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31-12-2021

Sayı: 797-TGD-2021
Konu: TGD Araştırma Projeleri Destek Bursu Hk.

TÜRK GASTROENTEROLOJİ DERNEĞİ SAYIN ÜYESİ
Dr. Prof. Dr. Serhat BOR

Türk Gastroenteroloji Derneği "Araştırma Projelerine Destek" faaliyetleri kapsamında yürütülen çalışmalarımız ile ilgilendiğiniz için teşekkür ederiz.

Yapılan proje başvuruları arasında "Laparoskopik Anti-Reflü Cerrahisi Öncesi ve Sonrası Özofagus Mukozasında Meydana Gelen İnflamatuar Değişikliklerin Araştırılması" konu başlıklı çalışmanız proje değerlendirme kurulu tarafından uygun görülmüş olup, bütçe desteği yönetim kurulu kararı ile kabul edilmiştir.

Tebrik eder, çalışmalarınızda başarılar dileriz.

Saygılarımla.

[redacted]

Prof. Dr. Dilek OĞUZ
TGD Yönetim Kurulu Başkanı

TÜBİTAK

Gastroözofageal Reflü Hastalığının Patogenezinde İnflamasyon Mekanizmalarının Araştırılması

Program Kodu: 1001

Proje No: 118S260

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HAZİRAN 2021

İZMİR

Abstract

Investigation of Inflammatory Mechanisms in the Pathogenesis of Gastroesophageal Reflux Disease

Gastroesophageal Reflux Disease (GERD) is frequently seen in the community and has no definitive treatment. There are two main views on the pathogenesis of the disease. The first is that epithelial damage starts from the surface and progresses to the basal layer as a result of acidic-peptic damage and the inflammatory response of granulocytes. However, this view is insufficient to explain patients without erosion and patients with mild acid exposure. The other view is that T lymphocytes atract from the basal layer with the chemoattractants, and granulocytes do not migrate until the damage occurs. The second theory has been supported by current studies. Based on this information, we aimed to investigate the inflammatory processes occurring in the esophageal epithelium of the phenotypes and the patients who recovered after laparoscopic reflux surgery at the molecular level and to examine the effects of these changes on tissue integrity. For this purpose, five phenotypes of GERD including mild erosive reflux (ERD A/B), severe erosive reflux (ERD C/D), non-erosive reflux (NERD), esophageal hypersensitivity (HS) and functional pyrosis (FP) and post-operative patients who had reflux surgery among these supgroups were included in the study.

Esophageal biopsies were taken during upper GIS endoscopy; inflammatory gene expressions, protein levels and tissue integrity were examined. As a result of the study, we showed that although there was chronic inflammation in the severe erosion group, the acute response was also triggered, and in the mild erosion group, these two processes worked together, but cytokines were also secreted, which preserved the cell homeostasis. In other groups, it was concluded that T lymphocytes were more dominant, supporting the current theory. In addition, it was understood that the inflammatory response was highly triggered in the HS and FP groups associated with mild reflux exposure and sensitivity, but sensitivity-related cells such as mast cells and anti-inflammatory cytokines were at higher levels. After reflux surgery, homeostatic parameters were dominant and anti-inflammatory cytokines had an intense effect.

This is the most comprehensive study of immune cells and cytokines/chemokines in the esophageal mucosa of GERD phenotypes and patients after reflux surgery. Many proteins in our study were demonstrated for the first time in the esophageal epithelium. Our results will shed light on the potential role of cytokine-directed therapies in the treatment of GERD.

Keywords; Gastroesophageal reflux disease, inflammation, cytokine, chemokine, esophagus