

Young Patients with Gastric Cancer

Heriberto Medina*

Department of Microbiology & Immunology, Medical University of South Carolina, USA

Abstract

Although it may be found earlier, gastric cancer is often an aging-related disease and is most frequently discovered after the sixth decade of life. Most nations and areas do not have screening programmes for the 5.0% of gastric cancer (GC) patients who are diagnosed before the age of 40. With the exception of the United States, the incidence of gastric cancer in young adults (GKYA) has decreased over time in most other nations. A young adult's susceptibility to GC may be influenced by lifestyle choices, environmental factors, and genetic changes. The majority of GKYA patients have cancers that are classified as either genomically stable or microsatellite stable/epithelial-Mesenchymal transition subtypes, with mutations in CDH1 being the most prevalent genetic abnormalities. The characteristics of GKYA include a higher frequency in females, more aggressive tumour behaviours, detection at late stages, less comorbidities and better treatment candidates, as well as survival outcomes that are comparable to or better than those of older patients. The two most efficient approaches to reduce GC mortality are lowering the prevalence of GC and identifying at a relatively early stage, taking into account the larger loss of life-years in younger individuals. To accomplish these objectives, there needs to be a change in the low level of knowledge of GKYA among the general public, policymakers, doctors, and researchers.

Keywords: Young Adenocarcinoma; Gastric Cancer; Survival; Hereditary gastric cancer; Treatment.

Introduction

With a higher frequency in middle-aged and older populations, gastric cancer is thought to be the second most common cause of cancer-related death. Despite a decline in the overall prevalence of the disease over the past few decades, the rate of stomach cancer in young patients has climbed. Although gastric cancer in young people is uncommon, it is generally accepted that these patients have more aggressive biological behaviour and a worse prognosis. Other writers, however, have noted that the tumour stage and survival of young patients with stomach cancer are comparable to those of older individuals [1].

Some features of gastric cancer in young patients, including a greater frequency of *Helicobacter pylori* infection, a noticeably higher frequency of diffuse intestinal type, and a significantly higher frequency. We conducted a retrospective cohort analysis to identify the causes of the distinct biological behaviour and prognosis in younger patients since data on the biological and clinical courses of gastric cancer in young patients are still debatable. The purpose of this study was to determine the independent risk variables that, when compared to older patients, affect mortality, morbidity, and prognosis in patients under the age of 50 and to pinpoint the cancer characteristics that are typical of a younger age [2].

Gastric cancer (GC) is the fifth most frequent cancer worldwide and the third largest cause of cancer death, making it one of the greatest medical challenges in the world. GLOBOCAN has recorded more than a million new cases of GC and approximately 783 thousand fatalities in 2018. The majority of these cases are found in East Asia, where the age-standardized incidence rate (ASR) is 32.1 per 100,000 for men and 13.2 per 100,000 for women. The highest rate among other South-East Asian nations, the ASR in Vietnam was estimated to be 16.3 per 100,000 individuals of both sexes. Only liver cancer and lung cancer had higher fatality rates in Vietnam than did GC, which affected both males and females equally. The high prevalence of *Helicobacter pylori* (up to 75% among Vietnamese adults) together with other risk factors like smoking, obesity, and socioeconomic level, has been suggested as the cause of this. According to a recent study, it would be crucial to evaluate the health-related quality of life (HRQOL) of GC patients in

order to improve treatment regimens and increase patient and medical professional knowledge. However, the majority of general research on GC in Vietnam has been on illness management and treatment to raise the 5-year survival rate, leaving a void in the patients' HRQOL [3].

Therefore, the 363 patients in our hospital who underwent gastrectomy and D2 lymphadenectomy from 1996 to 2007 were retrospectively examined for clinical and pathological traits. Following surgery, the patients' lymph node metastases were histologically negative, and the invasion depth reached at least the muscle layer (pT2-4N0M0). In this patient population, our goal was to find independent predictive indicators. In order to assess the effectiveness of postoperative adjuvant chemotherapy in this particular patient population, we divided these patients into two groups: a "surgery-only" control group and a "postoperative chemotherapy treatment" group, and we compared their survival rates. Additionally, we compiled and evaluated the risks and side effects of postoperative adjuvant chemotherapy in this patient population [4].

Material and Methods

Adenocarcinoma in the gastric residual that develops at least five years following partial gastric resection for PUD is referred to as GSC. Adenocarcinoma of the distal oesophagus and proximal stomach with the tumour core within 5 cm above to 5 cm below the anatomic EGJ was defined as PGC, while adenocarcinoma of the proximal third of the nonoperative stomach was characterised as EGJC. This study excluded cases of early primary cancer recurrences and metachronous

***Corresponding author:** Heriberto Medina, Department of Microbiology & Immunology, Medical University of South Carolina, United States; Tel: 18932107458; E-mail: Medina.heriberto@gmail.com

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adenocarcinomas. Between January 1980 and June 2012, information on patients who were diagnosed with upper gastrointestinal cancer and were admitted to our tertiary centre was collected in an electronic prospective database that was searched for patients with GSC, PGC, and EGJC [5].

