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AN INTRODUCTION TO ESOPHAGEAL CANCER: PATHOGENESIS, TYPES AND RISK FACTORS

SAURABH SINGH RATHORE*

Declaration

The Declaration of the author for publication of Research Paper in The Indian Journal of Research Anvikshiki ISSN 0973-9777 Bi-monthly International Journal of all Research: I, *Saurabh Singh Rathore* the author of the research paper entitled AN INTRODUCTION TO ESOPHAGEAL CANCER: PATHOGENESIS, TYPES AND RISK FACTORS declare that , I take the responsibility of the content and material of my paper as I myself have written it and also have read the manuscript of my paper carefully. Also, I hereby give my consent to publish my paper in Anvikshiki journal , This research paper is my original work and no part of it or it's similar version is published or has been sent for publication anywhere else. I authorise the Editorial Board of the Journal to modify and edit the manuscript. I also give my consent to the Editor of Anvikshiki Journal to own the copyright of my research paper.

Abstract

Cancer is a wide group of many diseases and Esophageal cancer is one of the member of this group. Esophageal cancer affects the esophagus, present in upper part of digestive system just below pharynx. Esophageal cancer is the 6th most common cancer of the world. It has features like rapid progression, late diagnosis and low survival rates. Esophageal cancer occurs in different parts of world with different prevalence. The two most common types of Esophageal cancers are the Esophageal squamous cell carcinoma and the Esophageal adenocarcinoma. This article discusses the pathogenesis of the esophageal cancer, the common types of esophageal cancer and the risk factors associated with it. It may be helpful to non-clinicians for increasing the understanding of this disease.

Introduction

Cancer or malignant neoplasm comprises a disease group wherein cells undergo division beyond the normal limits (uncontrolled growth), invasion (intrusion and destruction of adjacent tissues by cancerous cells), and occasionally metastasis (lymphatic or vascular movement and spread of cancerous cells to other parts of the body). Generally, adult somatic cells have cancer causing gene mutations. Therefore, cancer is as a genetic disease instead of its sporadic nature. The inherited genetic backgrounds are largely unknown and majority of cancers may originate by combinatorial effects of avoidable and unavoidable carcinogens. So, factors causing mutations along with the factors responsible for increasing cell replication can result in cancer development. Genetic and epigenetic changes in tumor suppressor genes, oncogenes and "caretaker" genes that favor expansion of the new clone over the old accumulate

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in multiple steps during cancer progression. Therefore cancer is believed as a clonal disease. These events result in the normal cell's transition into cancerous cell and develop capacity of proliferation irrespective of exogenous mitogens, refractory response to growth-inhibitory signals, resistant behaviour to apoptotic mechanisms, angiogenesis, immortality and metastasis [1].

Esophageal cancer (EC) is world's sixth most common cancer. It is unique features of rapid development, poor prognosis and less than 10% 5-year survival after diagnosis [2-4]. There occurs wide variation in the incidence of EC in different geographical areas of the world and the overall death rate is increasing [5-7]. The incidence of EC is expressed as number of new cases occurring/number of cases per year/rate per 100,000 persons per year. It is found to be very high in Belgium, Iran, North Central China, Japan, Kashmir, certain regions of South Africa, United Kingdom and Normandy district of France [7-9]. According to cancer statistics-2009 report, death rate of EC is up to 7.94/100,000 persons in males [6]. In Indian scenario, EC is second and fifth most common cancer in males and females respectively [10]. The reasons for variation in incidence rates, measures for early diagnosis of EC and predictors of prognosis of EC are yet to be explored in more detail. The development of EC has been linked to multiple factors: environmental risk factors (tobacco - smoking and chewing, alcohol intake, house hold combustible fuels, hot & salty beverages etc.) and genetic factors of the host [11]. Many of the low penetrance genes have been looked for their possible role and association with susceptibility and prognosis of EC [12].

Pathogenesis of Esophageal cancer

Esophageal cancer is formed in the tissues lining the esophagus (esophagus is an organ containing a muscular tube which connects pharynx to the stomach and allows food to pass through it). EC is formed in tissues lining the esophagus (the muscular tube through which food passes from the throat to the stomach). Following sequence of events characterise the pathogenesis of esophageal cancer:

(i) progression of normal epithelium to basal cell hyperplasia
(ii) dysplasia (carcinoma in situ)
(iii) invasive cell carcinoma

Origin of carcinoma occurs in mucosa of esophageal wall and subsequent invasion of the submucosa, muscular layers takes place. Involvement of contiguous structures like trachea-bronchial tree, the aorta, or the recurrent laryngeal nerve in the initial progression is also possible. In more advanced stages, tumor tends to metastasize and moves to peri-esophageal lymphnodes and, eventually, to the liver, lungs or other distant vital organs. To characterise and stage the disease following signs and symptoms are used:

(i) tumor invasion of the esophageal wall
(ii) involvement of lymphnodes
(iii) the presence of metastases

Most EC cases are diagnosed only when the tumor progression reaches to the later stages because significant symptoms occur only when tumor becomes fairly large and lumen becomes obstructed. Until such conditions the cancer spreads to lymphnodes (mediastinal, cervical or celiac).

