간담췌 질환에서 내시경의 역할

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Liver

Clinical manifestations of portal hypertension Gynecomastia Caput medusae Ascites Expandable stent -Hepatic vein Umbilical hernia Liver Edema (swelling) of legs Portal vein

Anatomy : The Portal Venous System







Cause of Portal Hypertension





Suprahepatic, hepatic, and infrahepatic



Development of Collaterals

Normal

- Systemic vein pressure > portal vein
- Systemic bed \rightarrow portal bed
- In portal HTN
 - Portal vein >systemic vein
 - Reversal of flow
 - To decompress the portal pressure
 - : angiogenesis, development of new collaterals, increase in the size of the collaterals, usually insufficient



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Pathophysiology: Gastroesophageal Varices

Development of varix

: HVPG at least 10 mmHg

Venous drainage of GEJ

- Gastric zone
- Palisade zone
- Perforating zone
- Truncal zone

Distal esophagus : coronary v. Fundus: short gastric v. \rightarrow splenic v.





Natural History and Epidemiology



- Variceal hemorrhage : 12% / year
 - 5% for small varices and 15% for large varices
 - Red wale marks and advanced liver disease

Recurrent variceal hemorrhage in 1 year : 60%

- 6 week mortality with bleeding episode: 15-20%
 - 0% in Child A patients, 30% in Child C patients

Diagnosis of Gastroesophageal Varices

- Esophagogastroduodenoscopy (EGD)
 - Gold standard
 - During withdrawal of the endoscope
 - Esophagus, maximally inflated, the stomach, completely aspirated
 - The size of the varices in the lower third of the esophagus is the most important

Diagnosis of Gastroesophageal Varices

EUS

- As good as EGD for detection of esophageal varices, but better than EGD to detect gastric varices
- To determine predictors for recurrence of varices after therapy
 - : presence and size of paraesophageal varices
- Echo-free or hypoechoic lumen in the esophageal submucosa



Diagnosis of Gastroesophageal Varices: EUS





Diagnosis of Gastroesophageal Varices

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- Portal hypertensive change: splenomegaly, reversal of flow in the portal vein, portosystemic collateral blood flow
- Portal vein diameter >13mm: presence of esophageal varices
- Transient elastography; liver stiffness correlate to the presence/ degree of esophageal varices
- СТ

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- Detection rate: 92% for large varices, 53%-60% for small varices
- MRI
 - Sensitivity of Gd-enhanced MRI: 81%





Classification of Esophageal Varices



- Several endoscopic classification systems for esophageal varices
 - Simple classification recommended by AASLD/EASL
 - Japanese Research Society for Portal Hypertension (JRSPH) system
 - Dagradi classification
 - Westaby classification

AASLD / EASL / APASL recommendation



- The size classification: as simple as possible
 - 2 grades : small (≤ 5mm) or large (>5mm)
 - 3 sizes
 - Small: minimally elevated veins above the mucosa
 - Medium: tortuous veins occupying less than 1/3 of the esophageal lumen
 - Large: occupying more than 1/3 of the lumen
- Presence or absence of red color signs

JRSPH system

- Six categories
 - Location (L)
 - Form (F)
 - Color (C)
 - Red color sign (RC)
 - Bleeding signs
 - Mucosal findings



Location



Ls

- Locus superior
- Upper part of the esophagus
- Lm

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- Locus medialis
- Middle part of the esophagus

- Locus inferior
- Lower part of the esophagus

Form



- F₀: no varicose appearance
- F₁: straight, small-caliber
- F₂: moderately enlarged, beaded
- F₃: markedly enlarged, nodular or tumor-shaped

Form



Color



Cw

- White varix
- Look like large folds of the esophageal mucosa
- Cb
 - Blue varix
 - Bluish-white or cyanotic, distended by blood
 - Cbv (violet), Cb-Th (thrombosed)





Red Color Signs

- Reddish changes immediately beneath the submucosa
- Reliable predictors of the risk of variceal bleeding
- Categories
 - Red wale markings (RWM)
 - : longitudinal dilated veins resembling whip marks
 - Cherry-red spots (CRS)
 - : small red spots on the mucosal surface (2-3mm)
 - Hematocystic spots (HCS)
 - : large (4mm or more), round, red projections look like blood blisters. blood coming from the deeper extrinsic veins straight out towards the lumen through a communicating vein into the superficial veins







