LETTER TO THE EDITOR

Clinics related to acute pancreatitis wonder whether IFN-γ can attenuate pancreatic injury or not

Ramazan Amanvermez[1]*, Gökhan Selçuk Özbalcı[2]

[1]Department of Medical Biochemistry, Faculty of Medicine, Ondokuz Mayıs University 55139 Samsun, Turkey. [2]Department of General Surgery, Faculty of Medicine, Ondokuz Mayıs University 55139 Samsun, Turkey.

Acute pancreatitis (AP) is an inflammatory disease of the pancreatic tissue associated with little or no fibrosis of the gland. Continued clinical and experimental studies/trials are important for understanding AP pathogenesis and its current treatment approaches. Meng et al. [1] contribute to our knowledge on NF-κB and cytokines IL-18 and IL-27 in experimental AP at 6 h, 12 h, 24 h and 48 h after IFN-γ treatment. However, the messages are implicit both in presentation of results and in the paper itself are complicated by the fact that is likely to be data interferences in relation to AP treated with IFN-γ. For instance, the conclusion that "*the increase in NF-κB and IL-18 may exert influence on pro-inflammatory cytokines to deteriorate inflammation in the pancreas. Thus, to control the IFN-γ might has promise to attenuate pancreatitis... IFN-γ treatment might be associated with JAK-STAT mediated transcription activation*" is based on a speculative opinion. There are just similar sentences taken from different articles excepting for cited references in introduction section, the amount of the application dose for IFN-γ is unknown and the authors are no interpretation their own findings on discussion section in this study. According to this study, serum amylase level, the edema, the NF-κB and TNF-α expression in the pancreas were significantly increased in the treatment of IFN-γ after AP. As a result of these findings, the application of IFN-γ can cause a deleterious effect within the pancreas in the course of AP. On the other hand, Hayashi et al. [2] reported that recombinant murine IFN-γ therapy markedly alleviated acute pancreatitis when administered 4 hours in mice, with reduced NF-κB activation and COX-2 expression. Thus, IFN-γ may possess anti-inflammatory effects on AP by repression of the proinflammatory consequences of NF-κB activation. In addition, Rau et al. [3] have indicated that immunostimulative treatment with recombinant rat IFN-γ attenuated the progression of intrapancreatic necrosis within the first 24 hours after AP induction along with a substantial reduction of subsequent neutrophil tissue infiltration and decreased TNF-α at the late stage of AP. Moreover, plasma IFN-γ concentration is known to increase at the early stage of disease in mild and severe AP patients compared with healthy controls. Therefore, immunostimulative regime could be more effective during the late stage of this disease when infectious complications and immunoparalysis might be a dominant cause of mortality in the course of AP. Actually, pharmacological therapies are limited in the treatment of AP, and none of the therapeutic agents used for therapy are effectively curative. Regardless of AP severity, hospitalization of the patients with suspected acute pancreatitis for observation and diagnostic study is generally mandatory. After the diagnosis, patients with moderate to severe disease should be transferred to the ICU for observation, and supportive treatments and interventions. Treatment may change depending on the etiology and severity of the disease. Antibiotic therapy, peritoneal lavage, sphincterotomy with ERCP and even surgical operations can be applied [4, 5]. Novel effective therapeutic strategies in the treatment of AP may be evolved.

DECLARATION OF INTEREST

The authors declare no conflict of interest.

REFERENCES

[1] Meng H, Gong J, Fang L, Song Z, Wu F, Zhou B, et al. Effect of interferon-γ on NF-κB and cytokine IL-18 and IL-27 in acute pancreatitis. Bosn J Basic Med Sci 2013; 13 (2): 114-118.

[2] Hayashi T, Ishida Y, Kimura A, Iwakura Y, Mukaida N, Kondo T. IFN-γ protects cerulein-induced acute pancreatitis by repressing NF-κB activation. The Journal of Immunology 2007; 178: 7385-7394.

[3] Rau BM, Krüger CM, Hasel C, Oliveira V, Rubie C, Beger HG, et al. Effects of immunosuppressive and immunostimulative treatment on pancreatic injury and mortality in severe acute experimental pancreatitis. Pancreas 2006; 33 (2): 174-183.

[4] Kilic E, Amanvermez R, Kefeli M, Polat C, Gunay M. Protective effects of etanercept and methylprednisolone on pancreatic damage in cerulein-induced acute pancreatitis. Saudi Med J 2010; 31 (4): 394-399.

[5] Brunicardi FC, Anderson DK, Billiar TR, Dunn DL, Hunter JG, Matthews JB, et al. Schwartz's principles of surgery (9th ed.), 2010. Chapter 33: Pancreas, pp: 1177-1186.

* Corresponding author: Ramazan Amanvermez,
Department of Medical Biochemistry, Faculty of Medicine,
Ondokuz Mayıs University 55139 Samsun, Turkey
Phone: +90 362 3121919, ext.: 2534
Fax: +90 362 4576041
e-mail: aramazan@omu.edu.tr