CODEN [USA]: IAJPBB ISSN: 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES
http://doi.org/10.5281/zenodo.2593214

Available online at: http://www.iajps.com *Research Article*

GENES ASSOCIATION IN COLORECTAL CANCER

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Article Received: December 2018 **Accepted:** February 2019 **Published:** March 2019

Abstract:
Colorectal cancer now a days most common disease in the world many strategies have been under investigated for the treatment of colorectal cancer. Colorectal cancer undergoes metastasis and genes responsible for tumor suppressor genes. B catenin genes inhibition involves in the cancer. epithelial cells transformed in to the oncogenes. Mutation in the WNT pathway leading to the formation of lesion and these polyps leads in the formation of mutation in the epithelial cells. TCF family is the main predominant in the colorectal cancer. TCF 4 is the main precursor of this family. while TCF1 is the main dominant negative key precursor of this family.

Keywords: *Colorectal Cancer, B catenin genes, TCF family, HNPCC syndrome*

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*Please cite this article in press ABDULRAHEEM et al., **GENES ASSOCIATION IN COLORECTAL CANCER.**, Indo Am. J. P. Sci, 2019; 06(03).*

INTRODUCTION:

Colorectal cancer disease is the deadly disease occurring in the world. Prevalence of this type of cancer increases with the increasing of age. [1] This type of cancer is related with lymph node sometimes it may not be related to it. [2] Colorectal cancer is also related to the HNPCC syndrome, study show that it is autosomal dominant pattern, hereditary cancer syndrome can be diagnosed by the presence of familial adenomateous polyposis .

Immunological problems:

Serum albumin is normally functioning. Ornithine decarboxylase activity is normal. Colorectal cancer undergoes metastasis and genes responsible for tumor suppressor genes [3] B catenin inhibition involves in the cancer. Epithelial cells transformed in to the oncogenes mutation in the WNT pathway leading to the formation of lesion and these polyps leads in the formation of mutation in the epithelial cells. [4]

genes family involves in the colorectal cancer:

TCF family is the main predominant in the colorectal cancer. [3] TCF 4 is the main precursor of this family while TCF1 is the main dominant negative key precursor of this family. [5] Adenoma size increase in this type of cancer leading to the apoptosis. [6] Constant use of aspirin leads to the increase rate of colorectal cancer. cyclooxygenase cycle involves in the tumour formation its level increases up too many leading to the adenoma. cyclooxygenase type 2 involves in the cell formation of tumour such as in colorectal carcinogenesis formation. [7] Cancer forming tumors have glycolysis cycle depends on oxygen more taking of ATP. [8] Tumor signal results in the progress of solid tumour response. [9] Disease regulation, study shows that, treatment was observe and patients are giving off treatment. [10] Patients of colorectal cancer are assigned to different therapies and bevacizumab- treatment therapy is one of these therapies. Patients who are taking these effective therapies are discontinue with the chemotherapy treatment because to show the effect of current therapy. [11] The primary results show the survival rate. There was no similarty , Secondary results measures their standard living such a way of living which food they take, and their standard living . Patients which are giving the therapy they also gone with the process of analyzing, data was generated to show the survival rate related to this therapy. Survival rate was recording by the time from randomization study to decline rate or death during the study, the analyses record the result by the therapy after a month survival records are analysed with the death rate for different therapies.

MATERIALS AND METHODS:

Table 1: Questionnaire to evaluate awareness about etiology of Colo Rectal Cancer

Hepatitis A is a	Yes	No
1. Viral disease		
2. Bacterial disease		
3. Fungal disease		
4. Genetic disease		
5. Metabolic disease		
Ever suffered from Colorectal Cancer		
6. You		
7. Your family		
8. Your relative		
9. Your neighbor		
10. Your friend		
Colorectal Cancer is transmitted by		
11. Contacts or blood transfusion		
12. From parents to offspring		
Colorectal Cancer may be treated by		
13. Medicines		
14. Surgery		
15. Do not worry, it is easily curable		

Table 1: Questionnaire to evaluate awareness about etiology of Colorectal Cancer

Colorectal Cancer is a	Yes	No
1. Viral disease		
2. Bacterial disease		
3. Fungal disease		
4. Genetic disease		
5. Metabolic disease		

Table 2: Questionnaire to evaluate views about prevalence of Colorectal Cancer

Ever suffered from Colorectal Cancer	Yes	No
1. You		
2. Your family		
3. Your relative		
4. Your neighbor		
5. Your friend		

3: Questionnaire to evaluate views about transmission of Colorectal Cancer

Colorectal Cancer is transmitted by	Yes	No
1. Contacts **Table** or blood transfusion		
2.		

Table 4: Questionnaire to evaluate views about Hope for Colorectal Cancer

Colorectal Cancer may be treated by	Yes	No
1. Medicines		
2. Surgery		
3. Do not worry, it is easily curable		

RESULTS:

Table 5: Awareness about etiology of ColorectalCancer

Questions	**Male**		**Female**		**Total**	
	Yes	**No**	**Yes**	**No**	**Yes**	**No**
1. Viral disease	**20%**	**80%**	**26.6%**	**73.3%**	**30%**	**74%**
2. Bacterial disease	**40%**	**60%**	**60%**	**40%**	**57%**	**42%**
3. Fungal disease	**20%**	**80%**	**53.3%**	**47%**	**48%**	**51%**
4. Genetic disease	**0%**	**100%**	**27%**	**73.3%**	**22%**	**77%**
5. Metabolic disease	**0%**	**100%**	**33.3%**	**67%**	**28%**	**71%**

DISCUSSION:

Results shows that people are not much aware about this disease. awareness is very important about some disease because it brings knowledge for curing this disease. It is also very important about awareness of chronic disease. This survey is important because this give views about disease. This survey consists of 200

students 100 are boys and 100 are girls of the BAHUDDIN ZAKARIYA UNIVERSITY Multan.

