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Esophageal Adenocarcinomas: A Rising Trend in Kashmir Histopathological Perspectives

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Abstract:

The Esophageal malignancies are one of the important causes of Deaths in cancer related deaths as they are usually detected at a late stage of disease and the survival rates after treatment of these tumors have not changed appreciably .Kashmir region is a hilly region lying in the north of India with unique lifestyle and dietary habbits. These tumors are mostly present at the lower end of esophagus .The article highlights the important points in the development of adenocarcinomas of esophagus in general and to kashmir region in specific.

Key Words:

Barret, Carcinogens, Plummer, metaplasia, glands, columnar

Introduction:

In general the cancers of upper gastrointestinal tracts are common in Kashmir. Specifically the Esophageal cancers are common in Kashmir . There is a rising trend of these cancers as evidenced by increasing number of cases detected with the added fact of improvised diagnostic techniques. This is because of the fact that the dietary styles of Kashmiri people os quite different from the related regions. The number of etiological factors implicated in causation is

multifactorial and is associated with other factors usually implicated in causation of esophageal cancers.

Text:

Epidemiologic studies have suggested that the cause is the presence of a large amount of carcinogenic nitrosamines in the soil of this region and contamination of foods by fungi, most often Geotrichum candidum, and yeast, which produce mutagens, ingestion of very hot tea are believed to injure the esophageal mucosa and lead to malignant degeneration, chewing tobacco ,drinking burning hot beverages and the use of tobacco and wine are believed to be etiologic factors in the development of esophageal carcinoma. Alcohol consumption and cigarette smoking seem to be the most consistent risk

factors . Specifically in Kashmir cancers are attributed to spicy foods, salt tea,repeated boiling of salt tea,brassica olerecea, dried vegetables, smoked fish or dried fish. The high concentration of nitrosamines, methylnitrosourea, nitrosoproline and nitrosoamide are particularly important for the development of esophageal Cancers .

The etiology of esophageal carcinoma is unknown, but as indicated earlier, certain nutritional factors and potential carcinogens have been incriminated: alcohol, tobacco, zinc, nitrosamines, malnutrition, vitamin deficiencies, anemia, poor oral hygiene and dental caries, previous gastric surgery, and chronic ingestion of hot foods or beverages¹. A number of esophageal lesions are premalignant, associated with an increased incidence of late carcinoma: achalasia, caustic burns, Plummer-Vinson syndrome, leukoplakia, esophageal diverticula, reflux esophagitis, and hiatal hernia, (columnar epithelial-lined) esophagus, Barrett's irradiation esophagitis, and ectopic gastric mucosa.

Adenocarcinomas occur most commonly in the distal third of the esophagus, in the sixth decade of lifeand with a higher male-to-female ratio and are on rise ²Esophageal adenocarcinoma may arise from (1) malignant degeneration of metaplastic columnar epithelium (Barrett's mucosa) or the heterotopic islands of columnar epithelium, or the esophageal submucosal glands. They can occur in cardia of stomach as well.³

Patients with a columnar-lined lower esophagus (Barrett's metaplasia)⁴ are 40 -120 times more likely to develop adenocarcinoma than the general population. Although the true incidence of Barrett's esophagus in the general population is unknown, it has been estimated that adenocarcinoma arises in 8% to 15% of patients with a columnar-lined esophagus. The finding ph nodes. Distant spread to liver and lungs is common.

Skinner and Belsey's Classification of Esophagitis

of dysplasia in Barrett's mucosa is an ominous prognostic sign of impending malignant degeneration, with severe dysplasia being virtually synonymous with carcinoma in situ and being an indication for resectional therapy.

As is the case with squamous cell carcinoma, adenocarcinoma of the esophagus exhibits an aggressive behavior, with frequent transmural invasion and lymphatic spread. Because many of these tumors arise in the distal esophagus, spread to paraesophageal, celiac axis, and splenic hilum lymph nodes is common. Metastases to the lung and liver are most frequent. The 5-year survival for esophageal adenocarcinoma is low with the presence of lymph node metastases exerting a significant negative effect on survival.

