LANDMARK CASES

Case based discussions on interesting clinical problems managed at Sakra in first 100 days of starting the institute of gastroenterology and hepatobiliary sciences
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He completed his MBBS from Kasturba Medical College, Mangalore. He did his post graduation- MD (General medicine) from the same institution & DM (Gastroenterology) from S.P.G.I, Lucknow.

Dr. Dinesh Kini has a greater vision to establish a state-of-the-art Gastroenterology and hepatobiliary unit with a comprehensive approach associated with SGE/Interventional GE radiology and GE pathology services.

Previous Appointments
- Assistant Professor of Medicine, Kasturba Medical College, Manglore (1994 - 1995).
- Senior Consultant Gastroenterologist/Hepatologist, Manipal Hospital, Bangalore (2001 - 2009).
- Head – Department of gastroenterology, MILDD, Bangalore (2010 - 2012).

Field of Expertise
- Liver, Biliary Diseases
- Advanced endoscopy interventional gastroenterology
- Particularly difficult bile duct stone extraction
- Bile duct cancer and stenting of complex bile duct blocks
- Pancreatic endoscopy treatment
- Small bowl enteroscopy

International Work Experience
- Part of multiple international Clinical Trials.
Dr. Sadiq S Sikora had his MBBS and General Surgical training from the prestigious All India Institute of Medical Sciences (AIIMS), New Delhi. Later he moved to the prestigious Sanjay Gandhi Post Graduate Institute of Medical Sciences (SGPGIMS) at Lucknow for his specialization and subsequent faculty position. He then completed the “Surgical Oncology Fellowship” from the University of Pittsburgh, USA.

As the Johns Hopkins Institute, Baltimore, Maryland with Prof. JL Cameron, he gained insight into the nuances of pancreatic and biliary surgery, while on a UICC sponsored ICRETT fellowship.

Dr. Sadiq Sikora has a vast experience of 25 years in conducting specialized surgical procedures, which includes living related liver transplantation, complex pancreaticobiliary surgery for benign and malignant diseases, liver resection, surgery for portal hypertension, advanced laparoscopic surgery for gastrointestinal cancers.

Dr. Sadiq’s key Achievements

- He was the key member of the team, which established the living related liver transplant program at Kochi, Kerala.
- Dr. Sikora started the successful Liver Transplant Program (cadaveric and living donor) at Manipal Hospital, Bangalore.
- His major contributions are in the areas of bile duct injury repairs, biliary and pancreatic cancers, innovative techniques in Esophagus resection, pancreatic surgery and hepaticojejunostomy.
- Has been invited as a faculty in national and international conferences to deliver talks on pancreatic disorders, bile duct injuries and GI cancers.
- Has published more than 150 articles in international and national peer reviewed journals with significant contribution in the fields of repair of bile duct injury and pancreatic diseases.
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**Case Presentation**

► A 60-years-old male, a known case of chronic liver disease, viral markers were negative.
► The patient had been diagnosed to have ascites year ago and it was difficult to treat. It was unresponsive to diuretic treatment and the patient had been requiring large volume paracentesis (LVP) every 2 weeks.
► The renal function was also compromised and he was diagnosed to have hepatic hydrothorax since 2 weeks.

**Investigations**

► Apart from the routine complete blood count, liver function tests and renal function tests; ascites fluid tapping was done to confirm transudate (high SAAG).
► Ascitic fluid analysis ruled out SBP.

**Management**

► The patient was advised Transjugular intrahepatic portosystemic stent shunt (TIPSS). Post procedure his ascites improved and the dose of diuretics was tapered.
► There was significant improvement in the pleural effusion after 2 taps in 2 subsequent weeks.
► The blood creatinine levels reached within normal levels.
► During the follow-up the patient suffered from hepatic encephalopathy which was managed with lactulose and LOA.

**Discussion**

Ascites is a common complication of liver cirrhosis associated with a poor prognosis. It has been classified into high grade (>1.1g/dl) or low grade (<1.1g/dl) according to the serum-ascites albumin gradient (SAAG). High grade ascites is generally found in patient with cirrhosis, alcoholic hepatitis,
congestive cardiac failure, BCS, VOD, fatty liver of pregnancy, FHF or massive liver metastasis. Low grade ascites is present in patient with peritoneal carcinoma, tubercular peritonitis, disease of pancreas, biliary tract or in nephrotic syndrome and collagen diseases.