Types of Esophageal cancer

There are many subtypes of EC but two of them are most common: ESCC or esophageal squamous cell carcinoma (originates from the cells lining the upper part of the esophagus) and EADC or esophageal

adenocarcinoma (originates in the glandular cells that are present at the junction of the esophagus and stomach and which make and release mucus and other fluids). Two distinct histopathologies are associated with these carcinomas. ESCC is the most common EC worldwide (90-95% of all ECs). EADC accounts for 50-80% of all ECs in the west, particularly in males [13]. More than 90% of the esophageal carcinomas were diagnosed as ESCC in the decade of 1960 [14]. However, there was considerable increase in the incidence of EADC during past 30-40 years and now it is more commonly observed EC than ESCC in the western world. However, in India SCC histopathology is observed in more than 90% of ECs. Less common histologies of EC are adenoid cystic, mucoepidermoid, adenosquamous, undifferentiated, and malignant melanoma. These show poor prognoses. Carcinoma of small cell has a course similar to that of lung carcinoma and it is also observed in esophagus [15]. Leiomyosarcoma is a rare non-epithelial tumor occurring in the esophagus leiomyosarcoma. Breast cancer is the most common source of metastasis for EC [14].

Risk Factors Associated with Esophageal Cancers

EC development is the result of Multifactorial etiologies. Previous reports demonstrate major risk factors [16,17]. In different histopathologies, the risk factors are also different and are listed as follows:

Esophageal squamous cell carcinoma (ESCC)

1. *Age between 60 to 70 years:* With increasing age, incidence and risk for EC development also increases as the mean age of EC patients is more than 55 years. The age group for 75% of the total cases is between 55 to 85 years and probability of EC development is less than 1/100,000 for persons below the age of 40 years [13].
2. *Alcoholism and tobacco chewing/smoking:* Long term heavy drinking of alcohol and tobacco intake (chewing as well as smoking in any form) is an important risk factor for ESCC. Alcoholism and habit of tobacco consumption in combination makes a person more susceptible for ESCC [18,19]. Smoking is particularly more risky for ESCC development as multiple carcinogens are present in cigarette smoke: polycyclic aromatic hydrocarbons (PAHs), N-nitrosamines, aromatic amines, heterocyclic aromatic amines, and aldehydes. These chemicals are the major risk factors for ESCC [20].
3. *Non-consumption of fruits and vegetables:* Phytochemicals are antioxidants, found abundantly in fruits and vegetables, which are potent modifiers of carcinogenesis. Antioxidants suppress the abnormal proliferation of early, preneoplastic lesions so a diet lacking antioxidants may become a risk factor for ESCC [21].
4. *History of head and neck squamous cell carcinoma:* A previous history of lung carcinoma or carcinoma of digestive system may pose a risk in such persons for ESCC development [22].
5. *Gender:* Incidence and mortality due to ESCC in males is about 3 times more than that in females [13].
6. *Achalasia:* The condition in which contraction of lower esophageal sphincter causes obstruction of flow of food and drinks to the stomach is known as Achalasia. It is caused by a probable defect in nerve cells present in lower esophagus. This defect prevents the relaxation of lower sphincter and makes swallowing difficult. There occurs dilation of esophagus above this narrow region and food is retained in this dilated region. Achalasia is a known risk factor for ESCC (6% of all Achalasia patients develop ESCC) but why it is so, is not clearly understood [23].
7. Race: Studies show that in comparison to Caucasians, African American race (Blacks) are at three fold increased risk of developing ESCC [24].
8. *Ingestion of carcinogen and corrosive chemical:* Irritant chemicals and carcinogens pose a risk for ESCC development on long term ingestion. Some corrosive chemicals like lye and very hot liquids can cause corrosive injury to esophagus thereby causing risk for ESCC development [25].
9. *Plummer-Vinson syndrome:* Plummer-Vinson or Paterson-Kelly syndrome is a triad of dysphagia, iron-deficiency anemia and esophageal webs. One of the constituent condition of this syndrome, "Esophageal webs" is associated with increased risk of ESCC. Esophageal webs interfere with swallowing of food and occur as abnormal protrusions of tissue in to the esophagus.
10. *Radiation therapy:* Radiations cause mutations in DNA therefore an earlier history of radiation therapy is considered as a risk factor for developing ESCC [13].

