



CODEN [USA]: IAJPBB

ISSN: 2349-7750

**INDO AMERICAN JOURNAL OF  
PHARMACEUTICAL SCIENCES**<http://doi.org/10.5281/zenodo.1401413>Available online at: <http://www.iajps.com>

Research Article

**ANALYSIS OF CBC IN GASTROINTESTINAL CANCER**<sup>1</sup>Dr. Muhammad Yasir, <sup>2</sup>Dr. Salman Haider, <sup>2</sup>Dr. Sidra Rana<sup>1</sup>Mayo Hospital, Lahore<sup>2</sup>Nishter Hospital, Multan**Abstract:**

**Introduction:** Cancer is a leading cause of death worldwide: it accounted for 7.9 million deaths (around 13% of all deaths) in 2008. The rising life expectancy means that the risk of developing cancer is also increasing. Gastric cancer is the second most common cancer worldwide and almost two-thirds of all cases occur in developing countries.

**Objectives of the study:** The basic aim of the study is to analyze the complete blood count (CBC) in gastrointestinal cancer patients.

**Methodology of the study:** The study was conducted at Mayo hospital, Lahore during 2017 with the help of concerned coittie of hospital. . The data was collected from both genders and the sample size is 70. Detailed history was taken from all patients with special reference to duration of disease, mode of infection, previous history of disease, HBV or HCV or any other kind of infection. A thorough clinical examination was carried out and stigmata of disease and histopathology was noted.

**Results:** According to analysis CBC of patients shows that the level of WBCs and RBCs become decreases in gastrointestinal cancer. It shows that GSH level become increases in patients as compared to control. But the GPx become decreases. All other antioxidants become increases in patients group as compared to control.

**Conclusion:** It is concluded that the level of antioxidants will be used as a important biomarker in the diagnosis and treatment of gastrointestinal cancer. And the level of blood cells become decreases in diseased condition.

**Keywords:** Analysis, Cbc, Gastrointestinal, Cancer.

**Corresponding author:****Dr. Muhammad Yasir,**

Mayo Hospital,

Lahore

QR code



Please cite this article in press Muhammad Yasir et al., *Analysis of CBC in Gastrointestinal Cancer.*, Indo Am. J. P. Sci, 2018; 05(08).

**INTRODUCTION:**

Cancer is a leading cause of death worldwide: it accounted for 7.9 million deaths (around 13% of all deaths) in 2008. The rising life expectancy means that the risk of developing cancer is also increasing. Deaths from cancer worldwide are projected to continue rising, with an estimated 12 million deaths in 2030 [1]. As most cancers appear in adults at an advanced age, the burden of cancer is much more important than other diseases in populations with a long life expectancy. The eight leading cancer killers worldwide are also the eight most common in terms of incidence. Together, they account for about 60% of all cancer cases and deaths. They are cancers of the lung, stomach, breast, colon-rectum, mouth, liver, cervix and oesophagus [2].

Gastric cancer is the second most common cancer worldwide and almost two-thirds of all cases occur in developing countries. It is the fourth most common cancer in men, while in women it is the fifth most common cancer (based on statistic in 2008)<sup>3</sup>. Although the incidence of gastric cancer is declining, it still remains a major health problem and a common cause of cancer mortality worldwide. Gastric cancer carcinogenesis refers to accumulation of genetic alteration of multiple genes such as oncogenes, tumour suppressor and mismatch repair genes [4].

The dynamic balance between cell proliferation and apoptosis is very important to maintain the homeostasis in human body and gastric carcinogenesis is related to this imbalance. Development of gastric cancer is believed to be a slow process with primary etiological determinants for gastric cancer being exposure to chemical carcinogens and/or infection with *Helicobacter pylori*. It has been reported that gastric cancer also expresses multidrug-resistance associated protein (MRP) and shows lower sensitivity to anti-cancer drugs. Gastric cancer is more common in older populations, usually occurring in the seventh and eighth decades of life [5]. The mean age at diagnosis was 67 years in one large series. Although suspected, there is current uncertainty as to whether gastric cancer in young patients is associated with a worse clinical outcome [6].

**Pathology of disease**

Gastric cancer is an aggressive disease that continues to have a daunting impact on global health. Despite an overall decline in incidence over the last several decades, gastric cancer remains the fourth most common type of cancer and is the second leading cause of cancer-related death worldwide [7].

Although the incidence is declining due to improved nutrition, food preservation, better prevention, earlier diagnosis and treatment, the disease still carries a poor prognosis. Gastric cancer is often diagnosed at an advanced stage. The cornerstone of therapy is surgical resection with adjuvant chemotherapy or chemo-radiation in appropriate cases. Such an approach has led to improved survival. Unfortunately, treatment of advanced or metastatic gastric cancer has seen little progress and median overall survival (OS) in this group remains <1 year. Gastric cancer is a heterogeneous disease that demands continued attention and research with regard to prevention, early detection and novel therapeutic options [8].

