



QUADRIPLEGIC SEQUELAE OF CONGENITAL CEREBRAL PALSY CASE WITH END-STAGE ESOPHAGEAL CANCER: CASE REPORT

Yusuf Adnan Güçlü¹, Mehmet Çiçek¹, Zübeyde Gülce¹, Ayşegül Savk¹, Haluk Mergen¹,
Kurtuluş Öngel²

¹Izmir Tepecik Training and Research Hospital, Family Medicine Clinic, Izmir, Turkey.

²Izmir Katip Celebi University, Faculty of Medicine, Department of Family Medicine, Izmir, Turkey.

Corresponding Author:- **Kurtuluş Öngel**

E-mail: kurtulusongel@gmail.com

Article Info	ABSTRACT
<p>Received 15/01/2015 Revised 27/01/2015 Accepted 22/02/2015</p> <p>Key words: esophageal cancer, stent, neoadjuvant therapy, palliative care.</p>	<p>Esophageal cancer is about 6th to 8th ranks worldwide among all cancers. The first signs of esophageal carcinoma in 90% of cases are dysphagia and weight loss. Esophageal cancer can be treated with surgery as long as no metastasis to other organs occur. In the reported case, tests are made according to the procedure; according to the pathology and staging it is considered end-stage, stent inserted to the patient for palliation, neoadjuvant chemotherapy and radiotherapy are recommended. His stent is clogged after a certain period with decreased oral intake and increased vomiting and hemorrhage, the patient is asked to be undergone interventional radiological intervention. With this case report, we have tried to emphasize the palliative care approach in such patients of quadriplegic sequelae of congenital cerebral palsy with end-stage esophageal cancer.</p>

INTRODUCTION

Esophageal cancer is about 6th to 8th ranks worldwide among all cancers [1]. It constitutes the 1.5-2% of all cancers, 5-7% of all digestive system cancers [2]. It ranks #6 in mortality. Esophageal cancer incidence is frequent in the 5 to 6 decades and is rare under 3rd decade ($\approx 0.2\%$). It is found about 3-5 times higher in men than women [2]. Esophageal cancer is very common in the East region of Turkey and it is common extending from south of the Caspian Sea, Afghanistan, to China and Japan [2,3]. Smoking and alcohol consumption, inadequate intake of fresh fruits and vegetables are the three most important risk factors for esophageal cancer [4]. Diet plays an important role in the development of squamous cell carcinoma. Tylosis with the hyperkeratosis of the palm and soles may be a characterized sign [5,6]. Drinking very hot tea in the eastern part of our country increases the squamous cell type of cancer development. Meanwhile the role of human papilloma virus (HPV) is not well shown in the development of esophageal cancer as it is in anogenital cancer. In France and Portugal in Europe, in Asia (Japan,

China, Hong Kong, India, Pakistan and Korea), South Africa, Alaska and Australia, HPV infection are reported esophageal squamous cell cancer in higher proportion [2,7]. The most important risk factor for adenocarcinoma is Barrett's esophagus. Prolonged bile and acid reflux increases the risk of adenocarcinoma of the esophagus [8]. Familial and genetic factors are also important in cancer development. Gastroesophageal reflux at young age, Barrett's esophagus in the middle ages returns to esophagus cancer when they reach to the 50-60 age [8]. The first signs of esophageal carcinoma in 90% of cases are dysphagia and weight loss [3]. The diagnosis of esophageal cancer is made by endoscopy and pathological examination of the piece taken during this process. All patients undergo thoracic and upper abdominal computed tomography (CT) [9]. This tomographic investigation gets information whether the disease is dispersed to the surrounding tissue, lungs and liver. Endoscopic esophageal ultrasonography is effective in detection of the esophageal wall and adjacent organs of the cancer and is better than



computerized tomography in detecting superior mediastinal lymph nodes. Positron emission tomography (PET) shows tumor size, relationship with the surrounding tissue, division rate and whether metastasis to the lymph nodes or other organs exists or not. All these tests become obvious clinical stage of esophageal cancer and the treatment is planned according to the step [9,10]. Barium enema, esophagogastroduodenoscopy, endoscopic ultrasonography and MR are of the diagnostic methods.

