from sites within the systemic circulation. One manifestation of this is reduced renal arterial perfusion, followed in turn by reduction in the glomerular filtration rate. Nephrotic syndrome (NS) may not be evident clinically unless renal arterial perfusion is such that significant amounts of protein are able to traverse the damaged glomerular endothelium.

Case: A 67 year old man with a 26 year history of myelofibrosis developed ascites; its characteristics were those of PHTN, i.e., low albumin and elevated serum ascites albumin gradient. Treatment with sodium restriction, and furosemide and spironolactone was not effective in controlling ascites; moreover, he developed azotemia (creatinine 3.2 gm%; normal range [NR] 0.6-1.3). Large volume paracentesis became necessary, and eventually on a weekly basis. He had sustained esophageal variceal hemorrage earlier the same year. Before consideration of transjugular intrahepatic portasystemic (TIPS) shunt insertion, the patient underwent hepatic venous pressure gradient (HVPG) assessment and liver biopsy. His HVPG was 17 mm Hg (NR: 2-5). The biopsy revealed stage 2 fibrosis, hepatic plate atrophy and evidence of extramedullary hematopoiesis. Thereafter, TIPS shunt insertion was undertaken without complication. The HVPG was reduced to 4 mm Hg. However, within one month the patient developed hepatic encephalopathy and required hospital admission; heavy proteinuria was demonstrated. Ten days after discharge, the patient had impressive, bilateral lower extremity edema, continued heavy proteinuria (3+) and further reduction in serum albumin concentration (2 gm%: NR 3.4-5). His serum creatinine concentration was 1.9 gm% (NR: 0.6-1.3). Discussion with his nephrologist confirmed that proteinuria had never been present hitherto. The 24 hour urine protein was 8 gm (NR: <200mg); renal biopsy confirmed the presence of non-AL amyloid deposits.

Conclusion: Clinical and laboratory evidence of NS emerged within one month of this man undergoing TIPS shunt insertion for refractory ascites formation. It is our contention that the presence of PHTN, and thus reduced renal blood flow had prevented these features from being clinically apparent until the HVPG was corrected by TIPS shunt insertion. The patient's clinical course has been dominated since by hepatic encephalopathy and worsening renal function to the point of renal replacement therapy. Although a few reports exist about the co-existence of myelofibrosis, PHTN and NS, we believe that this is the first account of NS emerging only after TIPS shunt insertion for a complication of PHTN.

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A Rare Cause of Recurrent Pain Abdomen Managed by ERCP

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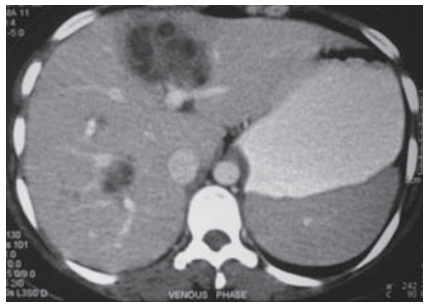
Purpose: Recurrent pain abdomen is common presentation in gastroenterology practice. Here, we report a rare case of recurrent pain right upper quadrant.

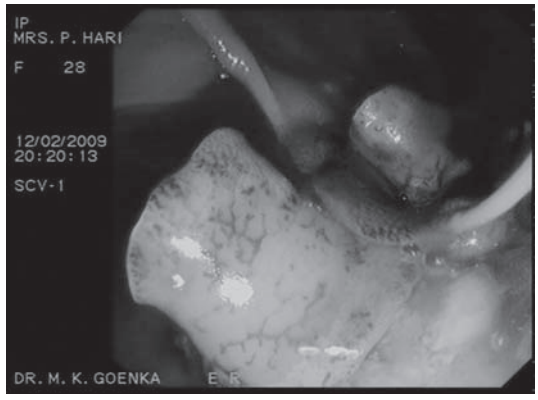
Methods: Case report

Results: A 28 years female presented with pain right upper abdomen of 2 months duration. There was no history of fever or jaundice. There was no history of bowel disturbance or urinary symptom. Ultrasonography showed multiple cystic lesions in both lobe of liver. Hemogram showed Hb- 9.4gm/dl, WBC count - 9100/dl with eosinophil- 36%. Liver function test showed AST - 64 IU/L, ALT - 84 IU/L and ALP - 746 IU/L. She was started on oral metrondiazole, ofloxacin and antispasmodics. Intermittent pain continued and she started to have low grade fever. CT abdomen revealed multiple complex cysts on both lobe of liver (Fig 1). Aspiration revealed brownish material. Bile pigment was noted on aspirated material and culture was sterile. ERCP was done, which showed irregular filling defects in bile duct with communication with on of the cyst. On balloon extraction, 3 live fasciola hepatica were removed (Fig 2). She received Triclabendazole and doing well on follow up.

Conclusion: The liver fluke, Fasciola hepatica presenting as recurrent pain abdomen is rare. Also, Fasciola hepatica infestation is not reported from this part of world. Finally, patient was managed by removal of live Fasciola at ERCP.



CT abdomen showing complex cysts.



Fasciola hepatica removed from bile duct at ERCP.

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Congenital Intrahepatic Shunt Presenting as a Pseudometastatic Liver

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Purpose: A 29 year old caucasian male, with no prior medical history, was referred by his general practitioner to the hepatology outpatient clinic with the presumptive diagnosis of diffuse hepatic metastization seen in abdominal ultrasonography (multiple hyperecogenic nodules over the hepatic parenchyma — 1-4 cm in diameter - suggestive of secondary lesions). The US had been requested due to abnormal liver test

Results: AST 64 U/L, ALT 98 U/L, Gamma GT (GGT) 270 U/L and Alkaline Phosphatase (Alk P) 145 U/L. Anorexia and fatigue had insidiously developed over the last 2 months and physical examination showed a nodular hepatic border 3 cm below the costal margin. No other changes were noticed. Blood test results confirmed elevated AST, ALT, GGT and Alk P with normal albumin level and coagulation tests. Serologic markers for hepatits A, B, and C were negative and tumor markers (carcinoembryonic antigen (CEA), Alpha Fetoprotein (AFP) and CA 19.9) were normal. An upper GI endoscopy and a colonoscopy proved normal too. An abdominal magnetic resonance was requested and revealed multiple hepatic nodules with different sizes, distributed predominantly over the left hepatic lobe. A magnetic resonance angiography showed a portosystemic intrahepatic shunt (between the right portal vein and the inferior vena cava - Park type 1). An ultrasound guided liver biopsy was performed and the histology was consistent with nodular regenerative hyperplasia of the liver. Nodular regenerative hyperplasia of the liver is a rare, benign condition in which there is hepatic micro-nodular regeneration with little or no fibrous septation. The hypothesis that this transformation may be associated with hepatic vascular defects is widely recognized. In this case a congenital vascular defect was the trigger for this transformation that can simulate hepatic metastization.