The Society for the Surgery of the Alimentary Tract

2001 Postgraduate Course

Concepts In GI Surgery: A Case-Based Management Approach

Sunday, May 20, 2001
Hyatt Regency Hotel
Atlanta, Georgia

Course Director: Michael G. Sarr, M.D.
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SESSION IV
PANCREAS/SMALL BOWEL

Moderator: Andrew L. Warshaw, M.D.

Extrahepatic Biliary Obstruction
Gregory G. Tsiotos, M.D.

Solid Masses of the Pancreas
Michael B. Farnell, M.D.

Cystic Masses of the Pancreas
Carlos Fernández-del Castillo, M.D.

Palliation of Pancreatic Cancer
Keith D. Lillemoe, M.D.

Necrotizing Pancreatitis
Markus W. Büchler, M.D.

Small Bowel Obstruction
Robert E. Brolin, M.D., F.A.C.S.

Crohn's Disease of the Small Bowel
Eric G. Weiss, M.D., F.A.C.S., F.A.S.C.R.S., F.A.C.G.

Extrahepatic Biliary Obstruction

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Lecture Objectives

By the end of this lecture, the listener will be able to:

- Understand the importance of appropriate triage of patients with extrahepatic biliary obstruction in today's context of very low morbidity and mortality of major hepatic and pancreatic resections.
- Clarify which are the crucial questions that need to be answered preoperatively according to the level of the obstruction, and how each diagnostic/staging modality addresses these issues.
- Implement an efficient, problem-directed, quick, and cost-effective algorithm for the optimal preoperative evaluation of patients with extrahepatic biliary obstruction.
- Understand the appropriate utilization of preoperative biopsy and preoperative biliary stenting.

Case History 1

A 72-year-old Caucasian female presents to her family physician with a four week history of progressively increasing jaundice and fatigue. She has lost five kilograms over the last two months and although she has no specific symptoms, she feels weak. On physical exam she is deeply jaundiced and she has palpable liver edge (1.5 cm below the costal margin). Two more weeks elapsed between presentation and completion of successive series of tests (as suggested by the primary care provider), including hepatitis serology, liver scan, and ultrasound which revealed intrahepatic biliary tract dilatation involving both the right and the left systems, but the hepatic and main bile duct were of normal size. Laboratory tests included: Hb: 13.1 g/dL (12–15.5 g/dL), total bilirubin: 19.8 mg/dL (0.1–1.1 mg/dL), direct bilirubin: 17.4 mg/dL (≤0.3 mg/dL), alkaline phosphatase: 749 U/L (119–309 U/L), GGT: 179 U/L (6–29U/L), AST: 148 U/L (12–31 U/L), ALT: 171 U/L (9–29 U/L). WBC was normal.

Case History 2

A 56-year-old Native American male presents with a four month history of mild jaundice and occasional episodes of melena after which jaundice disappears. He does not complain of pain or any other acute symptoms. Although he reports some occasional nonspecific upper abdominal discomfort after a meal and three episodes of vomiting, he has not lost any weight and his appetite and energy level are normal. Physical exam was essentially normal except icteric sclera. Laboratory tests included: Hb: 10.9 g/dL (13.5–17.5 g/dL), MCV: 72 fL (81.2–95.1 fL), total bilirubin: 4.1 mg/dL (0.1–1.1 mg/dL), direct bilirubin: 3.4 mg/dL (≤0.3 mg/dL), alkaline phosphatase: 312 U/L (98–251 U/L), GGT: 63 U/L (12–48 U/L). WBC was normal and AST and ALT mildly elevated. Abdominal ultrasonography revealed dilated intrahepatic and extrahepatic bile ducts.