**Objectives of the study**

The basic aim of the study is to analyze the complete blood count (CBC) in gastrointestinal cancer patients.

**METHODOLOGY OF THE STUDY:**

The study was conducted at Mayo hospital, Lahore during 2017 with the help of concerned coittie of hospital. . The data was collected from both genders and the sample size is 70. Detailed history was taken from all patients with special reference to duration of disease, mode of infection, previous history of disease, HBV or HCV or any other kind of infection. A thorough clinical examination was carried out and stigmata of disease and histopathology was noted.

**Blood investigation**

It includes Hemoglobin (Hb), total leucocytes count (TLC), and differential leucocytes count (DLC), platelet count, level of antioxidants and other related factors. We collected data on incidence of gastric cancer, incidence of oesophageal cancer, deaths from gastric cancer, deaths from any cause, and adverse effects arising due to therapy.

**Statistical analysis**

The data were sampled and entered into the SPSS worksheet for analysis. The alpha criterion was set at 0.05 (95% confidence interval [CI]). After constructing a 2x2 contingency table, chi-square without Yates correction was used to find the association between the potential risk factors and hepatitis status.

**RESULTS:**

Table 01 shows the CBC of control and diseased group. According to analysis CBC of patients shows that the level of WBCs and RBCs become decreases in gastrointestinal cancer.

**Table 01:** CBC in gastrointestinal cancer patients

Blood component	Abbreviation used	Reference range	Diseased values
White blood cells	WBC	4500-11,000/mm <sup>3</sup>	3.8-11.0 x 10 <sup>9</sup> /L
Red blood cells*	RBC	Male: 4.3-5.9 million/mm <sup>3</sup> Female: 3.5-5.5 million/mm <sup>3</sup>	3.9-4.0 million/mm <sup>3</sup>
Hemoglobin*	HGB	Male: 13.5-17.5 g/dL Female: 12.0-16.0 g/dL	9-11 g/dL
Hematocrit*	HT	Male: 41%-53% Female: 36%-46%	0.41-0.53
Mean corpuscular volume	MCV	80-100 μm <sup>3</sup>	70-80
Mean corpuscular hemoglobin	MCH	25.4-34.6 pg/cell	39-54 pg/cell
Mean corpuscular hemoglobin concentration	MCHC	31%-36% Hb/cell	4.81-5.58
Platelets	Platelets	150,000-400,000/mm <sup>3</sup>	150-400 x 10 <sup>9</sup> /L

Table 02 shows the antioxidants level of patients. It shows that GSH level become increases in patients as compared to control. But the GPx become decreases. All other antioxidants become increases in patients group as compared to control.

**Table 02:** level of antioxidants in patients

	group	N	Mean	Std. Deviation	Std. Error Mean
GSH	control	10	.93150	.283559	.089669
	patients	17	4.15765	.534536	.129644
GPx	control	10	.78900	.344970	.109089
	patients	17	.17750	.038108	.009242
Catalase	control	10	4.33600	.748750	.236776
	patients	17	3.69382	1.188499	.288253
SOD	control	10	.38020	.165513	.052340
	patients	17	.92471	.814660	.197584
MDA	control	10	2.57200	.814886	.257690
	patients	17	1.80729	1.379333	.334537

## DISCUSSION:

The development of gastric cancer is a multifactorial process and many conditions influence the likelihood of occurrence, of them, family history of gastric cancer, *Helicobacter pylori* infection (a common bacteria that can also cause stomach ulcers), history of an adenomatous gastric polyp larger than 2 centimetres, history of chronic atrophic gastritis, history of pernicious anemia, obesity, alcohol, smoking, red meat and low socioeconomic status are all believed to be important [9].

In 1994, the International Agency for Research on Cancer and The World Health Organization classified *Helicobacter pylori* as a type I carcinogen, the exact mechanism leading to gastric carcinoma is not clearly understood. The effects of *H. pylori* infection on gastric cancer appear multifactorial, involving host and environmental factors as well as differing bacterial strains. *H. pylori* is most closely associated with intestinal gastric cancers, which follow a stepwise pathway but

toward malignancy, similar to that in the colon [10]. In the Correa model of gastric carcinogenesis, gastric inflammation leads to mucosal atrophy, metaplasia, dysplasia, and, ultimately, carcinoma. Studies have shown that *H. pylori* infection is an independent risk factor for distal gastric cancer, with a 3- to 6-fold increased risk relative to those without the infection. In patients with *H. pylori*, the presence of specific gene polymorphisms increases the risk of developing gastric carcinoma [11]. Genes that encode tumor necrosis factor alpha (TNF-α), and interleukins IL-1, IL-8, and IL-10 have each been associated with higher cancer rates in the setting of *H. pylori*. While intestinal gastric cancer is strongly associated with chronic *H. pylori*, this strong link is not seen in diffuse gastric cancer [12]. Diffuse or cardia gastric cancer, however, has been associated with other risk factors such as higher socioeconomic class, obesity, and type A blood.