Esophageal Adenocarcinoma is known for its aggressivehistopathological behavior, rapidly infiltrating locally, involving adjacent lymph nodes, and disseminating widely by hematogenous spread. The Histological Lack of the serosal layer tends to favor local tumor extension. Anatomical location suggests the extension. The Upper- and middle-third tumors tend to involve the nearby structures like the tracheobronchial tree, aorta, and left recurrent laryngeal nerve as it loops around the aortic arch, whereastumors locvated at the lower-third may invade the diaphragm, pericardium, or stomach. The extensive mediastinal lymphatic drainage, which communicates with cervical and abdominal collateral vessels, is responsible for the finding of mediastinal. supraclavicular, or celiac lymph node metastases in at least 75% of patients with esophageal carcinoma. Cervical esophageal cancers drain to the deep cervical, paraesophageal, posterior mediastinal, tracheobronchial lymph nodes. Lower esophageal tumors spread to paraesophageal, celiac, and splenic hilar lym

- Grade I: Distal esophageal mucosal erythema (which may obscure the esophagogastric squamocolumnar junction)
- Grade II: Mucosal erythema with superficial ulceration, typically linear and vertical and with an overlying fibrinous membranous exudate that is easily wiped away, leaving a bleeding surface
- Grade III: Mucosal erythema with superficial ulceration and associated submucosal fibrosis on biopsy—a dilatable early stricture
- Grade IV: Extensive ulceration and fibrous luminal stenosis—may represent irreversible panmural fibrosis

Savary and Miller's Classification of Esophagitis

- Grade I: Single erosive or exudate lesion, oval or linear, involving only one longitudinal fold
- Grade II: Noncircular multiple erosions or exudate lesion involving more than one longitudinal fold, with or without confluence
- Grade III: Circular erosive or exudative lesion
- Grade IV: Chronic lesions: ulcer(s), stricture(s), or short esophagus isolated or associated with lesions of Grades I, II, or III
- Grade V: Islands, fingerlike forms or circumferential distribution of Barrett's epithelium isolated or associated with a lesion of Grades I through IV.

Barrett's mucosa: Here the metaplastic replacement of normal esophageal squamous mucosa by columnar epithelium is of particular importance in this context, because esophagoscopy with biopsy is the most sensitive and specific diagnostic test for this condition, and periodic surveillance is mandatory to detect progression of premalignant dysplastic change. 5,6,7,8 Because the squamocolumnar epithelial junction may normally be found within 2 to 3 cm. of the anatomic esophagogastric junction, by convention, the diagnosis Barrett's mucosa requires the histologic identification of the metaplastic columnar epithelium 3 cm. or more proximal to the junction of the tubular 9,10,11,12,13,14. esophagus with the stomach. development of Barrets esophagus should be carefully monitored for subsequent development of cancer especially the Adenocarcinoma.

Conclusion:

Early recognition and knowledge about the risk factors implicated in causation as well as the early diagnostic detection can significantly reduce the morbidity and mortality of these cancers .

Refrences:

- 1. Duranceau, A.: Epidemiologic trends and etiologic factors of esophageal carcinoma. In Delarue, N. C., Wilkins, E. W., Jr., and Wong, J. (Eds.): International Trends in General Thoracic Surgery. Vol. 4, Esophageal Cancer. St. Louis, C.V. Mosby, 1988, pp. 3–10.
- 2. Blot, W. J., Devesa, S. S., Kneller, R. W., and Fraumeni, J. F., Jr.: Rising incidence of adenocarcinoma of the esophagus and gastric cardia. JAMA, 265:1287, 1991.
- 3. Ferguson, M. K., and Skinner, D. B.: Carcinoma of the esophagus and cardia. In Orringer, M. D., and Zuidema, G. D. (Eds.): Shackelford's Surgery of the Alimentary Tract, 4th ed. Vol. I, The Esophagus.

