elevated transaminase levels. An increase in ALT, AST, or GGT level was detected in 545 (11.7%) who were negative for viral serology and had normal TS. In US steatosis was found in 430 (54%.) The grade of steatosis (I in 104, 24.1%, II in 111, 25.8%, and III in 18, 4.1%) correlated to ALT and trigyceride levels and BMI.
Conclusion: We found that hemochromatosis is very rare in this country and the frequency of hepatitis C is much lower in comparison to Hepatitis B. In this population with a low frequency of obesity and alcohol consumption, still, steatosis was the most frequent cause of elevated liver enzymes.

752 INTERVENTIONAL TREATMENT IN BUDD-CHIARI SYNDROME

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Background: Budd-Chiari syndrome (BCS) is an pathophysiologic process that results in interruption of the normal hepatic outflow. The goal of treatment should be decompression of the congested liver. TIPS and balloon angioplasty are the only minimally invasive procedures for that purpose. We presented the results of this procedures that were performed in 28 patients within the last 3 years.
Methods: Patients were subdivided according to the anatomic nature of the obstruction which was defined by MR-angiography and DSA. Group-1 patients have membranous obstruction of the hepatic veins (HV) or VCI and treated by balloon angioplasty. The rest of the patients are in Group-2 and TIPS procedure is the choice of treatment.
HV were catheterized through right jugular vein, if unsuccessfull, ultrasound guided percutaneous direct approach by Chiba needle through the right intercostal space was preferred. Those with complete obstruction were perforated by the needle and all were dilated with a balloon (18-24mm).
Those who have complete thrombosis of the HV, TIPS was performed. Liver punctures were performed above the narrowed portion of VCI, 2cm below the right atrium and 2 metalic stents (10x70mm) were placed between PV and suprahepatic portion of VCI.
Results: Group-1 (n: 18) The shunt was patent after a year in 14 cases and redilation was needed in 4 without mortality. Group-2 (n: 10). 2 patients died after unsuccessful TIPS in a year, 7 patients survived the year and 1 died of pulmonary bleeding.
Conclusion: Balloon angioplasty is a very effective treatment modality in selected cases but needs re-dilatation in some. TIPS is very hard to perform in BCS but if done properly is an excellent modality to decompress the liver.

753 TIPS FOR ACUTE AND CHRONIC BUDD-CHIARI SYNDROME:

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Background/Aim: TIPS provides decompression of the liver in Budd-Chiari syndrome (BCS) irrespective of inferior vena cava obstruction, obviating the surgical mortality particularly in acute BCS. Thus TIPS is considered the treatment of choice in many centres. We evaluated the long term outcome of TIPS in patients with acute and chronic BCS.
Patients: 16 patients (8 males, average age 38.2) with BCS underwent TIPS. Actiology: 6 myeloproliferative disease, 3 paroxysmal nocturnal haemoglobinuria, 1 antiphospholipid syndrome, 1 protein C and S deficiency, 1 hepatic metastasis, 4 cryptogenic. Indication: 9 acute (liver failure) and 7 chronic BCS (3 refractory ascites, 3 variceal bleeding, 1 progressive fibrosis).
Results: 1 had unsuccessful TIPS placement (percutaneous hepatic vein dilatation, alive). In the liver failure group, 4 of 9 died within few days after TIPS: 1 ruptured liver; 1 ruptured portal vein; 1 progressive liver failure; 1 acute pulmonary oedema. 2 technical complications: 1 portal vein rupture (died) (only 1 of 2 ruptures in 329 TIPS procedures) and 1 intrahepatic haemorrhage (resolved). Average follow up after TIPS (11 patients, all anticoagulated): 20 months (range 4-43). In all, ascites resolved and liver function improved. 1 died after 18 months because of the original metastatic disease. 5 TIPS dysfunctions, all resolved: 3 balloon dilatation, 2 repeat TIPS. No patient has required liver transplantation. Conclusion: TIPS can be performed in BCS obviating surgery and transplantation, and long term results are good. However mortality is high in acute BCS and in these cases liver transplantation could result in a better outcome.

754 DOES PELIOSIS HEPATIS ASSOCIATE WITH CASTLEMAN'S DISEASE?

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Castleman et al.described the disease known as giant lymph node hyperplasia. Peliosis hepatis is characterised by multiple bloodfilled cysts and dilated hepatic sinusoids in the liver. It was often noted in the immuncompromised host. The association between these two disorder has not been reported before. Here we report a case with both peliosis hepatis and Castleman's Disease.
Case Report: A 21 years old young man was hospitalised for growth retardation. Physical examination at that timedisclosed pallor and hepatomegaly. He has suffered from growth retardation and iron defficiency anemia since 4 years. He recovered iron many times. Laboratory findings at that time showed iron defieciency anemia and hypoalbuminemia. Abdominal Ultrasonography showed giant lymph nodes between the tail of pancreas and splenic hilum and hepatomegaly with a heterogenous echo pattern. Bone marrow examination, small bowel series and upper gastrointestinal endoscopy were normal. Liver biopsy showed sinusoidal dilatation. Computed tomography guided lymph node biopsy was performed but it wasn't diagnostic. Diagnostic laparotomy was performed and lymph nodes were excised and liver biopsy was repeated. Histopathologic examination showed peliosis hepatis and Castleman's Disease. 1 month after surgery his weight, albumin and hemoglobin levels improved.
Conclusion: Castleman's Disease is an immunoproliferative disease which the patient may be immuncompromised. Peliosis hepatis is usually seen in immuncompromised patients, therefore it may be a finding of Casteleman's Disease.

755 KAVA-INDUCED ACUTE LIVER INJURY: A REPORT OF 29 NOVEL CASES

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Botanical drugs are widely used and often contain highly active compounds. Numerous reports have shown the potential hepatotoxicty of certain herbals. Kava root (Piper methysticum rhizoma), frequently used as a remedy against anxiety, contains kavapyrones with sedative effects. Several case reports demonstrated the development of hepatitis after the intake of Kava. However, the awareness of Kava as a cause of liver damage is still low though new cases steadily occur. We analyzed 39 so far unpublished cases of adverse hepatic reactions after the intake of Kava which had been reported to the German Department of Pharmacovigilance (BfArM) between 1990 and 2002. Causality was evaluated using the CIOMS score.

Hepatic injuries were mainly necrotizing and cholestatic hepatitis. In 29 of 39 cases, a causal role of Kava was either 'certain' or 'probable' according to the CIOMS criteria. The majority of patients were women (25 female, 10 male). Both the dose and the latency to when the hepatotoxic reaction emerged were highly variable. Three patients died due to fulminant liver failure and 5 patients underwent liver transplantation while in the remaining individuals complete remission was noticed after withdrawal of Kava. Pathophysiologically, an immunoallergic reaction seems likely, since four patients responded well to corticosteroids and one patient experienced an accelerated relapse due to accidental reexposure. The present 29 novel cases of Kava-associated hepatotoxic reactions emphasize the potentially severe hepatotoxicity of Kava. Due to these data, the approval of Kava-containing drugs has been revocated on June 14th, 2002, by the German pharmacovigilance authorities.