제60차
한국췌장외과연구회
- KAHBPS 부울경지회 심포지움

일시: 2019년 6월 8일 (토)

장소: 부산대학교병원 권역호흡기전문질환센터(R동) 13층 강당

주최: 한국췌장외과연구회, 한국간담췌외과학회 부울경지회

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12:00-13:00	점심식사 및 등록		
13:00-13:10	개회사	KPSC 회장 **최인석**	
	축사 I	부산대학교병원 **윤명희** 과장	
	축사 II	KAHBPS 부울경지회 회장 **신동훈**	
13:10-13:20	KPSC 연구기금 수상자 발표 및 계획	**이현국**(이화의대)	
13:10-13:15	한국 가족성 췌장암의 유병률과 임상 양상 분석	**김홍범**(서울의대)	6
13:15-13:20	진행성 담낭암에서 사람상피세포증식인자수용체2형(HER-2)의 예후 예측인자로서의 의의 및 향후 치료와의 연계성	**이승은**(중앙의대)	9
13:20-13:50	Invited Lecture	**나양원**(울산의대)	
	Bile acid의 Enterocirculation이 여러가지 질환에 미치는 영향	**황성순**(연세의대)	12
13:50-15:10	Optimal Surgical Extent for IPMN: Current Guideline and Clinical Practice	**김송철**(울산의대), **이우정**(연세의대)	
13:50-14:05	Current international consensus guideline	**장진영**(서울의대)	16
14:05-15:10	Case Presentation	**신상현**(성균관의대)	18
	1) Multifocal IPMN		
	2) Main duct type IPMN		
	3) How to interpret the resection margin and treat		
	Panel Discussion	**김상걸**(경북의대), **장진영**(서울의대), **민석기**(이화의대), **강창무**(연세의대)	
15:10-15:30	Coffee Break		
15:30-16:50	Dilemmas in Treating Postoperative Complications of Pancreatectomy Part 1: Intra-abdominal fluid collection & POPF	**최성호**(성균관의대), **윤성수**(영남의대)	
15:30-15:45	Changing concept of POPF	**김희준**(전남의대)	46
15:45-16:50	Case Presentation	**이우형**(울산의대)	57
	1) Criteria of PCD insertion for intra-abdominal fluid collection		
	2) Which kind of conservative management for POPF		
	3) Timing of drain removal		
	4) Management for long-standing POPF: internal drainage, fistula tract embolization, etc		
	Panel Discussion	**한성식**(국립암센터), **황대욱**(울산의대), **서형일**(부산의대), **양재도**(전북의대)	
16:50-17:30	Case Discussion	**윤유석**(서울의대), **윤명희**(부산의대)	
16:50-17:00	Malignant paraganglioma mimicking pancreatic malignancy	삼성서울병원 **김나루**	
17:00-17:10	A case of multiple recurrence in liver and peritoneum after distal pancreatectomy with splenectomy for SPN	서울아산병원 **황경연**	
17:10-17:20	Complete remission after palliative chemotherapy for metastatic pancreatic cancer	서울아산병원 **이종우**	
17:20-17:30	Endoscopic lavage in pancreatic cyst	서울대병원 **변윤형**	
17:30-17:40	연구제안: SMA First Approach for Pancreatic Head Cancer	**한성식**(국립암센터)	
17:40-17:50	한국미세침습췌장수술연구회(MIPS)소개	**김송철**(울산의대)	
17:50-18:00	공지사항 및 폐회사	KPSC 회장 **최인석**	

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KPSC 연구기금 수상자 발표 및 계획

Moderator

이현국 (이화의대)

Curriculum Vitae

김홍범

서울의대

학 력

2001-2007	중앙대학교 의학부 학사
2014-2016	중앙대학교 대학원 의학과 석사
2017-2019	서울대학교 대학원 의학과 박사 수료

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2015-2016	서울대학교병원 간담췌외과 전임의
2016-2018	동국대학교 일산병원 외과 임상조교수
2018-2018	서울대학교병원 외과 진료교수
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대한내시경복강경외과학회 평생회원
대한외과대사영양학회 평생회원
대한종양외과학회 평생회원
간담췌외과학회 종양등록위원회 (KOTUS 관리자)
간담췌외과학회 정보위원회
간담췌외과학회 연구위원회
간담췌외과학회 보험위원회

한국 가족성 췌장암의 유병률과 임상 양상 분석

김홍범 서울의대

췌장암 생존율을 높일 수 있는 가장 좋은 방법은 치료 가능한 조기 췌장암을 발견 하는 것이다. 하지만 췌장암은 특징적인 증상이나 징후가 없기 때문에 조기 발견을 위해서는 위험인자를 갖고 있는 환자들이 영상의학적 검사를 실시할 수 밖에 없다. 췌장암 위험인자로는 흡연, 당뇨, 만성 췌장염뿐만 아니라, 췌장암 가족력도 위험인자로 알려져 있다. 다른 위험인자들은 생활 습관과 환경과 연관되어 있는 후천적인 요인이라 한다면, 췌장암 가족력은 선천적인 요인으로서 췌장암 조기 검진 대상자로 가족력이 있는 사람들을 고려할 수 있다. 가족력이 있는 췌장암을 연구하기 위해서는 가족성 췌장암에 대한 현황 파악을 최우선으로 실행해야 한다.

다양한 암에서 가족력은 암의 위험인자로 알려져 있고, 췌장암 역시 가족력이 있을 경우 발병 위험도가 증가한다고 알려져 있고 문헌 보고에 따르면 가족 중에 췌장암 환자가 있는 경우 승산비 (오즈비, odds ratio) 는 5.3, 상대위험도 (relative risk) 는 1.5-1.7까지 증가한다고 알려져 있다. 가족성 췌장암은, 1차 혈족 관계 (first degree relative)의 가족 구성원 중 2명 이상의 췌장암 환자가 있을 경우 정의할 수 있고 전체 췌장암 환자의 4%-10%를 차지한다고 알려져 있다.

가족성 췌장암에 대한 대표적인 연구는 The National Familial Pancreas Tumor Registry (NFPTR)으로 1994년 미국의 Johns Hopkins University 에서 시작되어, 현재는 24개 기관이 참여하고 있으며, 아시아에서는 일본이 참여하고 있다. NFPTR은 다양한 심포지움과 컨소시움을 통하여 췌장암 환자와 가족으로부터 정보를 수집하고, 고위험군에게 검진 방법을 추천해주어 췌장암을 조기 발견할 수 있게 하여 궁극적으로는 췌장암 생존율을 높이는 것을 목표로 하고 있다.

컨소시움이 형성된 해외에 비하여 가족성 췌장암의 현황 및 임상양상에 관한 국내 연구는 미비하다. 다만 BRCA mutation 의 빈도를 알아보는 연구에서 가족성 췌장암으로 정의할 수 있는 환자는 110명 중 8명 7.2% 였고, 산발성 췌장암와 차이를 보이는 임상적 특징은 없었다.

따라서 가족성 췌장암의 국내 현황을 파악하기 위하여, 대규모 단일 기관인 서울대학교병원 췌장암 환자의 가족력을 파악하고자 한다. 2007년부터 서울대학교병원에서 수술받은 췌장암환자 약 1000여명의 전자의무기록에 등록된 가족력 및 1차혈족 관계 가계도를 통하여 췌장암 가족력을 파악하여 가족성 췌장암 빈도를 알아보고 같은 기간 내의 산발성 췌장암 환자와의 비교를 통하여 가족성 췌장암의 임상 특징을 분석한다. 이 연구를 바탕으로 추후 대규모 국가규모의 대규모 연구를 진행할 때 유용한 참고 자료가 될 수 있다.