Role of MSI gene:

The role of MSI to colorectal cancer has been under investigated. MSI means microsatellite instability results by having mutation during the DNA replication and that results in the formation of tumour and this mutated repetitive sequence occurs in genomes. [12] Without disease progress shows results, records on progression-survival were recorded during or later at the time of infection. Patients have no response under the influence of characterized fellow program. [11]
p53 gene results in apoptosis gene p53 expression results the cell cycle arrest in the formation of p21 gene transcriptional depandant on the kinase activity. [13] Study shows that b catenin is present in the colon cancer cells. [5] p21 is the main modulator in the cell cycle arrest which enhance the cell division of colorectal cancer. mesenchymal cells also under goes differentiation under the influence of p21, which activates the B catenin. [5]

The process which is responsible for colorectal cancer formation are not truly understood.

Analyzing of genes involved in colorectal cancer formation process of analyzing normal cells with the tumour infected cells from patients of cancer, which shows and understanding the process of oncogenes. Study shows the protein MYC up regulated the process of understanding suppression and metabolically global metabolic process of colorectal tumors by enhancing its activity of metabolic process of tumour cells. Most important of this point conducting study, this metabolic reprogramming process of tumors suppression occurred in a specific site or receptor not connected to a specific gene mutations site in a colorectal tumors formation. Some, small- molecular biologic molecule which act as inhibitor of gene MYC required necessary for the process of study which involves analyses. Study demonstrate the knockdown process of MYC gene resulting in the downstream process of pyrimidine process of formation of nucleotide base synthesis genes takes part to the suppression of colorectal tumors cancer cells induction and proliferation and intact gene of MYC, and thus pyrimidine formation synthesis pathways may be perfect targets for colorectal cancer therapy. [14]

Study conducted to show the mutation in *KRAS gene* shows for no increase in results panitumumab therapy. The study conducted for a control test made to study the presence or absence of relative effect of panitumumab therapy in *KRAS* mutational pathway not depends on the significance of the potential presence of prognostic and antagonistic pathway give result of strongly influence of *KRAS* mutations on the possible results, enabling us to study conclusion that tumors suppression in the clinical benefit observed in the population of *KRAS* unselected population was perfectly taken from the *KRAS* population. Giving the crossover analyses design, conclusions are only limited to the influence of *KRAS* mutational.

Population on tumor and tumor response end points Indeed, the patients received panitumumab therapy on disease. [15] To characterize and summarize the cancer cell alterations in colorectal tumors, study show a genome wide based analysis of samples, analyzing exon sequence, DNA promoter methylation cells number and messenger RNA and microRNA expression. A subset of samples under goes deeply to the whole gene sequence.

Colo rectal tumors carcinomas cancer were exactly sure to be highly mutated containing mutation, some of these have the expected high marker microsatellite instability inaccuracy, [2] related with high amount of methylation and gene sequencing also affecting the somatic cancer cells related to the miss match-repair gene sequence and polymerase proof reading a mutation a mutations. [16] Not including in study the mutated cancers, colon rectum cancers were found to have considerably similar banding patterns of genomic alteration and sequencing. genes were definitely mutated altered sequence, and in addition to a mutations. [17] Frequent mutations related to cancer and mutated copy-number of KRAS mutation alterations and drug- targetable amplifications of and newly discovered cancer has been increased. [18] chromosomal mutation which specifically introduce translocation process include the fusion. [19] Study analyses suggest the new markers for the suppression of colorectal carcinoma cancer it is an important role for gene directed towards the activation and suppression related to the colorectal cancer. [20]

Study shows that mutated carcinomas of the colon rectum are distinguishing at the genomic sequence level. [13] Tumours from the colorectum cancer were consists of likely to be highly methylated and t contain mutation rates at elevated level in the tumors suppression process. [21] Study recognized the activation of gene responsible for tumors signaling pathway and in turns inactivating the signaling pathway, resulting in the increased activity of the many pathways which are responsible for the activity

of cancer. [21] Although a number of these genes could potentially be key players in the development of CRC, their actual role in promoting the growth of colon tumours remains to be proven. To identify relevant target genes that are important in the pathogenesis of CRC, we additionally investigated whether reducing the levels of these genes in an established colon cancer cell line might

alter its proliferation either in culture or when implanted in nude mice. Such results would be consistent with a possible role for these genes in the pathogenesis of colon cancer. Thus, it is important to consider possible interactions when using combination therapies that disrupt the functions of multiple distinct genes.

Genome sequencing targets the large number of sequence and proteins which are involves in the pathways of cancer formation process such as cell cycle arrest also specifically target the receptor tyro kinase. [17] Study shows the understanding of deadly disease and identifying the possible for treatment for colorectal cancer. [18]Disease progress initially in the phase of the analyses. [14]

Recommendation for early detection of colorectal cancer

Some tests and procedure for both gender purpose of test is to early detect colorectal cancer.

Table 1

Test	Sex	Age	Frequency
Sigmoidoscopy preferably flexible	M,F	50 and over	Every 3 to 5 years
Fecal Occult Blood Test	M,F	40 and over	Every year
Digital rectal examination	M,F	18 and over	Every year
Pap test	M,F	40 and over	Every year
Pelvic Examination	M,F	40 and over	Every year
Endometrial Tissue Sample	M,F	18 and over	Every year
Breast self-examination	F	18 to 50	Every month

CONCLUSION:

Many genes involving in the formation and signaling of cancer and it is the participation of different genes in colorectal cancer. [22]

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