The most common procedure carried out on EGJC patients was splenectomy via left thoracophrenolaparotomy, with total gastrectomy reaching the distal oesophagus. In PGC cases, a total gastrectomy was performed. GSC patients were given the option of undergoing a left thoracophrenolaparotomy or an abdominal approach for a complete gastrectomy. The typical procedure for these individuals was D2 lymphadenectomy accompanied with omentectomy (removal of nodal tissue along the main branches of the celiac axis and per gastric nodes). Selected patients with suspicious nodes in the splenic hilum and/or the mesojejunum, respectively, had en bloc splenectomy and/or prolonged resection of the jejunum [6].

A number of clinical and pathological factors were assessed. According to the above mentioned criteria, macroscopic appearance was divided into fungating and ulcerofungating, ulcerated and ulcer infiltrative, or infiltrative. To classify tumours into intestinal, diffuse, or unclassified kinds, Lauren employed hematoxylin-and-eosin-stained sections. Venous invasion was rated as either present or absent in orcein-stained sections. Additionally, lymphatic vessel invasion was graded as either missing or prominent [7].

Discussion

The size and colour of the gastric neoplasm can be used to distinguish between a gastric adenoma and early gastric cancer utilising C-WLI (conventional endoscopy with white-light imaging). Additionally, the use of indigo carmine dye also contributes some information. The malignancy risk of adenomas smaller than 2 cm in diameter ranges from 1% to 5%, but jumps to more than 50% for bigger adenomas [8]. The size of the adenoma has some bearing on the malignant potential. Malignant lesions are far more likely to be red, however the prevalence of redness in stomach cancer ranged from 60% to 75%. The microvascular and microsurface pattern seen by magnifying endoscopy with narrow-band imaging (M-NBI) is of some use in gastric neoplasms lacking red colouring. We discovered that information regarding the surrounding stomach mucosa serves as a helpful reference for distinguishing between gastric adenoma and early gastric cancer, in addition to the characteristics of the neoplasm itself. Gastric atrophy is a common feature of all gastric neoplasms, but the likelihood of developing early gastric cancer was significantly increased by the C-type pattern of endoscopic gastric atrophy [9].

As the disease progresses, Correa hypothesised the following ontogeny of gastric cancer: normal mucosa, superficial gastritis, atrophic gastritis, intestinal metaplasia, dysplasia, and cancer. Contrary to colon cancer, it is acknowledged that the adenoma-carcinoma sequence is not a prominent gastric cancer ontogeny, and numerous studies have demonstrated that intestinal metaplasia does not always indicate pre cancer status [10]. In our research, intestinal metaplasia was strongly related with both early gastric cancer and gastric adenoma, and we found no discernible difference between the two types of cancer in terms of this association. On the other hand, early gastric cancer was strongly related with localised gastric atrophy (C-type) of the surrounding stomach mucosa, whereas extensive gastric atrophy (O-type) was seen in individuals with either gastric adenoma or early gastric cancer. In other words, a gastric tumour with localised gastric atrophy (C-type) in the surrounding gastric mucosa is likely

to be diagnosed as gastric cancer. Our results support a prior study that found patients with significant gastric atrophy can develop gastric adenoma, and there is no evidence that these patients will eventually develop gastric cancer [11].

A well-designed randomised controlled prospective study should be used to test our conclusion since, first, it was a retrospective study conducted in a single centre. Second, because the study only involved Japanese patients, the findings could not be completely applicable to the Western population, where rates of *Helicobacter pylori* infection, atrophic gastritis, and gastric acid secretion may vary [12].

Conclusion

There are other, not previously addressed, processes that can result in chromosomal instability in stomach malignancies, such as telomere shortening, telomerase activation, relaxation of cell-cycle checkpoints, imperfect chromosomal replication, and so forth. The intricacy and heterogeneity of gastric tumours show that gastric cancer originates from a multi-step process involving various genetic and epigenetic alterations in several genes, in addition to the genetic makeup of the host and environmental variables [13]. There are countless changed genes linked to gastric carcinogenesis, however it is unclear how significant these alterations are. According to study, genetic instability is an early event in the development of stomach cancer and has a significant role in the onset and advancement of neoplastic diseases [14]. The precise driving forces of genomic instability have not yet been identified. The understanding of gastric aetiology is further complicated by complicated gene-gene interactions, gene-environment interactions, lifestyle factors, and ethnic background. Complete genotyping of stomach cancer patients may provide more knowledge about genetic variants and their interactions, allowing the identification of groups at higher risk of developing the disease. Additionally, algorithms that may explain all these alterations in people and their interactions need to be created. Large research and meta-analyses must also be conducted in order to identify panels of biomarkers for susceptibility assessment, diagnosis, prognosis, and treatment [15].

Conflicts of Interest

None

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None

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