Curriculum Vitae

이승은

중앙의대

경 력

2008-2010	서울대학교병원 임상조교수
2010-2014	중앙대학교병원 조교수
2014-현재	중앙대학교병원 부교수

KPSC 연구기금 수상자 발표 및 계획

진행성 담낭암에서 사람상피세포증식인자수용체2(HER-2)의 예후 예측인자로서의 의의 및 향후 치료와의 연계성

이승은 중앙의대

담낭암은 담도계에서 발생하는 가장 흔한 암이기는 하지만 전체 암종 중에서는 발생율 20위로 비교적 드문 암으로 알려져 있다. 담낭암은 전형적인 증상이 없어서 조기 진단이 매우 어렵고 따라서 진단 당시 이미 전신 전이 등 상당히 진행된 경우에 발견되는 경우가 많다. 그러나 담낭암이 매우 드문 암인데다가 담낭암만을 대상으로 한 연구가 매우 제한적이어서 이런 경우에 항암 치료의 효과는 잘 알려져 있지 않으며 표적 항암제의 효과에 대해서도 아직 이렇다 할 연구가 없는 상태이다. 따라서 담낭암의 예후를 향상시키기 위해서는 효과적인 치료 표적을 찾아내는 것이 매우 중요하다고 하겠다.

HER-2/neu유전자는 인체의 17번 염색체 장완(17q21)에 위치하는 원종양 유전자(proto-oncogene)로서 185KDa 크기의 당단백으로 HER-1, HER-3, HER-4와 함께 세포막 성장인자 수용체 단백질 그룹을 이루며 세포내 신호전달 체계에서 신호의 증폭에 중요한 역할을 한다. HER2는 여러 암의 발생에 관여함이 이미 널리 알려져 있고 담낭암과의 연관성에 대한 연구도 소수 발표되었다. 이러한 HER2 과발현과 담낭암과의 연관성에 대한 연구를 바탕으로 하여 최근 HER2 양성인 담낭암 환자의 경우 HER2 단일 클론 항체인 trastuzumab 에 좋은 반응을 보인다는 연구들이 발표되었고 이후 비로소 미국에서 HER2 양성 담낭암 환자를 대상으로 한 trastuzumab 치료 2상 임상 연구가 시작되었다. 그러나 안타깝게도 이 연구는 연구 참여자수 부족으로 조기 종료되었다. 따라서 이런 문제를 해결하기 위해서는 우리나라를 비롯한 담낭암 호발 국가 주도 하에 HER2양성 담낭암 환자에서 HER2 단일 클론 항체를 이용한 임상 시험이 필요하다고 하겠다. 그러나 이를 시행하기에 앞서 담낭암 환자에서 HER2 발현 정도와 이것의 임상적 의의, 예후 예측인자로서의 의미를 검토하는 일이 선행되어야 할 것이다.

본 연구에서는 진행성 담낭암 환자에서 HER2 과발현 정도를 살펴보고 HER2 과발현이 담낭암의 예후 예측 인자로서 의미가 있는지 분석하여 향후 HER2 단일 클론 항체를 이용한 담낭암 치료 임상 연구의 근거를 마련하고자 한다.

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Invited Lecture

Moderator

나양원 (울산의대)

Curriculum Vitae

황성순

연세의대

학 력

1995~1999	서울대학교 분자생물학과 이학학사
2003~2008	University of Illinois at Urbana-Champaign, 이학박사

경 력

2008~2015	Salk Institute, 박사후 연구원
2015~2017	세종대학교 바이오융합공학과 조교수
2017~현재	연세대학교 의과대학 의생명과학부 조교수

학회활동

한국세포분자생물학회 정회원
생화학분자생물학회 대의원
EMM 편집위원

Bile acid의 Enterocirculation이 여러가지 질환에 미치는 영향

황성순 연세의대

Comparisons of the gut microbiome of lean and obese animal, as well as those of lean and obese human individuals have revealed that obesity is associated with gut microbiome changes by numerous environmental factors, such as high fat diet. Here we report the human gut microbial composition in a population sample of 49 non-obese and 50 obese Korean individuals. We find that only a few bacterial species are sufficient to distinguish between individuals with various metabolic markers. The abundance of these bacteria is markedly reduced with increase of adiposity, BMI, WC, blood TG, and fatty liver while microbial gene richness has not been markedly changed between individuals. Animal studies reveal that oral treatment of those species enhances bile acid metabolism and increases OXPHOS in adipose tissues to protect against diet-induced obesity. Using comprehensive genomic analysis, we have noticed that gene expression profile of effector bacteria is strain-specific that contributes to differential metabolic responses in animal models. Furthermore, we have revealed that carbohydrate metabolic process is effector strain-specific to prevent against diet-induced obesity. Our findings clearly support that strain-specific metabolic processes of microbiota are responsible for host metabolic homeostasis to prevent against diet-induced obesity.

한국췌장외과연구회
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Optimal Surgical Extent for IPMN: Current Guideline and Clinical Practice

Moderator

김송철 (울산의대), **이우정** (연세의대)

Curriculum Vitae

장진영

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	서울대병원 인턴, 레지던트, 전임의
2002-2012	서울대병원 조교수-부교수
2008	미국 MD Anderson Cancer Center 교환교수
2013	서울의대 정교수
2015	서울대병원 암병원 진료부장

학회활동

IHPBA 학술위원, IAP 학술위원
IAP 췌장암 전구병변 가이드라인 제정위원
APHPBA 2019 학술 위원장

Current international consensus guideline

장진영 서울의대

Curriculum Vitae

신상현

성균관의대

학 력

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Case Presentation

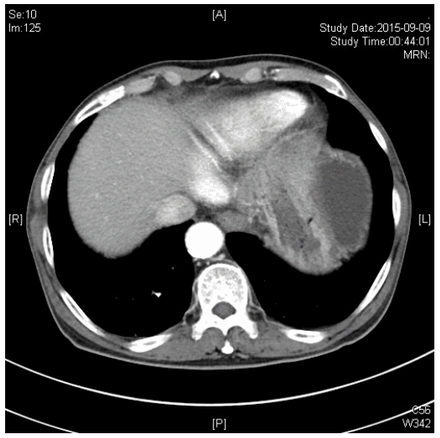
신상현 성균관의대

Multifocal IPMN

Case 1 (M/71)

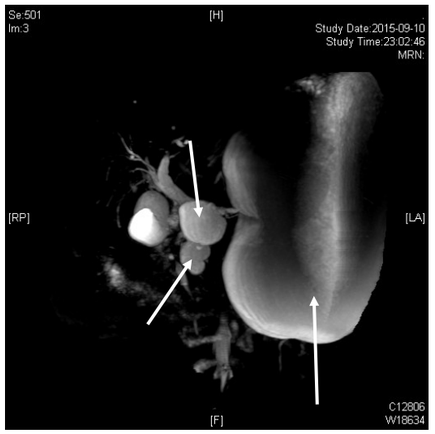
- pancreatic cyst detected during work-up for abdominal pain
- Underlying disease:
  - Atrial fibrillation (on medication)
  - Hypertension(on medication)
  - BPH (on medication)
- Lab findings:
  - CA 19-9: 36.98 U/ml
  - Mild decreased GFR (54.3 ml/min)

CT



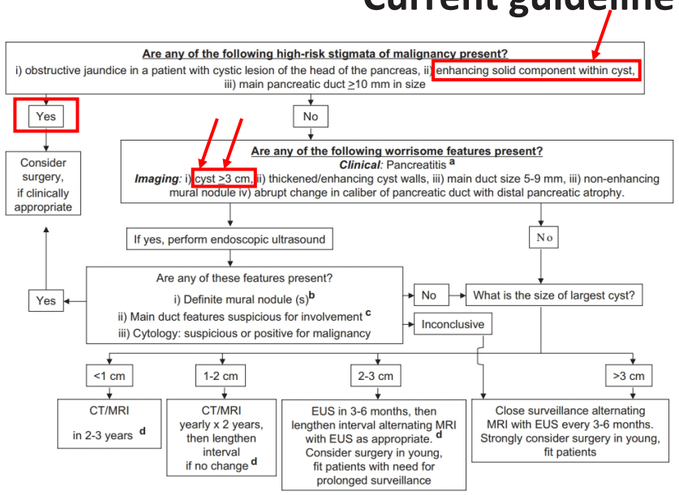
- Multiple (x4) pancreatic cysts.
  - largest cyst in the tail: 19.4 cm
  - Head & neck: 2.5 cm, 3.5 cm, 1.3 cm
- Preoperative diagnosis
  - R/O MCN (malignancy cannot be excluded)
  - IPMN