Case History 3

A 65-year-old Hispanic male presents with worsening jaundice over the last six weeks, weight loss of eight kilograms over the last three months, decreased appetite and some fatigue. Although he is a lean person, he developed non-insulin dependent diabetes mellitus about a year ago. He complains of some abdominal discomfort, but he denies abdominal or back pain, or any other acute symptoms. His internist observed him for three weeks as he expected results for hepatitis serology and as the ultrasound had shown cholelithiasis in addition to both

intrahepatic and extrahepatic biliary dilatation. On physical exam there is no palpable abdominal mass and no supraclavicular lymphadenopathy. Laboratory tests included: Hb: 13.9 g/dL (13.5–17.5 g/dL), MCV: 83 fL (81.2–95.1 fL), total bilirubin: 9.8 mg/dL (0.1–1.1 mg/dL), direct bilirubin: 8.4 mg/dL (≤0.3 mg/dL), alkaline phosphatase: 583 U/L (98–251 U/L), GGT: 98 U/L (12–48 U/L). WBC was normal and AST and ALT were elevated.

Introduction

Although in the majority of patients obstructive jaundice (OJ) is related to benign conditions (i.e., gallstones and their complications), the suspicion of a malignancy involving the biliary tract should be raised very early and considered probable until proven otherwise. Such malignancies are aggressive and intervention at the earliest possible stage is of paramount importance if an improved outcome is truly sought. Despite past nihilism, the last few years have been marked by a tremendous decrease in morbidity and mortality after major pancreatic and hepatic resections. Currently, in centers with a major interest in pancreatic and liver surgery operative mortality is consistently <5%, morbidity is substantially decreased, and 5-year survival is longer.[1] This reality has shifted the attitude of experienced surgeons toward major pancreatic and hepatic resections and explains why patients with OJ should undergo their evaluation and operation in centers with dedication to and a proven record in liver and pancreatic surgery. Today the low willingness to embark on a pancreatectomy or hepatectomy unless tissue diagnosis is available, the exhaustion of every diagnostic means to rule out conditions which may not absolutely necessitate resection (chronic pancreatitis, periampullary adenoma) are past. When faced with a patient with a mass causing OJ, today's surgeon is more willing to resect it without precise tissue diagnosis and extensive work-up to identify its pathologic nature; rather he/she is much more concerned whether this mass is indeed resectable. This is very important to recognize because the question of the past, *"what this mass is?"* (i.e., precise diagnosis) is now replaced by the question, *"Can I take it out?"* (i.e., preoperative clinical staging). Consequently, the way various tests are utilized has also changed. The framework of this presentation is based on the following principles:

1. *Early suspicion* of a tumor in patients presenting with OJ.

2. *Resectability* and *operative planning* are the concepts that should dominate preoperative evaluation. Diagnostic/staging modalities should be chosen with these specific concepts firmly in mind. Tumors causing OJ can be hilar, mid-duct, or periampullary; at each location resectability and operative planning depend on different, but very specific imaging findings; thus, each test should be pursued in order to address specific questions.

3. *Rational implementation* of preoperative biopsy and preoperative biliary decompression/stenting.

Initial Clinical Presentation

Careful history proves that OJ rarely is the sole symptom; it generally accompanies a constellation of either *acute* or *chronic* symptoms. Acute symptoms (fever, acute abdominal pain, chills) generally reflect gallstone-related disease. Chronic symptoms, usually nonspecific (weight loss, fatigue, vague abdominal/back pain, recent onset of diabetes[2]) reflect presence of a tumor. Most patients with OJ belong to the former group; this is why many physicians may generalize and manage all of them as having complicated gallstone disease; thus valuable time for the diagnosis and management of a tumor elapses, various treatments prove fruitless, and the tumor grows. *The lesson is that OJ in anyone >40 yrs should be assumed to be extrahepatic biliary obstruction until proven otherwise.* OJ truly secondary to gallstone disease can be usually ruled in or out based on history

alone. The characteristics of coexisting pain, fever, and leukocyrtosis may translate to choledocholithiasis; recent laparoscopic cholecystectomy may allude to the presence of an iatrogenic bile duct injury. Liver tests with a cholestatic pattern are not specific for a stone or tumor.

