Endoscopic biopsy: a simple guide for beginners

In routine endoscopic practice, usage of biopsy forceps is inevitable. But most of the times we have to work under time constraints, particularly in high volume centers and we are in a hurry to complete the procedure, especially when we need to take multiple biopsies. Taking a biopsy in a hurry may lead to mucosal trauma, bleeding and possible perforation.[1,2] It is sometimes difficult to observewhen to slow down the insertion of biopsy forceps through the working channel of the endoscope to decrease chances of trauma, particularly for beginners.

We have observed that the endoscopist has to push the biopsy forceps 16 to 20 times before it reaches the gastrointestinal lumen in the standard gastroscope. When the endoscopy assistant starts extending his elbow and when his arm crosses the level of trunk towards endoscopist, only 2 to 4 further pushes are needed for the biopsy forceps to reach the lumen (**Figures 1 & 2**). So by just observing assistant's movements which usually lie within the visual field of the endoscopist, we can slow the rate of pushing the biopsy forceps once the assistant's arm crosses the level of the trunk. Following this method reduces the chances of trauma and results in the smooth introduction of the biopsy forceps into the gastrointestinal lumen. This holds good for standard gastroduodenoscopes, colonoscopes and enteroscopes as well, when standard biopsy forceps recommended by endoscope manufacturers are used. We feel our observation or any other similar relevant observations by endoscopists can be helpful in avoiding trauma to the mucosa and damage to biopsy forceps, especially for the beginners.

Figure: 1 The assistant's elbow in flexion position and arm (black arrow) at the level of trunk (arrow with dotted outline) whilst initially introducing the biopsy forceps.

Figure: 2 After flexion shoulder along with extension at the elbow (black arrow) and once arm crosses the level of trunk (arrow with dotted outline) of the assistant, biopsy forceps (red arrow) reaches the lumen and comes into view.

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Successful living donor liver transplantation with partial nephrectomy for co-existing renal cell carcinoma and cirrhosis

Introduction

Patients with cirrhosis are at a high risk of liver failure and mortality following non-transplant surgery.[1,2] The presence of

co-existing extra-hepatic malignancy is generally considered a contraindication to liver transplantation thereby limiting the treatment options in such patients. There are only a few reports in the literature describing a combination of deceased donor liver transplantation with resection of extra-hepatic tumours.[3-5] We describe the evaluation and management of a case of hepatitis B virus (HBV) related cirrhosis who was incidentally detected with renal cell carcinoma (RCC) on pre-transplant imaging.

Case report

A 47-year-old lady, a known case of HBV related liver cirrhosis for 3 years, presented to us for evaluation for living related donor liver transplantation (LDLT). She had been previously treated medically for spontaneous bacterial peritonitis. She had no history of hepatic encephalopathy or variceal bleeding. The patient was taking antiviral treatment for HBV and diuretics for the last 3 years. Abdominal examination revealed the presence of ascites and splenomegaly. Her hematological and biochemical investigations were as follows: Hemoglobin 10.7 (normal, 11-15) g/dL, total leucocyte count 5100 (normal, 4000-10000) cells/mm^3, platelet count 54000 (normal, 150-450) cells/mm^3, international normalized ratio (INR) 1.48, total bilirubin 1.8 (normal, 0.2-1) mg/dL, aspartate aminotransferase (AST) 57 (normal, 0-42) U/L, alanine aminotransferase (ALT) 23 (normal, 0-60) IU/L, alkaline phosphatase 155 (normal, 39-117) IU/L, gamma-glutamyl transpeptidase 42 (normal, 0-64) IU/L, total protein 6.9 (normal, 6.6-8.7) g/dL, serum albumin 2.4 (normal, 3.5-5) g/dL and serum creatinine 1.9 (normal, 0.6-1.3) mg/dL. Renal dysfunction improved with human albumin infusion and cessation of diuretics. HBV-DNA was below detectable levels. Her Child-Turcotte-Pugh score was 10/15 (Child's C) and MELD at admission was 19. Contrast enhanced computed tomography (CECT) scan revealed cirrhosis, ascites, dilated but patent portal vein and splenomegaly (**Figure 1**). There was no evidence of hepatocellular carcinoma. Incidentally, a 2 cm size lobulated hypodense cyst with heterogeneous enhancement (non FDG avid on Fludeoxyglucose Positron Emission Tomography [FDG PET-CT], was noted in the right kidney (**Figure 2**). A diagnosis of HBV related cirrhosis (decompensation with ascites) with right renal cyst, Bosniak category III/IV (likely malignant) was made.

