A review of gastrointestinal cancer: the most important and prevalent

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Abstract: Gastrointestinal system cancers are one of the most prevalent cause of death in industrialized countries These cancers are more prevalent in regions with direct contact to foods or gastrointestinal products such as esophagus stomach, large and small intestines. Esophagus cancer is the third prevalent gastrointestinal cancer and the sixth cause for mortality in the world. Stomach cancer is the fourth prevalent which is, the most common cause for cancer in males is after lung cancer. In USA, small intestine cancer has devoted itself 3% of gastrointestinal cancers and %0/5 of all cancers. Due to low prevalence and non-specific presentation of small intestine cancer, the diagnosis is difficult and diagnosed in final stages because of late reference in the of patient which leads to have a poor prognosis. Large intestine cancer which is the most prevalent, cancerous cells begins to proliferate within intestinal tissue. The risk factors includes high age, male sexuality, smoking, drinking, long term reflux, barret esophagus, celiac disease, crohns disease, consisting nitrites and canned foods and eating salty fish and also hot and hot drinks, obesity and genetics background which seemingly can be prevented from gastrointestinal cancers by eating high fibrous food, no drinking or smoking and low consumption of canned and salty foods.

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1. Introduction

One of the major site for cancer is gastrointestinal tract its prevalence varies according to lesions. Many studies demonstrated that several factors can be implemented in cancer development such as race, age, sex, high fat and salty diets, alcohol and tobacco consuming, and immobility. Although, some of the gastrointestinal cancers have been associated with environmental factors, however, several investigators demonstrated that the patients who had familial background are at increased risk for gastrointestinal cancer and the risk of these cancers may decreased in this population under effect of environmental factors. Several treatment strategies have been recorded, however surgery and chemotherapy are widely used for treatment of gasterointestinal cancers. Radiotherapy also, may be applied before or after surgery in some cases. Several studies have indicated that life style modification have positive and satisfactory effects in prevention of gastrointestinal cancers.

2. Esophageal cancer

This cancer originated from esophageal cells and divided in two kinds depending on cancerous factors, esophagus squamous cell carcinoma (SCC) and esophageal adenocarcinoma (1). SCC is generated in upper and middle parts of esophagus whereas the second one fatten is produced in lower part (the attachment of esophagus and stomach) which they are similar in complications and treatment (2). Esophagus cancer is the third most common gastrointestinal cancer and the sixth cause of mortality from the cancer throughout the world (3). The probable relationship between esophageal cancer and genetic factors is remarkable due to its high presentation in some world regions. This cancer usually present in males and females above 50 years, and it is seen more among males (4).

2.1 Risk Factors

The precise cause for esophageal cancer is not known completely, but the studies shows that every following factor can enhance the probability of esophageal cancer (5).

- Age: most patients are above 50-60years
- Gender: this cancer is more common in males
- Smoking: smoking or even using tobacco without smoking is a most important risk factor to the cancer.

• Drinking; individuals who drink alcohol in high quantity continuously expose to esophageal cancer and people who use smoking and drinking together have higher risk of esophageal cancer (4, 6).

• Long term reflux and barret esophagus: if stomach acid goes up and enters esophagus, reflux of lower part of esophagus is inflamed, change gradually

and become like stomach sell. This condition is called barret esophagus considered as pre cancer state leading to esophageal adenocarcinoma (4).

• Diet: Consuming precursor carcinogenic materials like nitrites convert to carcinogenic substance like nitrosamine which is abundant in conserve, salty fish, sausages can increase severely the risk of various types of gastrointestinal cancers especially esophageal cancers (7).

• Hot drinks; hot tea or coffee increase the risk of esophageal cancer by burning and changing the epithelial cell of esophagus (8).

• Heredity and genetic mutations: The mechanism of hereditary factors is not clearly known in esophageal cancer however, many studies demonstrated that esophageal carcinoma is more prevalent in some regions such as china (1).

• Obesity: increase the risk 4 times more, in addition, the researchers suppose that there is a relationship between obesity and reflux.

• Concordant diseases: the individuals with other head and neck cancers are more susceptible for esophageal cancer (9).

1.2 Clinical presentations

Unfortunately this cancer may be asymptomatic in early stage. But, in next stages the patient may present with dysphagia while about %75 of esophageal diameter is obstructed. Dysphagia present firstly in solid foods and then gradually will be seen with liquids. Weight loss, felling of a foreign body in throat and odynophagia may be seen by developing the disease (10).