Adenocarcinoma (EADC)

1. *Age between 50 to 60 years:* In comparision to ESCC, EADC development takes place in comparatively low age group of 50-60 years [13].

2. *Alcoholism and Tobacco chewing/smoking:* Tobacco consumers, particularly smokers are at 2 to 3 fold higher risk for EADC development in comparison to non-smokers [20,26]. Like ESCC alcoholism for long periods is an independent risk factor for ESCC but with tobacco consumption in any form the risk is increased [26,27].
3. *Barrett's esophagus:* Barrett's esophagus (BE) is the result of prolonged gastro-esophageal-reflux-disease (GERD). BE is characterised by the conversion of normal squamous epithelium to metaplastic columnar morphology and predispose a patient for EADC development [28].
4. *Hiatal hernia:* In hiatal hernia, upper part of the stomach protrudes into the thorax through a tear or weakness in the diaphragm. Because of this protrusion, hiatal heria promotes reflux of gastric contents via its direct and indirect actions on the anti-reflux mechanism. Hiatal hernia is associated with all the symptoms and consequences of GERD- heartburn, esophagitis, Barrett's esophagus and EADC [21].
5. *Race:* Caucasians are shown to be at increased risk for EADC development as its prevalence is higher in them [29].
6. *Gastroesophageal reflux disease (GERD):* GERD is the long term acid irritation and/or upper abdominal discomfort. In this disorder, acid leaks from the stomach to esophagus and it causes 'heartburn'. GERD may occur without any symptom. However, 'heartburn' is considered to be the main symptom of GERD. A long term suffering from GERD increases the risk of EADC development as evidenced by the fact that about 30% of EADC cases can be linked to GERD [21,30].
7. *Gender:* Males are more prone to develop EADC in comparision to females [13].
8. *Obesity:* Obesity increases the risk of development of EADC four times as compared to non-obese individuals [31].
9. *Infection from Helicobacter pylori:* Infection from Helicobacter pylori causes induction of atrophic gastritis which makes reflux less acidic. The Helicobacter pylori itself produces ammonia and thus help in neutralisation of gastric acid [32]. Helicobacter pylori infection was found to be associated with reduced risk for EADC development by Ye et al. in a case-control study [33].

References

[1] Hanahan D, Weinberg RA (2000), The hallmarks of cancer. Cell 100: 57-70.

[2] Brenner DE (2004), Update on cancer-preventive therapeutics. Clin Adv Hematol Oncol 2: 25-26, 64.

[3] Hiyama T, Yoshihara M, Tanaka S, Chayama K (2007), Genetic polymorphisms and esophageal cancer risk. Int J Cancer 121: 1643-1658.

[4] McCabe ML, Dlamini Z (2005), The molecular mechanisms of oesophageal cancer. Int Immunopharmacol 5: 1113-1130.

[5] Layke JC, Lopez PP (2006), Esophageal cancer: a review and update. Am Fam Physician 73: 2187-2194.

[6] Jemal A, Siegel R, Ward E, Hao Y, Xu J, et al. (2009), Cancer statistics, 2009. CA Cancer J Clin 59: 225-249.

[7] Parkin DM, Bray F, Ferlay J, Pisani P (2001), Estimating the world cancer burden: Globocan 2000. Int J Cancer 94: 153-156.

[8] Lam AK (2000), Molecular biology of esophageal squamous cell carcinoma. Crit Rev Oncol Hematol 33: 71-90.

[9] Messmann H (2001), Squamous cell cancer of the oesophagus. Best Pract Res Clin Gastroenterol 15: 249-265.

[10] Nandkumar MA, Yamato M, Kushida A, Konno C, Hirose M, et al. (2002), Two-dimensional cell sheet manipulation of heterotypically co-cultured lung cells utilizing temperature-responsive culture dishes results in long-term maintenance of differentiated epithelial cell functions. Biomaterials 23: 1121-1130.

[11] Umar SB, Fleischer DE (2008), Esophageal cancer: epidemiology, pathogenesis and prevention. Nat Clin Pract Gastroenterol Hepatol 5: 517-526.

[12] Vallbohmer D, Lenz HJ (2006), Predictive and prognostic molecular markers in outcome of esophageal cancer. Dis Esophagus 19: 425-432.

[13] Parkin DM, Bray F, Ferlay J, Pisani P (2005), Global cancer statistics, 2002. CA Cancer J Clin 55: 74-108.