MRI



- Cyst in the tail: 19.4 cm
  - 1.5 cm-sized subtle enhancing nodule
- Cysts in the head & neck: 2.5 cm, 3.5 cm
  - suspicious minimal septum
  - no solid enhancing component

Current guideline



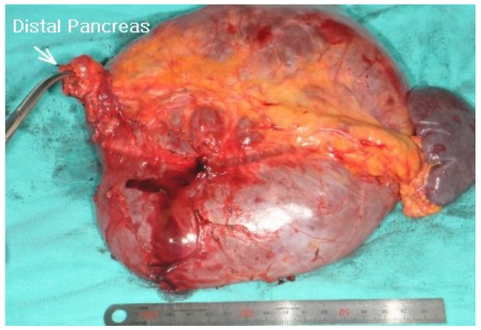
Cysts	High-risk stigmata	Worrisome features
Tail (19.4 cm)	• Enhancing nodule	• Size> 3cm
Neck (3.5 cm)	(-)	• Size> 3cm
Neck (1.3 cm)	(-)	(-)
Head (2.5cm)	(-)	(-)

Clinical question #1

- What kind of operation?
  - A. Distal pancreatectomy including a cyst in the tail (with high-risk stigma)
  - B. Subtotal pancreatectomy including a cyst in the neck (with worrisome features)
  - C. Total pancreatectomy
  - D. Other:

Operation

- Distal pancreatectomy including a cyst in the tail and splenectomy
- Frozen biopsy for pancreatic resection margin: negative



Pathology

Intraductal papillary-mucinous neoplasm, **low-grade**
(1) Tumor site: pancreas body/tail
(2) Tumor size: 9x3x3 cm (marked cystic change)
(3) Tumor limited to the pancreas
(4) N0: No regional lymph node metastasis (0/2)
(5) Pancreas intraepithelial lesion in pancreatic resection **margin: low-grade** (PanIN-I)

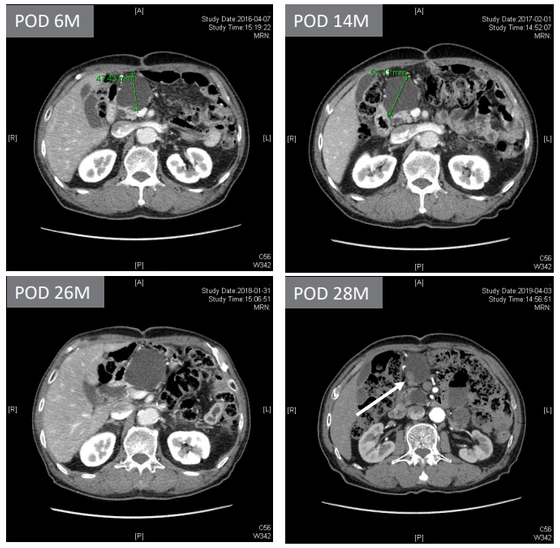
Clinical question #2

Postoperative Surveillance

- How frequent?
  - A. every 3 month
  - B. every 6 month
  - C. every 1 year
  - D. Other:
- How long?
  - A. 5 year
  - B. 10 year
  - C. Life long
  - D. Other:
- Which imaging modality?
  - A. CT
  - B. MRI
  - C. EUS
  - D. Other:

Follow-up CT

- Increasing size of a cyst in the neck
  - 3.5 cm → 6.1 cm
- Enhancing nodule (+)



Clinical question #3

- What is your treatment plan?
  - A. Surveillance
  - B. EUS-guided biopsy
  - C. Surgical resection without biopsy
  - D. Other:

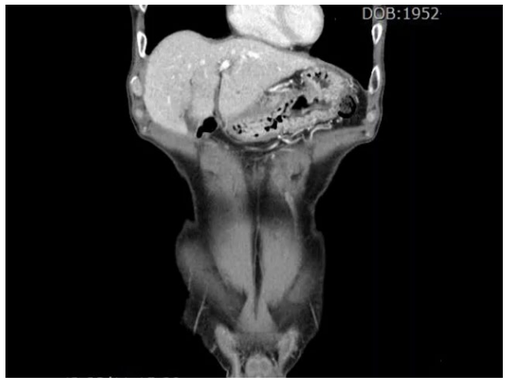
Clinical question #4

- What kind of operation if surgical resection is planned?
  - A. Segmental resection including a cyst in the neck if feasible
  - B. Completion total pancreatectomy
  - C. Other:

Case 2 (M/64)

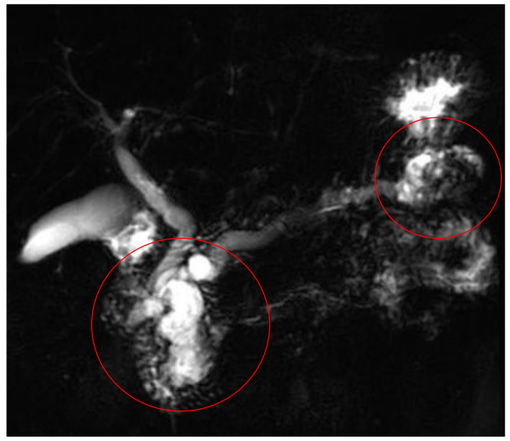
- pancreatic cyst detected during work-up for dyspepsia
- Underlying disease:
  - BPH (on medication)
- Lab findings:
  - CEA: 5.6 ng/ml
  - CA 19-9: 18.4 U/ml

CT



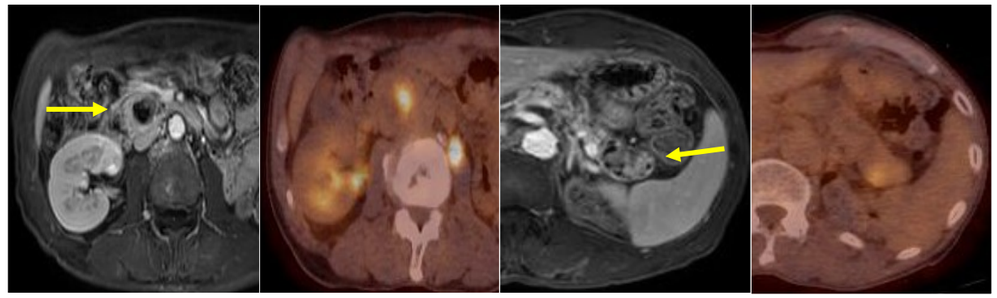
- Two cystic mass on pancreas
  - Head : 4.1 cm
  - Tail : 3.2 cm
  - Intracystic enhancing solid components
- Diffuse main p-duct dilatation
  - Maximum : 8mm

MR



- Two cystic mass with intracystic enhancing solid components at pancreas head **(3.5cm)** and tail tip **(3.7cm).**
- Main pancreatic duct dilatation **(8mm).**
- Small peripancreatic lymph nodes of pancreas head
- Preop Dx: Mixed type IPMN with invasive carcinoma at pancreas head and tail.