The primary goal of management on cirrhotic ascites treatment is the minimization of ascitic fluid volume and peripheral edema without intravascular volume depletion. It is achieved chiefly by limiting sodium intake to 88 mEq (2000 mg) per day (including all foods, liquids and medications). Most patients with cirrhosis and ascites are treated with dietary sodium restriction and diuretics and respond well to the treatment.

**Refractory ascites is defined as the one which:**

- is unresponsive to sodium restricted diet and diuretic therapy comprising of 400 mg spironolactone + 160 mg furosemide,
- spironolactone + 160 mg furosemide,
- rapidly after LVP and
- has complication of diuretic therapy (e.g., encephalopathy, S.Creat > 2mg, S.Na⁺ < 120, S.K⁺ > 6).

**There are 3 therapeutic options for patients with diuretic resistant or refractory ascites caused by liver cirrhosis:**

<table>
<thead>
<tr>
<th>Ref. No.</th>
<th>Inclusion Criteria</th>
<th>Method of Randomization and Analysis</th>
<th>N</th>
<th>Control of Ascites</th>
<th>Survival</th>
<th>Encephalopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>91</td>
<td>Tense ascites and failure of 4 weeks of therapy</td>
<td>No details</td>
<td>60</td>
<td>61% vs. 18% (p = 0.006)</td>
<td>69% vs. 52% (p = 0.11)</td>
<td>58% vs. 48%*</td>
</tr>
<tr>
<td>92</td>
<td>Ascites refractory to medical therapy</td>
<td>Sealed opaque envelope; Intention to treat</td>
<td>70</td>
<td>51% vs. 17% (p = 0.003)</td>
<td>41% vs. 35%*</td>
<td>All 77% vs. 66% (p = 0.29) Severe 60% vs. 34% (p = 0.03)</td>
</tr>
<tr>
<td>94</td>
<td>Refractory ascites</td>
<td>No details; Intention to treat</td>
<td>109</td>
<td>58% vs. 16% (p = 0.001)</td>
<td>40% vs. 37%*</td>
<td>Moderate- severe 38% vs. 12% (p = 0.058)</td>
</tr>
<tr>
<td>95</td>
<td>Refractory or recidivant</td>
<td>No details</td>
<td>66</td>
<td>79% vs. 42% (p = 0.001)</td>
<td>77% vs. 52% (p = 0.021)</td>
<td>Severe (p = 0.039)</td>
</tr>
</tbody>
</table>

*p value not significant.
Liver transplantation
Serial therapeutic paracentesis approximately every two weeks
Transjugular intrahepatic portosystemic stent shunt (TIPS)

Transjugular intrahepatic portosystemic shunt (TIPS) involves creation of a low-resistance channel between the hepatic vein and the intrahepatic portion of the portal vein (usually the right branch) using angiographic techniques and is used to treat the major consequences of portal hypertension such as variceal hemorrhage and diuretic resistant ascites. It is more advantageous that LVP because of the better control of ascites. Studies have demonstrated treatment free survival benefits with TIPSS however the major concern is the trend towards hepatic encephalopathy. Studies have also observed no change in quality of life when LVP was compared to TIPSS.

TIPSS though advantageous is contradicted in conditions like:
- Heart failure
- Severe tricuspid regurgitation
- Severe pulmonary hypertension
- Sepsis
- Intrinsic kidney disease
- Overt encephalopathy
- MELD >18
- CTP >12

Conclusion
The above mentioned case was an ideal candidate for TIPSS and responded to the treatment effectively.
CASE 2

An Unusual Cause of Ascites

Case Presentation
► A 39-year-old female presented to the emergency department with lower abdominal discomfort and mild ascites.
► She is a known case of diabetes mellitus.
► Had a history of LSCS twice (12 years and 7 years back).
► Also had a history of appendicectomy 13 years back.

Laboratory Investigations
► Hemoglobin: 10.7 gm%
► Total WBC count: 15,000/µl
► Platelets: 3,70,000/µl
► Liver function test: Normal
► Serum Creatinine: 0.79 mg/dl

Imaging Studies
Contrast Enhanced Computed Tomography (CECT) of abdomen showed (Figure 1):
► Gross ascites with subtle peritoneal thickening and mild omental thickening.
► No malignancy.
► No features to suggest cirrhosis/portal HTN.