Esophageal cancer has four stages: Stage I: cancer cells are found only in the uppermost layer of the inside of the esophagus. Stage II: cancer has spread to the esophagus or the amount of the deep layer adjacent lymph nodes. Stage III: cancer has invaded the deeper part of the esophagus or esophageal wall, to lymph nodes or tissues, there is no spread to other parts of the body. Stage IV: The cancer has spread to other parts of the body. Esophageal cancer, liver, lungs, it can be dispensed anywhere on the body including the brain and bones.

With this case report, we have tried to emphasize the palliative care approach in such patients of quadriplegic sequelae of congenital cerebral palsy with end-stage esophageal cancer.

CASE REPORT

Twenty-eight-year-old known cerebral palsy with quadriplegic sequelae patient addressing with widespread pain, nausea and vomiting to the center is diagnosed as esophagus adenocarcinoma by the endoscopic investigation. Diagnosis is made up three months ago and esophageal stent and neoadjuvant therapy is implemented because of highly spreading tumor. Liver and bone metastases detected at follow-up PET and CT scans and a metastatic cardioesophageal tumor is obtained. The patient is been accepted as terminal stage in staging. The stent is inserted into the esophagus to ensure nutrition. By the clogging of the stent, nausea, vomiting and feeding problems occurred and the patient is admitted to our palliative care center for feeding, pain, nausea palliation and radiotherapy indication. Radiotherapy was recommended by the radiation oncology consultant.

General condition of the patient was moderate conscious, non-cooperated. He had a pectus carinatum view in the thorax. Both lungs were participating equally to breathing and had a restrictive pulmonary dysfunction. Breath sounds were decreased. There was no additional voice in the cardiovascular system examination. Abdomen was free, defense and rebounding were not present.

In the laboratory values; haemoglobin (Hb): 11,9 gr/dl, WBC: 9,6 K/ μ L, hematocrite (Hct): 37%, mean corpuscular volume (MCV): 75,6 fl, platelets (PLT): 271000/ μ L, fasting blood glucose (FPG): 112 mg/dl, urea: 24 mg/dl, creatinine: 0,5 mg/g, aspartic aminotransferase (AST): 33 U/l, alanin aminotransferase (ALT): 12 U/l, alkaline phosphatase (ALP): 243 IU/l, gamma glutamyl transferase (GGT): 54 mg/dl, lactic dehydrogenase (LDH): 528 U/l; iron (Fe): 26 mg/dl; iron binding capacity: 212

mg/dl, ferritin: 193 ng/ml. Other findings in patients were in normal levels, mild anemia due to malignancy was detected.

CT of the brain was found to be compatible with corpus callosum agenesis. Left lateral ventricle assessed as moderate wide asymmetric; variational difference is thought to be secondary to the corpus callosum agenesis. In all abdomen and thorax CT and PET scanning malignancy and metastasis signs are viewed in esophagus, lungs, liver, pelvis and vertebra. According to the gastroenterology consultation regarding the bleeding points, no chance of hemorrhage control was found endoscopically, in this respect radiation oncology and general surgery consultation were advised. General surgery consult accepted the patient as inoperable and neoadjuvant chemotherapy and radiology were advised. After radiation oncology and medical oncology consultations, radiotherapy is advised for the patient and a total of 10 seances of radiotherapy were completed. After four months, the patient is called for the follow-ups.

After the radiotherapy séances, abundant hemorrhage occurred two times. Because his Hb value is decreased to 7.8% g/dl, two units of erythrocyte suspension were administered and oral intake is forbidden. After intravenous feeding was started, Hb value increased to 11.3 g/dl. Because oral intake and vomiting caused continuous bleeding, percutaneous endoscopic gastrostomy (PEG) was consulted by gastroenterology and general surgery. PEG is not considered appropriate because of widespread metastases. The inserted esophageal stent was suggested to be replaced. Interventional radiology unit is asked to determine the hemorrhage focus and coagulate it. Interventional radiology unit advised BT angiography in order to determine the bleeding focus. The treatment of the patient continues and radiologic intervention will be planned.

