Ampullary adenomas (AA) are benign, and if untreated, can undergo malignant transformation into ampullary adenocarcinoma. AA can occur sporadically or associated with familial adenomatous polyposis (FAP). The incidence of AA is increasing due to more frequent use of imaging and endoscopy. Management of AA includes Endoscopic ampullectomy (EA), local surgical excision and pancreaticoduodenectomy, depending on the size, lymph node involvement, ingrowth into bile or pancreatic duct and presence or absence of advanced duodenal polyposis. Accurate preoperative diagnosis and staging is essential in the management of AA. Endoscopic ultrasound (EUS) can aid in preoperative risk stratification of size, regional nodal metastasis and ductal and vascular invasion in high risk ampullary lesions.

INTRODUCTION
The incidence of ampullary adenoma (AA) is increasing with the ever-more-frequent use of imaging and endoscopy. (Figure 1) AA can occur sporadically or in the context of genetic syndromes such as familial adenomatous polyposis (FAP). Adenocarcinoma of the ampulla of vater (AV) is relatively uncommon and it accounts for 0.2% of gastrointestinal cancers. Ampullary adenoma can transform into ampullary adenocarcinoma. Intestinal mucosa near the ampulla is more prone to neoplastic transformation than any other site in the small intestine as there is a transition from pancreaticobiliary epithelium to small intestinal epithelium and it is constantly irritated chemically and mechanically. According to SEER data base, the incidence of ampullary cancer (AC) was 0.59 per 100,000 per year and more common in males than females. Accurate staging is important in preoperative assessment of adenoma. Endoscopic retrograde cholangiopancreatography (ERCP) and endoscopic ultrasound (EUS) can aid in the preoperative staging for AA. Endoscopic ampullectomy (EA) can be safe and effective in experienced endoscopist hands and can avoid surgical intervention in patients without ductal extension. This review article focuses on pathogenesis, diagnosis, indications, technique and outcomes of endoscopic management of AA.
ANATOMY
The AV is a spherical structure formed by the confluence of common bile duct (CBD), pancreatic duct (PD) and the distal aspect of the sphincter of oddi muscle. The duodenal papilla is a nipple-like structure located on the medial aspect of the second portion of the duodenum. The ampullary region is a transition from pancreaticobiliary epithelium to small intestinal epithelium. The AV is located behind the major duodenal papillae and is covered by small intestinal-type epithelium. The entry of bile into the second portion of the duodenum is controlled by the smooth muscle fibers of the sphincter of oddi that open at the duodenal papilla and allows bile to flow into the small intestine. Periampullary tumors can originate from pancreas, duodenum, distal CBD or structures of AV. Ampullary carcinoma can arise from within the AV.

Clinical Presentation
AA is usually found incidentally, and they are asymptomatic. However, they can present with jaundice, pruritus, abdominal pain, nausea, vomiting, anorexia, malaise, dyspepsia and melena. Obstructive jaundice is usually caused by compression of the distal bile duct by the tumor. Jaundice at initial presentation with pancreatic invasion and superior mesenteric lymph node can predict advanced stage ampullary carcinoma with poor prognosis. Iron deficiency anemia with blood loss can occur secondary to ulceration from ampullary tumors. Acute or recurrent pancreatitis can occur with the obstruction of pancreatic duct from ampullary tumor.

Pathogenesis
AC develops from preexisting adenomas or flat preneoplastic lesions. Most AA develop sporadically. Patients with familial adenomatous polyposis (FAP) are more prone to colorectal adenoma and AA. Yamaguchi and Enjoji, defined three macrotypes of AC based on macroscopic appearance. Intramural protruding (intraampullary), extramural protruding (periampullary) and ulcerating AC. The common channel is formed by the intestinal mucosa of the ampulloduodenum and mucosa of the ampullar-pancreatico-biliary duct. Histologically, AC has 2 main types, which includes the intestinal type and the pancreaticobiliary type. The intestinal type resembles tubular adenocarcinoma of stomach or colon and the pancreaticobiliary type is characterized by papillary growth with scant fibrous cores. The AA of intestinal type can be tubular, villous or tubulovillous and closely resemble adenoma of the intestine.