- Philadelphia, W. B. Saunders, 1996, pp. 305–332.
- 4. Enterline, H., and Thompson, J.: Pathology of the Esophagus. New York, Springer-Verlag, 1984.
- Atwood, S. E. A., Ball, C. S., Barlow, A. P., Jenkinson, L., Norris, T. L., and Watson, A.: Role of intragastric and intraoesophageal alkalinisation in the genesis of complications in Barrett's columnar lined lower oesophagus. Gut, 34:11, 1993.
- 6. Gillen, P., Keeling, P., Bryne, P. J., et al.: Barrett's oesophagus: pH profile. Br. J. Surg., 74:774, 1987.
- 7. Parilla, P., Ortiz, A., Martinez de Haro, L. F., et al.: Evaluation of the magnitude of gastroesophageal reflux in Barrett's oesophagus. Gut, 31:964, 1990.
- 8. Pera, M., and Duranceau, A.: Malignant degeneration of Barrett's esophagus: Epidemiology of Barrett's esophagus and esophageal adenocarcinoma. Dis. Esophagus, 8:86, 1995.
- 9. Riddel, R. H.: Dysplasia and regression in Barrett's epithelium. In Spechler, S. J., and Goyal, R. K. (Eds.): Barrett's Esophagus—Pathophysiology, Diagnosis, and

- Management. New York, Elsevier, 1985, pp. 143–153.
- Roth, J. A., Lichter, A. J., Putnam, J. B., and Forastiere, A. A.: Cancer of the Esophagus. In Devita, V. T., Hehman, S., and Rosenberg, S. A. (Eds.): Cancer: Principles and Practice of Oncology, 4th ed. Philadelphia, J. B. Lippincott, 1993, p. 776.
- 11. Roth, J. A.: Multimodality therapy of cancer arising from Barrett's epithelium. World J. Surg., 19:205, 1995.
- 12. Sarr, M. G., Hamilton, S. R., Marrone, G. C., and Cameron, J. L.: Barrett's esophagus: Its prevalence and association with adenocarcinoma in patients with symptoms of gastroesophageal reflux. Am. J. Surg., 149:187, 1985.
- 13. Spechler, J. S., Robbins, A. H., Robbins, H. B., Vincent, M. E., Heeren, T., Doos, W. G., Colton, W. G., and Schimmel, E. M.: Adenocarcinoma and Barrett's esophagus: An overrated risk? Gastroenterology, 87:927, 1984.
- 14. Vaezi, M. F., and Richter, J. E.: Synergism of acid and duodenogastric reflux in complicated Barrett's esophagus. Surgery, 117:699, 1995.

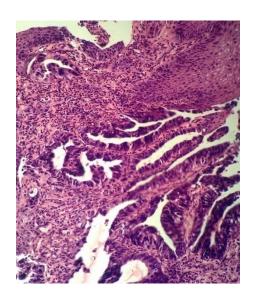


Fig 1: Barrets Esophagus

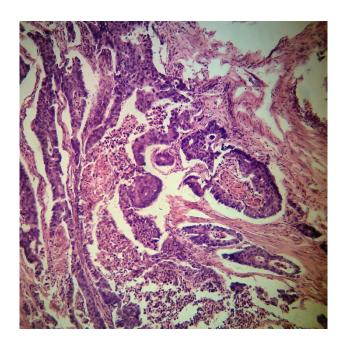


Fig 2: Adenocarcinoma Esophagus

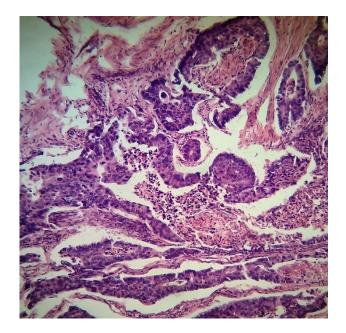


Fig 3: Adenocarcinoma Esophagus



Fig 4: CT Scan Cancer Esophagus



Fig 4: CT Scan Cancer Esophagus