Ultrasonography (US): After history taking, patients (still at the primary care level) generally undergo US, a sensitive test for presumed biliary obstruction and the most sensitive for gallstone disease. US defines the level of the obstructing lesion (stone or tumor) and reveals signs of acute inflammation (thickened bile duct wall, intramural fluid). In gallstone-related OJ, work-up is practically complete and management is initiated. If, however, gallstone disease is ruled out, an obstructing tumor is suspected, and a *critical point* is now reached, since one may continue with further tests, usually nondefinitive, sometimes not appropriate and generally time consuming, during which no management plan exists, time elapses and the tumor grows, or the patient may be referred to a center with a known track record in hepatobiliary disease, where further evaluation will be dictated by very specific guidelines based on modern principles.

Hilar Tumor

When US reveals intrahepatic, but not extrahepatic, biliary tract dilatation, the suspicion of a hilar tumor is raised. Specific issues that need to be addressed are:

1. Exclusion of distant disease (metastasis).

2. Definition of proximal (along the bile duct) as well as radial extension of the lesion. Precise definition of the proximal extension in relation to the hepatic duct bifurcation is critical because the surgeon can then know whether one or two bilio-enteric anastomoses are needed after resection (Bismuth type 1,2 versus 3,4), a hemihepatectomy is required as a part of a curative operation, or the tumor involves the contralateral ductal system and at what extent; extensive involvement of the contralateral ductal system as well, would make the tumor unresectable.

3. Radial extension to the hepatic artery proper and the portal vein (PV) or their main branches. Involvement of the contralateral hepatic artery and PV, or main PV thrombosis by tumor would again make the tumor unresectable.

Dynamic contrast-enhanced computerized tomoraphy (**CT-scan**) should be performed first. It will demonstrate the obstructing tumor and the extent of periductal hepatic parenchymal involvement, and reliably reveal signs of metastatic disease. Although CT gives information on the proximal and distant extent of the tumor along the hepatic ducts, a special study is required to specifically delineate ductal anatomy in detail. The relation of the tumor to the surrounding blood vessels can also be indicated, but not necessarily defined by CT because the fat plane between the duct and the hepatic artery and PV (especially at the level of the liver hilum) is normally very thin (unlike the normally thicker fat plane between PV and head of the pancreas). A good quality CT will demonstrate occlusion of the artery, the vein, or their branches, but will not reliably reveal possible infiltration of only their wall by the tumor.

Next, the anatomy of the biliary system and the pattern of its involvement with tumor have to be defined as accurately as possible. Of the two possible luminal imaging studies, percutaneous transhepatic cholangiography (PTC) and endoscopic retrograde cholangiography (ERC), the former yields by far most information. **PTC** will demonstrate accurately the ductal anatomy *proximal* to the lesion, which is precisely what is absolutely essential to assess resectability and form an operative plan. Questions that PTC is called to answer are: 1) Relation of the proximal extent of the tumor to the hepatic duct bifurcation, which alludes to the number of bilio-enteric

anastomoses that the surgeon should be prepared to perform after resection (≥1 cm margin beyond the tumor is required), 2) Level of extension of a tumor along one of the hepatic ducts, which indicates the need for ipsilateral hemihepatectomy for curative resection, 3) Possible extension along the contralateral hepatic duct, which may indicate the need for an intrahepatic contralateral bilio-enteric anastomosis (after ipsilateral hemihepatectomy) if the extension is limited, or may translate to unresectability if the tumor extends further inside the contralateral side, and 4) Precise delineation of the relation of the right posterior ductal system to the right anterior and to the main hepatic duct; often the former drains directly into the main hepatic duct and this is very important in planning the resection as well as the reconstruction.

After ductal anatomy is defined, possible vascular involvement should be investigated (reflected as obstruction, stenosis, or stricture). Hepatic **arteriogram** followed by splachnic venous phase is currently the test of choice. Thrombosis of the main PV, infiltration of the hepatic artery proper and PV by tumor, involvement of the contralateral hepatic artery and PV regardless of whether their ipsilateral (to the tumor) counterparts are involved are very significant findings and translate to unresectability. Angiography also reveals an aberrant left or aberrant right hepatic artery (present in about 20% of the patients each), which is essential in the appropriate planning of a major curative hilar and/or liver resection.