MR and PET-CT

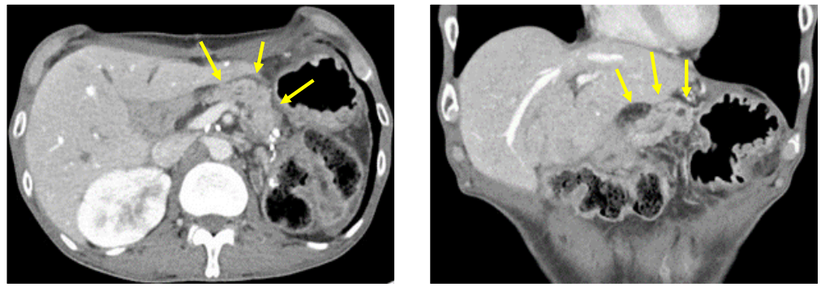


Clinical question #1

- What kind of operation?
  - A. Total pancreatectomy
  - B. Middle-segment–preserving pancreatectomy(MSPP)
  - C. Systemic chemotherapy
  - D. Other:

Operation

- Laparoscopic-assited middle-segment– preserving pancreatectomy (MSPP)



Pathology

P. Head

INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM associated with MUCINOUS CARCINOMA , involving main duct ("A")

1) Gross type: cystic
2) Location of tumor: head
3) Size of tumor : 2.5 x 1.5 x 1.0 cm
4) Extent of tumor: Tumor limited to the pancreas, more than 2 cm in greatest dimension **(T1)**
7) Surgical margins: free from tumor (safety margin: pancreatic neck: 1.5 cm, 8) Angiolymphatic invasion: not identified
9) Venous invasion: not identified
10) Perineural invasion: not identified
11) Tumor border: expanding
12) Stromal reaction: not identified
13) PanIN: present, multifocal, highest grade: 2
14) Chronic pancreatitis: absent
15)Lymph node: no metastasis in 12 lymph nodes

P. Tail

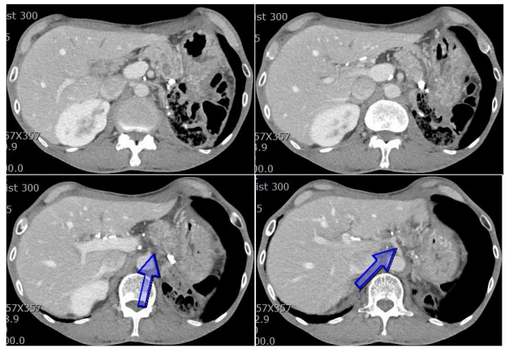
INTRADUCTAL PAPILLARY MUCINOUS NEOPLASM associated with MUCINOUS CARCINOMA , involving main duct (
1) Gross type: cystic
2) Location of tumor: tail
3) Size of tumor : 3.5 x 2.5 x 2.0 cm
4) Extent of tumor: Tumor extends beyond the pancreas but without involvement of the celiac axis or the superior mesenteric artery **(pT2)**
5) Splenic vein invasion: no invasion
6) Splenic artery invasion: no
7) Splenic parenchymal invasion : absent
8) Surgical margins: free from tumor (safety margin: pancreatic neck: 1.0 cm)
9) Angiolymphatic invasion: not identified
10) Venous invasion: not identified
11) Perineural invasion: present
12) Tumor border: infiltrative
13) Stromal reaction: not identified
14) PanIN: present, multifocal, highest grade: 2
15) Chronic pancreatitis: absent
16) Lymph node: no metastasis in 29 lymph nodes **(pN0)**

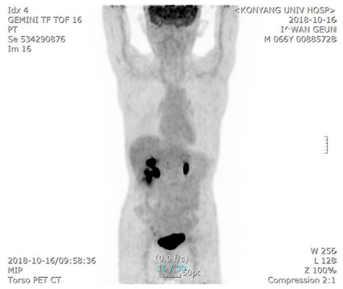
Clinical question #2

- What is your next plan?
  - A. Surveillance without adjuvant treatment
  - B. Chemotherapy
  - C. Chemo-radiotherapy
  - D. Other:

POD #19month ; PDCA Recurrence

Sx ; free, wt loss(-)
New onset DM ; Insulin tx
Ca19-9; 91.6ng/dl



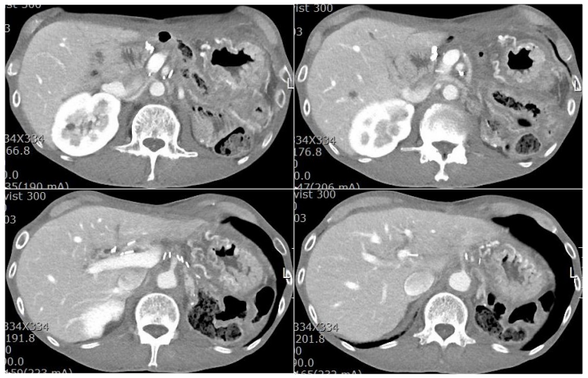


2nd Operation (POD#19month)

- Total pancreatectomy

  Invasive carcinoma with IPMN
  1. Size; 5.5x4x2.5cm
  2. Resection margin ; free
  3. LN metastasis ; 2/15
  4. Vascular invasion
  5. Lymphatic invasion

- CCRT+ Systemic chemotherapy (GEM-CIS)
- POD #3 month; no recurrence



Main duct type IPMN

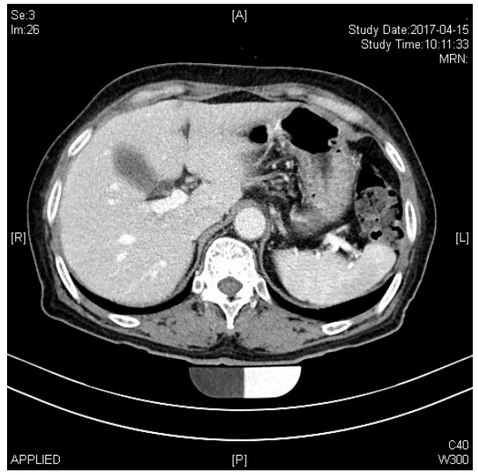
Case 3 (F/70)

- pancreatic cyst detected during work-up for elevated level of CEA

- 10 kg weight loss for 1 year
- Underlying disease?

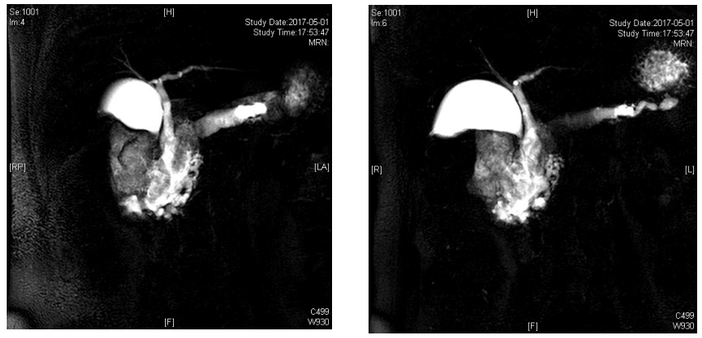
- Lab findings:
  - CA 19-9: 19.71 U/ml
  - CEA: 7.68 ng/ml
  - Fasting glucose: 131 mg/dl

CT



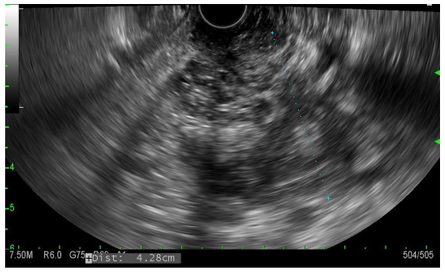
- 7.7 cm sized cystic mass in the pancreas head in communication with main p-duct
- Dilated main p-duct: max. 2.6 cm

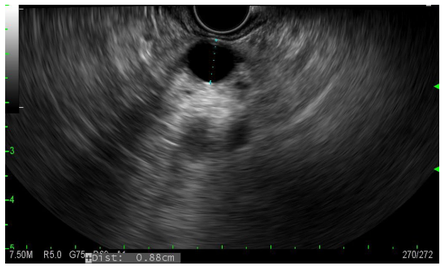
MR



- A 7.7 cm cystic mass in pancreas head with main p-duct dilatation.
- Enhancing mural nodules in the cystic lesion and dilated main p-duct
  → Mixed type IPMN with high risk stigmata.