Red Color Signs

Grade

- RC₀: absent
- RC₁: small in number and localized
- RC₂: intermediate between RC1 and RC3
- RC₃: large in number and circumferential



Red Color Signs

RC_o

226

RC₁(RWM)





tudy Date:2011-C Study Time:13:C



Bleeding Signs

- During bleeding / After hemostasis
- During bleeding
 - Gushing
 - Spurting
- After hemostasis
 - Red plug
 - White plug



Bleeding Signs







Mucosal Findings

- E : erosion
- Ulcer : Ul
- Scar : S



Classification of Gastric Varices - JRSPH

- Relation to the cardiac orifice
 - Lg-c: adjacent to the cardiac orifice
 - Lg-cf: extension from the cardiac orifice to the fornix
 - Lg-f: localized to the fornix
 - Lg-b: located in the body
 - Lg-a: located in the antrum
- Size : similar to esophageal varices
 - F₁,F₂,F₃

Description

- In the order of the six main categories
 - (L, F, C, RC, bleeding signs, and mucosal findings)
 - Esophageal varices with RWM and CRS
 - : Ls, F₃, Cb,RC₃ (RWM, CRS)
 - Spurting bleeding from EV
 - : Lm, F₂, Cb, RC₁ (CRS), spurting bleeding
 - Esophageal varices and fundic varices
 - : Ls, F_3 , Cb, RC₂(RWM, CRS), Lg-f, F_2 , RC₀
 - Spurting bleeding from GV extending from the cardiac orifice to the fornix: Lg-cf, F₃, spurting bleeding



Example

Lm, F2, Cb,RC2 (RWM, CRS)











Lm, F3, Cb, RC2 (RMW), spurting bleeding,S





Westaby classification



Grade I

- : Varices flush with the wall of the esophagus
- Garde II
 - : Varices protrude no more than half way to the center of the esophageal lumen
- Grade III

: Varices protrude more than half way to the center of the esophageal lumen

Classification of Gastric Varices – Sarin's

- Sarin's classification
 - GOV1: continuation of esophageal varices and extend for 2 to 5 cm below the GE junction along the LC side of the stomach
 - GOV2: continuation of esophageal varices and extend into the fundus
 - IGV1: isolated gastric varices located in the fundus
 - IGV2: isolated ectopic varices anywhere in the stomach



Classification of Gastric Varices

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- Small: less than 10mm in diameter
- Medium: 10 to 20 mm in diameter
- Large: large greater than 20mm in diameter

Gastric Varices

GOV2





Gastric Varices



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Ectpoic varix





Screening Policy

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- All patients with a diagnosis of cirrhosis should be screened for varices with an EGD
 - No varices, repeat EGD in 2-3 years
 - Small varices, repeat EGD in 1-2 years
 - EGD once a year in patients with decompensation
 - No EGD follow up in patients on β-blockers

Therapeutic Options





Risk Stratification of Esophageal Varices

NIEC (North Italian Endoscopy Club) Index



NEJM 1988;31:983-9

Therapies to Prevent the First Bleeding

- Pharmacological therapies
 - Nonselective β-blockers (NSB)
 - β₂ effect: splanchnic vasoconstriction, portal inflow↓
 - β₁ effect: cardiac output ↓, portal inflow↓
 - Propranolol or nadolol
- Endoscopic therapies
 - Endoscopic variceal ligation (EVL)

β -blockers : The evidences



589 Patients from 4 RCTs, NSB (286) vs Placebo (303)



NEJM 1991;19:475-505

Response Guided Therapy

HVPG and bleeding



- Propranolol
 - Starting dose of 20mg Bid
 - Increase to maximal tolerated dose until heart rate is 55/min
 - Indefinite duration
- Nadolol
 - Starting dose of 40mg daily



β-blockers : Side Effects



- Common adverse effects
 - Dizziness
 - Breathlessness
 - Fatigue
- Contraindication
 - Reactive airway disease
 - Peripheral vascular disease
 - DM

Endoscopic Variceal Ligation



- First introduced in 1989
- The blood flow is completely interrupted, producing ischemic necrosis of the mucosa and submucosa
- Granulation takes place, leaving shallow ulcerations that heal in 14 to 21 days
- EVL sessions are repeated monthly interval until eradication
- For large varices

EVL: The Evidences



Meta-Analysis of EVL vs No Treatment (5 trials, 601 subjects)

