fluoroscopy very difficult. The coiling of the guide wire into the collection becomes the most critical step, as without fluoroscopic or EUS guidance there is a risk of perforation of the cyst wall. To prevent this complication, we pushed the guide wire in slowly and ensured that once the tip of the guide wire was seen exiting the needle tip, only 10 cm of the guide wire was pushed further.

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References

1. Sharma V, Rana SS, Bhasin DK. Endoscopic ultrasound guided interventional procedures. *World J Gastrointest Endosc*. 2015;7:628-642.
2. Panamonta N, Ngamruengphong S, Kijsirichareanchai K, Nugent K, Rakvit A. Endoscopic ultrasound-guided versus conventional transmural techniques have comparable treatment outcomes in draining pancreatic pseudocysts. *Eur J Gastroenterol Hepatol*. 2012;24:1355-1362.
3. Rana SS, Bhasin DK. Non-fluoroscopic endoscopic ultrasound-guided transmural drainage of pseudocysts: A pictorial technical review. *Endosc Ultrasound*. 2015;4: 92-7.
4. Rana SS, Bhasin DK, Rao C, Gupta R, Singh K. Non-fluoroscopic endoscopic ultrasound-guided transmural drainage of symptomatic non-bulging walled-off pancreatic necrosis. *Dig Endosc*. 2013;25:47-52.
5. Rana SS, Sharma V, Sharma R, Gupta R, Bhasin DK. Endoscopic ultrasound-guided transmural drainage of calcified pseudocyst in a patient with chronic calcific pancreatitis. *Ann Gastroenterol*. 2015;28:290.

Primary systemic amyloidosis presenting as an intrahepatic space-occupying lesion

Amyloidosis is associated with a number of inflammatory and inherited disorders in which extracellular deposits of fibrillar proteins cause tissue damage and functional compromise. The abnormal fibrils are produced by aggregation of misfolded proteins which are insoluble unlike their normally folded counterparts.[1]

In both systemic and localized forms, the gastrointestinal system is the most commonly involved with the colon being the most common site. The liver is often involved; there may be hepatomegaly, and CT scan may show decreased attenuation of liver.[2] To our knowledge, only one case of primary systemic amyloidos is presenting as a liver mass has been described previously.[3] We hereby report a case of primary systemic amyloidosis presenting with abdominal pain and jaundice with CT showing a space-occupying lesion in the liver.

Case Report

A 57-year old man presented with dull aching pain in the right hypochondrium, anorexia, weight loss of 5 kg over 6 months, and jaundice with generalized itching for 2 months. Physical examination revealed hepatomegaly. Haemogram showed a haemoglobin of 11.4 gm/dl, total leukocyte count 16300/mm^3 and platelets 400000/mm^3 and ESR was 53 mm.

Liver biochemistry revealed serum total bilirubin of 8.2 mg% with direct bilirubin of 6.4 mg%, aspartate aminotransferase 121 U/L, alanine aminotransferase 37 U/L and alkaline phosphatase 465 U/L. Serum total proteins were 6.4 gm% and serum albumin was 2.8 gm%. PT INR was 1.3. Renal biochemistry was normal and viral markers were negative. Electrocardiogram showed low voltage complexes with left axis deviation and echocardiography was normal. Ultrasound examination of the abdomen showed a liver span of 16.8 cm; there was an ill-defined hypoechoic lesion in segment IV of the liver,

measuring 3 cm x 2.8 cm x 2.5 cm. Contrast enhanced CT of abdomen and chest showed an ill-defined hyperdense lesion near the gall bladder fossa in segment IVB of the liver with no post contrast enhancement. There was no lymphadenopathy (**Figure 1**). CT scan-guided biopsy of the liver lesion showed deposition of eosinophilic fibrillar acellular material in the space of Disse with atrophy of adjacent hepatocytes. It showed Congo-red and cresyl violet positivity suggesting amyloid deposition (**Figure 2**). Serum protein electrophoresis (SPEP) showed the presence of an M band at beta location with a predominance of lambda chains. Liver tissue immunohistochemistry confirmed amyloidosis. Rectal biopsy also showed amyloid positivity. Bone marrow biopsy revealed 7% plasma cells. The patient was diagnosed as a case of AL related amyloidosis and advised a chemotherapy regime: Cyclophosphamide + Bortezomib + Dexamethasone (CyBorD). One month after the diagnosis of AL related amyloidosis, the patient died of progressive liver failure.