EUS





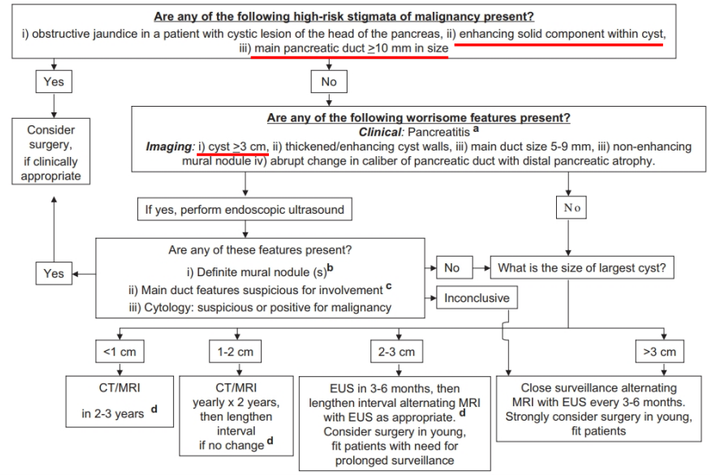
- 6cm sized irregular shaped cystic and solid mass in head
- Communicated with MPD
- MPD was diffuse dilated also in **body and tail without intraductal mass.**
- Duodenal papilla was patulous and mucin spilled out. Several fistulas was seen in the duodenum.

FNA

- Biopsy: inadequate for evaluation
- Cytology: ABNORMAL CELLS
  Intraductal Papillary-Mucinous Neoplasm
  (low-to-intermediate grade)

Current guideline

Mixed IPMN with high-risk stigmata (x2) and worrisome features (x2)



Pancreatic cancer IPMN malignancy & invasiveness calculator

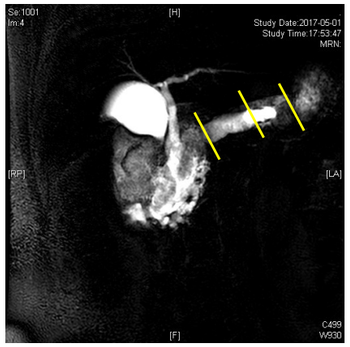
Clinical question #1

- What is your plan?
  - A. Short-term surveillance
  - B. EUS re-biopsy
  - C. Surgical resection
  - D. Other:

Clinical question #2

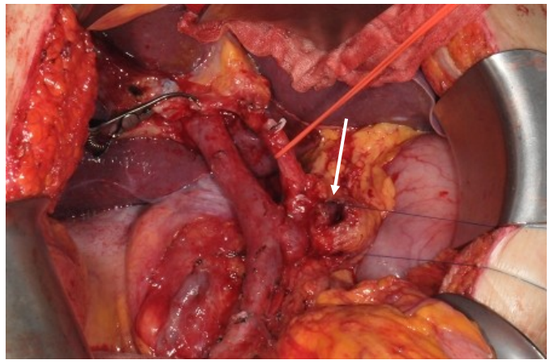
- What kind of operation?
  - A. PPPD & intraoperative evaluation of resection margin by frozen biopsy
  - B. Total pancreatectomy
  - C. Other:

- If A, in which finding of resection margin, will you finish operation?
  - A. Negative
  - B. Low-grade dysplasia
  - C. Intermediate-grade dysplasia
  - D. High-grade dysplasia



Operation

- PPPD
- Frozen biopsy of pancreas resection margin: negative



Pathology

Invasive colloid carcinoma, arising from IPMN, high grade dysplasia

(1) Tumor site: pancreas head

(2) Tumor size: 7x6 cm

(3) Histologic type of invasive carcinoma: intestinal

(4) T3: Tumor extends beyond the pancreas to duodenum mucosa (fistulous growth)

(5) N0: No regional lymph node metastasis (0/19)

(6) cM0 : Clinically No distant Metastasis

(7) Margin Status

- Pancreas neck **margin: high-grade dysplasia**
- Common bile duct, retroperitoneal resection margins: negative

Clinical question #3

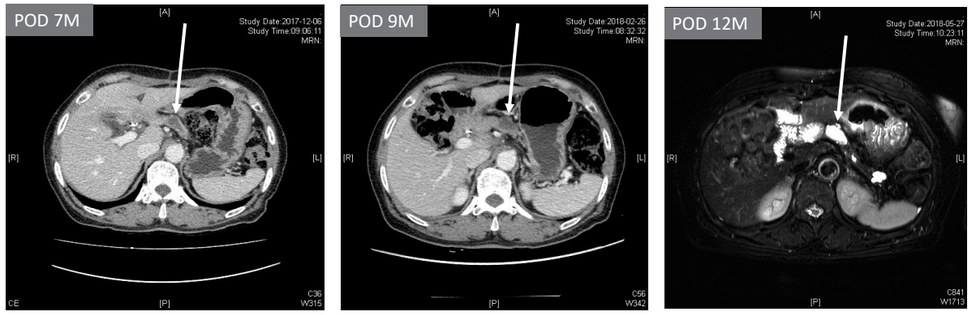
- What is your next plan?
  - A. Surveillance without adjuvant treatment
  - B. Chemotherapy
  - C. Chemo-radiotherapy
  - D. Reoperation for further resection
  - E. Other:

Clinical question #4

Postoperative Surveillance

- How frequent?
  - A. every 3 month
  - B. every 6 month
  - C. every 1 year
  - D. Other:
- How long?
  - A. 5 year
  - B. 10 year
  - C. Life long
  - D. Other:
- Which imaging modality?
  - A. CT
  - B. MRI
  - C. EUS
  - D. Other:

Postoperative F/U

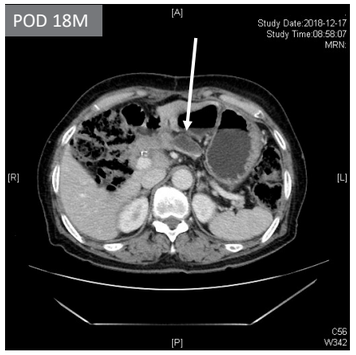


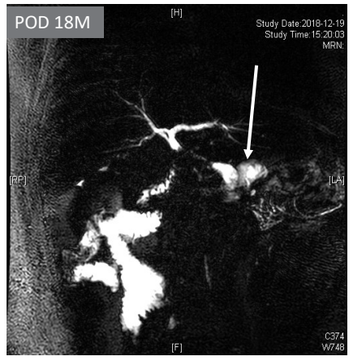
Recurrent main duct IPMN/colloid carcinoma vs.
Anastomosis site p-duct stricture/dilatation

Clinical question #5

- What is your next plan?
  - A. Short-term follow-up
  - B. EUS
  - C. Surgical resection
  - D. Other:

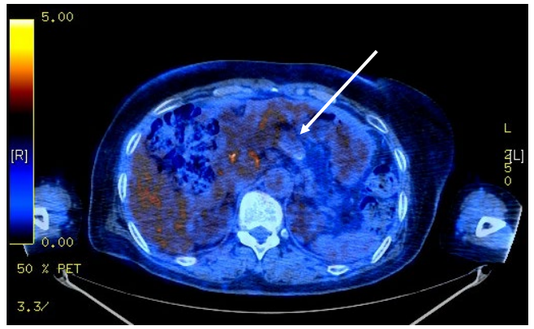
Further F/U





Progressed localized dilatation of main pancreatic duct with **irregular ductal wall thickening**: Tumor **recurrence, more likely** rather than anastomotic stricture.

PET



mild FDG uptake (SUVmax = 3.1)

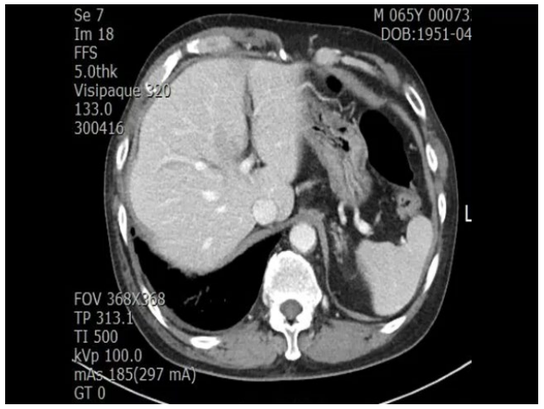
Clinical question #6

- What is your plan?
  - A. Short-term follow-up
  - B. EUS biopsy
  - C. Surgical resection
  - D. Other:

Case 4 (M/66)