[14] Koshy M, Esiashvilli N, Landry JC, Thomas CR, Jr., Matthews RH (2004), Multiple management modalities in esophageal cancer: combined modality management approaches. Oncologist 9: 147-159.

[15] IMAI T, SANNOHE Y, OKANO H (1978), Oat cell carcinoma (apudoma) of the esophagus: a case report. Cancer 41: 358-364.

[16] WONG A, FITZGERALD RC (2005), Epidemiologic risk factors for Barrett's esophagus and associated adenocarcinoma. Clin Gastroenterol Hepatol 3: 1-10.

[17] FREEMAN HJ (2004), Risk of gastrointestinal malignancies and mechanisms of cancer development with obesity and its treatment. Best Pract Res Clin Gastroenterol 18: 1167-1175.

[18] CASTELLSAGUE X, MUNOZ N, DE STEFANI E, VICTORA CG, CASTELLETTO R, et al. (1999), Independent and joint effects of tobacco smoking and alcohol drinking on the risk of esophageal cancer in men and women. Int J Cancer 82: 657-664.

[19] CUI R, KAMATANI Y, TAKAHASHI A, USAMI M, HOSONO N, et al. (2009), Functional variants in ADH1B and ALDH2 coupled with alcohol and smoking synergistically enhance esophageal cancer risk. Gastroenterology 137: 1768-1775.

[20] TOH Y, OKI E, OHGAKI K, SAKAMOTO Y, ITO S, et al. (2010), Alcohol drinking, cigarette smoking, and the development of squamous cell carcinoma of the esophagus: molecular mechanisms of carcinogenesis. Int J Clin Oncol 15: 135-144.

[21] KAMANGAR F, CHOW WH, ABNET CC, DAWSEY SM (2009), Environmental causes of esophageal cancer. Gastroenterol Clin North Am 38: 27-57, vii.

[22] GARAVELLO W, NEGRI E, TALAMINI R, LEVI F, ZAMBON P, et al. (2005), Family history of cancer, its combination with smoking and drinking, and risk of squamous cell carcinoma of the esophagus. Cancer Epidemiol Biomarkers Prev 14: 1390-1393.

[23] ECKARDT VF (2001), Clinical presentations and complications of achalasia. Gastrointest Endosc Clin N Am 11: 281-292, vi.

[24] BLOT WJ (1994), Esophageal cancer trends and risk factors. Semin Oncol 21: 403-410.

[25] YIORIS N, IVANKOVIC S, LEHNERT T (1984), Effect of thermal injury and oral administration of N-methyl-N'-Nitro-N-nitrosoguanidine on the development of esophageal tumors in Wistar rats. Oncology 41: 36-38.

[26] FREEDMAN ND, ABNET CC, LEITZMANN MF, MOUW T, SUBAR AF, et al. (2007), A prospective study of tobacco, alcohol, and the risk of esophageal and gastric cancer subtypes. Am J Epidemiol 165: 1424-1433.

[27] SUN L, KONIG IR, HOMANN N (2009), Manganese superoxide dismutase (MnSOD) polymorphism, alcohol, cigarette smoking and risk of oesophageal cancer. Alcohol Alcohol 44: 353-357.

[28] VAN DER WOUDE CJ, JANSEN PL, TIEBOSCH AT, BEUVING A, HOMAN M, et al. (2002) Expression of apoptosis-related proteins in Barrett's metaplasia-dysplasia-carcinoma sequence: a switch to a more resistant phenotype. Hum Pathol 33: 686-692.

[29] BLOT WJ, DEVESA SS, KNELLER RW, FRAUMENI JF, Jr. (1991), Rising incidence of adenocarcinoma of the esophagus and gastric cardia. JAMA 265: 1287-1289.

[30] LAGERGREN J, BERGSTROM R, NYREN O (1999), Association between body mass and adenocarcinoma of the esophagus and gastric cardia. Ann Intern Med 130: 883-890.

[31] MERRY AH, SCHOUTEN LJ, GOLDBOHM RA, VAN DEN BRANDT PA (2007), Body mass index, height and risk of adenocarcinoma of the oesophagus and gastric cardia: a prospective cohort study. Gut 56: 1503-1511.

[32] RICHTER JE, FALK GW, VAEZI MF (1998), Helicobacter pylori and gastroesophageal reflux disease: the bug may not be all bad. Am J Gastroenterol 93: 1800-1802.

[33] YE W, HELD M, LAGERGREN J, ENGSTRAND L, BLOT WJ, et al. (2004), Helicobacter pylori infection and gastric atrophy: risk of adenocarcinoma and squamous-cell carcinoma of the esophagus and adenocarcinoma of the gastric cardia. J Natl Cancer Inst 96: 388-396.

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