2nd Annual Congress on
MICROBIOLOGY AND MICROBIOLOGISTS
&
6th International Conference on
MYCOLOGY AND FUNGAL INFECTIONS
October 07-08, 2019 | Madrid, Spain

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Candida-associated gastric ulcer until yesterday, today and from tomorrow

Candida-associated gastric ulcer occurs not only in debilitated but healthy individuals. Though had been reported to demonstrate nothing but nonspecific endoscopic features, it occasionally exhibits a typical finding the author designated a candidarium. The natural history of the disease had not been clarified and the recurrence had not been described: the fungus had been reported to become undetectable once the ulcers were healed. However, the author demonstrated that the ulcer not only occurs but also recurs in a different site with a different shape in a non-diabetic, *Helicobacter pylori*-negative patient, who has not been given non-steroidal anti-inflammatory drugs, antibiotics, antineoplastic agents, or systemic corticosteroids, advocating that, contrary to the prevailing opinion, *Candida* (C.) is no innocuous bystander but an etiologic perpetrator: intragastric inoculation of *C. albicans* causes epithelial necrosis through activation of IL-23/IL-17 pathway in mice. In the oropharyngeal field, the fungus has recently been shown to secrete a cytolytic pore-forming toxin (PFT), candidalysin, into a pocket in the epithelium after penetrating into it to activate mitogen-activated protein kinase (MAPK)/MAPK phosphatase 1 (MKP1)/c-Fos pathway, triggering release of damage as well as immune cytokines. While candidalysin, exerting an effect even on the adjacent cells, directly injures the tissue with damage cytokines, immune counterparts activate polymorphonuclear leukocytes to eventually terminate inflammation. Though the epithelial response to the fungus is different from organ to organ, it invades into and induces necrotic cellular damage to the intestinal mucosa through the toxin to translocate: the action of candidalysin is proven not only on the stratified squamous mucosa but on the single layer of the columnar epithelium. Since, by analogy with intestinal candidiasis, it is never difficult to speculate that the PFT inflicts such damage to the gastric mucosa, a theoretically strong possibility has come up that *Candida*-associated gastric ulcer is actually *Candida*-induced ulcer.

Biography

Kenji Sasaki received his MD in 1973 and PhD in 1977 from Tohoku University. He is a JGES Board Certified Fellow and Preceptor, JSGE Board Certified Gastroenterologist, JSIM Board Certified Member and CRIM Editorial Board Member. He has given presentations at international 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 2012 Aug 28; 18 (32): 4450-4453, which was featured in the section of Infection and Immunity of World Biomedical Frontiers in September, 2013 and recommended by an associate research scientist on PubAdvanced in December, 2013. Invited to participate in a special issue, he published a review article "*Candida*-associated gastric ulcer until yesterday, today, and from tomorrow --- In quest of the etiology" in SciTz Gynecol Reprod Med 2017; 1(1): 1002.

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