Outcomes*	RR (CI)	RR Reduction	NNT (CI)
First esophageal variceal bleed	0.36 (0.26-0.50)	64%	4 (3-6)
Bleed-related mortality	0.20 (0.11-0.39)	80%	7 (5-11)
All-cause mortality	0.55 (0.43-0.71)	45%	5 (4-9)

Hepatology 2001;33:802-807

The Clinical Scenario of Primary Prophylaxis

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- Patients with cirrhosis and without varices
- Patients with cirrhosis and small varices
 - With high risk of bleeding (Child B/C or RCS)
 - Without high risk of bleeding
- Patients with cirrhosis and medium/large varices
 - With high risk of bleeding
 - Without high risk of bleeding

Patients without Varices



Development of varix or variceal hemorrhage



NEJM 2005;353:2254-2261

Patients with small varices

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- With high risk of bleeding (Child B/C or RCS)
 - : Nonselective $\boldsymbol{\beta}$ blockers should be used
- Without high risk of bleeding
 - : β blocker can be used (long-term benefit, not proven)

or repeat EGD in 1-2 years

- Nonselective β-blockers in the prevention of first varceal hemorrhage in patients with small size varices
 - : 7% vs 2% over 2 years, not significant

Patients with medium/large varices



- Medium/large varices with high risk of bleeding
 - : EVL or nonselective β blockers
- Medium/large varices w/o high risk of bleeding
 - : EVL or nonselective $\boldsymbol{\beta}$ blockers
 - : β blockers are preferred and EVL in patients with contraindication or intolerance or non-compliance to β blockers (AASLD)

Patients with medium/large varices



Meta-Analysis of EVL vs β blockers (16 trials, 1318 subjects)

First variceal bleeding **Risk Ratio** Study, year (Reference) Events. n/n (95% CI) EVL **B**-blockers Chen et al., 1998 (76) 0.58 (0.06, 6.00) 1/26 2/30 De et al., 1999 (66) 2.00 (0.20, 19.78) 1/15 2/15 Sarin et al., 1999 (67) 0.33 (0.11, 0.93) 4/45 12/44 Song et al., 2000 (77) 0.83 (0.32, 2.18) 6/31 7/30 De La Mora et al., 2000 (78) 0.50 (0.05, 4.81) 1/12 2/12 Lui et al., 2002 (68) 0.50 (0.14, 1.74) 3/44 9/66 Gheorghe et al., 2002 (79) 0.26 (0.08, 0.80) 13/28 3/25 López-Acosta et al., 2002 (80) 0.67 (0.12, 3.69) 2/28 3/28 Abulfutuh et al., 2003 (81) 0.60 (0.20, 1.79) 4/44 10/66 Patients with medium/large varices Lo et al., 2004 (69) 0.56 (0.20, 1.54) 5/50 9/50 Schepke et al., 2004 (70) 0.89 (0.52, 1.50) 19/75 22/77 Jutabha et al., 2005 (71)-0.11 (0.01, 1.98) 0/31 4/31 Thuluvath et al., 2005 (72) 1.88 (0.19, 18.60) 2/16 1/15 Psilopoulos et al., 2005 (73) 0.22 (0.05, 0.94) 2/30 9/30 0.47 (0.15, 1.47) Drastich et al., 2005 (82) 4/40 7/33 Gill et al., 2006 (84) 0.46 (0.19, 1.22) 6/50 13/50 Lay et al., 2006 (74) 0.70 (0.29, 1.69) 10/50 7/50 Abdelfattah et al., 2006 (83) 0.31 (0.11, 0.90) 4/51 13/52 Norberto et al., 2007 (75) 0.67 (0.12, 3.72) 2/31 3/31 Tripathi et al., 2009 (59) 2.18 (1.00, 4.75) 17/75 8/77 Overall fixed-effects model ⊘ 0.62 (0.49, 0.79) 94/769 158/815 Heterogeneity chi-square = 23.6: p = 0.21 | ² = 20% Overall, excluding Tripathi et al. (59) \diamond 0.54 (0.42, 0.69) 77/694 150/738 Heterogeneity chi-square = 13.4: p = 0.77 $|^{2} = 0\%$ 0.01 1 20 Risk Ratio Favors EVL Favors B-blockers