With the information gathered by these three tests in this order, all issues relevant to resectability and operative planning should be sufficiently addressed and a complete management plan can be implemented. Because PTC and angiography (both invasive) carry some risk of complications (~5% together), magnetic resonance imaging (MRI), supplemented by MR cholangiography and MR angiography, seems the ideal diagnostic/staging modality for hilar tumors, since it can replace all three aforementioned tests and it may yield all the information required. Indeed, MR has shown excellent results in small retrospective series; several prospective studies are under way to define MR's accuracy in operative planning compared to operative findings, as well as to CT, PTC, and angiography. It is conceivable that in the near future MRI (along with MRC and MRA) may well replace all current staging modalities. This would not only protect patients from the risks of two invasive tests, but it would also prove to be overall cheaper and faster (very important factors in today's era of cost- and resource-containment).

Mid-duct Tumor

Conceptually, from the surgeon's perspective, a mid-duct tumor is identical to a hilar tumor *not involving* the bifurcation. Consequently, criteria of resectability and operative planning, and thus preoperative evaluation, are esssentially the same. **CT** will demonstrate the mass and its distance to the head of the pancreas, and will identify possible distant disease. The ductal luminal study that is required may be **ERC** in this case, since exhaustive information of the ductal anatomy cephalad to the bifurcation is not essential, except for the extent of the tumor along the duct and the distance between its proximal margin and the bifurcation. **Angiography** is focused on involvement of the hepatic artery proper and the main PV, the level of their respective bifurcation, and possible presence of aberrant left or right hepatic artery. As with hilar tumors, MRI, MRC, and MRA, all in one setting, may soon replace CT, ERC, and angiography.

Periampulary Tumor

When US reveals intrahepatic and extrahepatic biliary tract dilatation, a periampullary mass should be strongly suspected. In this case, resectability and operative planning depend on a different set of issues:

1. Presence of distant disease (metastasis to the liver, lungs, distant lymph nodes, ascites)

2. Relation of the tumor to the peripancreatic vessels (superior mesenteric artery/vein [SMA/SMV], PV)

3. Presence of peritoneal surface implants (as in pancreatic cancer)

Thus, a well-organized algorithm[3] is needed to address these issues and the choice amongst the various diagnostic/staging modalities should be based on the efficacy of each, its risk, and *whether it will yield information that will directly impact the management of the patient.*

"Spiral" or "helical" CT has emerged as the gold standard. Not only does it delineate a periampullary mass, but it also provides information about liver metastases, peripancreatic nodal spread, ascites, and involvement of the SMV/PV confluence. Sensitivity of CT in demonstrating liver metastases depends on their size. Lesions ≥2 cm are evident as hypodense defects; metastases <1 cm are usually missed. This precisely is the argument for preoperative laparoscopy. Detection of nodal metastases is much less accurate. While enlarged peripancreatic lymph nodes, especially along the common hepatic artery, are often evident, it cannot be determined whether they are metastases or just reactive (often seen with extrahepatic biliary obstruction). Peritoneal metastases are rarely imaged directly; their presence may be inferred by the presence of ascites, which should precipitate percutaneous aspiration cytology.

If distant disease is ruled out by spiral CT, the next important factor is possible vascular involvement. Again, spiral CT can accurately evaluate the normally present "fat" plane between the SMA/SMV/PV and the head/uncinate region of the pancreas. Preservation of this plane strongly suggests no vessel involvement by the tumor. Preservation of the SMV/PV contour throughout its extent but loss of the fat plane is an indeterminate finding. Occlusion of the SMV/PV junction, especially with the presence of collateral vessels, is an absolute sign of unresectability and precludes abdominal exploration for resection. Compression of these veins with loss of the smooth contrast column within is consistent with, but not necessarily diagnostic of, tumor involvement. Recently, grading systems (grades: 0–4) based on the degree of CT-assessed vascular contour impairment and circuferential contiguity of tumor to vessel have been tested;[4] they seem to predict resectability very accurately. Lower grades (0–1: preserved perivascular fat plane or loss of fat plane with smooth displacement of the vessel) are associated with resectability close to 100% and higher grades (3–4: encased narrowed vessel >50% of the circumference or occluded vessel) to unresectability of 90%–95%. The middle grade (2: irregularity of one side of the vessel <50% of its circumference) is associated to 40% resectability. This approach provides for rationalized and objective vascular involvement by CT criteria and supports the argument that formal angiography is not necessary.

