tratamente chirurgicale recente cu anestezie.
- Anamneză litiazică.
- Consum de alcool, colangită (durere, febră, colangită)

# ICTER- Ce facem?

- **Teste de bază:**
- Reticulocitoză (hemoliză).
- Indicele prothrombinic.
- **Screening-ul hepatitic** (titrul viral sau antigenic pentru A, B, C, CMV, EBV).
- **Teste imunologice**: anti-smooth muscle antibodies (hepatită cronică fază activă) și Ac anti-mitocondriali (ciroză biliară primară).

# Teste funcționale hepatice în Icter

| | Haemolytic | Hepatocellular | Obstructive |
|---|---|---|---|
| Unconjugated bilirubin | Increased | Increased | Normal |
| Alkaline phosphatase | Normal | Normal | Much increased |
| γ glutamyl transferase | Normal | Increased | Much increased |
| Transaminases | Normal | Increased | Normal |
| Lactate dehydrogenase | Normal | Increased | Normal |

# ICTER- Ce facem?

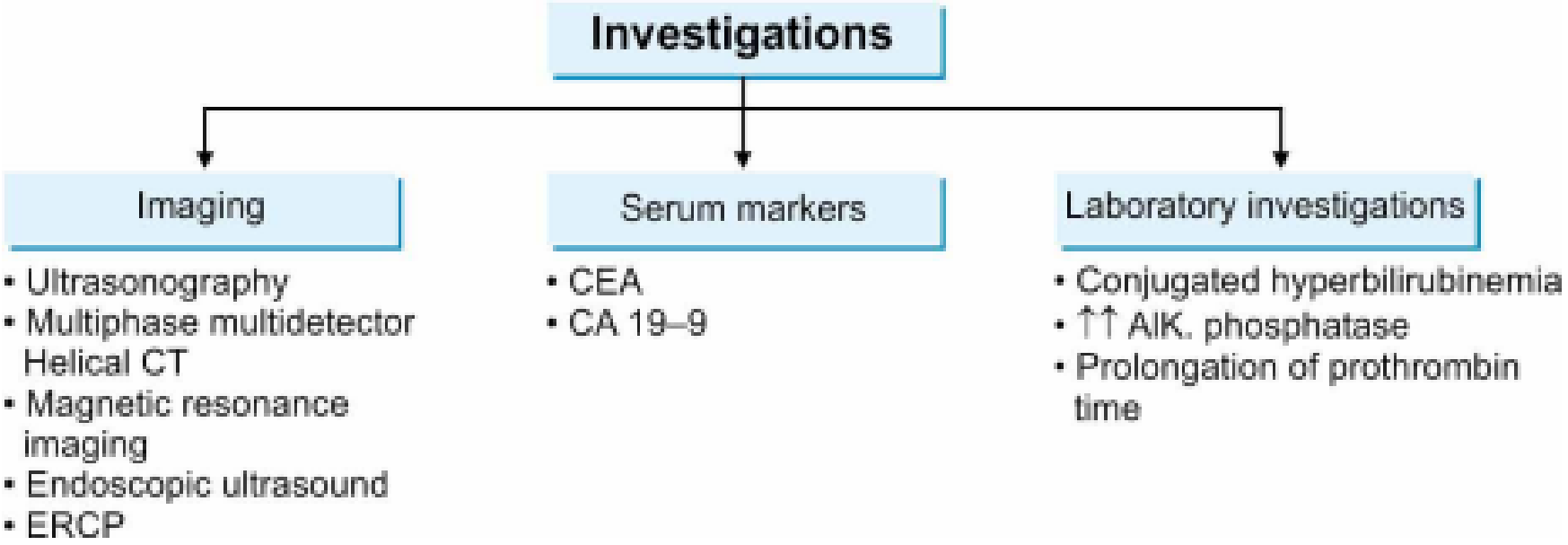
- **USG (ultrasonografie)**
- Examinarea ficatului, veziculei biliare, ducturilor biliare și pancreasului.
- Excluderea obstrucției extrahepatice (ducturi biliare dilatate).
- Localizarea posibilă a obstrucției.
- Examinarea parenchimului hepatic.
- **MRCP (colangiopancreatografie prin rezonanță magnetică)**
- Localizarea blocului biliar în cazul în care USG este ineficace.
- **Biopsie hepatică ghidată prin USG**
- Prelevarea de țesut hepatic pentru examinare.

# Complicațiile icterului:

- **Insuficiență hepato-renală**
  - Afectarea simultană a funcțiilor ficatului și rinichilor, frecvent întâlnită în cazuri avansate de icter mecanic. Poate duce la insuficiență renală acută.
- **Colangită**
  - Inflamația canalelor biliare cauzată de infecții bacteriene, autoimune sau obstrucții. Simptome: durere abdominală, icter și febră.
- **Dereglarea sistemului de coagulare**
  - Disfuncții în coagularea sângelui care pot duce la hemoragii sau formarea de trombi. Cauze: deficit de proteine de coagulare sau utilizarea anumitor medicamente.
- **Imunosupresie**
  - Reducerea activității sistemului imunitar, făcând organismul mai vulnerabil la infecții. Apare adesea ca rezultat al bolii sau tratamentelor specifice.

