

Eosinophilic pancreatitis need an attention.

Murli Manohar*

School of Medicine, Tulane University, New Orleans, USA

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Inflammatory diseases include a vast array of disorders and conditions that are characterized by inflammation, such as allergy, asthma, eosinophilic gastrointestinal disorders including pancreatitis [1-6]. The data indicates, 13 to 45/100,000 people in United States are having acute pancreatitis (AP) annually, whereas, chronic pancreatitis (CP) varies from 4.4 to 11.9/100,000 [1-3]. Pancreatitis is the inflammation of pancreas and arises due to several reasons such as alcohol abuse, mutation in trypsinogen gene, gallstones, and several drugs and food induced. Pancreatitis is of two types; the initial shock of pancreatitis is called acute pancreatitis and when these episodes frequency occurs several time it leads chronic pancreatitis. Pancreatic inflammation is characterized by destruction of acinar cells, which lead activation of several inflammatory cells like mast cells, neutrophils, macrophages and granulocytes such as basophils and eosinophils that secrete several pro-inflammatory cytokines [2,3]. Eosinophils develop during hematopoiesis in the bone marrow before going into bloodstream and about 1-6% of white blood cells. They have about half-life of 8 to 18 hours in the bloodstream, and mostly reside in the tissues where they can live for several weeks [7]. Eosinophils play key role in the mucosal immune system of the gastrointestinal tract during normal and inflammatory conditions. In normal conditions, the mucosa of the digestive tract is the only organ harboring a substantial number of eosinophils, which, if need be, get activated and exert several effector and immunoregulatory functions [8]. Eosinophils promote tissue fibrosis by secreting pro-fibrotic TGF- β 1 and other cytokines [9]. Several cases reports have been cited in the literature indicating the accumulation of eosinophils in pancreatitis patients and termed this condition as “Eosinophilic Pancreatitis (EP)” [10-14] but detailed mechanistic analysis of eosinophilic pancreatitis is ignored and not well explored.

We recently reported an important role of eosinophils in cerulein-induced chronic pancreatitis. Even we first time located eosinophils accumulation and degranulation followed by induced mast cells and its degranulation and acinar cells atrophy in the pancreas of cerulein-induced murine model of pancreatitis. Our data also indicates that the eosinophils deficient GATA 1 mice show reduced pancreatic remodeling compared to wild type or IL-5 gene deficient mice following cerulein-induced chronic pancreatitis [15]. These experimental data indicate that eosinophils accumulation and degranulation may be critical in promoting pancreatitis pathogenesis including fibrosis that is the major concern for the treatment options. Further, we extended our study to see the role of eosinophils in human pancreatic malignant, non-malignant and healthy biopsies and confirmed the accumulation and degranulation of eosinophils, mast cells,

induced IgE level and induced collagen deposition in malignant pancreatic biopsies as compared to healthy control [16]. Taken together, our experimental and translational approaches indicate that indeed eosinophilic pancreatitis may be an independent disease entity that needs appropriate attention to understand the mechanism of eosinophils accumulation in the pancreas and in promoting pancreatic fibrosis including malignancy.

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***Correspondence to:**

Murli Manohar, Ph.D.
School of Medicine
Tulane University
New Orleans
USA
E-mail: mmanohar@tulane.edu