Figure 1: (A) Axial image of contrast enhanced CT scan shows a middle mediastinal cystic structure(arrows) compressing the trachea(arrow head). (B) Coronal reconstruction of the same shows the extent of oesophageal mucocele, involving the stomach (asterisks).

graft is based on the pattern of blood supply, while the type of anastomosis is determined by the stricture level and the part of colon used for reconstruction.[3] The most important early postoperative complication is a cervical anastomotic leakage. Satisfactory long-term results following oesophageal exclusion for corrosive injuries has been reported.[3]

Oesophageal mucocele is a rare post-operative complication. Usually, the oesophageal mucosa is almost completely destroyed by recurrent chronic inflammation and scarring and this probably accounts for the rarity of the condition.[4] The mucous glands may also atrophy due to high intraluminal pressure, and hence the mucocele fails to attain substantial size.[4] It usually occurs within two months after surgery, although, there are reports of presentation as late as 10 years.[5] Most of the mucoceles remain small and asymptomatic. When symptomatic, they present with chest pain, dysphagia and / or abdominal pain. Respiratory distress, although extremely rare, may be seen in patients with larger lesions, as was seen in this case. Secondary infection of the mucocele may occur which may result in fistula formation or septicaemia.

The definitive treatment of a symptomatic oesophageal mucocele is transthoracic oesophagostomy.[4] There are reports that describe CT-guided percutaneous drainage or tube drainage through thoracotomy as a temporary procedure.[5]

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References

1. Havanond C. Clinical features of corrosive ingestion. *J Med Assoc Thail*. 2003;**86**:918-24.
2. Haddad R, Teixeira Lima R, Henrique Boasquevisque C, Antonio Marsico G. Symptomatic mucocele after oesophageal exclusion. Interact Cardiovasc *Thorac Surg*. 2008;**7**:742-4.
3. Knezević JD, Radovanović NS, Simić AP, Kotarac MM, Skrobić OM, Konstantinović VD, Pesko PM. Colon interposition in the treatment of oesophageal caustic strictures: 40 years of experience. *Dis Esophagus*. 2007;**20**:530-4.
4. Kamath MV, Ellison RG, Rubin JW, Moore HV, Pai GP. Oesophageal mucocele: a complication of blind loop oesophagus. *Ann Thorac Surg*. 1987;**43**:263-9.
5. Olsen CO, Hopkins RA, Postlethwait RW. Management of an infected mucocele occurring in a bypassed excluded oesophageal segment. *Ann Thorac Surg*. 1985;**40**:73-5.

ALPPS for a large hepatocellular carcinoma in hepatitis C patient

Resection is the only curative option for patients with large primary liver tumours and select secondary tumours. The limiting factor for cure is an adequate future liver remnant (FLR) and many patients cannot be taken up for resection because of low FLR. There have been several measures to increase the FLR to obviate the risk of post-

hepatectomy liver failure (PHLF). The most commonly practised being portal vein embolization (PVE).[1] The shortcomings of the procedure are firstly, inability to achieve adequate volume increase in all and secondly, the delay of 4-6 weeks for the hypertrophy to occur with the fear of progression of disease in this period. Associating liver partition and portal vein ligation for staged hepatectomy (ALPPS) is a new concept developed in Germany by Schnitzbauer et al.2 This involves a two-step procedure of combining parenchymal division with deportalisation of the right lobe and segment-IV in the first stage followed by completion hepatectomy in the second stage after a short interval of 7-10 days. The advantage is rapid and increased hypertrophy thus overcoming the disadvantages associated with PVE. ALPPS is being used both as a primary strategy for increasing the FLR and as a salvage procedure following failed PVE.[2,3] We present one case where ALPPS was performed.

Case Report

A 52 year old male presented elsewhere with right hypochondrial discomfort, anorexia and weight loss since 5 months. Abdominal ultrasound and a triphasic CT showed a large diffusely infiltrating lesion (12.8x11.4x6.0 cm) occupying segments V-VIII, with multiple well defined lesions in segment-IV (**Figure 1a and 1b**). A differential diagnosis of HCC or metastatic intrahepatic-cholangiocarcinoma(CC) was entertained. Tumour markers were within normal limits (CEA-0.93ng/ml, AFP-3.66ng/ml, and Ca19-9-10.36U/ml). He was deemed inoperable and a biopsy of the lesion was performed which was reported as consistent with CC. He then presented to us with same complaints. A PET-CT reported non FDG-avid infiltrative cystic-nodular mass involving most of right lobe, and segment-IV, suggestive of HCC (**Figure 1c and 1d**). There was no evidence of extra-hepatic disease. He was evaluated for surgical resection. He was found to be hepatitis-C positive. On upper gastrointestinal endoscopy there were no varices, platelet counts and liver functions were normal except for the raised aminotransferases.

