



A rare complication of IgG4-related disease in an ulcerated gastric cancer

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ABOUT THE STUDY

IgG4-Related Disease (IgG4-RD) is a chronic multi-organ fibro inflammatory condition characterized by tumefactive lesions, elevated serum IgG4 levels (often but not always), and tissue infiltration by dense lymphocytes and IgG4-positive plasma cells. Clinically and radio logically, IgG4-RD is frequently confused with neoplasm, infectious, and inflammatory disorders. The pancreas, biliary duct, lacrimal/salivary glands, lymph node, lung, liver, kidney, thyroid gland, gastrointestinal tract, prostate, hypophysis, stomach, skin, aorta, joint, retro peritoneum, meninges, and pleura are all known to be involved in IgG4-RD. We are not aware of any reports of IgG4-RD of the stomach associated with fungal infection. We present a case of IgG4-RD that manifested as ulcerated gastric cancer complicated by gastric mucormycosis.

IgG4-RD is a systemic disorder characterized by tumefactive lesions, elevated serum IgG4 levels (often but not always), and tissue infiltration of dense lymphocytes and IgG4-positive plasma cells in the affected organs. One of the most common disease symptoms is Autoimmune Pancreatitis (AIP). IgG4-RD has been found in almost every organ system, including the pancreas, biliary duct, lacrimal/salivary glands, lymph nodes, lung, liver, kidney, thyroid gland, gastrointestinal tract, prostate, hypophysis, stomach, skin, aorta, joint, retro peritoneum, meninges, and pleura.

Hereditary susceptibility, autoimmunity, and infectious agents are all thought to be risk factors for IgG4-RD. The DRB1*0405-DQB1*0401 mini-haplotype and the ABCF1 gene area appear to control the HLA-linked genetic basis for AIP, according to their findings. Furthermore, the peptides of *Helicobacter pylori* Plasminogen-Binding Protein (PBP) and Ubiquitin-Protein Ligase E3 Component N-Recognin 2 (UBR2) expressed by pancreatic acinar cells are homologous. Antibodies against the PBP peptide were detected in 94% of

autoimmune pancreatitis patients (33/35), but only 5% of pancreatic cancer patients (5/110), indicating that infectious factors and molecular mimicry may initiate IgG4-RD.

Furthermore, the innate and adaptive immune systems may work together to accelerate the progression of IgG4-RD and related tissue or organ fibrosis. Microenvironment disorders, including immune cells such as regulatory T cells, regulatory B cells, T follicular helper 2 cells, and M2 macrophages, are critical for the pathogenesis of IgG4-RD. Inflammatory cytokines such as IFN-, IL-4, IL-10, IL-5, IL-13, and TGF- are also involved in the IgG4-RD. In fact, IgG4-RD has no specific pathogenic factors, and its cognitive process is dynamic. Although genetic factors, infections, and autoimmunity disorders may all play a role in the pathogenesis of IgG4-RD, none of them are sufficient to explain it.

Because the incidence of IgG4-RD is quite low (1.4 cases per 100,000 people), diagnosis may be missed or incorrect at times. The multidisciplinary approach may be beneficial in the diagnosis of IgG4-RD. There are no specific clinical symptoms or signs for IgG4-RD, which are usually common in other diseases or exerting symptoms of complications. Males are more likely to experience abdominal pain, nausea, vomiting, and jaundice, whereas females are more likely to experience swelling of the lacrimal and submandibular glands. Male salivary glands, pancreas, and lacrimal glands are the most commonly involved organs, while female salivary glands, lacrimal glands, and sinuses are the most commonly involved organs.

Most patients with increased ESR, CRP, serum IgG level, and total IgE level have elevated serum IgG4 levels, but some patients have normal serum IgG4 levels (which vary greatly between studies, ranging from 2.5% to 40%). Increased IgG4 levels, on the other hand, can occur in other diseases such as allergy and lymphoma. The most

common imaging findings are organ enlargements of involved tissues. Honey-combing or ground-glass pacification, pulmonary nodules, and broncho vascular bundle thickening may be found if the lungs are involved. To some extent, both clinical and imaging manifestations are atypical, which is likely to be misleading and confusing, especially for single or infrequently involved organs. Pathological examination is required and required for the diagnosis of IgG4-RD.

The most common microscopic findings are diffused lymphoplasmacytic infiltration with irregular and storiform fibrosis, as well as obliterative phlebitis. Sometimes there is phlebitis without lumen obliteration and an increase in eosinophil's, but no sensitive or specific effects for the diagnosis of IgG4-RD. The presence of necrosis, discrete granuloma, and extensive neutrophilic infiltration all point to IgG4-RD. Immunohistochemical staining for IgG and IgG4 is critical. The absolute number of IgG4 positive plasma cells in corresponding organs or tissues should meet a certain standard, and the IgG4/IgG positive plasma cell ratio should be greater than 40%. Although pathological examination is important for the diagnosis of IgG4-RD, it cannot be confirmed by microscopy alone; multidisciplinary collaboration and comprehensive consideration of clinical and imaging manifestations, as well as laboratory tests, is critical, especially in cases where only one or a few organs are involved.

To our knowledge, IgG4-RD rarely involves stomach. In the few reports available, almost every case is presenting

multiple organs involvement accompanied by stomach related, and no case with fungal infection. We summarize all literature on the gastric involvement of IgG4-RD. In the above table, the majorities of cases are multiple organs involved or confirmed by surgical specimen; no patient associates fungal infection. Microscopically, there are four zones in the base of the ulcer of surgical specimen: inflammatory exudates, fibroid necrosis, granulation tissue and mature fibrous tissue. However, pathologists always accept biopsy tissues surrounding the base of the ulcer in the clinical practice for fear that gastric perforation and unnecessary operation, which undoubtedly brings benefit for patients.

At the same time, biopsy specimens make diagnosis of IgG4-RD involving the stomach difficult, particularly when accompanied by an ulcer lesion. Pathologists must form broad conclusions based on local findings. In this case, fungal infection complicates and complicates diagnosis. Under a microscope, gastric mucormycosis may cause secondary changes for ulcers; additionally, the identification of specific fungus will influence clinical decision and treatment methods.

According to our knowledge, this is the first report of an IgG4-related disease manifesting as ulcerated gastric cancer complicated by gastric mucormycosis. A multidisciplinary team discussion involving gastroenterology, radiology, and pathology departments is required and required for disease diagnosis.