Diagnosis
Accurate preoperative diagnosis and staging is essential in the management of AA. The side-viewing endoscope (SVE) allows better visualization of the morphological features of ampullary lesion and aids in acquisition of the tissue for biopsy during the procedure. To improve accuracy of diagnosis, atleast six biopsies from the
## Table 1. Complications of EA

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<tr>
<th>Author (year)</th>
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Intraductal ultrasound (IDUS) allows EUS from inside the biliary and pancreatic ducts. IDUS probes can be inserted through the accessory channel of a duodenoscope during ERCP into either the biliary or the pancreatic duct. IDUS is superior to EUS in visualization of tumors of major duodenal papillae with accuracy of 100% vs 59.3% (IDUS vs EUS); Sensitivity of 100% vs 75% (IDUS vs EUS) and specificity of 62.5% vs 50% (IDUS vs EUS) respectively.

Despite these benefits, IDUS is rarely performed given the additional cost and the need for a second EUS processor. Computed tomography (CT) and transabdominal ultrasound (US) are not adequate for staging for ampullary tumors but they can identify biliary and pancreatic duct dilation. CT can also identify locoregional lymph nodes and distant metastasis. Magnetic resonance cholangiopancreatography (MRCP) can assess the extent of intraductal involvement non-invasively and identify pancreas divisum, in addition to the identification of biliary and pancreatic dilation but small ampullary lesions are often missed on MRI with MRCP.

Endoscopic ultrasound (EUS) aids in the assessment of depth of mucosal invasion, infiltration of the periampullary wall layers and pancreatobiliary ducts preoperatively. The role of routine EUS preoperatively when the size of the AA is less than 1 cm or no suspicious signs of malignancy like ulceration, induration or bleeding is not clear but is often performed per protocol at many centers. EUS has modest sensitivity of 77% and specificity of 78% for T1 lesions; sensitivity of 70% and specificity of 74% for nodal invasion. Many endoscopists use EUS universally before performing ampullectomy to evaluate the lesion thoroughly, others use it selectively, and some others do not use it at all.

Intraductal ultrasound (IDUS) allows EUS from inside the biliary and pancreatic ducts. IDUS probes can be inserted through the accessory channel of a duodenoscope during ERCP into either the biliary or the pancreatic duct. IDUS is superior to EUS in visualization of tumors of major duodenal papillae with accuracy of 100% vs 59.3% (IDUS vs EUS); Sensitivity of 100% vs 75% (IDUS vs EUS) and specificity of 62.5% vs 50% (IDUS vs EUS) respectively. Despite these benefits, IDUS is rarely performed given the additional cost and the need for a second EUS processor. Computed tomography (CT) and transabdominal ultrasound (US) are not adequate for staging for ampullary tumors but they can identify biliary and pancreatic duct dilation. CT can also identify locoregional lymph nodes and distant metastasis. Magnetic resonance cholangiopancreatography (MRCP) can assess the extent of intraductal involvement non-invasively and identify pancreas divisum, in addition to the identification of biliary and pancreatic dilation but small ampullary lesions are often missed on MRI with MRCP.

Management and Ampullectomy Technique: Management of AA depends on the size, presence of concurrent duodenal adenomatosis, characteristics of the adenoma, endoscopic expertise and willingness of the patient to undergo surveillance after papillectomy. In general, AA less than 2-3 cm are more amenable to endoscopic removal, but there are case reports of EA for lesions less than 4.5 cm if there is no intraductal growth or
malignancy. Endoscopic characteristics of the AA including firmness, ulceration, and non-lifting after submucosal saline injection are suggestive of possible malignancy and patients with these findings may not be candidates for endoscopic removal.

The goal of endoscopic management of patients with AA should be complete excision of all adenomatous tissue when feasible. (Figure 2) En bloc or piecemeal resection may be performed. The advantages of en bloc excision include accurate histological assessment because of clear margins, an increased likelihood of complete removal of the AA, and potentially decreased procedural time. However, for large AA or lesions with limited endoscopic accessibility, en-bloc resection may not be feasible. Piecemeal excision is usually performed in these cases.

