HEMORRHAGIC SHOCK AS A PRIMARY AND UNUSUAL MANIFESTATION OF GASTRIC CANCER: A CASE REPORT

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Abstract

Gastric cancer represents a major health burden worldwide. The cause of gastric cancer is multifactorial, although infection with Helicobacter pylori is considered to be the primary cause. The early stages of gastric cancer are usually asymptomatic or associated with nonspecific symptoms, such as dyspepsia. Advanced stages and ulcerated tumors may be associated with signs of upper digestive bleeding and hemorrhagic shock. Upper gastrointestinal endoscopy is the first preferred diagnostic procedure. The only chance for cure of gastric cancer is surgical removal of the tumor and the adjacent lymph nodes. Case report: 67 year old male presented with melena and haematemesis. On examination pale, covered with cold sweat, advnamic, hypotensive TA = 60/30 mmHg in hemorrhagic shock. After initial stabilization esophagogastroduodenoscopy was performed and showed irregular and longitudinal ulceration with elevated and subminated edges, necrotic bottom of the lesion with a fixed coagulum. Gastric cancer was suspected and the patient was transferred to Digestive Surgery where a total gastrectomy was performed. The pathohistological finding is in addition to gastric adenocarcinoma. Conclusion: Epidemiologic studies suggest that eating more fresh fruit and vegetables, eating less salted food and eradicating Helicobacter pylori can decrease the risk of gastric adenocarcinoma. The best way for prevention is to carry out an effective screening program in high-risk groups and in high-incidence areas, so that early discovery, early diagnosis and early treatment can be achieved.

Key words: Gastric cancer, Helicobacter pylori, Hemorrhagic shock, Prevention, Early detection

Introduction

Gastric cancer is a major health burden worldwide. It is the second cause of cancer deaths after lung cancer (Ferlay et al., 2008; Jemal et al., 2011). More than 90% of the tumors are adenocarcinomas. The prognosis is dismal, with an average 5-year survival rate of less than 20%, mainly because of late diagnosis, because the early stages are clinically silent. The cause of gastric cancer is multifactorial, although infection with Helicobacter pylori is considered to be the primary cause; its effects are modulated by microbial, environmental, and host factors. Gastric cancer is one of a few types of neoplasms directly linked to an infectious agent. In 1994, the International Agency for Research on Cancer (IARC) classified infection with *H pylori* as a class I human carcinogen for gastric cancer (IARC, 1994). Infection with *H pylori* is very prevalent; it has been estimated that at least 50% of adults worldwide harbor the infection. However, a small minority (less than 1%) ever develop gastric cancer. The presence of Epstein-Barr virus (EBV) has been found in between 5% and 16% of gastric cancers, implying that it may possibly play a causative role. The virus is more frequently found in men than in women, in tumors of the cardia or gastric body and in tumors found in gastrectomy specimens (Murphy et al., 2009). Tobacco use has been found to be a risk factor for gastric cancer and precancerous lesions (IARC, 2004). High dietary salt consumption increases cancer risk (Joossens et al., 1996). Consumption of processed meat has also been associated with a high cancer risk (Gonzalez et al., 2006). Several studies have reported an association between cancer risk and genetic polymorphisms of genes linked to the inflammatory response, such as the interleukins IL1B, IL1RN, *IL10*, and tumor necrosis factor-α, *TNF* (Camargo et al., 2006; Loh at al., 2009; Persson et al., 2011). Several of these are tumor suppressors of gastric acid secretion, which may facilitate bacterial colonization

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of the gastric corpus. The IL1B-511T allele is a risk factor for gastric adenocarcinoma (Camargo et al., 2006). The early stages of gastric cancer are usually asymptomatic or associated with nonspecific symptoms such as dyspepsia. Advanced stages may be accompanied by persistent abdominal pain, anorexia, and weight loss. Ulcerated tumors may be associated with hematemesis. Persistent vomiting may be a sign of pyloric stenosis. The lack of specific symptoms may lead to a delayed diagnosis. Approximately 80% of patients are diagnosed at advanced stages in most countries where no early detection programs are in place. The location of the tumor dictates the anatomic classification: cardial and distal. It is frequently difficult to assign the location of origin in tumors of the gastroesophageal junction as esophageal or gastric, especially when the tumor has reached a considerable size. The American Joint Commission on Cancer Classification (AJCC) decided to classify tumors of the gastroesophageal junction and those involving the proximal 5 cm of the stomach as esophageal carcinomas (Edge et al., 2009). The degree of invasion dictates the classification of gastric cancer as early or advanced. Early cancers are limited to the mucosa and submucosa, irrespective of lymph node metastasis. Beyond those layers, tumors are classified as advanced. Advanced cancer cases are classified according to the gross morphology as Bormann groups: (1) polypoid, (2) ulcerated with well-defined borders, (3) ulcerated with ill-defined borders, (4) infiltrating diffuse without evidence of mass or ulceration, which is frequently called linitis plastica. The most frequently used classification is the Lauren classification, which recognizes two types: intestinal (with intercellular junctions) and diffuse (without intercellular junctions), representing two different nosologic entities. Both types are associated with H pylori infection (Lauren, 1965). Endoscopy and radiographic examination are the two main methods to diagnose gastric carcinoma. Endoscopic ultrasonography, computed tomography and laboratory studies are also helpful in the diagnosis of gastric carcinoma.