Clin Liver Dis 2010;14:231-250

Prophylaxis of First Variceal Bleeding:Summary

	Clinical Situation	Treatment	Goal	Duration	Follow up EGD
	No varices	No			every 2-3 years
	Small varices	β blockers may be recommended	Increase to maximal tolerated dose or until HR 55/min	Indefinite	Every 1 year Not in β blocker users
	Small varices + RCS or child B/C	β blockers		Indefinite	No
	Medium/Large varices	β blockers		Indefinite	
		EVL	Variceal eradication	Until eradication	3Mo, 6Mo, 1yr

Upper GI Bleeding in Cirrhosis

- Cirrhosis with upper GI bleeding (465 patients)
 - Esophageal varices (68%)
 - Gastric varices (11%)
 - Portal hypertensive gastropathy (6%)
 - Duodenal ulcer (3%)
 - Gastric ulcer (2%)
- Upper GI bleeding in a cirrhotic patient must be presumed to be variceal origin until proven otherwise!!

Acute Variceal Hemorrhage (AVH)



- Gold standard of diagnosis : endoscopy
- Definition of AVH
 - In a patient with known or suspected portal hypertension
 - Presence of hematemesis within 24 h of presentation, and/or ongoing melena, with melena within last 24 h
- Recent bleed
 - Clinically significant bleed occurred within 6 weeks of presentation

Treatment Algorithm of AVH





Endoscopic Management of AVH



- Endoscopy as soon as the patient is hemodynamically stabilized in a monitored unit (at least 12 hrs)
- In endoscopy unit
 - : Hemodynamically stable patient, no hepatic encephalopathy, no cardiopulmonary dysfunction
- Otherwise, in ER or ICU by portable device

Check List Before Endoscopy



- Patient
 - : Vital sign, two IV lines, fluid (blood), O2 supplement, consent
- Endoscopy unit
 - : Check endoscope (suction), emergency cart, patient monitor, accessories, SB tube, at least 3 medical persons
- Preparation prior to endoscopy
 - : SBP>80 mmHg, intubation if necessary, IV drugs (vasoconstrictors, antibiotics, PPI)
- Routine sedation is not recommended

Diagnosis of Variceal Bleeding

- Bleeding from esophageal varices
 - Direct visualization of bleed issuing from an esophageal varix, usually spurting
 - Presence of a sign of recent bleed on a varix
 - : white nipple sign or overlying clot
 - Presence of esophageal varices with red color signs and blood in the stomach in the absence of another source of bleeding
 - Presence of esophageal varices with red color signs and clinical signs of upper GI bleeding (hematemesis or melena), without blood in the stomach



Role of Endoscopic Therapy in AVH

- Endoscopic band ligation
- Endoscopic sclerotherapy (ES)



- Injection of a sclerosing agent (ethanolamine up to 10-15cc) into the variceal lumen (intravariceal) or adjacent to it (paravariceal)
- Cause a thrombosis of the varix and inflammation of the surrounding mucosa
- Disadvantage: esophageal ulcer, bacteremia
- EVL is more effective than ES with greater control of hemorrhage, lower rebleeding, and lower adverse events but without differences in mortality

EVL is better than ES



• Therapeutic failure 24% vs 10%, RR=2.4, 95% CI 1.1-4.9

• Failure to control bleeding 15% vs 4%, p = 0.02

J Hepatol 2006;45:560-567

Endoscopic Sclerotherapy



1

Study Date:2011 Study Time:16

Study Date:2011 Study Time:16

Failure to Control AVH

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- 15-20% of patients with AVH
 - : early rebleed or failure to control bleeding
- Failure to control AVH(within 48hr)
 - Direct visualization during endoscopy
 - Fresh hematemesis after 2hr of combination therapy
 - 2(or 3) drop in hemoglobin within 24h period without transfusion
 - Development of hypovolemic shock

Rescue Therapy – Balloon Tamponade Sengstaken-Blakemore (SB) tube





Rescue Therapy – Balloon Tamponade

- Hemostasis by direct compression of bleeding varices
- Uncontrolled bleeding or massive and profuse bleeding
- Temporary 'bridge' therapy: maximum 24 hrs
- Pneumatic compression of the fundus and the lower esophagus, stop bleeding in 85% of cases
- Recurrence in half of the patients following deflation
- Complication (20-30%)
 - : aspiration pneumonia, esophageal perforation
- If hemostasis is not achieved within 2 hrs, other therapeutic options should be tried

