Peri-portal fibrosis of the liver due to natural or experimental infection with schistosoma mansoni occurs in the Kenyan baboon

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Abstract

The chronic granulomatous inflammation that occurs during schistosomiasis mansoni and its reparative healing lead to hepatic fibrosis, with subsequent portal hypertension (a life-threatening sequela). IIi the murine model, granuloma modulation invariably leads to formation of fibrous tissue and deposition of extracellular matrix. Typically, < 10% of patients infected with Schistosoma mansoni progress to clay-pipe¬stem fibrosis. Similar fibrosis occurs in chimpanzees during experimental infections. Although pre~~qus studies of schistosomiasis mansoni in Kenyan baboons have failed to demonstrate appreciable liver fibrosis, classical peri-portal fibrosis has now been observed in the livers of three yellow baboons (Papio cynocephalus cynocephalus) with natural S. mansoni infections and three olive baboons (P. c. anu~is) with experimental infections after each was challenged with 1000 S. mansoni cercariae. The peri-portal fibrosis was indicated by marked fibroblast accumulation, increased collagen deposition, bile-duct hyper¬plasia and blood-vessel proliferation. The lesions were more severe in the naturally infected baboons than in those experimentally infected. No accompanying portal hypertension, ascites or portocaval anastomosis was noted in any of the animals. The development of the baboon as a model for chronic human schistosomiasis mansoni may be feasible.