The ascitic fluid was obtained for analysis and it showed:
► Albumin: < 1gm/dL
► Serum-ascites albumin gradient (SAAG): > 1.1
► Cell count: 83/µL
► Amylase: 74 U/L
► Cytology: Negative

Other tests results were as follows:
► ANA: Negative
► CA: 125 - Normal
► TSH: Normal
► UPT: Negative
► Viral marker: Negative
► OGD: Normal
► ECHO: Normal

Management

The review of ascitic fluid was not suggestive of any diagnosis and we were not convinced about its relationship with peritoneal thickness and high levels of SAAG in the fluid. We decided to re-examine the ascitic fluid and do a liver biopsy.

Meanwhile the patient was started on a salt restricted diet and diuretics. Ascites worsened with the treatment and warranted therapeutic tapping. The patient also developed oliguria/anuria over 12 hours. The serum creatinine level increased to 4.5 mg/dl. A Foley’s catheter was placed and with fluid challenge the oliguria improved. The ascitic fluid was sent for re-examination. The ascitic fluid creatinine increased to 7.99 mg/dl whereas the serum creatinine was 4 mg/dl. She was suspected to be having urinary ascites as her ascitic fluid creatinine was more than serum creatinine. Repeat CT scan done with injection of contrast through the Foley’s catheter.

The findings suggested that there was contrast leak from the posterosuperior right lateral wall of the
dome of the urinary bladder. Contrast leaks into the peritoneal cavity with moderate urinary ascites noted.

The patient was conservatively managed with Foley's catheter insertion for 4 weeks. Repeat imaging before removing Foley's catheter was done. A diagnosis of urinary ascites was made.

Discussion

Urinary ascites occurs when there is rupture of either the ureter or bladder, leading to leakage of urine into the peritoneal space. The causes include blunt trauma to the abdomen or iatrogenic, such as nicking the ureter during an abdominal surgery. Urinary ascites should be considered after usual causes of ascites, such as cirrhosis or nephrotic syndrome, have been excluded. An ascites fluid creatinine:serum creatinine ratio > 1.0 is highly suggestive of an intraperitoneal urine leak. The peritoneal fluid is typically bland with few WBCs. Smaller leaks are usually managed with a conservative approach while the larger leaks require surgery.
Case Presentation
A 76-years-old male was seen in the outpatient department with complaint of pain in abdomen. He had a history of open cholecystectomy 10 years back. Before coming to the current speciality he had been evaluated by 2 different multispecialty hospitals and was diagnosed to have large common bile duct (CBD) stone. He underwent 2 attempts of ERCP but the stone could not be extracted.

The challenge before the treating physician was whether to choose CBD exploration (surgery) or repeat the ERCP (non surgical method).

Discussion and Management
The currently available treatment options for large CBD stones (>1.5cm) include:

- EST followed by large balloon dilation (ESLBD)
- Mechanical lithotripsy
- ESWL
- Laser lithotripsy through Spyglass system
- Biliary endoprosthesis (stenting)
- LAP CBD exploration

Peroral cholangioscopy was performed in this patient and large stone was fragmented with laser. Stone fragments were cleared using basket and balloon sweeping. However, he required two session to clear all the CBD stone fragments.

Figure 1: Single operator system “Spyglass”
Conclusion

In this elderly man surgery was avoided and large CBD stones was extracted with endoscopic method.
Case Presentation

► A 36-year-old male who was a known case of extrahepatic portal venous obstruction (EHPVO) had a splenectomy done with splenorenal shunt surgery in 2011.

► He also had a history of variceal bleed for which esophageal varices ligation was done in 2012.

► He developed obstructive jaundice in 2013 and underwent endoscopic retrograde cholangio-pancreatography (ERCP) and common bile duct (CBD) stenting for portal biliopathy (CHD stricture).

► He also had a history of chronic kidney disease and the biopsy was suggestive of malignant proliferative glomerular nephritis.

► The presenting complaints included cholangitis with septicemia.

► He underwent ERCP in middle East with change of stent however the sepsis persisted and the serum creatinine levels were 5 mg/dl.

Investigations and Management

The patient was hospitalized and stabilized in an intensive care unit (ICU). The laboratory investigations indicated:

► Total cell count: 28,800/mm³

► Total bilirubin: 3.2 mg/dl

► ALT: 34 units/L

► Alkaline phosphatase: 574 IU/L

► Serum creatinine: 2.04 mg/dl

USG abdomen showed intrahepatic biliary dilatation (IHBRD) with stent in CBD. The physician decided that the patient must undergo either a magnetic resonance cholangiopancreatography (MRCP) or ERCP for the probable stent block.

