conferenceseries.com

J Gastrointest Dig Syst 2018, Volume 8
DOI: 10.4172/2161-069X-C2-068

Novel therapeutic targets for acute pancreatitis

Madhav Bhatia
University of Otago, New Zealand

Acute pancreatitis is a common clinical condition. Excessive Systemic Inflammatory Response Syndrome (SIRS) in acute pancreatitis leads to distant organ damage and Multiple Organ Dysfunction Syndrome (MODS), which is the primary cause of morbidity and mortality in this condition. Development of in vivo experimental models of acute pancreatitis and associated systemic organ damage has enabled us to study the role played by inflammatory mediators in the pathogenesis of acute pancreatitis and associated systemic organ damage. Using these models, recent studies have established the critical role played by inflammatory mediators in acute pancreatitis and the resultant MODS. Hydrogen sulfide (H2S) plays an important role in cardiovascular, central nervous and gastrointestinal systems and has been shown to act as a vasodilator. We have also shown that H2S acts as a mediator of inflammation. Substance P is an 11 amino acid neuropeptide that is released from nerve endings in many tissues. Subsequent to its release, substance P binds to neurokinin-1 (NK-1) receptors on the surface of effector cells. Using experimental models, recent studies in our laboratory have established the critical role played by H2S and substance P in acute pancreatitis. Furthermore, early results point to the clinical relevance of this research. Studies with experimental animal models of disease will therefore help define the role of these mediators in the pathogenesis of acute pancreatitis and can lead to the development of novel therapeutic approaches for this condition.

madhav.bhatia@otago.ac.nz

Notes: