failure in some patients,[6] but the optimal duration of prophylaxis remains controversial. Current European guidelines recommend antiviral prophylaxis for 12 months following chemotherapy.[7]Recent American and Australian Society guidelines recommendpatients with high baseline HBV DNA (>2,000 IU/ml) to continue prophylaxis until they reach treatment end points for chronic hepatitis.[8]It is clear from this case that even prolonged prophylaxis does not protect against withdrawal flares in some settings. It is also very important to perform frequent ALT and viral load testing after cessation of antiviral therapy to detect early flares.

In summary, we have presented here a case where frequent quantitative monitoring of HBsAg, anti-HBs and HBV DNA helped in optimising post-transplant HBV prophylaxis.This is a unique case of successful liver transplant for HBV reactivation precipitated by cessation of prolonged HBV prophylaxis forchemotherapy. We recommend lifelong continuation of antiviral prophylaxis following chemotherapy in patients with high baseline viral load.

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Unsuspected hepatic schistosomiasis in a liver explant

A 48-year-old man from the Middle-East presented to us with a history of severe portal hypertension, leading to refractory GI bleed not responding to therapy. On examination there was massive splenomegaly with minimal ascites. Magnetic resonance imaging revealed minimally shrunken liver with nodular regenerative hyperplasia. All autoimmune and viral markers were negative. Serum alpha-feto protein and CA 19-9 were within normal limits. Orthotopic live related liver transplant was done. We received a firm enlarged liver weighing 1175 grams and measuring 27×17×6cm. The liver showed a thickened capsule and bosselations on its inferior surface (**Figure 1A, 1B**). On cut section, there were round to stellate shaped,thick, grayish-white tracts of collagen around major

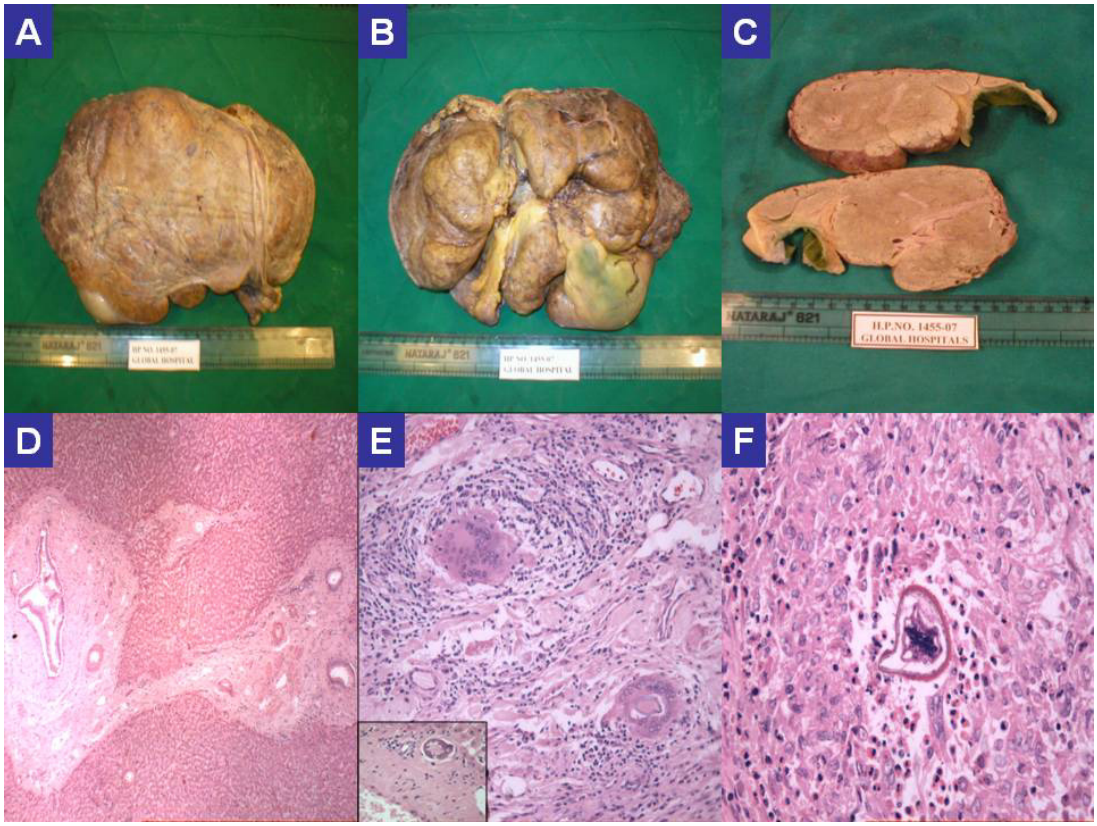

Figure 1: (A) Explant liver;(B) explant liver with bosselations on inferior surface;(C) cut sections displaying clay-pipe stem fibrosis;(D) light microscopy with rounded and expanded portal tracts (hematoxylin and eosin x100);(E) higher magnification demonstrating granulomatous inflammation, and a dead calcified egg (inset) (hematoxylin and eosin x200);(F) live egg in a granuloma rich in eosinophils (hematoxylin and eosin x400,)

portal tracts, which is described as Symmers' clay-pipe stem fibrosis (**Figure 1C**). The liver architecture was preserved and there was no cirrhosis. Histology revealed portal expansion with fibrosis. Within the fibrous septa tortuous arterioles and venules, entrapped bile ductules, along with granulomatous eosinophil-rich inflammation and dead calcified eggs were noted (**Figure 1D, 1E**). Occasional granuloma containing live eggs with shell and spine and the circular ring of nuclei were also identified (**Figure 1F**).Schistosomal haemozoin pigment phagocytosed by macrophages was visible within the portal tracts and sinusoidal Kupffer cells. Few portals tracts also showed evidence of venosclerosis with loss of venules with or without proliferation of abnormal vessels with focal cavernous transformation. We labeled the explant liver with hepatic schistosomiasis. Schistosomiasis or bilharzia is a tropical parasitic disease caused by blood-dwelling flukes of the genus Schistosoma. S. mansoni, S. japonicum, S. mekongi and S. intercalatum infections are associated with chronic hepaticfibrosis.[1] The main pathogenic lesions in chronic infection are not due to the adult worms but because of eggs that are trapped in tissues due to peri-intestinal migration or embolization to liver, spleen, lungs, or the cerebrospinal system. The eggs secrete proteolytic enzymes that provoke typical eosinophilic inflammatory and granulomatous reactions, which are progressively replaced by fibrosis.

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