ERCP and stent exchange was done. However, he continued to have fever. The laboratory values showed a change as follows:

► Total cell count: 33000 /mm³ to 26800/mm³

► Total bilirubin: 4.2 mg/dl to 2.3 mg/dl

► Alkaline phosphatase: 422 IU/L to 584 IU/L
Total cell count: 14,000/mm³
Total bilirubin: 1.1 mg/dl
Alkaline phosphatase: 456 IU/L
Serum creatinine: 1.39 mg/dl

The laboratory values were as follows:

Patient had good clinical improvement

Repeat imaging done which revealed:

- Post stenting status with stents in the left biliary ductal system.
- Hepatomegaly with dilatation of the right biliary ductal system secondary to portal biliopathy. Repeat ERCP was done.
- Multiple stents were placed into right and left ductal systems.

The next steps of management had to be one of the following options:

- Biliary bypass (HJ) vs Endoscopic treatment
- Liver transplant

A surgical gastroenterologist opinion was sought, who opined that he requires liver transplant at a later date. The patient was electively admitted when he was asymptomatic and the laboratory tests were normal (TB – 1.1 mg/dl, ALP – 210 IU/L, TC - 10,500/mm³). An USG of abdomen was done which showed:

- Residual dilatation of the right lobe intrahepatic biliary ducts noted. All the right lobe hepatic ducts are seen communicating with each other.
Left biliary ducts are collapsed.
Small calculus noted in the gall bladder.
Post splenectomy status.

**Discussion**

Portal hypertensive biliopathy (PHB) refers to abnormalities of the entire biliary tract including intra and extrahepatic bile ducts, cystic duct and gallbladder in patients with portal hypertension. It is not confined to EHPVO, but is also seen in patients with portal hypertension due to cirrhosis of liver and NCPF.

Prospective studies indicate that 81% to 100% of patients with EHPVO have PHB on ERC however only minority has symptoms.

**PHB caused by:**
- Pressure on the bile ducts from collaterals
- Ischemic injury to bile ducts during portal vein thrombosis

**The clinical picture is generally asymptomatic however may include:**
- Chronic cholestasis, likely to be caused by biliary stricture
- Biliary pain or acute cholangitis, likely to be caused by stricture and secondary biliary stones
- Secondary biliary cirrhosis

**Conclusion**

The management of PHB includes treatment of portal hypertension and relief of obstructive jaundice by shunt procedure by endoscopic method, Hepaticojejunostomy (HJ) or Liver transplantation if already there is secondary biliary cirrhosis.
Case Presentation

► A diabetic patient (DM since 7 years) presented with complaints of weight loss and intermittent fever since 4 years.

► He also had history of osteoporosis with a fracture of D12 and L3 vertebrae.

► He was investigated in several hospitals and his USG abdomen revealed bulky pancreas.

► His liver function tests were normal, Cancer antigen (CA) 19-9 was 420.

► Contrast enhanced computed tomography scan (CECT) of abdomen with MRI and MRCP was already done.

► He was evaluated in many hospitals with CECT/MRCP/EUS/PET CT/DOTANAC SCAN.

In April 2011 the patient underwent endoscopic ultrasound (EUS) to rule out cystic neoplasm and intraductal papillary mucinous neoplasm (IPMN). Fine needle aspiration cytology (FNAC) suggested NET. The physician suspected acute pancreatitis and the investigated further. The serum chromogranin levels were 542 and IgG4 was normal. A PET CT was done which revealed:

► Bulky, heterogenous somatostatin receptor avid whole of the pancreas, suggestive of diffuse neuroendocrine tumor.

► No evidence of distant metastates.

In March 2014 the liver function tests showed mildly elevated ALP and GGTP. CECT abdomen was repeated and showed mildly dilated biliary system. The serum chromogranin levels were 900. The physician reviewed all the older images with suspicion of autoimmune
pancreatitis (AIP). The patient was managed conservatively with steroids.

After a month of treatment the patient was readmitted with abdominal pain, pruritis and fever. The liver function tests revealed cholestatic picture. ERCP was done which revealed lower CBD stricture (benign) and managed with two stents placement.

**Discussion**

AIP is a distinct chronic inflammatory and sclerosing disease of the pancreas characterized by dense infiltration with lymphocytes and plasma cells many of which express IgG\(_4\) on their surface. Studies from Japan indicate that 6% of all patients evaluated for chronic
Pancreatitis have autoimmune pancreatitis.