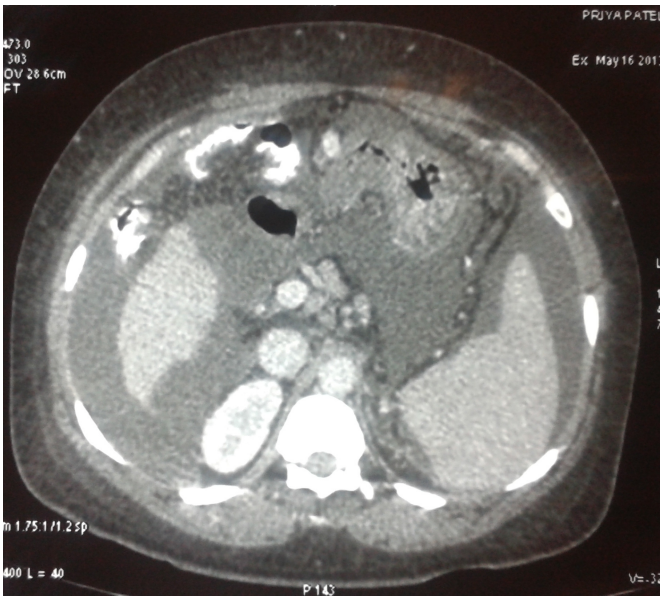

Figure 1: CT showing a shrunken nodular liver ascites splenomegaly and patent portal vein

DTPA scan revealed a total glomerular filtration rate (GFR) of 52.92 mL/minute. Differential GFR was 24.5 mL/minute in the left kidney (46.3%) and 28.44 mL/minute (53.7%) in the right kidney.

The patient underwent right lobe LDLT with partial right nephrectomy. During the surgical procedure, recipient hepatectomy was done first followed by a temporary portocaval shunt. Partial right nephrectomy was done next followed by disconnection of temporary portocaval shunt. Right lobe implantation was done. Graft weight was 870g. Graft Recipient Weight Ratio (GRWR) was 1.1. Frozen section for the partial nephrectomy specimen confirmed the presence of malignancy. The margins of the lesion were free of tumor. The patient was extubated on post-operative day 2. Serum creatinine during the first post-operative week ranged between 1.6-2.2 (normal, 0.6-1.3) mg/dL. Apart from steroids, rest of the immunosuppressive drugs had to be withheld (due to thrombocytopenia and abnormal serum creatinine values) till day 7 by when the creatinine normalized. Post-operative course was also marked by ascites and hyperkalemia which responded to medical management. The patient was discharged on post-operative day 21 with normal serum potassium, creatinine and liver functions.

Histopathological examination (HPE) of the explanted liver revealed mixed macro- and micro-nodular cirrhosis. HPE of the partial nephrectomy specimen revealed a well circumscribed tumour composed of small groups of large polyhedral cells with clear cytoplasm with abundant fibrovascular stroma and focal lymphomononuclear infiltrate (**Figure 3**). The margins were free of tumor. Tumor cells were positive for cytokeratin. The final diagnosis of the renal lesion was right renal cell