Regurgitation of indigested food with mal odor respiration and hiccup with vomit and pneumonia aspiration are other disease presentation. If the cancer disperses out of esophagus, firstly it compass lymphatic nodes then developed in liver, lungs, brain and bones. Esophageal cancer has poor prognosis especially in late stages (11).

2.3 Diagnosis

The diagnosis is based on clinical findings and the presence of concordant disease. As well as, the following evaluations are essential: modified barium swallow, upper endoscopy, CT scan (to diagnose the metastases of cancer to other parts of body), bone scan (to diagnose cancer metastases), bronchoscopy (to diagnose developing the cancer to lungs) (12).

2.4 Prevention

Several nutritional and environmental factors have been reported to be contributing in esophageal cancer. Accordingly the best prevention strategies include no smoking , avoiding drugs and drinking alcohol and hot tea and salty foods (7). The individual who have problem in swallowing solid food but not with liquids or gastrointestinal disorder for more than 5 days should refer to physician (4). The best prevention way of esophageal cancer is discontinuing smoking and alcohol consumption (13).

2.5 Treatment

If treatment is not begun immediately, the cancer rapidly develops to lungs and liver. The most important factor to predict patient survival affecting the treatment strategy is tumor staging (14).

For end stage patients in whom esophageal tumor is not resectable via surgery, supportive therapy including dysphagia treatment, malnutrition and fistula to esophagus are should be considered (15).

To salve the complications, it needs the frequent dilatation (esophagus) through endoscopy, surgical operation including gastrostomy or jejunostomy should be considered (3).

Pain reliever drugs to remove pain, sedatives to less anxiety and anticholinergics or calcium channel blockers for esophageal spasm are prescribed (1).

Also there are two additional methods: radiotherapy and surgery. Radiotherapy is generally used in upper parts of esophagus while the second in more applicable in middle and lower parts (14).

3. Stomach cancer

The prevalence of gastric cancer is two times more in males than females and it is the fourth most common cancer. Gastric cancer is the second cause of death due to cancer in man after lung cancer (16).

This cancer is more prevalent in low socio-economic groups. The immigrant from high prevalent regions to low prevalent have maintained their previous susceptibility to the cancer but the cancerous risk for children will be similar to new residents. This finding shows that probably environmental factor affecting the person in childhood, is responsible for stomach cancer and (17).

3.1 Risk factor

Long term consumption of high concentration of nitrites in smoked, dry and salty foods

- Infection by helicobacter pylori
- Previous gastrectomy.
- decreased stomach acidity
- atrophic gastritis
- gastric ulcer and polyp

Peoples with blood group A: the prevalence of gastric cancer in this group is more than those with blood group O (5).

3.2 Clinical presentations

Initial signs of gastric cancer are deceiving and similar to other gastrointestinal upsets so that the

person tries to eliminate the signs and symptoms using dietary intake. Weight loss and anorexia are important signs in diagnosing stomach cancer. In initial stages, gastrointestinal disorders like anorexia, weight loss, mild intermittent abdominal pain and nausea in early stages and in developed stages, melena and bloody vomit will be present (18).

In initial stages of stomach cancer, there are no physical symptoms and palpable abdominal mass in most cases is an indicator of long term growth and local aggression of tumor. However the tumoral cell may spread to liver through the blood. Iron deficiency anemia in males and fecal occult blood in both male and female should raise suspicion of gastrointestinal cancer (19).

3.3 Diagnosis

Stomach cancer is usually diagnosed by clinical findings and par-clinic studies such as upper endoscopy and biopsy. In addition feeling defect can be seen in barium swallow. Treatment is effective while the cancer is diagnosed in initial stages. The people who have Dyspepsia or have other clinical features like anorexia and weight loss need to further analysis (20).

Due to higher prevalence of stomach cancer in the over 45 year old individual, the patients with or without dyspepsia or serious symptoms should be evaluated by upper endoscopy (21).

3.4 Prevention

Decreasing foods consumption like bread, various meat products, and foods with high salt concentration can decrease the incidence of gastric cancer in society (16).

Decreasing consumption of sugarless beverage, sugarless sweets, fried foods, hot tea have significant role in decreasing the risk of gastric cancer (22).

3.5 Treatment

Partial gastrectomy and total gastrectomy seems to be the best treatment strategies. The operation is helpful, if the patient be in early stages (23).

If the cancer affects the little site in stomach, so that it can be removed wholly, the patient will have more chance to be alive, but the operation will not be beneficial in developed stages and other treatments like chemotherapy and laser therapy have important role to control the patients' symptoms (24).