The most common extrapancreatic conditions includes:

- Biliary strictures,
- Hilar lymphadenopathy,
- Sclerosing sialadenitis,
- Retroperitoneal fibrosis,
- Tubulointerstitial nephritis.

AIP generally overlaps with an unusual variant of Sjögren’s disease.

Epidemiology

- More common in men (2:1) and usually in middle age.
- More than 85% of patients are above 50 years.

Most common presentations include

- Painless obstructive jaundice -by enlarged pancreas/Infiltration of the biliary tree by inflammatory process.
- Pancreatic mass which is often confused with pancreatic carcinoma or lymphoma. Present in 85% of patients in an early report of 26 patients.
- Weight loss, vomiting, and glucose intolerance.
- Abdominal and referred back pain may occur.

The investigations recommended include

- Elevated serum immunoglobulins in 50-66%, especially in IgG_4.
- A large study US, using an IgG_4 cut-off of 140 mg/dl as normal and 280 mg/dl as diagnostic for AIP.
- Found a sensitivity of 76% and a specificity of 93.

<table>
<thead>
<tr>
<th>Table 1: Comparing AIP-1 and AIP-2</th>
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<tbody>
<tr>
<td>AIP -1</td>
</tr>
<tr>
<td>• IgG_4 elevations</td>
</tr>
<tr>
<td>• LPS (Lymphoplasmacytic sclerosing pancreatitis)</td>
</tr>
<tr>
<td>Older age</td>
</tr>
<tr>
<td>More likely to have biliary tract disease, retroperitoneal, renal, or salivary gland disease</td>
</tr>
<tr>
<td>High relapse rate</td>
</tr>
<tr>
<td>Less likely associated with IBD</td>
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</tbody>
</table>

Older age

More likely to have biliary tract disease, retroperitoneal, renal, or salivary gland disease

Without systemic involvement

High relapse rate

Do not experience relapse

Less likely associated with IBD

More frequently associated with inflammatory bowel disease
### Table 2: Criteria for diagnosis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Mayo CLINIC HISORt CRITERIA</th>
<th>ASIAN CONSENSUS CRITERIA</th>
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<tbody>
<tr>
<td>Histology</td>
<td>At least of the following: Lymphoplasmacytic infiltration with fibrosis and abundant IgG4 – positive cell infiltration</td>
<td>Lymphoplasmacytic infiltration with fibrosis and abundant IgG4 – positive cell infiltration</td>
</tr>
<tr>
<td></td>
<td>Periductal lymphoplasmacytic infiltrate with obliterative phlebitis and storiform fibrosis</td>
<td></td>
</tr>
<tr>
<td>Imaging</td>
<td>Lymphoplasmacytic infiltrate with storiform fibrosis with abundant IgG4 – Positive plasma cells(≥ 10/HPF)</td>
<td>Both of the following: Diffuse segmental, or focal enlargement gland, with or without a mass or hypoattenuated rim</td>
</tr>
<tr>
<td></td>
<td>Typical: Diffusely enlarged gland with delayed rim enhancement, diffusely irregular and attenuated pancreatic duct</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Other: Focal pancreatic mass or enlargement, focal pancreatic duct stricture; pancreatic atrophy, calcification or pancreatitis</td>
<td>Diffuse segmental, or focal pancreatic narrowing with or without stenosis duct</td>
</tr>
<tr>
<td>Serology</td>
<td>Elevated serum IgG4 level</td>
<td>One of the following: High levels of total IgG or IgG4, Detection of autoantibodies (ANA, RF)</td>
</tr>
<tr>
<td>Other organ involvement</td>
<td>Hilar or intrahepatic biliary strictures, distal (intrapancreatic) bile duct stricture, parotid or lacrimal gland involvement, mediastinal lymphadenopathy, retroperitoneal fibrosis</td>
<td>Not included in criteria</td>
</tr>
<tr>
<td>Response of glucocorticoid therapy</td>
<td>Resolution or marked improvement of pancreatic or extrahepatic manifestations</td>
<td>Included as an optical criteria, but only in patients with both imaging criteria above with a “negative workup” for pancreatobiliary cancer</td>
</tr>
</tbody>
</table>

- Histologically proven AIP may have a normal serum level of IgG4.
- 10% of patients with adenocarcinoma may have elevations in IgG4 levels.

