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Biography

Kenji Sasaki received his MD in 1973 and PhD in 1977 from Tohoku University. He is a Board Certified Fellow and Preceptor of the Japan Gastroenterological Endoscopy Society, Board Certified Gastroenterologist of the Japanese Society of Gastroenterology, Board Certified Member of the Japanese Society of Internal Medicine and Editorial Board Member of CRIM. He has given presentations at international medical congresses and published papers on gastroenterology in international journals. Acclaimed by Prof Tarnawski at DDW 2012, he published his article Candida-associated gastric ulcer relapsing in a different position in a different appearance in *World J Gastroenterol*. He served as a reviewer for CRIM, JMM, JPP and J Gastrointestinal Dig Syst.

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CANDIDA-ASSOCIATED GASTRIC ULCER UNTIL YESTERDAY, TODAY, AND FROM TOMORROW- IN QUEST OF THE ETIOLOGY

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), candida lysin, into a pocket in the epithelium which penetrates 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 Candida-induced ulcer. Therefore, the disease should be reinvestigated in the light of the recent immunological, microbiological, and molecular biological findings.

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