Rescue Therapy

- Second endoscopy
 - Carefully planned (if bleeding is mild and the patient is hemodynamicall y stable)
 - Need caution and more expertise

TIPS

- Shunt (surgical or TIPS) has clinical efficacy as salvage therapy
- In patients whom hemorrhage from esophageal varices cannot be controlled
- Rebleeding despite combined pharmacological and endoscopic therapy



Pancreas



ORIGINAL ARTICLE: Clinical Endoscopy

Linear-array EUS improves detection of pancreatic lesions in high-risk individuals: a randomized tandem study **P**

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Baltimore, Maryland; Rochester, Minnesota; Boston, Massachusetts; Houston, Texas; New Haven, Connecticut, USA











- 25세 여자
- 특이 과거력 없음

 내원전 시행한 타병원 screening U/S상 splenic hilum에서 pancreatic tail로 abutting하는 약 2.3 cm크기의 mass로 내원함
Outside LGP U/S image







MR pancreas





What's your impression?

Impression

1. Outside LGP US

→ Nodular lesion around splenic hilum abutting to pancreas tail Imp) Large accessory spleen

2. CT scan at our hospital

→ A 2.5cm sized low attenuating mass in pancreas tail; delayed enhancement가 의심되며 p-duct dilatation은 없음.

--DDx> microcystic serous cystadenoma, MCN, SPT Imp)?

3. MR pancreas at our hospital

DDx> R/O Solid pseudopapillary tumor R/O Solid endocrine tumor R/O Accessory spleen



Radial and linear EUS



EUS

→ oval-shape , 2.5cm x 1.9 cm 크기의 heterogenous hypoechoic mass-like lesion이 관찰됨.Echotexture가 spleen과 비슷한 양상 으로 관찰됨

Imp) Intrapancreatic accessory spleen

Differentiation of Intrapancreatic Accessory Spleen from Small Solid Pancreatic Tumor





Accessory spleen, also referred as splenunculi.

- Common congenital defect
- 10-30% of population
- Easily misdiagnosed as PNET because of their hypervascular appearance.



Signal Intensities of IPAS and Small Solid Tumors of the Pancreas Compared with Spleen Parenchyma

Imaging and Intensity	IPAS ($n = 20$)	Small Solid Tumor ($n = 22$)	P Value			
TI weighted			.050			
Hyperintensity	0 (0)	1 (4)				
Isointensity	17 (85)	11 (50)				
Hypointensity	3 (15)	10 (46)				
T2 weighted			.001			
Hyperintensity	1 (5)	8 (36)				
Isointensity	19 (95)	9 (41)				
Hypointensity	0 (0)	5 (23)				
Gadoxetic acid enhanced						
Arterial phase			< .0001			
Hyperintensity	0 (0)	6 (27)				
Isointensity	20 (100)	4 (18)				
Hypointensity	0 (0)	12 (55)				
Portal phase			< .0001			
Hyperintensity	0 (0)	9 (41)				
Isointensity	20 (100)	5 (23)				
Hypointensity	0 (0)	8 (36)				
Late phase			< .0001			
Hyperintensity	0 (0)	9 (41)				
Isointensity	20 (100)	9 (41)				
Hypointensity	0 (0)	4 Rediology 2013; 266:159-167				



EUS-guided FNAB



Pancreas, tail, EUS-guided needle biopsy:: Solid pseudopapillary neoplasm



M/61 Pancreatic mass로 내원







검 사 결 과	! 과						🔍 시계열 조회		
검사일시	검 사 명	결 과	+/-	Max	Min	Rmk	수정		
2014-07-01 08:35	CEA <ascitic fluid=""> EUS guided pancreati c cyst drainage</ascitic>	2048 ng/mL							
2014-07-01 08:35	CA 19-9 <ascitic fluid=""> EUS guided pancr eatic cyst drainage</ascitic>	>10000 U/m1							







K: mesocolic mass DIAGNOSIS : Pancreas, distal pancreatectomy: 1. Mucinous cystic neoplasm with low grade dysplasia size: 2.5 cm - confined to the pancreas - lymphovascular invasion: (-) - perineural invasion: (-) - clear surgical resection margin 2. Chronic pancreatitis with fibrosis and atrophy Spleen, splenectomy: No diagnostic abnormality recognized

58/M 우연히 발견된 췌장낭종









2014-10-22 Distal pancreatectomy

DIAGNOSIS : Pancreas, body, distal pancreatectomy: Intraductal papillary mucinous neoplasm with intermediate-grade dysplasia, with clear resection margin