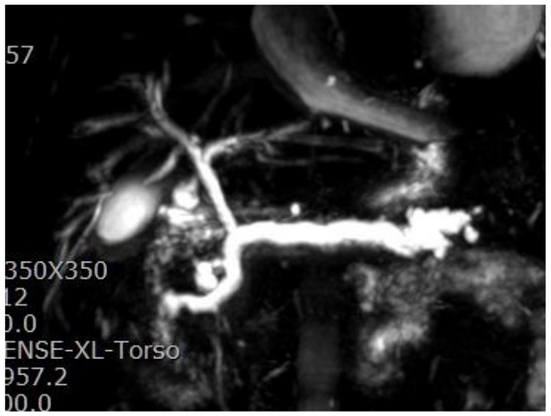
- pancreatic cyst detected during medical checkup
- Past medical history
  - Lung cancer (18YA)
  - Laryngeal cancer (5YA)
- Lab findings:
  - CEA : 3.7 ng/mL
  - CA 19-9: 14.6 U/mL

CT



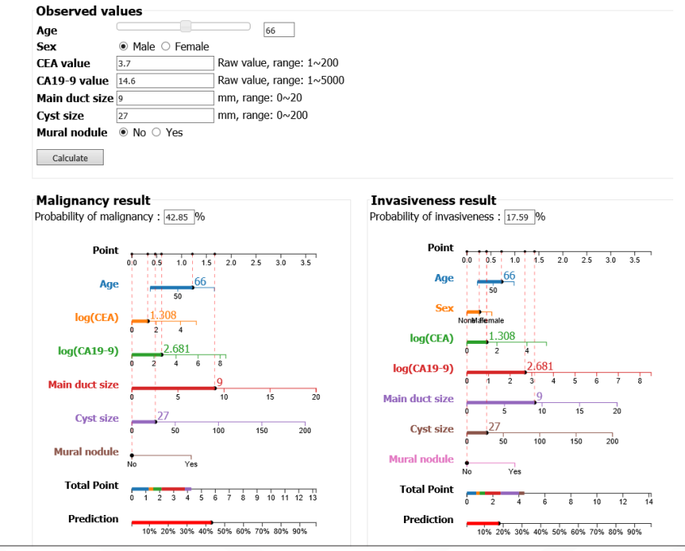
- Diffuse dilatation of main p-duct
  - Maximum size : 9 mm
  - Without obstructive lesion
  - No intraductal lesion

MR



Pancreas tail IPMN 5 cm
Pancreas neck IPMN 2.3 cm
No moral nodule
Main duct ; 6-9mm

Pancreatic cancer IPMN malignancy & invasiveness calculator

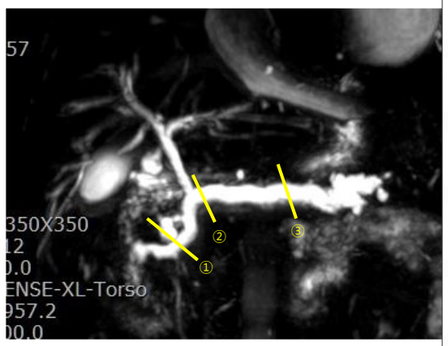


Clinical question #1

- What is your treatment plan?
  - A. Surveillance
  - B. Surgical resection
  - C. Other:

Clinical question #2

- If surgical resection is planned, which level of resection in case of partial pancreatectomy?



- What kind of operation?
  - A. Distal pancreatectomy with splenectomy
  - B. Spleen preserving distal pancreatectomy
  - C. Total pancreatectomy
  - D. Other:

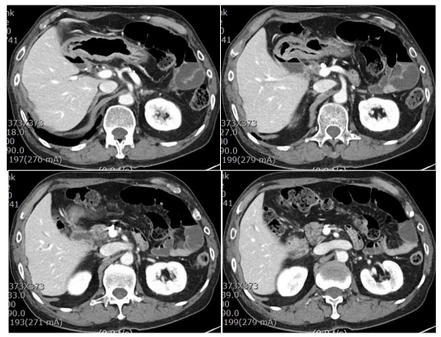
Operation

- ❖ Resection level: ②
- ❖ Laparoscopic spleen preserving distal pancreatectomy

Pathology

IPMN with high grade dysplasia
1. Site ; pancreas tail
2. Size; 5.3x1.5x1.5
3. Mixed type
4. Resection margin ; Low grade dysplasia(Pan IN 1B)
5. LN ; negative 0/1

POD# 24 month



2cm BD IPMN, No moral nodule

Pathology

Operation: Laparoscopic spleen preserving DP

IPMN with high grade dysplasia

1.Site ; pancreas tail

2.Size; 5.3x1.5x1.5

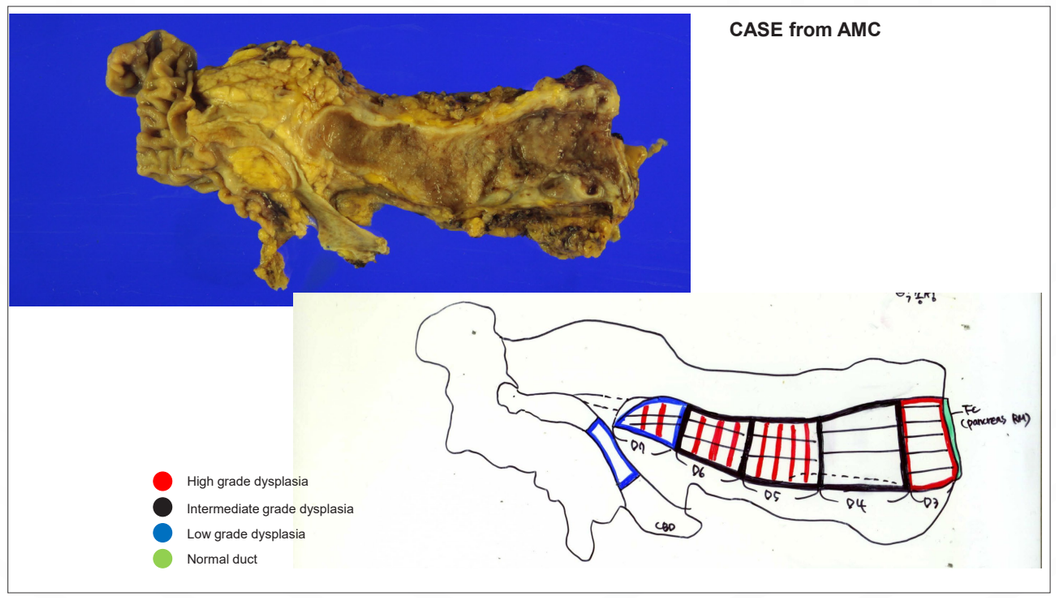
3.Mixed type

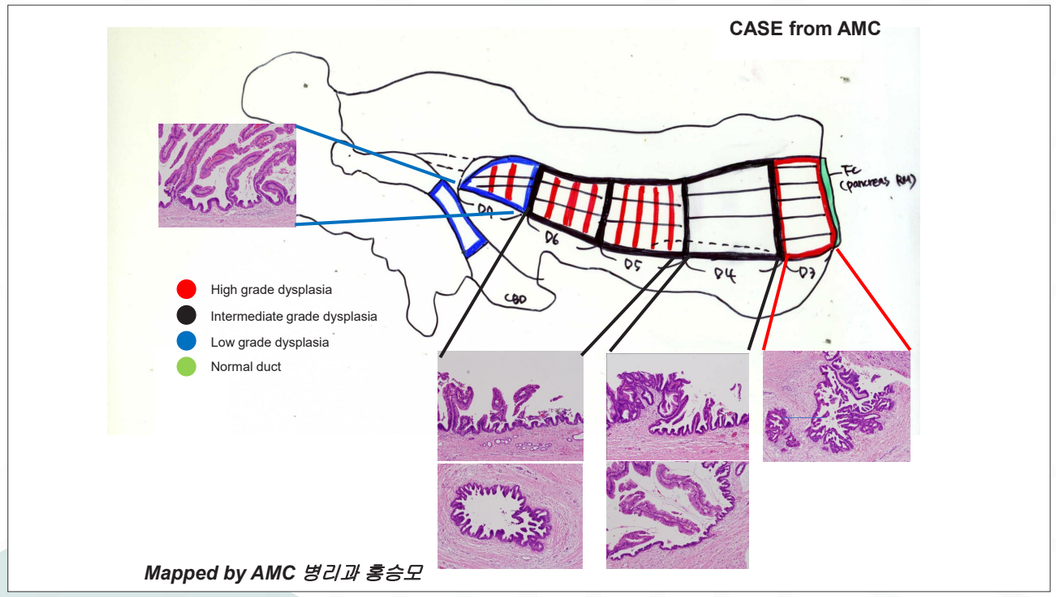
4.Resection margin ; Low grade dysplasia(Pan IN 1B)