In the past angiography was considered the test which best detects vascular involvement. However, three prospective studies[3] compared each one's efficacy in demonstrating vascular involvement and concluded that spiral CT is at least as effective (if not more) as angiography. Interestingly, one of them[5] showed that more than one third of the patients with vessel "encasement" on angiography proved to have resectable disease, whereas in one fourth of those with normal angiograms, the tumor proved unresectable. A major peripancreatic vessel appearing occluded on angiogram represents direct invasion by the tumor, but this finding is usually recognizable on a spiral CT. Current data suggests that angiography is not justified. It is an unreliable test and adds little to a good quality spiral CT. Another argument for angiography has been that it demonstrates vascular anomalies (aberrant right or left hepatic artery) in 30% of patients, so that injury to these vessels may be prevented. However, these anomalies are readily apparent; the replaced right hepatic artery is appreciated as an arterial pulse posterior to the hepatoduodenal ligament and the replaced left hepatic artery traverses the lesser omentum. These variations are easy to recognize intraoperatively. The surgeon exploring patients with a periampullary tumor with intent to resect, should be familiar with the variations of the region's normal anatomy and should not need to depend on a "road map."

After the issues of tumor delineation, absence of distant disease, and relation to the SMA, SMV, PV are sufficiently addressed by spiral CT, the last crucial issue is possible presence of peritoneal and small liver micrometastases, which are usually multiple, widespread and quite small (1–3 mm). Since they are present in 18%–40% of patients with cancer of the head of the pancreas[6,7] and these patients' survival does not exceed 6 months,[8] it is crucial to identify this substantial subgroup before a noncurative celiotomy. **Laparoscopy** and

biopsy of suspicious lesions should be a routine staging procedure for patients with periampullary tumor that looks resectable in CT. This minimally-invasive procedure can identify the patients in whom an endobiliary stent is all what is needed, save them an unnecessary laparotomy, and help improve the quality of their remaining life. There is preliminary evidence that peritoneal cytology during laparoscopy may reveal yet another subgroup in whom celiotomy and even resection will not add any survival benefit. The presence of malignant cells in peritoneal washings can predict not only tumor unresectability,[6] but also a shorter survival even when the tumor is resected.[9] Interestingly, patients with positive peritoneal cytology seem to have the same survival whether they have grossly visible metastases or not,[10] alluding to the fact that the significance of positive peritoneal cytology is similar to the presence of gross metastatic disease. If these findings are corroborated by others, peritoneal cytology will probably assume a more important role in patients with suspected pancreatic cancer. Today, spiral CT followed by laparoscopy are the two essential diagnostic/staging modalities that are only required for optimal assessment of resectability and operative planning in patients with a periampullary tumor.

ERCP is often performed "automatically" in nonreferral centers after OJ is diagnosed. Although very sensitive for periampullary tumors causing OJ (long, irregular strictures of the pancreatic and/or the bile duct ["double duct" sign]), with the current sophistication of spiral CT, its necessity and routine practice are highly questionable. There is a definite role for ERCP when the diagnosis is equivocal: in patients with OJ in whom no mass is evident in CT, or in those with known chronic pancreatitis in whom development of a pancreatic cancer is suspected. Generally, for the good-risk patient with new-onset OJ in whom CT clearly shows a periampullary mass, ERCP offers no further therapeutically useful information, does not usually alter the therapeutic approach, and only subjects the patient to a small risk; most pancreatic surgeons believe it is unnecessary in most patients with a periampullary tumor.

