from sites within the systemic circulation. One manifestation of this is reduced
renal arterial perfusion, followed in turn by reduction in the glomerular fil-
tration 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 ele-
vated serum aspartate amino transferase. 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 varical hemorrhage earlier the same
year. Before consideration of transjugular intrahepatic portosystemic (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
dema, continued heavy proteinuria (3+) and further reduction in serum
albumin concentration (2 gm%; NR 3.4-5). His serum creatinine concentra-
tion 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 for-
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Case:

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 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 nodu-
lar 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
hepatitis 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 consist-
tent 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 hypoth-
sis 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 metastatization.