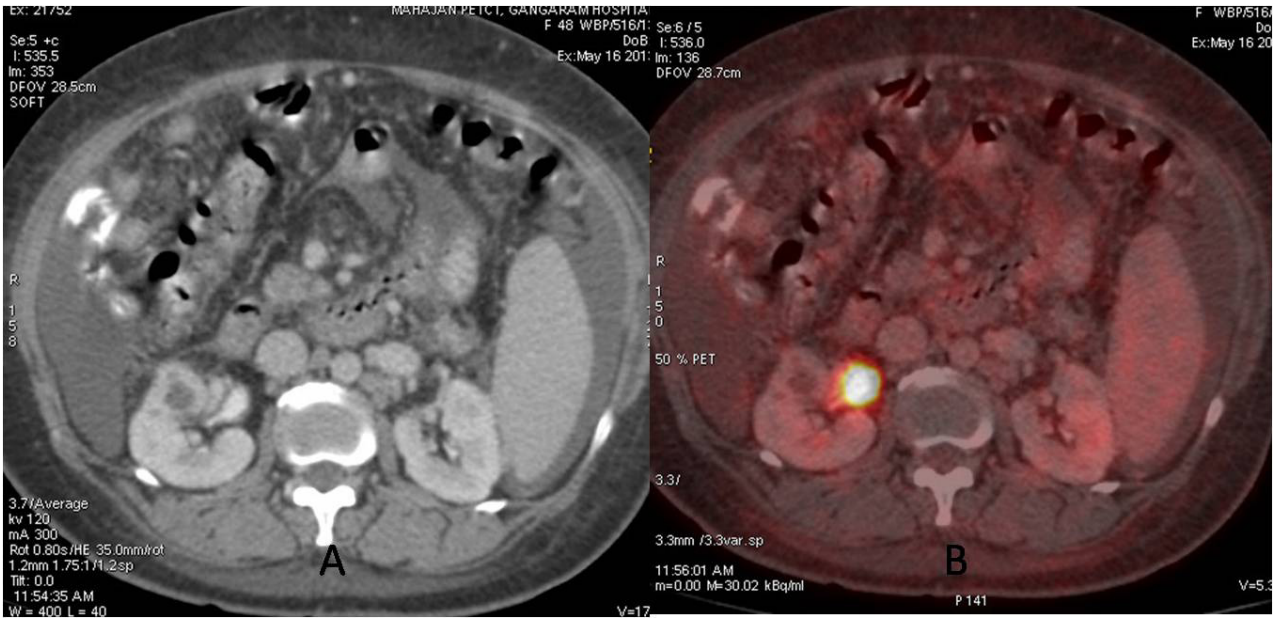

Figure 2: CT showing a heterogeneously enhancing cystic lesion (Bosniak type IIIIV) in right kidney (A) which was not FDG avid on FDG PETCT (B)

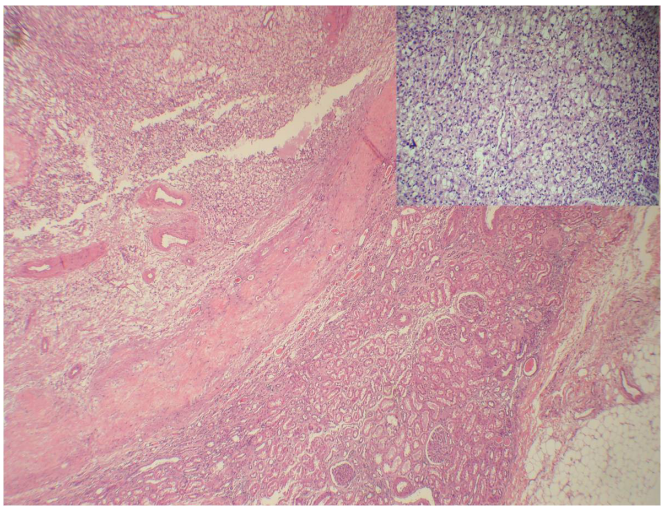

Figure 3: Photomicrograph showing a well encapsulated tumour with normal renal parenchyma in the periphery (HE 100x) Inset shows the polyhedral tumour cells with abundant clear cytoplasm (HE 200x).

carcinoma, clear cell variant, Fuhrmann grade I (Stage T1aN0M0).

Patient has been followed for 15 months with normal liver and renal functions without any evidence of recurrence of renal cell cancer on follow-up imaging.

Discussion

Patients with cirrhosis and extra-hepatic malignancy present a unique challenge. The risk of bleeding and decompensation with non transplant surgery has to be balanced against the risk of tumor recurrence with immunosuppression following simultaneous liver transplantation and resection.

