EuroSciCon

6th Annual European Conference on

Gastroenterology

June 19-20, 2018
Paris, France

Kenji Sasaki, J Clin Gastroenterol Hepatol 2018, Volume: 2
DOI: 10.21767/2575-7733-C1-001

CANDIDA-ASSOCIATED GASTRIC ULCER UNTIL YESTERDAY, TODAY AND FROM TOMORROW

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Candida-associated gastric ulcer, though formerly thought to affect only debilitated persons, has been reported to occur in apparently healthy individuals. Though had been reported to demonstrate nothing but nonspecific endoscopic features, the disease occasionally exhibits an apparently typical finding designated a candidarium. The natural history of the disease had been unknown and the fungus had been reported to be no longer detected once the ulcers were healed and no recurrence of the disease had been described. However, the ulcer is shown to not only occur but also recur in a different site with a different shape in a non-diabetic, *Helicobacter pylori*-negative patient without antecedent ulcers, who has not been given non-steroidal anti-inflammatory drugs (NSAIDs), antibiotics, or antineoplastic agents, which implies that, contrary to the prevailing opinion, Candida is no innocuous bystander but an etiologic perpetrator. Immune deficiency has recently been reported in relation to candidiasis, which is considered to explain the cause of intractable or recurrent Candida-associated gastric ulcer. In the oropharyngeal field, Candida albicans has recently been shown to secrete a hitherto unknown cytolytic peptide pore-forming toxin (PFT), candidalysin, into a pocket in the epithelium which penetrates into and to activate mitogen-activated protein kinase (MAPK)/MAPK phosphatase 1 (MKP1)/c-Fos pathway, triggering release of damage as well as immune cytokines. While the PFT, exerting an effect even on the adjacent cells, directly injures the tissue with damage cytokines, immune counterpart activates polymorphonuclear leukocytes (PMN) to eventually terminate inflammation, which results in restoring the fungus to the commensal state or eradicating it. Since it cannot be negated that such a phenomenon occurs in the gastric mucosa, a theoretically strong possibility has come up that the so-called Candida-associated gastric ulcer is actually Candida-induced ulcer. Therefore, the disease should be reinvestigated in the light of the recent immunological, microbiological, and molecular biological findings.

Biography

Kenji Sasaki has completed his MD and as an Immunologist, he completed his PhD at Tohoku University School of Medicine. He was trained at Miyagi Cancer Center. He is a Board Certified Fellow and Preceptor of Japan Gastroenterological Endoscopy Society, Board Certified Gastroenterologist of Japanese Society of Gastroenterology, Board Certified Member of the Japanese Society of Internal Medicine and Editorial Board Member of CRIM. He has published several papers on Gastroenterology in international journals and served as a Reviewer for Journal of Medical Microbiology, Journal of Pharmacology & Pharmacotherapeutics and Journal of Gastrointestinal & Digestive System.

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