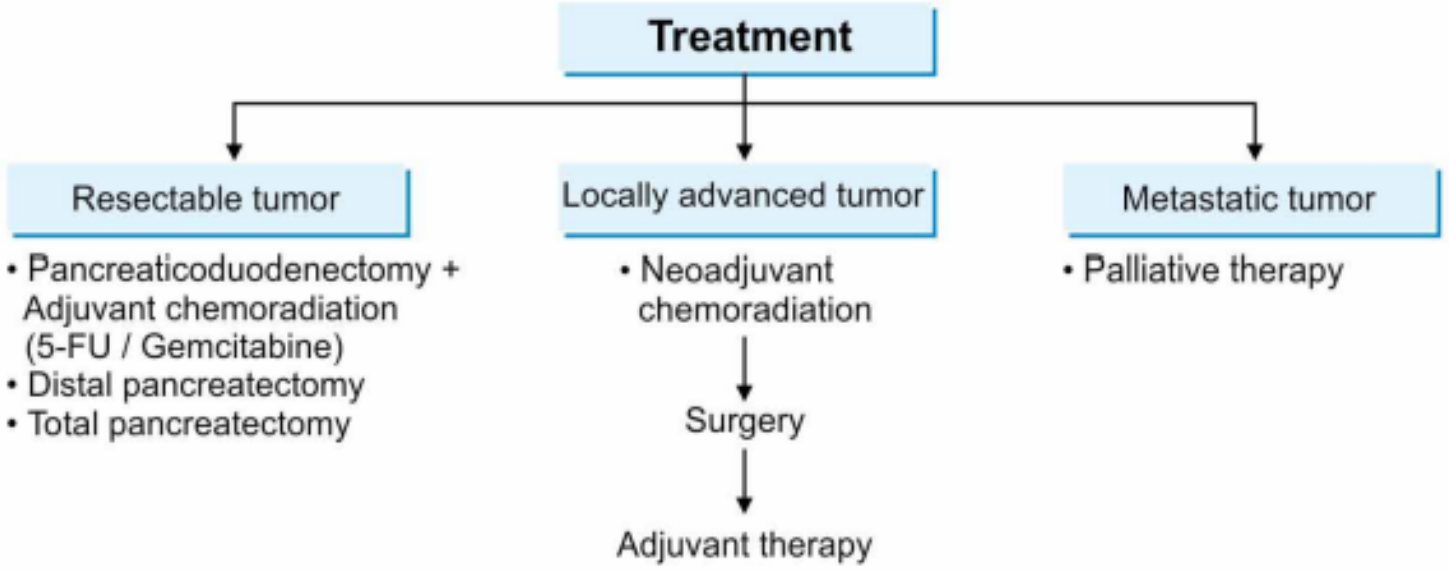
# Tratament și gestionare pentru icterul mecanic

- **Corectarea deshidratării**:
  - Administrează până la 1000mL soluție cristaloidă IV dacă nu există boală hepatică preexistentă.
  - Monitorizarea atentă a sodiului în cazurile de boală hepatică preexistentă și consultarea unui specialist.
- **Monitorizarea debitului urinar**:
  - Cateterizare uretrală.
  - Monitorizarea orară a debitului urinar.
- **Tratamentul infecțiilor**:
  - Prelevarea culturilor de sânge dacă pacientul este febril.
  - Administrarea de antibiotice IV conform protocolului local (ex. cefuroximă 750mg IV tds, gentamicină IV, ciprofloxacină PO sau IV 500mg).
  - Tratarea urgentă a obstrucției căilor biliare (ex. drenaj ghidat radiologic, ERCP, rar chirurgie).
  - Luarea în considerare a antibioticelor profilactice.
- **Verificarea timpilor de coagulare**:
  - Măsurarea timpului de protrombină (PT) și a timpului de tromboplastină parțial activată (APTT).
  - Administrarea vitaminei K 10mg IV în cazul unui PT prelungit.
- **Asigurarea unei nutriții adecvate**:
  - Revizuirea dietei pacientului.
  - Hrănirea enterală este optimă, dar poate necesita un NGT fin sau, ocazional, gastrostomie sau jejunostomie chirurgicală.
- **Rezolvarea obstrucției**:
  - Lichidarea obstrucției biliare pentru tratarea icterului.