As the lesion occupied most of right lobe with extension to segment-IV, curative procedure would mandate an extended right hepatectomy. The FLR volume

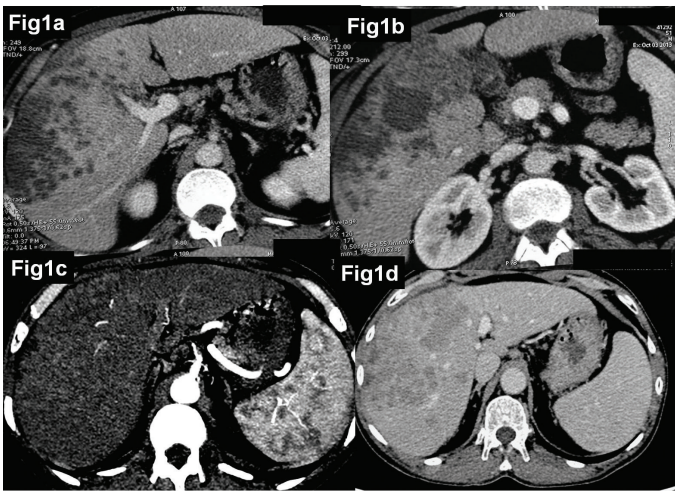

Figure 1: (a and b) Triphasic CT abdomen showing a large 12 x 8 x 12cm lesion occupying segments V, VI, VII, VIII, with multiple well defined lesions in segment VII, VIII and IV. (c and d) PET-CT scan showing infiltrative poorly marginated complex cystic nodular pattern mass involving most of the right lobe, another subcentimeter lesion in segment IVa. This lesion was enhancing in arterial phase and showed a washout in equilibrium phase.

was calculated to be 251cc (19% of total liver volume), hence a safe resection was not possible in one stage. Percutaneous PVE would mean a delay of 4-6 weeks for the FLR to become 25% of the whole liver. We planned for a staged hepatectomy (ALPPS). On CT-scan there was a suspicion of omental nodule, there was another area on the right lobe where the lesion seemed to have ruptured with omental adhesions. An initial diagnostic laparoscopy ruled out extra-hepatic or left lateral segment disease. Laparotomy confirmed the CT findings. First stage of ALPPS was performed. The right lobe was mobilised, portal structures were dissected, the right hepatic artery was replaced from the superior mesenteric artery, the right portal vein was identified, divided and suture ligated, segment-IV artery was also ligated. Liver partition was performed using a Cavitron Ultrasonic Surgical Aspirtaor (CUSA) and bipolar diathermy. Once the middle hepatic vein was identified before it joined the left hepatic vein the parenchymal transection was stopped. Gelfoam and surgicel was placed in the transection plane, and wound was closed over an abdominal drain. Patient did not require any transfusion. Two weeks after first

stage surgery, CT-scan of the abdomen was repeated. The FLR had increased to 423cc (41% of total liver volume, an overall increase of 69%) (**Figure 2**). Second stage surgery was performed 15-days after first stage. At exploration there were perihepatic adhesions. The left lateral segment had hypertrophied. After adhesiolysis, the right hepatic artery, right hepatic duct, right hepatic vein, and the middle hepatic vein were divided and ligated. The parenchymal transection was completed (**Figure 3**). Patient was discharged in a stable condition after 7 days. The histopathological report was a well differentiated multifocal HCC. Although lymphovascular emboli were present, the margins were free. The HCV RNA viral load was found to be 6,24,000 IU/ml after surgery. He was started on interferon and ribavarin therapy by the hepatologist 3 weeks after surgery. He had an uneventful recovery and returned to his routine activities 4-weeks later. At 6-months follow-up, patient was asymptomatic and his serum alpha-fetoprotein level was 4.3ng/ml. Follow-up CT-scan showed a hypertrophied FLR. There were two sub-centimeter lesions in the remnant liver. He underwent transarterial chemoembolisation (TACE) for these lesions and was started on chemotherapy (Sorafenib). Fourteen months post-surgery, he is completely asymptomatic, gained weight and is tolerating chemotherapy.