### Treatment

Autoimmune chronic pancreatitis may progress rapidly, from the initial symptoms to end-stage chronic pancreatitis, within months. Glucocorticoids therapy causes rapid resolution of both symptoms and radiographic abnormalities. 30 to 40 mg of prednisone orally per day for four to eight weeks is recommended. Pancreatic imaging should be repeated at four weeks, to assess clinical response. Once response is as desired, prednisone dose is tapered at a rate of 5 mg per weeks. Generally, 50-75% patients responds to glucocorticoids, but about 25 percent requires a 2nd course of treatment, while a smaller proportion needs continuous treatment. Time to response is variable but usually 2 weeks to 4 months CT findings of diffuse swelling and a halo (a hypoattenuating rim) are predictive of a favorable response to glucocorticoids. Predictors of a suboptimal response include ductal strictures and a focal mass-like swelling that persists after resolution of diffuse changes.
Endoscopic Treatment for Esophageal Perforation

Case Presentation

A 35-year-old male was referred to a specialist for esophageal perforation. The presenting symptoms consisted of tachypnea and left pleural effusion.

CECT of chest and abdomen was done.

The findings were as below:

- Leakage of contrast through the lower end of esophagus with associated hydropneumothorax and passive collapse of left lung.
- Normal distal passage of oral contrast agent in duodenum and jejunum.

Intercostal drain (ICD) was placed into the left side of chest and further evaluated.

Esophageal covered self-expandable metal stents (SEMS) was placed for leak. He had multiloculated collection which was drained under CT guidance. Antibiotics were started as per the culture and sensitivity of the pleural fluid and slowly the patient was started on oral feeds.

The patient again had non-bilious vomiting. Stent block, migration or reflux was thought to be the possible reasons. Dilute barium swallow studies were conducted which revealed reflux of contrast by the side of the stent.

Nasojejunal (NJ) feeds were given and prokinetics were added to his treatment. His ICD volume decreased over time and after a week NJ tube got dislodged.

He was slowly started on oral feeds and discharged. After 7 weeks he came back for stent removal, stent was removed endoscopically. Previous rent has completely healed.

Discussion

Esophageal perforation is a rare condition. Cases are often
iatrogenic secondary to esophageal instrumentation (60%); 15-30% cases are because of Boerhaave syndrome (spontaneous rupture of the esophagus). Other causes include trauma, foreign body ingestion, and operative injury. The goals of therapy are:

► Closure of the perforation
► Drainage of associated contamination
► Establishment of enteric nutrition
Operative therapy was the surgery of choice for the treatment of esophageal perforation however new endoscopic techniques have expanded the management options. The endoscopic therapy consists of:

- Esophageal stenting
- Endoscopic clips

**Conclusion**

Esophageal stenting was done by SEMS in this patient and perforation was sealed. Later rent was found to be completely healed after removing the stent (after 7 weeks).
Case Presentation

- A 49-year-old diabetic female with a history of parathyroid surgery (a year back) and hypothyroidism presented with dyspepsia and marginal weight loss of 6 months duration.
- She had no other clinical signs and symptoms.
- Her endoscopy was performed in October 2013, which showed a small (8 mm) submucosal lesion in the first portion of the duodenum.

Investigations and Management

- HPE and Immunohistochemistry suggested neuroendocrine tumor.
- A contrast enhanced computerized tomography scan of abdomen was done which revealed fatty liver.

According to World Health Organization (WHO) (2000/2004), the neuroendocrine tumor is classified as follows:

- well differentiated neuroendocrine tumor (Benign behavior),
- well differentiated neuroendocrine tumor (Uncertain behavior),
- well differentiated neuroendocrine carcinoma (Low grade malignant),
- poorly differentiated neuroendocrine carcinoma.

The criteria for classification include:

- Size
- Functioning/ non-functioning
- Local Invasion

Figure 1: Histological Findings

- Vascular invasion
- Ki67
- Metastases

In the case presented above the serum chromogranin levels were measured and found to be 61.41 ng/ml in October 2013 and 130 ng/ml in March 2014.

The management had to be decided between endoscopic management, surgery or routine
medical management and follow-up. The endoscopic approach was chosen. EMR was done and tumor was resected. However, she had a rent which was closed immediately with clips.

- Post procedure CT abdomen with oral contract was done and no leakage of the contrast or pneumoperitoneum was seen.
- The patient was further managed with NPO, IV antibiotics and analgesics.
- She was discharged after 5 days.
- The patient visited the hospital after 4 weeks for follow-up and an endoscopy was done which was suggestive of complete cure.