The equipment used includes thin wire snare of approximately 0.3mm size and microprocessor-controlled electrosurgical generator. There is no specific type of snare that is universally recommended when performing EA. Many snares have been used to perform ampullectomy. Snare size should fit the size of the target lesion, if possible. Depending on the size and morphological characteristics of the lesion, a variety of stiff-type snares can be used. The Spiral snare (20-mm spiral SnareMaster; Olympus, Tokyo, Japan) is preferred by some endoscopists to enable more tissue capture. The mini oval Acusnare (15 x 30-mm mini oval, Cook Medical, Brisbane, Australia) can be used to remove the residual tissue from the margin. For large exophytic lesions, Acusnare (25 x 55-mm AcuSnare [standard oval], Cook Medical, Brisbane, Australia) can be used. The use of thin wire snare maximizes the current density for swift transection and minimizes the risk of dispersion of the energy to the pancreatic orifice and thereby theoretically reduces the risk of late stenosis. Final snare selection is left to the endoscopist.

The role of submucosal injection of saline in EA is not clear. The anatomy is such that, the duodenal papilla is continuous with the AV and the AV is a confluence of the terminal part of the pancreatic and common bile duct that extends deep in to the muscularis propria layer of the duodenum. As a result, when submucosal saline injection is

(continued on page 24)
used for excision of the duodenal papilla, there can be tethering of the duodenal papilla to the ductal structures and can theoretically lead to incomplete resection as the ampullary lesion may not lift as expected which can make effective snare placement for en bloc resection difficult. However, many authors prefer to perform submucosal injection of saline prior to ampullectomy as it may reduce the risk of perforation and facilitate tissue removal. Most centers use normal saline, although some can add indigo carmine (0.04%) and epinephrine (1:100000) for submucosal injection.

All specimens after EA should be ideally retrieved for histological evaluation. Anti-peristaltic agents like glucagon or hyoscine butylbromide can be used to prevent migration of the specimen into the intestine. Commercially available retriever net or endoscopic suction can be used to retrieve the tissue but aspiration through accessory channels of duodenoscope can lead to fragmentation of tissue. However, sometimes the specimen may be lost if it rapidly passes beyond the reach following ampullectomy. Pinning of the specimen after flattening on to cork board or polyesterene block can prevent curling of the specimen and can aid the pathologist for accurate assessment of lateral and deep margins. The duodenoscope is typically reintroduced after retrieving the specimen to examine for any signs of bleeding stigmata or active bleeding and residual adenomatous tissue. Ablation therapies including monopolar coagulation, bipolar coagulation, Nd:YAG laser, photodynamic therapy and Argon plasma coagulation (APC) can be used to treat residual adenomatous tissue based on the institutional availability and preference of the endoscopist. The benefit of ablation therapy is controversial, and some authors prefer APC than other modalities as it can limit the depth of tissue injury with the setting of 40-50 W. In general, APC is the most commonly used ablation method given its ease of use through the duodenoscope and widespread availability.

The role of routine prophylactic pancreatic stenting after ampullectomy to prevent pancreatitis is also not clear. Some authors advocate pancreatic stenting with 5 French stents only if pancreatic orifice is not visible after EA. Harewood et al. showed in a randomized study that patients who underwent pancreatic duct (PD) stenting after EA had decreased rates of post-ampullectomy pancreatitis when compared to those who did not undergo PD stenting. To prevent cholangitis from hemobilia when there is major bleeding and to ensure bile drainage, when there is concern for retroduodenal perforation, biliary stenting is recommended. To minimize the risk of pancreatic ductal injury after ampullectomy, the pancreatic stent should be removed in relatively short timeframe. Also, any residual visible adenomatous tissue can be removed at the time of pancreatic stent removal. While pancreatic duct stents are widely used when performing ampullectomy, not all endoscopists use them in this context.

Complications

Common early complications after EA include pancreatitis, bleeding, perforation, and cholangitis. Late complications include papillary stenosis, pancreatic duct stricture, bile duct stricture, and adenoma recurrence. Outcomes of EA are discussed in detail in Table 1. Pancreatitis can develop in up to 3-30% following EA. Prophylactic pancreatic duct stent can reduce the risk of developing pancreatitis and can reduce the severity of pancreatitis if it develops. Routine prophylactic pancreatic duct stent placement is advocated by some authors to prevent pancreatitis after EA, although some studies showed no difference among patients who underwent EA with or without a pancreatic stent. As EA constitutes a high-risk
ERCP, rectal indomethacin is recommended unless the patient has a contraindication.\textsuperscript{36,37}

Bleeding can be intraprocedural or delayed and it accounts for 2-30\% of adverse events following EA.\textsuperscript{30,38} Intraprocedural bleeding can usually be controlled with adrenaline injection, balloon tamponade, coagulation forceps, stenting, and/or hemoclip placement. Delayed bleeding can be mild and self-limited or severe and life threatening, however endoscopic intervention might be needed when there is hemodynamic compromise. Massive bleeding unresponsive to endoscopic intervention usually warrants angiographic embolization.