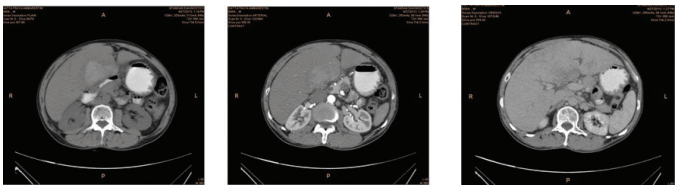

Figure 1: **From left to right: Plain scan showing ill defined hyperdense lesion in segment IV B near the gall bladder fossa. Both arterial and delayed phases showing no post contrast enhancement.**

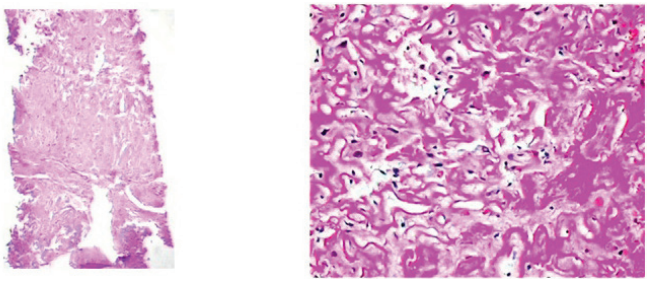

Figure 2: **From left to right: Hematoxylin and eosin stain (x100) - Deposition of abundant eosinophilic extracellular material around sinusoids. Congo red stain (x400) - Extracellular material deposited in perisinusoidal region is positive on Congo red.**

Discussion

Liver involvement is seen in both primary (AL) and secondary (AA) amyloidosis. In an autopsy series of AL amyloidosis, the liver was involved in 70% cases[4] and the median survival was only nine months (five-year survival of 17%). Radiological findings of hepatic amyloidosis are non-specific with biopsy being the confirmatory test. An enlarged liver with heterogeneous decreased attenuation is often seen on CECT abdomen. The hepatic parenchyma infiltrated with amyloid produces focal hypoattenuating areas due to impaired blood flow secondary to vascular involvement. Delayed phase shows delayed enhancement with focal hypoattenuating areas. This delayed phase picture is due to delayed passage of contrast into the hepatic parenchyma. Magnetic resonance imaging has also been used for evaluation of primary hepatic amyloidosis. Although T2 value of hepatic parenchyma is unchanged, the T1 value is increased and it can be used to monitor chemotherapy.[5]

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References

1. Kumar V, Abbas A, Aster J. Robbins and CotranPathologic Basis of Disease. 9thed. *Philadelphia: Saunders / Elsevier*; 2015.
2. Georgiades CS, Neyman EG, Barish MA, Fishman EK. Amyloidosis: review and CT manifestations. *Radiographics*. 2004;24:405-16.

3. Son RC, Chang JC, Choi JH. Primary hepatic amyloidosis: report of an unusual case presenting as a mass. *Korean J Radiol*. 2011;12:382-5.
4. Buck FS, Koss MN. Hepatic amyloidosis: morphologic differences between systemic AL and AA types. *Hum Pathol*. 1991;22:904-7.
5. Monzawa S, Tsukamoto T, Omata K, Hosoda K, Araki T, Sugimura K. A case with primary amyloidosis of the liver and spleen: radiologic findings. *Eur J Radiol*. 2002; 41:237-41.