Discussion
Management of carcinoid tumors is a challenge and often the following is suggested:

- Endoscopic excision of primary duodenal carcinoids appears to be appropriate for tumors < 1 cm.
- Duodenal carcinoids smaller than 2 cm may be excised locally; for tumors between 1 cm and 2 cm, complete resection is ensured by operative full-thickness excision. Follow-up endoscopy is indicated.
- Appropriate management of tumors larger than 2 cm can be problematic. These tumors can be treated with operative full-thickness excision and regional lymphadenectomy.

Conclusion
Endoscopically carcinoid tumors < 1 cm can be resected. However, in this case there was perforation. Timely detection and closing the perforation with clips helped us to prevent surgical exploration and patient was managed conservatively.
Case presentation

► A young male had syncope when he was in market and found in the midst of altered blood pool.
► He was resuscitated in the emergency department and shifted to MICU.

Course of the patient and management

► On investigation his hemoglobin (Hb) was 9 gm%.
► Gastroduodenoscopy was normal.
► Colonoscopy showed few aphthoid erosions in the terminal ileum.
► Contrast enhanced computer tomography scan (CECT) of abdomen with angiography was normal.
► He remained stable during the ICU stay with no further drop in the Hb levels. However, our patient who had a major bleed requires further evaluation to known the hidden site of bleeding (Obscure GI bleed).

The causes of OGB include:

► Angiodysplasia,
► Dieulafoy’s lesions,
► Erosions/ulcers,
► Crohn’s disease,
► Small bowel varices,
► Tumors,
► NSAID enteropathy,
► Radiation enteritis,
► Small bowel diverticulosis,
► Small bowel polyp,
► Aortoenteric fistula,
► Meckel’s diverticulum,
► Infectious diseases (Whipple disease, mycobacterium avium intracellulare, tuberculosis, cytomegalovirus, AIDS, helminthiases),
► Eosinophilic enteritis,
► Infiltrative diseases (amyloidosis, sarcoidosis),
► Acute graft-versus-host disease after bone-marrow transplant,
► Portal hypertensive enteropathy,
► Ischemic enteropathy.

Various diagnostic methods can be used for such patients which include

ENDOSCOPIC METHODS:
► Re-look routine upper GI endoscopy and colonoscopy
► Push enteroscopy (PE)
► Double balloon/Single balloon enteroscopy
► Capsule endoscopy (CE)
► Intraoperative enteroscopy

IMAGING TECHNIQUES:
► Small bowel series and enteroclysis
► CT Enterography and Enteroclysis
► MR Enteroclysis
► CT Angiography
► Radionuclide imaging
► Catheter directed angiography/Digital subtraction angiography

Figure 3: Enteroscope (a and b) and capsule endoscope (c)
Conclusion

In our patient we did deep enteroscopy for oral and aboral end which revealed deep ulcers in proximal ileum, which could not be reached through regular colonoscope. Biopsies from the ulcer were non-specific. Widal test was negative. The patient was managed conservatively and after a follow-up of 2 months there was no repeat episodes of bleeding.
Case presentation

► An elderly female was brought to the emergency department and hospitalized with complaints of 2 episodes of melena and one episode of hematemesis.
► She was resuscitated and managed in medical intensive care unit (MICU).
► Her hemoglobin was 10.5 gm% and there was history of NSAID use.

Further investigations and management

Emergency OGC was done in ICU and results showed no major lesions seen (Ulcer, Vx, Mw tear, growth etc). However altered blood was seen in duodenum.

The possible causes included:

► Hemobilia
► Dieulafoy’s
► GIST in a blind area

A CECT abdomen with CT angiography was done which revealed a duodenal diverticulum and a suspected vessel in diverticulum.

The endoscopy was repeated with side viewing scopy which revealed duodenal diverticulum with a ulcer and visible vessel which was clipped endoscopically.

Discussion

Duodenal Diverticular (DD) bleed:

Duodenum is second most common site of diverticular in alimentary tract after colon. Second part is most common site with 85 to 90% of total DD. These occur mainly in later decades of life with peak
incidence between 50 and 60 years of age and it increases with age. Most of the DD are asymptomatic and only about 10% of duodenal diverticula produce symptoms.