F/U lab : WNL F/U tumor marker : WNL (CA 19-9 7.31)

Endoscopic ultrasound-guided celiac plexus blockade











M/58 pancreatic cancer





EUS-guided choledochoduodenostomy









Biliary tract



Case presentation (F/85)

• C.C : RUQ pain

- Present illness:
 - 7년전 liver abscess에 대한 치료력있는 분으로 내원전 수일전 부터 시작된 우상복부 동통을 주소로 타 병원 들린뒤 APCT check후
 CBD stone with cholangitis imp.하에 외래
 - 로 의뢰됨



Case presentation (F/85)









Acute suppurative Cholangitis

- Prognosis: poor (when it is untreated)
- Conservative treatment with antibiotics (24 48 hr) in mild courses: can be tried but, who can guarantee ?
- **Biliary decompression** by ERCP or PTC is essential for life saving: decreased mortality from 100% to 40%



Anatomy





The View of the Ampulla of Vater

















B

-

А





Е

G



в











The structures of ampulla of Vater





















Case presentation (M/79)

• C.C : Epigastric pain

- Present illness:
 - 본원에서 AMI 로 진료력 있으시고 10년전에 서 CVA 있었으나 현재 medication받고 있지 않으심

복부의 전반적인 동통으로 타병원 들린뒤

APCT check하고 pancreatic mass 의심되어 제 외래로 내원하심

Case presentation (M/79)



EUS-guided FNA







Cytology results

CYTOPREPARATION: 26 Wet-fixed smears stained with Papanicolaou's

MACROSCOPIC OBSERVATION: about 0.2 ml, bloody clot material

CYTOLOGIC DIAGNOSIS : Pancreas, EUS-guided fine needle aspiration: Malignant Carcinoma, poorly differentiated.

CBD obstruction due to Pancreatic head cancer





ERCP가 필요한 경우

- Imaging study (US, CT)에서 CBD stone이 보일 때
- Imaging study에서 CBD dilatation이 있으면서 LFT abnormality (특히 ALP상승)이 있을 때
4

Biliary obstruction rise in intraductal pressure interruption of bile flow to the gut Biliary drainage















ERBD (Plastic stent)















Classification of Cholangiocarcinoma





Blechacz et al. Nat Rev Gastroenterol Hepatol.; 8(9): 512–522

Anatomic classification



Sleisenger and Fordtran's Gastrointestinal and Liver Disease, 9th edition

Clinical consideration

What is the size of a normal bile duct?

- Varies at different levels
- US 6-8 mm
- CT 8-10 mm
- Essentially unknown

What makes up a biliary stricture?

- Proximal dilatation
- Intrahepatic BD> 40% of parallel intrahepatic portal vein

Main questions for the doctor and the patient?

" Is this truly cancer ?"





4

Challenge in differentiating benign from malignant causes

• Biliary strictures are frequently a diagnostic dilemma

 Pre-operative diagnostic testing can establish a diagnosis in most patients, but indeterminate lesions still account for up to 20% of cases

• Should reduce unnecessary surgeries on benign strictures

Pathophysiology

Benign strictures

- Damage to the bile ducts during surgery or trauma to the abdomen
- Recurring condition, such as pancreatitis or bile duct stones
- Chronic disease, such as primary sclerosing cholangitis (PSC)

Fibrosis and Narrowing of the Bile Duct Lumen



Autoimmune cholangiopathy

PSC

Pathophysiology

Malignant strictures

- The result of either a primary bile duct cancer Narrowing of the bile duct lumen and obstructing the flow of bile
- Extrinsic compression of the bile ducts by a neoplasm in an adjacent organ Gallbladder, pancreas, or liver



External compression by gallbladder cancer

Etiology of bile duct stricture

Benign	Iatrogenic (liver transplant, cholecystectomy)Primary sclerosing cholangitisChronic pancreatitisAutoimmune pancreatitisIgG4 related cholangiopathyAutoimmune cholangitisMirizzi SyndromeInfections (tuberculosis, viral, parasitic,HIV cholangiopathy)IschemiaVasculitisTraumaDediction thereare
	Radiation therapy Pancreatic cancer
Malignant	Pancreatic cancer Cholangiocarcinoma Metastatic disease with external compression (lymph nodes)