4: Small intestine cancer

Diagnosing this cancer is difficult due to low prevalence and non – specific presentations. This cancer has accounted % 3 of all gastrointestinal cancers and % 0.05 of all cancers in USA. Due to delay in referring, this cancer usually diagnosed in late

stage and consequently has poor prognosis. Small intestine cancer includes various malignant (Carcinoid tumor, adenocarcinoma, sarcoma and lymphoma) and benign (adenoma, lipoma and leiomyoma) tumors (25).

4.1 Risk factors

Often the patient suffers from abdominal pain with nausea, flatulence, anorexia in the first stages and with advancement, different complications may appear like weight loss fatigability, and hematemesis, hematochezia, anemia, intestine obstruction, bile duct obstruction symptoms like jaundice and itching (26).

4.2 Diagnosis

Diagnosis is generally based on history and clinical findings. Whoever confirmation is based on paraclinical evaluations.

• Fecal occult blood test is a diagnostic test for diagnosing cancer in early stage in high risk individuals (27).

• Barium swallow; In most cases, swallowing barium is used for diagnosis in which the presence of filling defect confirms the diagnosis (28).

• Upper endoscopy of gastrointestinal system has more utilization in diagnosing the duodenal cancer; Capsule endoscopy can be helpful by providing pictures from gastrointestinal system Structure (29).

• CT scan or abdominal sonography can also helpful for the diagnosis

• Colonoscopy also widely used for diagnosis of lower gastrointestinal cancer (28).

4.3 Prevention and Treatment

The individual with familial cancer history need to be under supervision and underwent diagnostic evaluation such as fecal occult blood tests annually. Also they can prevent it in next years by more consumption of fibrous food and limited consumption of salty and high fat diets (30).

Treatment is different based on tumor type and stages and generally included operation, chemotherapy and radiotherapy. The main determinant factor to treatment is tumor size and its place and involvement of lymphatic glands, bone, liver and other organs (30).

5: Colon cancer

In this cancer, cancerous cells in intestinal tissue begin to proliferate. Although, this cancer is one of the most common cancers in human, the number of new cases and death from it has decreased considerably due to improving in screening and diagnosis methods. This disease presents in every age but it is more prevalent in individuals over 50 year old. It is remediable in the initial stages of diagnosis but this cancer may be asymptomatic in the initial stages and may affect the treatment (31).

5.1 Risk factors

Family or genetic history, previous history of large intestine cancer, the history of large intestine, the history of large intestine disease like ulcerative colitis, the age of over 40 years, high fat diet (specially with animal sources), low fiber diet, low physical activity, obesity, smoking and alcohol drinking are the most risk factors to present large intestine cancer (32).

5.2 Clinical presentations

In early stages, the disease is asymptomatic but the following symptoms may be presented gradually; hematochezia, abdominal cramp, feeling satiety, narrowing stool diameter, weight loss, and felling mass in stomach (33).

5.3 Prevention

Consuming vegetable and soy diet, fruits and avoiding high fat diets have an important role in preventing large Intestine cancer. Also, a daily regular exercise for 30 min is advised and seems to be useful(34).

The individuals with mentioned risk factors should be evaluated under gradual medical tests in youth age in which, fecal occult blood can be important for early diagnosis and treatment (35).

5.4 Treatment

Like other cancers, the early diagnosis of cancer is more beneficial and good prognosis.

Generically, There are three main treatment including surgery, radiotherapy and chemotherapy. In which two or more methods depending on cancer stage may be implemented one by one or together (36).

Operation is the major treatment for large intestine cancer by which colostomy along with colectomy considered.

Pre operation radiotherapy for large tumors can be implemented to decrease tumor size and facilitate the operation and also it may be performed post operation for cancerous cells invasion that would not be removed by surgeon during operation(37).

The basic implementation of radiotherapy in large intestine cancer is for removing probable remaining cancerous cells in intestinal tissue (38).