Among other sophisticated staging modalities, endoscopic US in patients with a periampullary mass evident in CT, appeared promising in assessing peripancreatic vascular involvement, but four prospective studies, each with a similar number of patients, reached contradictory results. More experience needs to be gained with this highly operator-dependent technique. MRI can depict the tumor, the peripancreatic vessels and the biliary tree by MRA and MRCP, but no study has compared it to spiral CT. Currently, MRI does not seem to add to the preoperative evaluation of patients with suspected periampullary tumor.

Preoperative Biopsy

Once a mass causing OJ is demonstrated in CT, traditionally there has been an "urge" to obtain tissue diagnosis, usually by CT- or US-guided fine needle aspiration cytology or endoscopically obtained brushings. Contrary to this common practice in nonreferral centers, there are problems associated with this "knee-jerk" reaction and many reasons to resist it. First, although positive predictive value of these techniques may approach 100%, negative predictive value is generally lower (60%–70%). While a histologic diagnosis of carcinoma is reliable, when the pathologist cannot find malignant cells, malignancy cannot be excluded. Second, while percutaneous biopsy is generally quite safe, several potentially serious complications can occur (~1%), including hemorrhage, pancreatitis, fistula, abscess, and rarely death. Third, and more concerning, are several reports of tumor seeding along the subcutaneous track of the needle or intraperitoneally.[3] Because of all these, *preoperative biopsy has little or no role in the evaluation of the good risk patient with a clinically resectable hilar or periampullary mass.*[11] A negative biopsy would not prevent operative exploration and resection. If the results of the percutaneous biopsy will not alter management, there seems no reason to perform it. However, there is a definite role for biopsy *when resection is not possible:* poor-risk patients in whom a major hepatic or pancreatic resection may not be tolerated, but who can receive chemoradiation and need tissue confirmation, as well as those with unresectable cancer, in whom a percutaneous biopsy confirms the diagnosis and leads to placement of a biliary endoprosthesis and/or palliative chemoradiation.

Preoperative Biliary Decompression

Endoscopic stent placement to relieve OJ is another "automatic" response just after an obstructing mass is diagnosed. This invasive procedure, very common recently with the wide availability of ERCP, usually takes place prior to having assessed whether the tumor is resectable and has been adopted by many as part of the initial work-up and preparation of the patient with a presumed malignant stricture of the biliary tree. The major reason for this practice is that it quickly and relatively safely palliates patients with OJ from their most dramatic symptom. Also, it generally reflects pessimism on the feasibility of definitive treatment, since it is commonly believed that this palliative measure may be the only procedure that most patients will ever need and for the few who will undergo exploration for resection, placement of a biliary stent "does not burn any bridges" and may decrease perioperative morbidity and mortality. The true benefits of preoperative biliary decompression however, are related to: 1) reversal of jaundice-induced immunosuppression (allegedly decreasing perioperative morbidity and mortality) and 2) level of postoperative liver function. With today's evidence, the rational criterion for preoperartive biliary decompression is *whether a major hepatectomy will be a part of the planned curative resection.*

For a hilar tumor involving the bifurcation and extending towards one of the hepatic ducts, where a *hemihepatectomy should be performed,* the functional status of the remaining half of the liver is of paramount importance. Hemihepatectomy reduces the available parenchyma around 50%, which needs to be at its optimal functional status (i.e., decompressed bile canaliculi and hepatocytes) to meet the patient's metabolic needs; this is especially true when preoperative liver function is worse than Childs-Pugh class A. Also, patients with profound jaundice are more prone to develop portal and peripheral endotoxemia with renal function compromise.[12] For these reasons, patients in whom a liver resection is planned, should undergo preoperative biliary decompression, since this will improve the function of the remaining hepatic parenchyma and decrease the risk of postoperative hepatic and renal failure and sepsis.[13]