Clinical practice of IBDS



No mass on cross-sectional imaging

- Typically contrast-enhanced CT or MRI

Conventional histopathology is non-diagnostic

- ERCP with brush cytology



Ideal endoscopic sampling technique: High sensitivity → few false negatives Perfect specificity → no false positives

Harewood GC. Curr Opin Gastroenterol 2008





Andrew Y. Wang Hepatobiliary ACG regional PG course, 2013

Assessment and management of patients with IBDS



Key steps

Characterization of the stricture pathogenesis

History

Laboratory studies

Cross-sectional imaging

Invasive imaging and tissue sampling

Relief of biliary obstruction

Defnitive treatment or palliation of the pathologic process

Medical, endoscopic, percutaneous, or surgical means

Historical feature of IBDS

A. Historical features suggestive of benign etiologies

History of right upper quadrant surgery

Trauma

- Ulcerative colitis or Crohn disease
- Chronic pancreatitis
- Difficult biliary stone disease
- Stable weight
- Fluctuating labs

B. Historical features suggestive of malignant etiologies

- Never-operated abdomen Absent history of abdominal illness Weight loss Short course without antecedent illness
- Decompensation of known primary sclerosing cholangitis

Clinical Clue

A. Reassuring

Younger patient h/o pancreatitis h/o biliary stone Normal CA 19-9 (?) Elevated IgG4 Stable weight Prior hepatobiliary surgery (ex. Cholecystectomy)

B. Concerning

Weight loss Elevated CA 19-9 (?) Long-term PSC Longer stricture (>1cm) Asymmetric stricture AUPBD Choledochal cyst

Testing modalities



"Despite a large number of tests available to establish the benign or malignant nature of biliary strictures, no single test has sufficient sensitivity to be considered adequate."

"Up to 20% of indeterminate biliary strictures are determined to be benign following surgical intervention"

"Typically, noninvasive laboratory and imaging tests are part of an initial evaluation often leading to endoscopic approaches with tissue sampling."

Christopher L. Bowlus et al. Nat Rev Gastroenterol Hepatol. 2016 Jan; 13(1): 28-37



81 y.o. F AST 90 IU/L TB 9.0 GB in situ Itching symptom

Perihilar cholangiocarcinoma-typical

After injection of gadolinium

Ring-shaped or heterogeneous enhancement with persistent





John L. Gollan, et al. Liver MRI



Tissue Acquisition and Pathologic Investigations Brush Cytology



7

Todd H. Baron et al. ERCP, 2nd edition

Cholangiopancreatoscopy

- Endoscopic Retrograde
 Cholangiopancreatography
- Radiographic images (similar to black and white x-rays) are taken to document findings





Cholangiopancreatoscopy-

Cholangioscopy is the examination of the bile ducts using an endoscope to enable direct visualization of the biliary tree during ERCP, can help obtain biopsy specimens, lead to the diagnosis of abnormalities, and guide stone therapy.





SpyGlass[™] DS System image showing LHD villous lesion in same patient





Indeterminate Stricture Diagnosis

26 Patients, Peter Draganov, MD, et al, *GIE*, Vol. 75(2), 2012

Key Results: Demonstrated 76.5% sensitivity using SpyBite[™] Forceps performing cholangioscopy with the SpyGlass System vs 29.4% sensitivity using blind biopsy and 5.9% sensitivity using brushings.





Benign Strictures

The following images demonstrate benign post-operative strictures treated with plastic st vascular abnormalities, nodules or exophytic tissue. Click on an image for a larger view





Benign Stricture Post-

Liver Transplant



Scattille



Malignant Strictures

The following images depict various characteristics of malignant strictures. Click on an image for a larger view



Indeterminate perihilar biliary stricture

Single operator fiberoptic choledochoscopy

68 y.o. M AST 105 IU/L TB 11.4 mg/dL s/p cholecystectomy Presenting with worsening RUQ pain and fever



EUS 유도 담도배액술(유두부 접근시) (rendezvous technic)





(*A*) The extrahepatic bile duct was punctured from the **second portion of the duodenum** under EUS guidance.

(*B*) A cholangiogram was taken through the needle to determine the configuration of the biliary ducts.

(C) A guide wire was placed though the needle, biliary duct, obstruction, and ampulla, deeply into the duodenum.

(D) Deep biliary cannulation was achieved **over the guide wire**.

(*E*) A metallic stent was deployed at the stricture.

Thank You for Your Attention I