Case report

We report a case of a 67 year old male presented with melena and haematemesis. On examination pale, covered with cold sweat, adynamic, hypotensive TA = 60/30 mmHg in hemorrhagic shock. On a rectal examination melena was present. Laboratory tests: Le= $12.2x10^{9}/l$, Er= $2.1x10^{12}/l$, Tr= $185..139x10^{9}/l$, Hgb=68 g/l, CK=176 U/L, CKMB=98.8 U/L, Creatinine=122 umol/l, Urea=20.3mmol/l, Glikemia=8.9 mmol/l, K=3.6 mmol/l,Na=137mmol/l,LDH=155 U/L,Fe=3.9 umol/l, crp=5.0mg/l. After initial stabilization with filtered Erythrocytes, Isogroup plasma, Sol. Gelofusine, Sol. 5% Dextrose and 0.9% NaCl, Amp.



Figure1: Esophagogastroduodenoscopy – shows irregular and longitudinal ulceration with elevated and subminated edges, the bottom of the lesion is necrotic with a fixed coagulum.

Pantoprasole and Tranexamic acid. We performed esophagogastroduodenoscopy: The entrance to the lumen of the stomach is suppressed from the medial to laterally, and the lumen of the corporate segment is filled with a large coagulum and thicker hematinic contents. At 2 to 3 cm distal from gastroesophageal

junction and along the small curve of the stomach greater irregular and longitudinal ulceration is detected, with elevated and subminated edges, and the bottom of the lesion necrotic and with a fixed coagulum. Several edge biopsies were taken for HP. The antral segment with blood stains. On inversion the angulus and fundus of the stomach are neat, while subcardial was detected greater exulceration. Pylorus and duodenum without bleeding lesions. Based on the endoscopic findings – Exulceratio ventriculi regio subcardialis, gastric cancer was suspected. The patient was transferred to Digestive Surgery where a total gastrectomy was performed. The pathohistological finding is in addition to gastric adenocarcinoma.

Discussion

Gastric cancer continues to be an important healthcare problem from a global perspective. Most of the cases are diagnosed at late stages when the treatment is largely ineffective. Helicobacter pylori (H. pylori) infection is a well-established carcinogen for gastric cancer (IARC, 1994). While lifestyle factors are important, the efficacy of interventions in their modification, as in the use of antioxidant supplements, is unconvincing (Kato et al., 2006; Kobayashi et al., 2002; Poydock et al., 1979; Palace et al., 1999; Rousseau et al., 1992). Although several screening approaches have been proposed, including indirect atrophy detection by measuring pepsinogen in the circulation, none of them have so far been implemented, and more study data is required to justify any implementation (Leja et al., 2009; Mizuno et al., 2009). Mass eradication of H. pylori in high-risk areas tends to be cost-effective, but its adverse effects and resistance remain a concern (,Leung et al., 2004; Wong et al., 2012). Searches for new screening biomarkers, including microRNA and cancer-autoantibody panels, as well as detection of volatile organic compounds in the breath, are in progress. The early stages of gastric cancer are usually asymptomatic or associated with nonspecific symptoms such as dyspepsia. Advanced stages may be accompanied by persistent abdominal pain, anorexia, and weight loss. Ulcerated tumors may be associated with hematemesis. Persistent vomiting may be a sign of pyloric stenosis. The lack of specific symptoms may lead to a delayed diagnosis. Approximately 80% of patients are diagnosed at advanced stages in most countries where no early detection programs are in place. Upper gastrointestinal endoscopy is the first preferred diagnostic procedure. The advantages of endoscopy are that biopsies can be taken, small lesions evaluated more fully and cancer can be localized and mapped (Yao, 2012). Gastric carcinoma of intestinal type occurs more often in the antrum which is the area prone to Helicobacter pylori. Cancer can be detected as mucosal ulceration, polypoid mass, infiltrating lesion or large gastric folds. When ulcers are observed, at least six biopsy specimens, from both the edge and the base of the ulcer, need to be obtained to enhance diagnostic yield (Yao, 2012). Chromoendoscopy and magnifying endoscopy may improve the detection rate of early gastric cancer (Kikuste et al., 2013; Rami et al., 2012). Endoscopic ultrasonography (EUS) is used to assess the extent and stage of tumor, including wall invasion and local lymph node involvement and to help guide aspiration biopsies to determine their features. Usually CT scan is combined with EUS to evaluate the staging of gastric tumors (Rami et al., 2012). The only chance for cure of gastric cancer is surgical removal of the tumor and the adjacent lymph nodes. Even when gastric cancer is not suitable for cure, palliative surgical resection is still an effective method to relieve symptoms (Tan et al., 2019). Subtotal gastrectomy is often chosen for patients with distal cancers, but most proximal tumors as in our case need total gastrectomy. The prognosis after surgical resection depends on the staging of tumor, lymph node involvement, distant metastases and DNA aneuploidy. Early gastric cancer can be treated by endoscopic mucosal resection (EMR) or endoscopic submucosal dissection (Tan et al., 2019). Gastric adenocarcinoma is resistant to radiotherapy. Radiotherapy may be applied for management of tumor bleeding, obstruction, and pain as a palliative therapy (Tey et al., 2017). Chemotherapy usually adopts postoperatively or preoperatively as an adjuvant therapy in patients with advanced gastric cancer because gastric carcinoma has a low survival rate and high recurrence rates. The effect of chemotherapy with single agent treatment is limited with a low response rate of 20-30%, so combination regimens are often applied for gastric carcinoma which increases the response rate to 40% (Joshi et al., 2021; Choi et al., 2015).

Conclusion

Epidemiologic studies suggest that eating more fresh fruit and vegetables, eating less salted food and eradicating Helicobacter pylori can decrease the risk of gastric adenocarcinoma. The best way for prevention is to carry out an effective screening program in high-risk groups and in high-incidence areas, so that early discovery, early diagnosis and early treatment can be achieved.

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