5.LN ; negative 0/1

How to interpret the resection margin and treat

Discussion





KPSC Since 2004 한국췌장외과연구회 Korean Pancreas Surgery Club

제60차 한국췌장외과연구회
- KAHBPS 부울경지회 심포지움

Dilemmas in Treating Postoperative Complications of Pancreatectomy Part 1: Intra-abdominal fluid collection & POPF

Moderator

최성호 (성균관의대), **윤성수** (영남의대)

Curriculum Vitae

김희준

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학 력

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Changing concept of POPF

김희준 전남의대

Introduction

Postoperative pancreatic fistula (POPF) is a main cause of morbidity and mortality after pancreatic resection. POPF is associated with major morbidity including intra-abdominal sepsis and post-pancreatectomy hemorrhage (PPH), carrying a mortality risk of 1% of all POPF and 25% of grade C POPF.[1] In this session, we will discuss the change concepts in the understanding of POPF pathophysiology and management.

Changed definition of POPF

The consensus definition of POPF was reviesed in 2016 mainly to restrict the definition of POPF to only those that were "associated with a clinically relevant development/consideration related directly to the postoperativ pancredatic fistula" (previously defined grade B and C). A grade A POPF has been redefined the term "biochemical leak", as it does not affect the clinical course of the patient. Grade B and C were defined more specific to clarify the distinction between the two categories (table 1).[2]

Table 1 2017 ISGPF definitions and grades of postoperative pancreatic fistula[6]

Event	Biochemical leak	Grade B POPF	Grade C POPF
Drain amylase concentration >3× upper limit of normal serum value	Yes	Yes	Yes
Persisting peripancreatic drainage >3 weeks	No	Yes	Yes
Clinically relevant change in the management of POPF	No	Yes	Yes
Percutaneous or endoscopic drainage of POPF-associated collections	No	Yes	Yes
Angiographic procedures for POPF-associated bleeding	No	Yes	Yes
Reoperation for POPF	No	No	Yes
Signs of infection related to POPF	No	Yes (without organ failure)	Yes (with organ failure)
POPF-related organ failure	No	No	Yes
POPF-related death	No	No	Yes

Abbreviations: ISGPF, International Study Group on Pancreatic Fistula; POPF, postoperative pancreatic fistula.

Changing concepts in pathophysiology of POPF

Traditionally, pathophysiology of POPF was assumed tobe due to a gradual loss of mechanical integrity of the pancreatoenteric anastomosis leading to "leakage" of pancreatic fluid. Therefore, many surgical methods were described to ameliorate the POPF, such as reinforcement, fibrin selants, autologous tissue patches, bioabsorbable meshes, and various methods of pancreatoeneteric anastomosis. However, despite these strategies, the rates of POPF have not significantly diminished.

Recently, relationship between infection and POPF have been reported. Yamashita et al have reported that pseudomonas aeruginosa activate trypsinogen to typsin, and the activated protease is correlated to development and severity of POPF.[3] Yang et al also have been reported that positive drain culture after PD was associated with higher incidence of POPF. Therefore, infection could be a major factor associated with development and severity of POPF.[4]

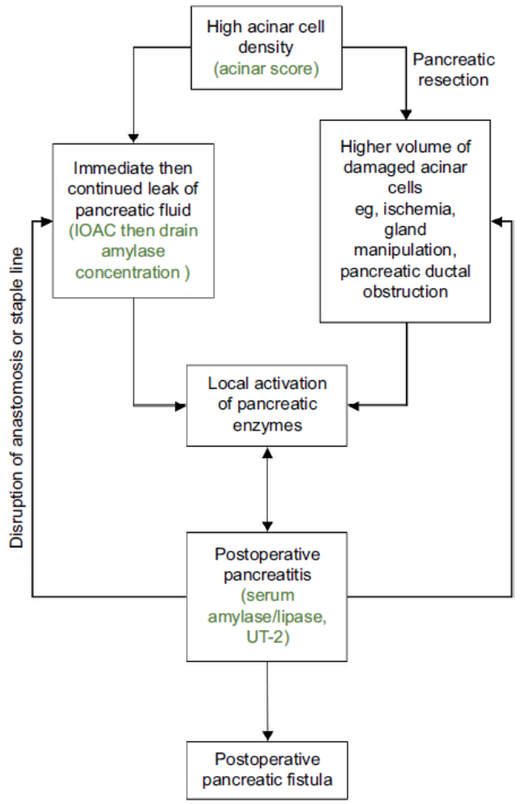

Figure 1 Hypothesized mechanism for the development of postoperative pancreatitis and POPF.
Abbreviations: IOAC, intraoperative amylase concentration; UT-2, urinary trypsinogen-2; POPF, postoperative pancreatic fistula.

Reuver et al[5] and Nahm et al[6] demonstrated that high intraopeative amylase concentration (IOAC) is highly predictive of the development of POPF. These data suggested for the first time that the underlying pathophysiological events that lead to the eventual recognition of a POPF as per the ISGPF definition occur at the time of pancreatic resection. The density of acinar cells at the pancreas resection margin has been demonstrated to correlate with the IOAC and the development of postoperative pancreatitis (POP, as measured by urinary trypsinogen-2 and serum amylase/lipase on POD 1). The IOAC and postoperative pancreatitis are, in turn, strongly associated with the development of POPF.[7] The interaction between IOAC, acinar cell density, POP, and POPF has yet to be definitevely elucidated; however, the authors have hypothesized a potental mechanism (Figure 1) whereby high-risk pancreata with a high acinar cell density are prone to both immediate leakage of protease-rich pancreatic fluid (IOAC) and the development of pancreatitis in the remnant gland as aresult of ischemia and/or glandular manipulation. Several studies have suggested that focal ischemia may be involved with development of POP. Ansorge et al[8] have demonstrated that there was a higher perianastomotic lactate/pyruvate ratio indicating local ischemia, and also significantly higher levels of perianastomotic TAP and plasma amylase, indicating that pancreatitis was associated with the development of POPF.

Prediction of POPF

Proponents of surgical drians cite controlled drainage of effluent, mitigation of the clinical severity of POPF, as well as early detection of POPF as motives to place a drain.[9] Others raise concern about ascending infection as well as drain erosion leading to anastomotic complications as reasons to omit drain placement.[10,11] Various strategies have been proposed to help predict the development of POPF, in orderto guide surgeons as to which patients to place a drain, and when to consider their early removal. The fistula risk scoring (FRS) system is the most widely used predictive tool (Table 2).[12] The factors most consistently shown to be predictive of POPF after PD include soft gland texture, non-PDAC, non-chronic pancreatitis pathology, small pancreatic duct

Table 2. Fistula Risk Scoring (FRS) system for the prediction of POPF.

Risk factor	Parameter	Points*
Gland texture	Firm	0
	Soft	2
Pathology	PDAC or chronic pancreatitis	0
	Ampullary, duodenal, cystic, islet cell, etc	1
Pancreatic duct diameter	≥5 mm	0
	4 mm	1
	3 mm	2
	2 mm	3
	≤1 mm	4
Intraoperative blood loss	≤400 mL	0
	401–700 mL	1
	701–1000 mL	2
	>1000 mL	3

Note: *Out of 10.
Abbreviation: PDAC, pancreatic ductal adenocarcinoma.

diameter (<3 mm), and high intraoperative blood loss (>1000 mL). In a muti-institutional validation study of the FRS evaluating 594 patients who underwent PD, the incidence of grade B/C POPF was 6.6% for low risk, 12.9% for moderate risk, and 28.1% for high-risk patients.[13] McMillan et al. advocate for omission of prophylactic drain placement in patients deemed to be negligible or low risk as calculated by the FRS.[14,15]

Table 3. FRS zones and probability of POPF after PD - Results from a multi-institutional validation study of 594 PD patients.