Conclusion

Gastrointestinal system cancers are of the most prevalent cancers with high mortality. According cancer international research organization at least % 35 all cancers and more than % 50 gastrointestinal system cancers have related to nourishment type meaning that foods stimulating cancer like oxidated fat in frying oil, rotten foods. Very hot drinks and foods, obesity and overweight are very important factors cancer development (39). Also, precursor carcinogenic materials like nitrites convert to carcinogenic substance like nitrosamine which is abundant in conserve, salty and smoked fish, sausages increase severely the risk of affection of various cancer including gastrointestinal cancers. The probability of cancer is remarkably decreased by healthy foods and appropriate nutrition which in order to attain the goal, the different dietary factors as cancerous simulation should be recognized and avoided. The nourished food with fiber, calcium, vitamin D, E, C, A, B12, fresh fish and fat acid Ω 3 and sufficient quantity of vegetables, fruits specially dark, green vegetables, or near yellow like sugar beet, carrot, onion and garlic can a decrease incidence significant role to of gastrointestinal cancers (34).

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References

- [1] Enzinger PC, Mayer RJ. Esophageal cancer. New England Journal of Medicine. 2003;349(23):2241-52.
- [2] Kato J, Kuwabara Y, Mitani M, Shinoda N, Sato A, Toyama T, et al. Expression of survivin in esophageal cancer: correlation with the prognosis and response to chemotherapy. International journal of cancer. 2001;95(2):92-5.
- [3] Oyama T, Tomori A, Hotta K, Morita S, Kominato K, Tanaka M, et al. Endoscopic submucosal dissection of early esophageal cancer. Clinical Gastroenterology and Hepatology. 2005;3(7): 67-70.
- [4] Holmes RS, Vaughan TL, editors. Epidemiology and pathogenesis of esophageal cancer. Seminars in radiation oncology; 2007: Elsevier.
- [5] Kelley JR, Duggan JM. Gastric cancer epidemiology and risk factors. Journal of clinical epidemiology. 2003;56:1-9(1).
- [6] Engel LS, Chow WH, Vaughan TL, Gammon MD, Risch HA, Stanford JL, et al. Population attributable risks of esophageal and gastric cancers. Journal of the National Cancer Institute. 2003;95(18):1404-13.
- [7] Corley DA, Kerlikowske K, Verma R, Buffler P.
 Protective association of aspirin/NSAIDs and esophageal cancer: a systematic review and meta-analysis.
 Gastroenterology. 2003;124(1):47-56.
- [8] Castellsague X, Munoz N, De Stefani E, Victora

- [9] Brown LM, Hoover R, Silverman D, Baris D, Hayes R, Swanson GM, et al. Excess incidence of squamous cell esophageal cancer among US Black men: role of social class and other risk factors. American journal of epidemiology. 2001;153(2):114-22.
- [10] Shaheen N, Ransohoff DF. Gastroesophageal reflux, Barrett esophagus, and esophageal cancer: scientific review. Jama. 2002;287(15):1972-81.
- [11] Brücher BLDM, Stein HJ, Bartels H, Feussner H, Siewert JR. Achalasia and esophageal cancer: incidence, prevalence, and prognosis. World journal of surgery. 2001;25(6):745-9.
- [12] Flamen P, Lerut A, Van Cutsem E, Cambier J, Maes A, De Wever W, et al. The utility of positron emission tomography for the diagnosis and staging of recurrent esophageal cancer. The Journal of thoracic and cardiovascular surgery. 2000;120(6):1085-92.
- [13] Umar SB, Fleischer DE. Esophageal cancer: epidemiology, pathogenesis and prevention. Nature Clinical Practice Gastroenterology & Hepatology. 2008;5(9):517-26.
- [14] Hofstetter W, Swisher SG, Correa AM, Hess K, Putnam Jr JB, Ajani JA, et al. Treatment outcomes of resected esophageal cancer. Annals of surgery. 2002;236(3):376.
- [15] Fujita H, Sueyoshi S, Yamana H, Shinozaki K, Toh U, Tanaka Y, et al. Optimum treatment strategy for superficial esophageal cancer: endoscopic mucosal resection versus radical esophagectomy. World journal of surgery. 2001;25(4):424-31.
- [16] Brenner H, Rothenbacher D, Arndt V. Epidemiology of stomach cancer. Cancer Epidemiology: Springer; 2009. p. 467-77.
- [17] Brenner H, Bode G, Boeing H. Helicobacter pylori infection among offspring of patients with stomach cancer. Gastroenterology. 2000;118(1):31-5.
- [18] Buller H ,Van Doormaal F, Van Sluis G, Kamphuisen P. Cancer and thrombosis: from molecular mechanisms to clinical presentations. Journal of Thrombosis and Haemostasis. 2007;5(s1):246-54.
- [19] Yang C-J, Hwang J-J, Kang W-Y, Chong I-W, Wang T-H, Sheu C-C, et al. Gastro-intestinal metastasis of primary lung carcinoma: clinical presentations and outcome. Lung Cancer. 2006;54(3):319-23.
- [20] Nakajima T. Gastric cancer treatment guidelines in Japan. Gastric cancer. 2002;5(1):1-5.

