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Chronic liver disease and hemostatic disturbances

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The liver is the primary site of synthesis of most of the clotting factors and the proteins involved in the fibrinolytic system. These include all the vitamin K-dependent coagulation proteins (factors II, VII, IX, X, ATIII, protein C, protein S and protein Z), as well as factor V, XIII, fibrinogen, antithrombin, and plasminogen. Decreased plasma concentrations for all proteins except for factor VIII are observed in patients with hepatic failure. Besides low levels of clotting factors due to impaired synthesis capacity, also dysfunctional proteins are found in patients with liver failure. Additionally, liver plays a vital role in the regulation of anticoagulation. Removal and clearance of activated clotting and fibrinolytic factors, especially tissue plasminogen activator (tPA), is mediated through the hepatic reticuloendothelial system. Multiple alterations in platelet number and function can be found in patients with liver disease. A mild to moderately reduced platelet count is frequently present in patients with acute or chronic liver failure. The net outcome of these alterations in the hemostatic system is a bleeding diathesis, although thrombosis of the portal vein is also frequently seen in patients with cirrhosis. As liver disease has multiple effects on the hemostatic system, it is difficult to determine which factors contribute most to the bleeding diathesis. No single coagulation test is predictive of hemorrhage or thrombosis in patients with chronic liver disease. A prolonged PT or international normalized ratio (INR) is a key indicator of hepatic dysfunction and commonly used as a trigger for liver transplantation. Thrombophilia tests: levels of the naturally occurring anticoagulants (ATIII, protein C, protein S) may all be reduced as a consequence of liver disease. In conclusion: Because of the hemostatic problem of liver disease is multifactorial, the follow up and management of chronic liver disease may ameliorate the bad prognosis.

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