When *hemihepatectomy is not part of the intended curative operation,* preoperative biliary decompression offers no advantage since the available liver parenchyma remains intact. On the contrary, it converts a sterile biliary system to a colonized one, increasing the risk for cholangitis and postoperative infectious complications. In a recent large study with 567 patients undergoing Whipple, it was demonstrated that preoperative stenting was associated with significantly higher wound infection and pancreatic fistula and offered no benefit whatsoever.[14] Three other prospective studies have failed to show any significant benefit of preoperative biliary decompression.[3] For these reasons, before an endobiliary stent is placed, patients should be thoroughly staged and the decision of tumor resectability should be made first. In the good-risk patient in whom the tumor causing OJ is deemed resectable with no need for hepatectomy based on the appropriate staging, preoperative biliary decompression *has no place*. Some have suggested that a stent can be helpful during bile duct dissection, serving as a "guide intraoperatively." It is doubtful, though, whether an experienced hepatobiliary surgeon needs a biliary stent to identify and safely dissect the bile duct. Moreover, the intraluminal foreign body incites an inflammatory reaction in the hepatoduodenal ligament making dissection in fact more difficult. A dilated bile duct makes for an easier biliary-enteric anastomosis, a finding not as prominent after decompression.

In patients with *unresectable hilar and mid-duct tumors,* biliary stenting may be in fact the only intervention needed. In those with *unresectable periampullary tumors,* the presence or absence of gastric outlet obstruction as well as life expectancy should be considered. Gastric outlet obstruction dictates operative gastrojejunostomy, at which point a proximal, wide, and durable hepaticojejunostomy should be constructed; preoperative biliary stenting would be of no benefit. On the contrary, short life expectancy (<6 months, such as with liver metastases) without duodenal obstruction makes biliary stenting the ideal palliative measure. The same is true for patients with serious co-morbid conditions, who are unsatisfactory candidates for resection. This subgroup will have the best quality of life with endoscopic palliation. In summary, preoperative biliary decompression should be used selectively and not routinely in patients with biliary obstruction. This decision often requires the experience and judgment of a referral center with a special interest in pancreatic disease.

Summary

The role of the primary care physician is crucial in the optimal management of patients with OJ. Increased awareness and a low threshold of suspicion are the most important means to decrease the delay in diagnosis of an obstructing tumor. Triage is equally important. It cannot be over-emphasized that patients with OJ, not due to gallstone disease (based on absence of acute symptoms and US findings), should undergo the final evaluation and operation by experienced surgeons with dedication to and a proven record of low morbidity and mortality in hepatobiliary and pancreatic surgery. In such a setting, where operative resection is so safe, precise preoperative pathologic diagnosis of a tumor causing OJ is less important than appropriate clinical staging. US gives the rough location of an obstructing tumor based on the level of bile duct dilatation and decompression. Consequently, a very organized algorithm should be implemented to accurately assess resectability and develop the operative plan.

- For a hilar tumor spiral CT, PTC, and arteriogram with venous phase address all pertinent issues.
- For a mid-duct tumor, spiral CT, ERC, and arteriogram with venous phase are the tests required?
- For a periampullary tumor spiral CT will address distant metastasis and relation to the peripancreatic vasculature, and laparoscopy will identify peritoneal implants.
- ERCP is not indicated when a periampullary tumor is evident in CT, but is important when the cause of OJ can not be demonstrated in CT.
- Preoperative biopsy: No, when the tumor is clinically resectable; yes, when the tumor is clinically unresectable.
- Preoperative biliary decompression: Yes, when a hemihepatectomy is part of the intended curative procedure and for most unresectable tumors; no, when the liver remains intact (i.e., periampullary and mid-duct tumors, hilar tumors not requiring hepatectomy).

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2001 SSAT POSTGRADUATE COURSE

CONCEPTS IN GI SURGERY:
A CASE-BASED MANAGEMENT APPROACH

May 20, 2001

Hyatt Regency Hotel, Atlanta, Georgia

7:55 a.m. **Welcome**
Michael G. Sarr, M.D.