This patient had an incidentally detected early stage unilateral renal lesion suspected for malignancy. FDG PET-CT was done in this patient to clarify the nature of this lesion and also to rule out disseminated disease. The lesion was not FDG avid and there was no evidence of abnormal FDG uptake elsewhere. It is well known that FDG is excreted through the urine and therefore small primary lesions may be missed on FDG-PET. Therefore, accuracy of FDG-PET is less than CT for the primary tumor. However, FDG-PET is more sensitive than CT for detection of metastatic disease.[6]

In our patient, the risk of decompensation and mortality associated with nephrectomy in the setting of cirrhosis, and a possibly higher risk of recurrence after liver transplantation (in view of the need for immunosuppression post transplant) were considered and discussed between the treating team, patient and patient's family members following which a decision was taken to perform partial right nephrectomy with right lobe LDLT.

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Pancreatic endotherapy in management of pancreato-pericardial fistula

Acute or chronic pancreatitis is associated with complications like pseudocyst, pancreatic necrosis, splenic vein thrombosis, pancreatic ascites and pleural effusion. Rarely do we find a patient presenting with cardiac tamponade and gross pericardial effusion due to a pancreaticopericardial fistula. Here we report a case of pancreaticopericardial fistula complicating alcoholic chronic pancreatitis, managed successfully by ERCP and stenting.Surgery, generally considered treatment of choice, was avoided.

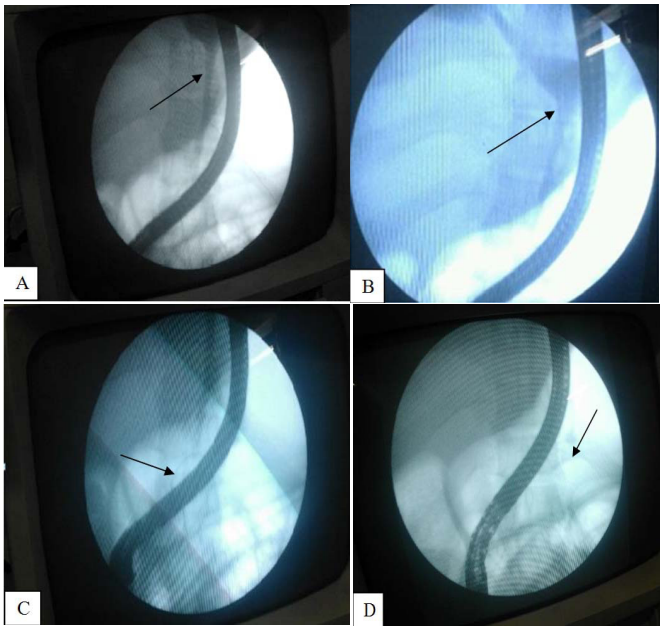

Figure: 1 Upper panel (A & B) shows pancreatopericardial fistula during ERCP. (C) Arrow shows guidewire in the pancreatic duct. (D) Arrow pointing towards pancreatic stent bridging the leak

Case report

A 38-year-old male patient presented to the emergency department with a 2-day history of severe breathlessness, chest pain and abdominal pain. His symptoms had gradually progressed over 1month with dyspnea progressing from NYHA class 1 to class 4.Hewas admitted in medical intensive care unit with hypotension, tachycardia, pallor, and tachypnea. Examination revealed muffled heart sounds, elevated JVP, normal respiratory examination. Abdominal examination revealed epigastric tenderness and shifting dullness. His hemoglobin was 9.8g/dL, TLC 15,000,serum creatinine 0.7mg/dL, BUN 8mg/dL, random sugar 84mg/dL, total protein 5.6g/dL and serum albumin 2.9g/dL and normal serum electrolytes. Serum bilirubin, SGPT, SGOT, prothrombin time with INR were normal. Bedside X-ray chest revealed water bottle shape heart which was suggestive of pericardial effusion. Urgent pericardiocentesis was performed and 1000mL fluid removed. The next day the patient again complained of dyspnea, CT thorax revealed gross pericardial effusion with collapse of right atrium and right ventricle. Patient was retapped and percutaneous drain left in situ. Ultrasound abdomen showed