Depending on the size and location of the primary tumor, the preferred means of therapy is surgical

resection with total or subtotal gastrectomy. Chemotherapeutic interventions have been evaluated in the neoadjuvant, adjuvant, and metastatic settings [13]. The SWOG 9008 trial evaluated postoperative chemo-radiation after resection of gastric or G-E junction tumors. Patients were randomized to observation versus 5-FU/leucovorin and radiation up to 45 Gy. The 10 year follow-up revealed significant improvement in OS in the treatment arm; however, postoperative chemotherapy is generally poorly tolerated in this population. The MAGIC trial evaluated perioperative chemotherapy with epirubicin, cisplatin, and 5-FU (ECF) in adenocarcinoma of the G-E junction or lower esophagus and found that the chemotherapy group had significantly higher OS and progression-free survival (PFS) than those who received surgery alone. In the metastatic or recurrent setting any of the combination chemotherapy regimens such as ECF, DCF, FOLFIRI or best supportive care are reasonable options [14].

#### CONCLUSION:

It is concluded that the level of antioxidants will be used as a important biomarker in the diagnosis and treatment of gastrointestinal cancer. And the level of blood cells become decreases in diseased condition.

#### REFERENCES:

1. Ferro A, Peleteiro B, Malvezzi M, Bosetti C, Bertuccio P, Levi F, et al. Worldwide trends in gastric cancer mortality (1980-2011), with predictions to 2015, and incidence by subtype. *Eur J Cancer*. 2014;50:1330-44.
2. Bang YJ, Kim YW, Yang HK, Chung HC, Park YK, Lee KH, et al. Adjuvant capecitabine and oxaliplatin for gastric cancer after D2 gastrectomy (CLASSIC): A phase 3 open-label, randomised controlled trial. *Lancet*. 2012;379:315-21.
3. Wang R. The epidemiological study of subtype risk factors of gastric cancer. *Zhonghua LiuXingBingXue ZaZhi*. 1993;14:295-299.
4. Sun X, Mu R, Zhou Y, Dai X, Qiao Y, Zhang S, Huangfu X, Sun J, Li L, Lu F. 1990-1992 mortality of stomach cancer in China. *Zhonghua ZhongLiu ZaZhi*. 2002;24:4-8.
5. Correa P. Human gastric carcinogenesis: a multistep and multifactorial process--First American Cancer Society Award Lecture on Cancer Epidemiology and Prevention. *Cancer Res*. 1992;52:6735-6740.
6. Rugge M, Correa P, Dixon MF, Hattori T, Leandro G, Lewin K, et al. Gastric dysplasia: The Padova international classification. *Am J Surg Pathol*. 2000;24:167-76.
7. Smith MG, Hold GL, Tahara E, El-Omar EM. Cellular and molecular aspects of gastric cancer. *World J Gastroenterol*. 2006;12:2979-90.
8. Yasui W, Sentani K, Motoshita J, Nakayama H. Molecular pathobiology of gastric cancer. *Scand J Surg*. 2006;95:225-31.
9. Graziano F, Humar B, Guilford P. The role of the E-cadherin gene (CDH1) in diffuse gastric cancer susceptibility: From the laboratory to clinical practice. *Ann Oncol*. 2003;14:1705-13.
10. Censini S, Lange C, Xiang Z, Crabtree JE, Ghiara P, Borodovsky M, et al. Cag, a pathogenicity island of *Helicobacter pylori*, encodes type I-specific and disease-associated virulence factors. *Proc Natl Acad Sci U S A*. 1996;93:14648-53.
11. Covacci A, Censini S, Bugnoli M, Petracca R, Burroni D, Macchia G, et al. Molecular characterization of the 128-kDa immunodominant antigen of *Helicobacter pylori* associated with cytotoxicity and duodenal ulcer. *Proc Natl Acad Sci U S A*. 1993;90:5791-5.
12. Cunningham D, Starling N, Rao S, Iveson T, Nicolson M, Coxon F, et al. Capecitabine and oxaliplatin for advanced esophagogastric cancer. *N Engl J Med*. 2008;358:36-46.
13. Dikken JL, van Sandick JW, Maurits Swellengrebel HA, Lind PA, Putter H, Jansen EP, et al. Neo-adjuvant chemotherapy followed by surgery and chemotherapy or by surgery and chemoradiotherapy for patients with resectable gastric cancer (CRITICS) *BMC Cancer*. 2011;11:329.
14. Bang YJ, Van Cutsem E, Feyereislova A, Chung HC, Shen L, Sawaki A, et al. Trastuzumab in combination with chemotherapy versus chemotherapy alone for treatment of HER2-positive advanced gastric or gastro-oesophageal junction cancer (ToGA): A phase 3, open-label, randomised controlled trial. *Lancet*. 2010;376:687-97.