FRS points (out of 10)	Risk zone	Risk of POPF* (%)
0	Negligible	**
1–2	Low	6.6
3–6	Moderate	12.9
7–10	High	28.1

Notes: *Clinically relevant. **No patients in this validation cohort were of negligible risk.

Abbreviations: FRS, Fistula Risk Score; PD, pancreaticoduodenectomy; POPF, postoperative pancreatic fistula.

More recently, many studies, including the aforementioned publication, discuss the value of postoperative day 1 drain amylase (POD1DA) as a predictive factor for POPF.[16,17] In a systematic review, Liu et al. have reported 0.85 of sensitivity and 0.80 of specificity of POD1DA for all POPF, and 0.70 of sensitivity and 0.88 of specificity for CR-POPF.[17] Bertens et al. compared the predictive value of FRS and POD1DA, and they concluded that FRS and POD1DA are equally accurate in predicting CR-POPF.[18] FRS and POD1DA has been used to direct early drain removal by POD3~5 by predicting the development of CR-POPF. McMillan et al. recommended to omit drain in negligible/low risk patients by FRS, and early removal of drain (on POD3) in moderate/high risk group, if POD1DA was lower than 5,000IU/L.[15]

Efforts to overcome POPF

Anastomosis

In the largest multicenter RCT to date of PG versus PJ during PD (RECONPANC study), there was no significant difference in the rate of POPF (20% vs 22%, p=0.617).[19] Two recent RCTs comparing duct-to-mucosa vs invagination PJ revealed no significant difference in the rate of POPF between the two techniques.[20,21]

Suture materials for capsule-to-bowel layer

Several studies comparing polydioxanones (PDO) suture versus polyester (PE) sutures for capsule-to-bowel layer demonstrated that the use of PE suture for PJ is associated with a significant reduction of CR-POPF(PE vs PDO, 16.7% vs 83.3% in FRS high risk zone, p<0.01).[22,23]

Stent

Results of studies evaluated the impact of stents to POPF were heterogenious. In a randomized multicenter trial, external stent was associated with a higher rate of clinically relevant POPF than internal stent.[24] In the other hand, a propensity score matched retropsective cohort analysis by Ecker et al of 522 patients with FRS high-risk group demonstrated a reduction in POPF rates with the use of external stents, and an increase in POPF rates with internal stents compared with no stents.[25] Some studies demonstrated an increased POPF rates with the use of internal stents especially in high-risk pancreata.[26,27] Several RCTs and on retrospective study with propensity score matched analysis have reported a reduction in POPF rates with the use of external stents.[25,28,29] However, a Cochrane review evaluating three RCTs comparing internal versus external stents failed to show superiority of one form of stent over the other in terms of POPF reduction.[30]

Drain

As aforementioned, drain is associted with ascending infection. In a RCT of 179 patients who underwent pancreatic resection, randomized to having a drian or no drain placed at the time of surgery, patients with a drain had a higher incidence of POPF, intra-abdominal abscess and/or collection.[10] Recently, the 2016 PANDRA trial evaluated 395 patients undergoing PD randomized to either receiving an intra-abdominal drain or not at the time of surgery. This demonstrated a significant reduction in POPF rate (5.9% vs 11.9%, p=0.030) and fistula-associated complications (13.0% vs 26.4%, p=0.0008) in patients who did not receive a surgical drain.[31] Patients who do receive an intra-abdominal drain may benefit from its early removal by reducing the risk of secondary infection.[11,14,15,32] It may be appropriate to remove drains early in patients who are at a low risk of POPF.

Somatostatin analogues

Octreotide binds to G-coupled somatostatin receptors, thus exerting an inhibitory action on both exocrine and endocrine functions of the pancreas. Becuase of this action, octreotide has long been used in pancreatic surgery. However, there has been no clear benefit demonstrated in the use of somatostatin analogs to prevent or treat POPF. Meta-analysis of RCTs about prophylactic use of somatostatin analogs demonstrated that somatostatin analogue did not improve the post-operative outcomes following PD.[33,34] Despite it, the use of smatostatin analogue is popular,

possibly as a result of a number of early single armed case series reporting a high rate of spontaneous pancreatic fistula closure.[35,36] A meta analysis of seven RCTs evaluating treatment effect of somatostatin analogues in established POPF showed that rates of POPF resolution were not increased with the use of somatostatin analgues.[37] In 2014 Allen et al. have reported that paresiotide decreased the rate of CR-POPF.[38] However, the result of following reserches for validation of this drug was heterogenous.[39-42]

Protease inhibitor

Two RCTs evaluating ulinastatin in PD demonstrated that ulinastatin reduced drain amylase level, postoperative pancreatitis, and POPF.[43,44]

Management of Grade B/C POPF

Where the patient remains clinically stabel, a "Step-up" approach to POPF management i usually acceptable. Where a CT scan has detected an intra-abdominal peripancreatic collection that is safely accessible by percutaneous route, radiologically guided drainage of such collections has been demonstrated to be both effective and safe.[45,46] If the fluid collection cannot be reached by percutaneous route, EUS may be utilized to drain these collections.[47] A match-controlled study, conducted by Al Efishat et al., demonstrated that comparable success rates and outcomes of endoscopic drainage of post-operative peripancreatic fluid collection, compared to percutanesou drainage.[48]

Conclusion

POPF is a complex problem. Understading the pathophysiology of POPF, evidence-based strategy, and multidisciplinary approach is mandatory to overcome this complex problem.

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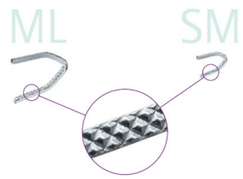

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with a unique jaw shape that reduces the need to use a separate dedicated dissecting instrument

Unparalleled strength

with a blade design that delivers more secure seals, even in the most challenging conditions

Optimal efficiency

from increased sealing speed, multi-functionality, and simplified steps for use

*Design Validation Study with surgeons (n=33) operating in simulated procedures in an animate porcine laboratory model. #051950-160425
†In a design validation study with surgeons (n=33) operating in simulated procedures in an animate porcine laboratory model (26/33) #053344-160516
‡In a pre-clinical study, for both iliac dissection and lymph node dissection, the HD 1000i was significantly superior to the predicate devices in dissecting capability ($p<0.001$ in all cases). #051950-160425
*In a pre-clinical study, 100% (56/56) of porcine blood vessels remained hemostatic over a 30-day survival period. #049339-160315
†In a benchtop study with 5-7 mm porcine carotid arteries that compared median burst pressure, HARMONIC® HD 1000i (1878 mmHg) vs. competitor product A (1224 mmHg) ($p<0.0001$). #049305-160315.
‡In a benchtop study with 5-7 mm porcine carotid arteries that compared median burst pressure, HARMONIC® HD 1000i (1878 mmHg) vs. competitor product B (1171 mmHg) ($p<0.0001$). #049315-160315
*In a porcine study comparing sealing times of HARMONIC ACE®+7 and HARMONIC® HD 1000i, HARMONIC® HD 1000i Shears transected vessels faster than HARMONIC ACE®+7 (mean vessel transection time of 9.186 vs 15.291). #051753-160420
†In a design validation study with surgeons (n=33) operating in simulated procedures in an animate porcine laboratory model (26/33) #053344-160515
‡Design Validation Study with surgeons (n=33) operating in simulated procedures in an animate porcine laboratory model (33/33) #053346-160516
§Seal reliability at 240 mmHg of 98.2% vs. 98.4% for HARMONIC ACE®+7 MIN button. Speed based on average time to transect 150 mm of porcine jejunum ($p=0.0000$). #050508-160401
||Device measurements based on a metrology study (median cut length of 18.87 mm vs. 14.56 mm). #050283-160329
#Based on average device tip grasping force (distal 5 mm of the jaw). #050295-160329

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Shaping the future of surgery