MONONUCLEAR CELL INFILTRATION ACCOMPANIED BY THE PROGRESSION OF FIBROTIC AND CIRRHOTIC TRANSFORMATIONS IN THE LIVER TISSUE

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Abstract. *Fibrosis is a complex, multicellular wound-healing response, where myofibroblasts—key producers of extracellular matrix (ECM)—actively interact with both liver-resident cells and those recruited from the circulation. Macrophages and infiltrating monocytes contribute to fibrogenesis through multiple pathways, including cytokine secretion and the production of reactive oxygen species. Interestingly, these same macrophages also play a crucial role in fibrosis resolution by facilitating ECM degradation. T lymphocytes influence fibrosis progression by interacting directly with myofibroblasts and releasing various cytokines. Typically, a Th2-skewed immune response supports fibrotic activity, whereas Th1-associated cytokines may exert antifibrotic effects. Natural killer (NK) cells help to restrain fibrosis and promote its regression, partly by inducing apoptosis in activated fibrogenic cells. Recent studies also point to a potential role for NKT and B cells in modulating liver fibrogenesis. Overall, mononuclear immune cells serve as key regulators of the fibrotic process and represent promising targets for therapeutic intervention.*

Keywords: *Liver fibrosis, chronic liver disease, Morphological, genotype, pathological process, mononuclear immune cells, chronic viral virus hepatitis B (HBV).*

ИНФИЛЬТРАЦИЯ МОНОНУКЛЕАРНЫХ КЛЕТОК, СОПРОВОЖДАЮЩАЯСЯ ПРОГРЕССИРОВАНИЕМ ФИБРОЗНЫХ И ЦИРРОТИЧЕСКИХ ПРЕОБРАЗОВАНИЙ В ТКАНИ ПЕЧЕНИ

Аннотация. *Фиброз — это сложная многоклеточная реакция заживления ран, при которой миофибробласты — основные производители внеклеточного матрикса (ВКМ) — активно взаимодействуют как с клетками, находящимися в печени, так и с клетками, рекрутированными из кровотока. Макрофаги и инфильтрирующие моноциты способствуют фиброгенезу через множество путей, включая секрецию цитокинов и продукцию активных форм кислорода. Интересно, что эти же макрофаги также играют решающую роль в разрешении фиброза, способствуя деградации ВКМ. Т-лимфоциты влияют на прогрессирование фиброза, взаимодействуя напрямую с миофибробластами и высвобождая различные цитокины. Как правило, иммунный ответ, смещенный по Th2, поддерживает фиброзную активность, тогда как цитокины, связанные с Th1, могут оказывать антифиброзное действие.*

Естественные клетки-киллеры (NK) помогают сдерживать фиброз и способствуют его регрессу, отчасти путем индукции апоптоза в активированных фиброгенных клетках. Недавние исследования также указывают на потенциальную роль NKT- и B-клеток в модулировании фиброгенеза печени. В целом, мононуклеарные иммунные клетки служат ключевыми регуляторами фиброзного процесса и представляют собой перспективные цели для терапевтического вмешательства.

Ключевые слова: *Фиброз печени, хроническое заболевание печени, Морфологический, генотип, патологический процесс, мононуклеарные иммунные клетки, хронический вирусный гепатит B (HBV).*

Introduction

The liver possesses a uniquely complex immunological environment, housing the largest reticuloendothelial system in the body and serving as a major producer of innate immune components such as acute-phase proteins, complement factors, inflammatory cytokines, and chemokines. In addition to its central role in host defense, the liver is also highly susceptible to immune-mediated injury triggered by infectious agents, autoimmune mechanisms, and malignant processes.

Recent research has highlighted the liver as a key site for innate immune activity. The innate immune system acts as the body’s first line of defense, responding rapidly to environmental challenges including microbial invasion and physical or chemical insults.

Effective clearance of pathogens and proper immune regulation rely on the sequential activation of both the innate and adaptive immune systems, a process largely coordinated by dendritic cells that bridge the two arms of immunity.

The liver hosts a specialized array of immune cells, including dendritic cells, natural killer T (NKT) cells, and regulatory T cells, which collectively shape the local immune response.

Moreover, non-immune hepatic cells—such as hepatocytes, sinusoidal endothelial cells, and biliary epithelial cells—also participate in immune regulation, contributing to the liver’s unique immunological capacity.

These immune and non-immune components act both synergistically and independently to determine the liver's response to various immunological stimuli. Furthermore, individual variations in genetic background significantly influence immune responsiveness to both external and internal agents. Understanding the genetic underpinnings of immune function may offer novel insights into the pathogenesis, diagnosis, and treatment of liver diseases.

Materials and Methods

The aim of this study was to analyze morphological changes in the liver across different HBV genotypes in Uzbekistan.

Mononuclear infiltration is closely associated with the development of fibrotic and cirrhotic changes in the liver. However, the exact mechanisms linking infiltration to fibrosis—similar to those observed in pneumosclerosis—remain poorly understood. The rate and extent of fibrotic transformation in areas of inflammatory infiltration can vary significantly.