DISCUSSION

Esophageal cancer accounts for 90-95% of squamous cell carcinoma, but an increase is observed in the incidence of adenocarcinoma in recent years. When examined settlements; cervical esophagus 8%, upper thoracic esophagus 3%, middle thoracic esophagus 32%, the lower thoracic esophagus 25% and the cardia 32% [2]. The case presented here is the cardio-esophageal located.

Different symptoms could be occurred by the esophageal cancer and the spread of the disease due to local progression. Physical examination is usually normal but can be detected with cervical and supraclavicular lymphadenopathy. In this patient were oral intake disorders, weight loss, there is pain and nausea vomiting.

In the presented case, tests conducted appropriately to the procedure. According to the agreed end-stage pathology and staging [11], a stent was inserted for palliation, neoadjuvant chemotherapy and radiotherapy is advised. After clogging of the stent, increased vomiting and hemorrhage caused by oral feeding, a radiological



intervention is thought to be planned. A new stent insertion or PEG is not advised to the patient who cannot be fed orally due to spreaded metastases and lesion invasion to cardia region of the stomach.

The follow-up of such patients reported here is very difficult. Family brings material and spiritual burden.

Admissions, dischargings, the examination and the intervention, treatment and cure exhaust the patient and his relatives. Palliative care activities on the agenda in Turkey are proceeding rapidly. Such care services should be more thoroughly multiplying.

REFERENCES

1. Mao WM, Zheng WH, Ling ZQ. (2011). Epidemiologic risk factors for esophageal cancer development. *Asian Pac J Cancer Prev*, 12, 2461-2466.
2. Huang J, Bashir M, Ianettoni MD. Carcinoma of the Esophagus. General Thoracic Surgery. Eds. Shields TW, Lo Cicero III J, Reed CE, Feins RH in. Lippincott Williams &Wilkins, Philadelphia 2009, 7th Edition, 1984-2016.
3. Tuncer I, Uygan I, Kösem M ve ark. (2001). Van ve çevresinde görülen üst gastrointestinal sistem kanserlerinin demografik ve histopatolojik özellikleri. *Van Tıp Derg*, 8, 10-13.
4. Morita M, Kumashiro R, Kubo N et al. (2010). Alcohol drinking, cigarette smoking, and the development of squamous cell carcinoma of the esophagus: epidemiology, clinical findings and prevention. *Int J Clin Oncol*, 15, 126-134.
5. Risk JM, Mills HS, Garde J, Dunn JR, Evans KE, Hollstein M, Field JK. (1999). The tylosis esophageal cancer (TOC) locus: more than just a familiar cancer gene. *Diseases of the Esophagus*, 12, 173-176.
6. Gupta N, Barwad A, Rajwanshi A, Kochhar R. (2012). Prevalence of human papilloma virus in esophageal carcinomas: a polymerase chain reaction-based study. *Acta Cytol*, 56, 80-84.
7. Heath EI, Limburg PJ, Hawk ET, Forastiere AA. (2000). Adenocarcinoma of the esophagus: risk factors and prevention. *Oncology*, 14, 517-524.
8. Okten I. (1999). Özofagus kanserleri. *Güncel Gastroenteroloji*, 3, 94-105.
9. Cook MB, Shaheen NJ, Anderson LA et al. (2012). Cigarette smoking increases risk of Barrett's esophagus: an analysis of the Barrett's and Esophageal Adenocarcinoma Consortium. *Gastroenterology*, 142, 744-753.
10. Başoğlu A, Çelik B, Aydın O. (2005). Özofagus kanserinde endoskopik lugol boyama yöntemi ile proksimal cerrahi rezeksiyon sınırının tespiti. *Türk Göğüs Kalp Damar Cerrahisi Dergisi*, 13(1), 24-30.
11. Barut Y, Toprak H, Güleç AK ve ark. (1996). Özofagus kanseri: Wallstent ile Palyatif tedavinin ilk sonuçları. *İstanbul Tıp Dergisi*, 4, 1-4.