SESSION I – ESOPHAGUS

Moderator: Tom R. DeMeester, M.D.

8:00 a.m. **Squamous Carcinoma of Esophagus**
Carlos A. Pellegrini, M.D.

8:10 a.m. **Adenocarcinoma of Esophagus**
Jeffrey H. Peters, M.D.

8:25 a.m. **Gastroesophageal Reflux Disease**
John G. Hunter, M.D.

SESSION II – STOMACH

Moderator: Daniel Dempsey, M.D.

8:40 a.m. **Carcinoma of the Stomach**
Kevin Conlon, M.D.

8:55 a.m. **Perforated Duodenal Ulcer**
Theodore N. Pappas, M.D.

9:05 a.m. **Bleeding Duodenal Ulcer**
Michael W. Mulholland, M.D.

9:15 a.m. **DISCUSSION**

9:45 a.m. **BREAK**

SESSION III – LIVER/BILIARY TREE

Moderator: William C. Meyers, M.D.

10:00 a.m. **Solid Masses of the Liver**
Yuman Fong, M.D.

10:15 a.m. **Cystic Masses of the Liver**
Kevin E. Behrns, M.D.

10:30 a.m. **Liver Transplantation**
Stuart J. Knechtle, M.D.

10:45 a.m. **Post Laparoscopic Cholecystectomy Injuries of the Bile Duct**
John H. Donohue, M.D.

11:00 a.m. **Management of Common Bile Duct Stones**
Bruce D. Schirmer, M.D.

11:10 a.m. **Biliary Pancreatitis**
Michael L. Steer, M.D.

11:20 a.m. **Biliary Neoplasms**
J. Nicholas Vauthey, M.D.

11:35 a.m. - 12:00 p.m. **DISCUSSION**

12:00 p.m. **LUNCH**

12:30 p.m. **Laparoscopic Colectomy for Cancer**
Tonia Young-Fadok, M.D.

SESSION IV – PANCREAS/SMALL BOWEL

Moderator: Andrew L. Warshaw, M.D.

1:00 p.m. **Extrahepatic Biliary Obstruction**
Gregory G. Tsiotos, M.D.

1:10 p.m. **Solid Masses of the Pancreas**
Michael B. Farnell, M.D.

1:25 p.m. **Cystic Masses of the Pancreas**
Carlos Fernandez del Castillo, M.D.

1:35 p.m. **Palliation of Pancreatic Cancer**
Keith D. Lillemoe, M.D.

1:45 p.m. **Necrotizing Pancreatitis**
Markus W. Buchler, M.D.

1:55 p.m. **Small Bowel Obstruction**
Robert E. Brolin, M.D.

2:05 p.m **Crohn's Disease of Small Bowel**
Eric G. Weiss, M.D.

2:20 p.m. **DISCUSSION**

2:45 p.m. **BREAK**

SESSION V – COLON/RECTAL

Moderator: Ira J. Kodner, M.D.

3:00 p.m. **Crohn's Colitis**
Robin S. McLeod, M.D.

3:10 p.m. **Chronic Ulcerative Colitis**
Merril T. Dayton, M.D.

3:25 p.m. **Anorectal Fistula and Abscess**
Herand Abcarian, M.D.

3:35 p.m. **Hemorrhoids**
James Fleshman, M.D.

3:45 p.m. **Rectal Cancer**
Heidi Nelson, M.D.

4:00 p.m. **Bariatric Surgery**
Harvey J. Sugerman, M.D.

4:15 p.m. **Repair of Incisional Hernia**
B. Todd Heniford, M.D.

4:25 p.m. **Small Bowel Transplantation**
David Grant, M.D.

4:35 p.m. **DISCUSSION**

5:00 p.m. **ADJOURN**

Postgraduate Course

Concepts in GI Surgery: A Case-Based Management Approach

Sunday, May 20, 2001
Hyatt Regency Hotel
Atlanta, Georgia

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FACULTY DISCLOSURE

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