- **Markerii Tumorali**
- **CA 19-9**:
  - Crește în aprox. 75% dintre pacienții cu cancer pancreatic.
  - Poate fi crescut în patologii benigne pancreatice, hepatice sau biliare.
  - ↑↑CA 19–9 indică un stadiu avansat al bolii.
  - ↑CA 19–9 post-operator indică recurența sau avansarea bolii.

# Opțiuni de Tratament




- **Chirurgical**: Îndepărtarea chirurgicală a tumorii și, uneori, a țesuturilor înconjurătoare afectate.
- **Chimioterapic**:
  - **Neoadjuvant**: Administrat înainte de chirurgie pentru a micșora tumoarea.
  - **Adjuvant**: Administrat după chirurgie pentru a distruge celulele canceroase rămase.
- **Radioterapic**: Utilizarea radiațiilor pentru a distruge celulele canceroase sau a micșora tumorile.

# Tratament chirurgical:

**Criterii de Rezecabilitate**

- **Absența metastazelor**: Nu există metastaze la momentul evaluării.
- **Tumora nu implică axul celiac sau SMA**: Tumora nu invadează axul celiac sau artera mezenterică superioară (SMA).
- **Lipsa implicării SMV sau PV**: Radiologic, nu există implicare a venei mezenterice superioare (SMV) sau a venei portale (PV).

**Criterii pentru Rezecție Tip Borderline**

- **Implicarea parțială a SMV și PV**: <180° din circumferința venei mezenterice superioare (SMV) și a venei portale (PV).
- **Lipsa implicării directe a trunchiului celiac sau SMA**: Tumora nu implică direct trunchiul celiac sau artera mezenterică superioară (SMA).
- **Evaluarea radiologică a SMV și PV**: Dislocări ale acestor vene, tromboze venoase etc.

# Tratament curativ

**Tipuri de Rezecție Chirurgicală**

- **Rezecția radicală proximală**: Procedeu Whipple.
- **Pancreatectomie totală**: Îndepărtarea întregului pancreas.
- **Pancreatectomie distală**: Îndepărtarea părții distale a pancreasului.
- **Doar 10-20%** dintre pacienți sunt eligibili pentru acest tip de tratament.

# Tratament paliativ:

**Rezolvarea Icterului**

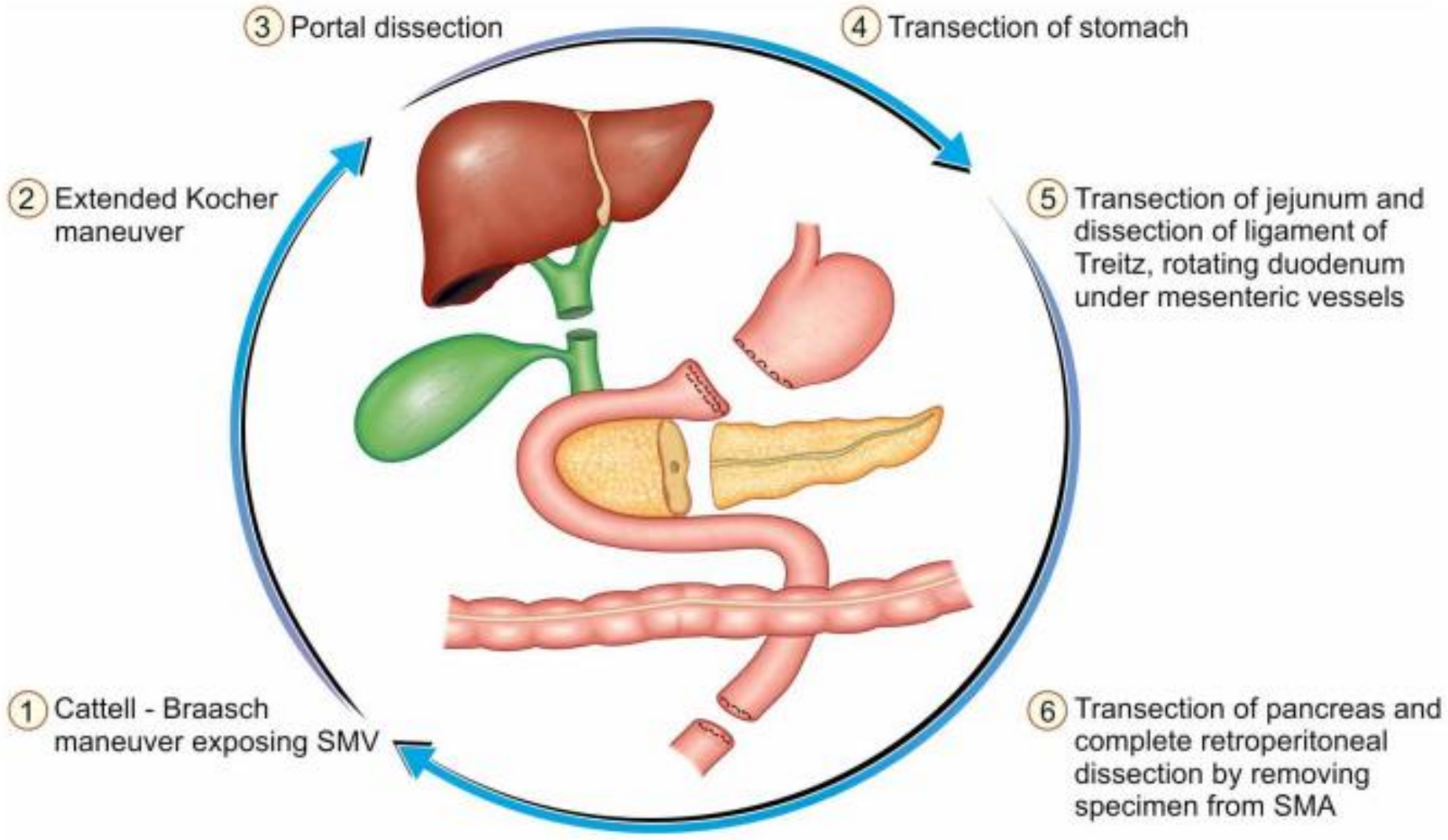
- **ERCP**: Intervenție endoscopică pentru drenarea bilei.
- **Drenaj transparietohepatic**: Drenaj al arborelui biliar prin piele.
- **By-pass biliodigestiv sau gastro-enteral**: Crearea unei rute alternative pentru drenajul biliar, evitând obstrucția.

