Enhanced expression of transforming growth factor-β1 in inflammatory cells;α- smooth muscle actin in Stellate cells;and collagen accumulation in experimental granulomatous hepatitis caused by Toxocara canis in mice.

林永和

Wu MS;Liao CW;Du WY;Kao TC;Su KE;Lin YH;Chang CC;Fan CK*

摘要

Abstract

Although toxocaral granulomatous hepatitis (TGH) characterized with a dominant-Th2 type immune response is a self-limiting disease, little is known concerning the role of fibrosis-related cytokine transforming growth factor-beta 1 (TGF-β1) in pathogenesis of TGH. A detailed histological and quantitatively immunohistochemical analysis of TGF-β1, α-smooth muscle actins (α-SMA), and collagen was performed on the liver tissues from mice infected with Toxocara canis as assessed between day 1 and 42 weeks post-infection (DPI or WPI).

TGF-β1 was detected mainly in infiltrating leukocytes in lesions with strong expressions from 4 to 16 WPI. Larvae per se also exhibited strong TGF-β1-like molecule expressions in the trial. Alpha-SMA was detected predominantly in hepatic stellate cells (HSC) which surrounded the lesions with moderate expressions largely throughout the period of the entire experiment. Collagen was observed to accumulate in inflammatory lesions and biliary basement with moderate to strong expressions from 1 WPI onwards in the trial. Since many evidences have indicated that leukocytes have the potential to influence HSC by producing TGF-β1 which can affect HSC to increase collagen synthesis in various liver diseases, we may propose that persistently elevated TGF-β1 expression in infiltrating leukocytes and active HSC with marked α-SMA expressions may contribute to healing of injured sites through up-stimulation of collagen deposition; in contrast,

abnormally persistent collagen accumulation may cause irreversible fibrotic injury in the TGH.