Perforation can be guidewire-induced, periampullary during sphincterotomy or luminal (usually occurring during the actual ampullectomy maneuver) and accounts for 2-10\% of EA resections.\textsuperscript{34,38} Guidewire perforations are usually not causes of significant clinical injury. Early recognition of perforation and conservative management with intravenous (IV) antibiotics, bowel rest, and IV fluids are often all that is needed in the case of small perforations, and many of these can be managed nonoperatively. Most perforations are small and retroperitoneal, and do not warrant surgery. In some cases, endoscopic closure can be accomplished with endoscopic clips. Surgical intervention is required if the patient shows signs of acute abdomen or decompensation. For distal common bile duct (CBD) or periampullary injuries, fully covered Self- expandable metal stent (SEMS) can be beneficial.\textsuperscript{39}

Cholangitis is uncommon after ampullectomy if a biliary stent is placed and can be managed with IV antibiotics. ERCP with stent placement or replacement may be necessary for biliary drainage if conservative management fails. Papillary stenosis (2-17\%) is usually a late complication after EA and it includes biliary and pancreatic duct stenosis.\textsuperscript{40,41} Papillary stenosis can arise as a consequence of scarring from the ampullectomy procedure itself. The treatment of papillary stenosis includes sphincterotomy, stent placement and balloon dilation. Catalano et al. showed in their study that papillary stenosis was seen more in patients who did not have pancreatic duct stent and they recommended prophylactic pancreatic duct stent to prevent post EA pancreatitis and pancreatic duct stenosis.\textsuperscript{30}

**Recurrence Rates and Follow Up**

Recurrence rates after EA vary from 11-30\%.\textsuperscript{25,42} Risk factors for recurrence include large size, genetic predisposition, possibly absence of adjuvant thermal ablation (laser, APC) during initial EA to treat residual tissue.\textsuperscript{30} Recurrence is usually treated with endoscopy and if there is intraductal invasion or cancer, then surgical intervention is recommended. (Figure 3)

There are no specific guidelines on the follow up after EA, however there is some consensus on initial follow up endoscopy at 3 months and once every 6 months for a period of 2 years. Once there is complete eradication or no recurrence after 2 years of follow up, yearly endoscopy afterwards is recommended.\textsuperscript{43,44} Some authors recommend that after 2 years of initial endoscopy follow up, FAP patients should undergo endoscopic surveillance every 2-3 years for the rest of the life as there is 100-330 fold of developing duodenal cancer.\textsuperscript{45} For sporadic AA, endoscopic surveillance can be performed as clinically indicated.\textsuperscript{25} In patients with genetic predisposition like FAP or Gardner syndrome, the goal of surveillance is to detect high grade dysplasia and large lesions are more likely to have high grade dysplasia and in patients with sporadic AA, the goal of surveillance is to detect recurrence at the excision site.\textsuperscript{46} The severity of duodenal adenomatosis is graded by spigelman (0-IV) and with grade III, IV, there is more risk of recurrence of AA and high-grade dysplasia which makes endoscopic papillectomy less feasible.\textsuperscript{41} In patients with complex histories or unusual situations, follow up can be individualized.

**CONCLUSION**

Endoscopic management of AA is safe and effective when appropriately selected in the hands of experienced advanced endoscopist. A multidisciplinary team including gastroenterology, radiology, pathology, oncology and surgery is key in management of AA. Surveillance for recurrence should be individualized based on pathology, risk factors like large size and genetic predisposition. Surgery is recommended when there is intraductal extension in to common bile duct and/or pancreatic duct with invasive cancer on biopsy or large ampullary lesions that cannot be endoscopically treated.
Clinical Update on the Endoscopic Management of Ampullary Adenoma

References

33. Patel R, Davitie J, Varadarajulu S, Wilcox CM. Endoscopic resection of ampullary adenomas: compli-
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