**Cuparea Sindromului Dureros**

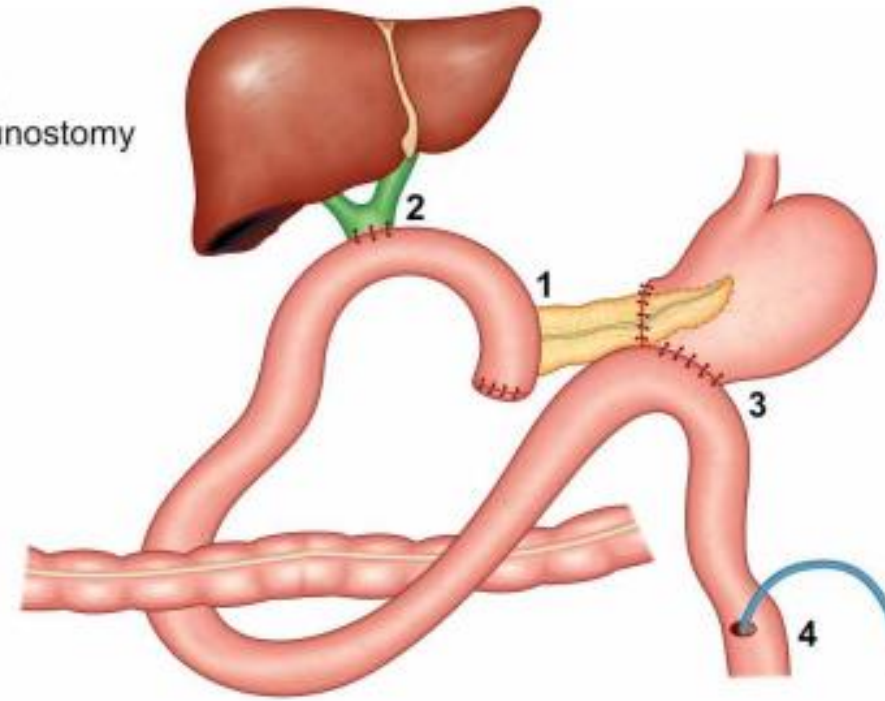
- **Morfina orală (oramorph sau MST)**: Utilizată pentru ameliorarea durerii severe.
- **Ablația chimică a trunchiului celiac**: Procedură pentru reducerea durerii intense prin distrugerea nervilor care transmit durerea.
- **Blocurile nervoase**: Injecții pentru a bloca semnalele de durere din anumite regiuni.
- **Analgezice adjuvante**: Medicamente precum gabapentin sau pregabalin pentru a trata durerea neuropatică.
- **Radioterapia paliativă**: Utilizată pentru a reduce dimensiunea tumorilor și a ameliora durerea.

**Suport emoțional și social**: Asistență pentru pacient și familia acestuia, inclusiv consiliere psihologică și grupuri de suport.

## Steps of resection




## Steps of reconstruction




(2) End to side hepaticojejunostomy

(1) End to side pancreaticojejunostomy

(3) End to side gastrojejunostomy

(4) Jejunostomy tube placement

Complications of pancreaticoduodenectomy:

- Delayed gastric emptying
- Hemorrhage
- Pancreatic fistula

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# Pancreatic Adenocarcinoma

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