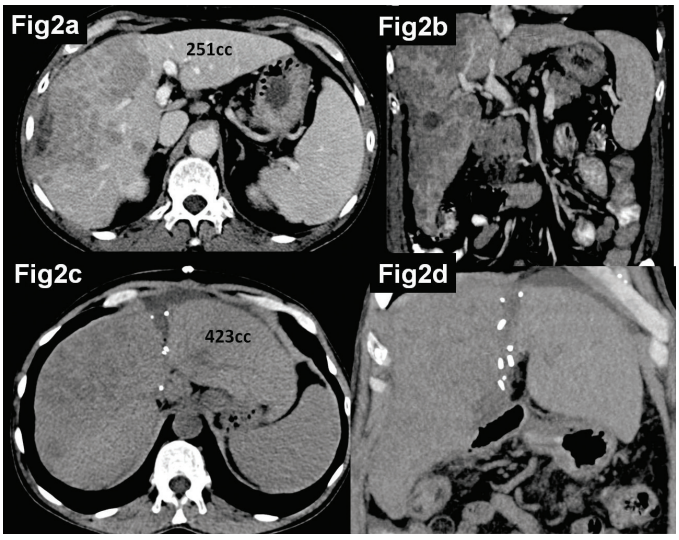

Figure 2: **(a and b) Preoperative CT scan showing FLR volume of 251cc, 19% of total liver volume. (c and d) CT scan after 13 days of first stage hepatectomy showing the liver partition and hypertrophied left lateral segment with a volume of 423cc, 41% of total liver volume.**

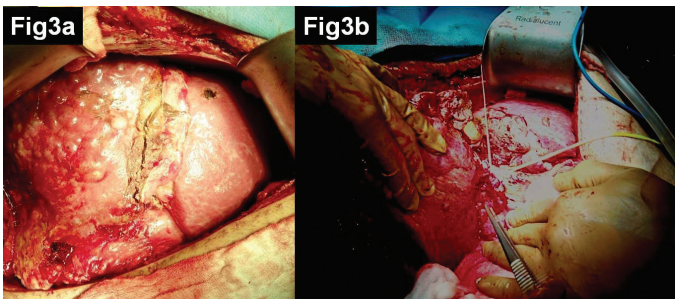

Figure 3: **(a and b) Second stage hepatectomy showing the liver partition, hypertrophied left lateral segment, and the portal structures being dissected for division.**

Discussion

For patients with primary or metastatic liver tumours, requiring extended resections, it is important to ensure adequate FLR to prevent PHLF. After the initial introduction of ALPPS in 2012, it was adapted by hepatobiliary surgeons as a useful tool for patients who were deemed unresectable because of; low FLR, bilateral liver tumours, thrombosed portal vein (PVE not possible), and where PVE failed.[2,3]

About 400 cases of ALPPS have been reported from various centres.[4,5] The indications for this procedure were colorectal liver metastases, CC, HCC, gall-bladder cancer, hemangioendothelioma, non-colorectal non-neuroendocrine liver metastases, sarcoma, metastases from ovarian tumor, gastric cancer, and one case for liver cyst. Indications were either as primary or as a salvage treatment. The reported increase in FLR volume varies between 23.8-200% (mean 84.16%) after an interval of 4-30 days (mean11.6 days) between the two stages of ALPPS. In our case there was a 69% increase in FLR after an interval of 14-days. The morbidity and mortality associated with ALPPS has been an area of concern. Mean postoperative morbidity reported is 35% (range 22-90%) and the mean operative mortality is 12%(range-0-28.7%).[4,5] There is a concern regarding increased incidence of recurrence (upto 20%) in the remnant liver, probably as a result of rapid hypertrophy.[4,5] Our patient was found to

have recurrent disease in the remnant liver, which was treated with TACE and Sorafenib.

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References

1. Makuuchi M1, Thai BL, Takayasu K et al. Preoperative portal embolization to increase safety of major hepatectomy for hilar bile duct carcinoma: a preliminary report. *Surgery*. 1990;**107(5)**:521-7.
2. Schnitzbauer AA, Lang SA, Goessmann H et al. Right portal vein ligation combined with in situ splitting induces rapid left lateral liver lobe hypertrophy enabling 2-staged extended right hepatic resection in small-for-size settings. *Ann Surg*. 2012;**255**:405-414.
3. Soumil J. Vyas, Neil Davies, Lee Grant et al. Failure of Portal Venous Embolization. ALPPS as Salvage Enabling Successful Resection of Bilobar Liver Metastases. *J Gastrointest Canc*. DOI 10.1007/s12029-014-9643-6.
4. Ielpo B, Caruso R, Ferri V et al. ALPPS procedure: our experience and state of the art. *Hepatogastroenterology*. 2013;**60(128)**:2069-75.
5. Nadalin S, Capobianco I, Li J et al. Indications and limits for associating liver partition and portal vein ligation for staged hepatectomy (ALPPS). Lessons Learned from 15 cases at a single centre. *Z Gastroenterol*. 2014;**52(1)**:35-42. doi: 10.1055/s-0033-1356364.