[21] Tajiri H, Doi T, Endo H, Nishina T, Terao T, Hyodo I, et al. Routine endoscopy using a magnifying endoscope for gastric cancer diagnosis. Endoscopy. 2002;34(10):772-7.

http://www.sciencepub.net/stem

- [22] Yang L. Incidence and mortality of gastric cancer in China. World Journal of Gastroenterology. 2006;12(1):17.
- [23] Otsuka K, Murakami M, Aoki T, Tajima Y, Kaetsu T, Lefor AT. Minimally invasive treatment of stomach cancer. The Cancer Journal. 2005;11(1):18-25.
- [24] Kunisaki C, Shimada H, Nomura M, Hosaka N, Akiyama H, Ookubo K, et al. Lymph node dissection in surgical treatment for remnant stomach cancer. Hepato-gastroenterology. 2001;49(44):580-4.
- [25] Barker N, van Es JH, Kuipers J, Kujala P, van den Born M, Cozijnsen M, et al. Identification of stem cells in small intestine and colon by marker gene Lgr5. Nature. 2007;449:1003-7.
- [26] Hemminki K, Li X. Incidence trends and risk factors of carcinoid tumors. Cancer. 2001;92(8):2204-10.
- [27] Fletcher CD, Berman JJ, Corless C, Gorstein F, Lasota J, Longley BJ, et al. Diagnosis of gastrointestinal stromal tumors: a consensus approach. International Journal of Surgical Pathology. 2002;10(2):81-9.
- [28] Pidhorecky I, Cheney RT, Kraybill WG, Gibbs JF. Gastrointestinal stromal tumors: current diagnosis, biologic behavior, and management. Annals of Surgical Oncology. 2000;7(9):705-12.
- [29] Cobrin GM, Pittman RH, Lewis BS. Increased diagnostic yield of small bowel tumors with capsule endoscopy. Cancer. 2006;107(1):22-7.
- [30]Yamamoto H, Kita H, Sunada K, Hayashi Y, Sato H, Yano T, et al. Clinical outcomes of double-balloon endoscopy for the diagnosis and treatment of small-intestinal diseases. Clinical Gastroenterology and Hepatology. 2004;2(11):1010-6.
- [31] O'Brien CA, Pollett A, Gallinger S, Dick JE. A human colon cancer cell capable of initiating tumour growth in immunodeficient mice .Nature. 2006;445(7123):106-10.
- [32] Giovannucci E. Modifiable risk factors for colon cancer. Gastroenterology Clinics of North America. 2002;31(4):925-43.
- [33] Jasperson KW, Tuohy TM, Neklason DW, Burt RW. Hereditary and familial colon cancer. Gastroenterology. 2010;138(6):2044-58.
- [34] Brady LJ, Gallaher DD, Busta FF. The role of probiotic cultures in the prevention of colon cancer. The Journal of nutrition. 2000;130(2):410S-4S.
- [35] Corpet DE, Pierre F. Point: From animal models

to prevention of colon cancer. Systematic review of chemoprevention in min mice and choice of the model system. Cancer Epidemiology Biomarkers & Prevention. 2003;12(5):391-400.

- [36] Twelves C, Wong A, Nowacki MP, Abt M, Burris III H, Carrato A, et al. Capecitabine as adjuvant treatment for stage III colon cancer. New England Journal of Medicine. 2005;352(26):2696-704.
- [37] André T, Boni C, Mounedji-Boudiaf L, Navarro M, Tabernero J, Hickish T, et al. Oxaliplatin, fluorouracil, and leucovorin as adjuvant treatment for colon cancer. New England Journal

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of Medicine. 2004;350(23):2343-51.

- [38] Lacy AM, García-Valdecasas JC, Delgado S, Castells A, Taurá P, Piqué JM, et al. Laparoscopy-assisted colectomy versus open colectomy for treatment of non-metastatic colon cancer: a randomised trial .The Lancet. 2002;359(9325):2224-9.
- [39] Veldkamp R, Kuhry E, Hop W, Jeekel J, Kazemier G, Bonjer HJ, et al. Laparoscopic surgery versus open surgery for colon cancer: short-term outcomes of a randomised trial. Lancet Oncol. 2005;6(7):477-84.