**The symptoms include:**

- Epigastric pain, nausea and vomiting
- Pressure – jaundice/ cholangitis/ pancreatitis /obstruction
- Diverticulitis, Abscess, Perforation and Bleed
- Bezoar formation
- Adenocarcinoma

It has been reported that bleed from a duodenal diverticulum occurs in 7% of people in DD. It is caused by ulceration because of ectopic gastric mucosa or inflammation. NSAIDs have also been attributed to development of ulcerations. Treatment options include endoscopic haemostasis, embolization and surgery.
**Facilities: Gastroenterology and Hepatopancreatico Biliary Sciences**

### Medical Gastroenterology Services

#### ESOPHAGEAL DISEASES

**Diagnostic services**
- Upper GI endoscopy for
  - Retrosternal burning sensation and reflux disease
  - Difficulty in swallowing
- Endoscopy with NBI for
  - Early detection of esophageal cancers
- Manometry for difficulty in swallowing
- pH Metry for reflux disease
- Endoscopic Ultrasound for
  - Evaluation of submucous lesions/ growth
  - Assessment of resectability of tumors

**Therapeutic services**
- Control of GI bleeding – EVL/EST
- Foreign body removal
- Esophageal stricture dilatation
- Esophageal stent placement
- Ballon dilatation for Achalasia
- EMR/ ESD for early esophageal cancer

#### STOMACH DISEASES

**Diagnostic services**
- Upper GI endoscopy for evaluation of
  - Ulcer disease
  - Bleeding
  - Cancers/ polyps
  - Anemia
  - Vomiting

**Endoscopic Ultrasound for**
- Evaluation of submucosal lesions
### Medical Gastroenterology Services

#### Therapeutic services for
- Bleeding ulcer – Injection therapy, Clipping of vessel and thermal ablation
- Glue injection for gastric varix bleed
- Stent placement for gastric outlet obstruction
- Feeding tube/ PEG tube placement
- Foreign body removal
- APC for GAVE
- EMR/ESD for early gastric cancer
- Ballon placement for treatment of obesity

#### PANCREATICO BILIARY DISEASES

##### Diagnostic services
- **Endoscopic Ultrasound for**
  - Detection of small bile duct stones and tumors
  - Diagnosis of early Chronic Pancreatitis
  - Evaluation of Pancreatic Cancers
  - Tissue acquisition from lymph nodes, and pancreatic lesions
- **Choledochoscope/Spyglass**
  - For evaluation of bile duct stricture
  - To rule out bile duct cancers

##### Therapeutic services
- **ERCP for**
  - Metal and plastic stent placement for biliary obstruction/ portal biliopathy
  - Bile duct stone removal
  - Endoscopic management of bile duct injuries
  - Pancreatic duct stone removal
  - Accessory pancreatic duct sphincterotomy and stenting for Pancreas divisum
  - Drainage of pancreatic pseudocyst
  - Pancreatic duct stenting for -
    - Pancreatic duct stricture
    - Pancreatico pleural fistula
    - Traumatic pancreatic duct disruption
- **Endoscopic necrosectomy**
- **Endoscopic Ultrasound for**
  - Drainage of pancreatic pseudocyst
  - Celiac gangion block/neuronolysis
## Medical Gastroenterology Services

### SMALL INTESTINAL DISEASES

**Enteroscopy for diagnosis and management of small intestinal**
- Bleeding
- Tumors

### COLONIC DISEASES

**Diagnostic services**
- Colonoscopy for
  - Blood in stool
  - Long standing diarrhea
  - Colonic obstruction
  - Detecting cancers
- Endoscopy with NBI for
  - Early detection of colonic cancers
- Manometry for
  - Severe constipation (due to pelvic floor dysfunction)
  - Fecal incontinence
- Endoscopic Ultrasound for
  - Evaluation of anal sphincter defects

**Therapeutic services for**
- Polypectomy for colonic polyps
- Endoscopic mucosal/ submucosal dissection for precancerous lesions and early cancers
- Colonic decompression
  - Tumor related obstruction (endoscopic metal stent placement)
  - Non tumor related (Volvulus and pseudo obstruction)
- Colonic/ piles bleeding( Banding/ injection therapy/ Clip placement)
- Biofeedback for severe constipation and fecal incontinence

### SPECIAL OPD SERVICES

**Hepato pancreato biliary clinic for**
- Iver, gall bladder, bile duct and pancreatic diseases

**Luminal clinic for**
- Chronic diarrhea, Ulcerative colitis, Crohn’s disease, Swallowing disorders, Dyspepsia and Reflux disease
Facilities: Gastroenterology and Hepatopancreatico Biliary Sciences

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