To date, little is known about the general patterns of collagen synthesis and degradation in the liver under normal conditions and during various stages of chronic inflammation. There is reason to believe that collagen synthesis is upregulated even at early stages of mononuclear infiltration. Nevertheless, in such cases, either collagen accumulation does not occur, or its degradation proceeds simultaneously at an accelerated rate.

Interestingly, during the phase of progressive inflammatory infiltration, collagenase activity in liver tissue increases sharply, whereas during advanced, irreversible fibrosis, its activity markedly decreases [Okazaki L. et al., 1976]. This may be due to suppression of Kupffer cells (KC), the main source of hepatic collagenase [Fujiwara H. et al., 1973], or due to impaired recruitment and retention of macrophages (Mφ) within inflammatory foci (see Chapter 3).

It is also plausible that Kupffer cells, along with macrophages from a mobile pool—similar to pulmonary macrophages—may under certain conditions produce not only collagenase [Decker K. et al., 1982], but also fibroblast-stimulating factors (FSFs). Supporting this, studies show that following CCl_4-induced hepatotoxicity, Kupffer cells become rapidly activated, followed by activation of hepatic stellate cells (HSCs), the precursors to fibroblasts, within a few days. It is thus likely that KCs serve as a source of factors that promote the proliferation of HSCs, with all the downstream consequences [Geerts A. et al., 1984].

Further evidence of a functional connection between hepatic macrophages and HSCs comes from our own experiments. When rats undergoing CCl_4 intoxication were treated with prodigiosan, mononuclear infiltration in the liver was enhanced. At the same time, however, the progression from infiltration to unbalanced fibrogenesis was significantly delayed [Kutina S.N., Mayansky D.N., 1981].

In progressive mononuclear infiltrates, collagen is not only actively synthesized by fibroblasts, but also degraded by collagenases produced by mesenchymal cells. Initially, collagen synthesis and degradation remain balanced. However, as collagenase activity decreases, fibrotic remodeling of the liver stroma becomes stabilized. As long as the liver retains a sufficient number of macrophages (Mφ) capable of producing collagenase and breaking down preformed collagen, the fibrotic process tends to shift toward regression and resorption of fibrous septa.

According to our data, the number of functionally active macrophages within fibrotic septa gradually declines as fibrosis progresses. At the stage of stable, irreversible fibrotic transformation, their presence becomes minimal (see Fig. 30). This decline likely plays a decisive role in the transition from balanced fibrogenesis to persistent liver fibrosis.

Therefore, hepatic macrophages may serve as a central regulatory element, mediating various signals that either enhance or suppress fibrogenesis. They can be seen as "conductors" of the fibroplastic process, whereas fibroblasts—the main producers of collagen—act as its "performers." It is also important to consider that mononuclear infiltrates or granulomas include a proportion of T lymphocytes, whose number varies depending on the intensity of antigenic stimulation originating from the site of inflammation.

For instance, T cells are abundantly present in hepatic granulomas induced in rats by streptococcal group A peptidoglycan-polysaccharide complexes [Wahl S. et al., 1986] or by *Schistosoma mansoni* cercariae [Weinstock V., Blum A., 1986]. The latter model is widely used for studying cellular mechanisms involved in the transition from infiltration to fibrosis within granulomatous zones. It has been shown that the transformation of “active” granulomas into “resolving” ones—characterized by connective tissue ingrowth—depends on the activity of both macrophages and T cells.

In the experiments of S. Wahl et al., fibrogenesis within delayed-type hypersensitivity (DTH) granulomas was clearly dependent on antigen-stimulated T lymphocytes. Both whole granulomas and isolated cells cultured in vitro produced large amounts of soluble factors that stimulated fibroblast proliferation. The T cell dependence of fibrosis was further supported by the fact that fibrotic development was markedly reduced in athymic nude mice and in animals treated with cyclosporin A, a potent T cell inhibitor.

Additionally, factors isolated from cirrhotic human liver were shown to increase collagen synthesis in fibroblast cultures by more than five-fold [Rojkind M., 1983]. It would be of great interest to compare these with the factors secreted by macrophages and T cells. Necrotic hepatocyte products also act as strong stimuli for hepatic fibrosis. Although the exact mechanisms remain unclear, they may involve the activation of macrophages by proteases, leukotrienes, and other so-called “necrohormonal” fractions.

CONCLUSION:

In summary, during hepatic inflammation—where hepatocytes represent the primary targets of immune-mediated injury—non-parenchymal liver cells play a pivotal role in shaping the inflammatory and immunological landscape. Through mechanisms such as chemokine secretion, facilitation of lymphocyte adhesion and migration, antigen presentation, and modulation of T cell responses, these cells not only amplify inflammatory processes but also

contribute to maintaining immune homeostasis under physiological conditions. Their active participation highlights the complexity of liver immunobiology and underscores their significance in both the progression and potential resolution of immune-mediated liver diseases.

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