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Gut-the Trojan horse of systemic autoimmunity

Aaron Lerner[1] and **Torsten Matthias** [2]
[1]Technion-Israel Institute of Technology, Israel
[2]AESKU KIPP Institute, Germany

Objectives & Study: In suitable circumstances, the human gut possesses all the components necessary to start the autoimmune cascade. The aim of the study was to characterize the multiple gut-remote organ autoimmune axes.

Methods: A systematic review was performed to identify studies referred to gut-gut, brain, joint, bone, endocrine, kidney, lung, liver, heart and skin exes using Medline, Google and Cochrane Library databases.

Results: The specific dysbiota and tight junction dysfunction seems to be a primary defect in autoimmune diseases. Intestinal permeability is decreased in many: Ulcerative colitis, Crohn's disease, celiac disease, inflammatory joint disease, ankylosing spondylitis, juvenile onset arthritis, psoriatic arthritis, type 1 diabetes mellitus and primary biliary cirrhosis. The end result of the passage of those non-self- proteins, from the luminal compartment to the sub epithelial one, initiates the autoimmune cascade. The richness of the mucosal milieu in immune components, cells and systems; blood and lymphatic vessels; entero-neuronal and endocrine network; and mural endo-mesoderm cohabitation constitute an ideal place to initiate, maintain and propagate the autoimmune process. The mucosal committed immune cells, post translation modified proteins, proinflammatory cytokines and lymphokines have the capacity to circulate via the local vessels, to bring the autoimmune message to remote organs, thus creating gut-extra intestinal organ axes of autoimmunity. Each one of the remote organs: Brain, joint, bone, endocrine, kidney, lung, liver, heart and skin, is directionally relayed to the intestinal events taking place in the genetically susceptible individuals

Conclusions: The immune system carefully distinguishes between self and non-self-components. The intestine is a major site of changing tolerance to autoimmunity. The disease specific dysbiota, its post translational capacity to modify proteins, the plethora of substrates, the leaky gut, the local adjacent immune, neuroendocrine, vascular and lymphatic systems make the intestine a prime candidate to drive systemic autoimmunity

aaronlerner1948@gmail.com

No-fistula vs. fistula anorectal malformation: Outcome comparative study

Abdullah Sarkar
Alfaisal University, Saudi Arabia

Anorectal malformations (ARM) refer to a wide variety of congenital anomalies, most commonly referring to imperforate anus. Occurring on average of 1:3500 live births, imperforate anus is described as the failure of the rectum to descend through the external sphincter complex. In surgical history, the first form of management for imperforate anus began in the 7th century with a highly morbid procedure by Paulus Aegineta, and until current day pediatric surgical intervention of ARM has evolved with modifications and updates periodically; attempting to improve our pathophysiological understanding and surgical outcome. Anorectal malformation is a congenital defect that exists in varying presentations. These are of two types, high and low anomalies owing to its severity. The high-type anorectal malformation is divided into two types, with (fistula) and without fistula (no-fistula). Of the categorical presentations, no-fistula type ARM has recently been investigated and reported on as its own disease in the literature; with increasing evidence of distinct associations, risk factors, and anatomical differences with surgical management implications and outcomes. As pediatric surgeons globally have identified unique characteristics associated with no-fistula type ARM, we aim to report our experience with management of this anomaly and its outcome, compared to the more common fistula type ARM.

asarkar1992@gmail.com