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"A Review on Cancer"

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ABSTRACT: -
Cancer is a cluster of cells that start dividing and cannot stop. Cancer begins in cells, which are the building blocks of our body. Normally, body forms new cells as we need them, and old cells don't die when they should. These extra cells can form a mass called tumor. So, in easy words, cancer is cell functioning gone wrong. Sometimes, this tumor only forms a mass and stop, also known as benign tumors. While sometimes, this tumor starts dividing and cannot stop, this type is known as malignant tumor.
They can break the nearby tissues and start spreading. Cancer is not just abnormal division of cells; it is also an invitation to many diseases. One defining feature of cancer is the spread of cancer from one part of the body to another, commonly known as metastasis. Mostof the cancers are named from where they start. Example: lung cancer starts in lungs, brain tumors are formed in brain etc. The agents causing the cancers are carcinogens, which can be physical, chemical, or biological.
Cancer usually remains undetected for a long time. But now, due to rapid advancement in technologies, in the last decades, it is now possible to analyze the molecule are makeup of different cancer types. Symptoms and treatment depends on the cancer type and how advanced it is. Most treatment plants may include surgery, radiation or chemotherapy. Now come on you in treatments may also include hormone therapy, or stem cell transplantation.
Key Word: - Cancer, Malignant Tumor, Division of Cells, Stem Cell.

I. INTRODUCTION:

A tumor is defining as a swelling or morbid enlargement those results from an overabundance of cell growth & division. The NATIONAL CANCER INSTITUTE define a tumor as "an abnormal mass of tissue that results when cells divided more than they should or do not die when they should." A tumor is a mass or lump of tissue that may resemble swelling (1).

A tumor develops when cells reproduce too quickly & without control. Tumor can vary in size from a tiny nodule to a large mass depending on the type.Not all tumors are cancerous, but it is a good idea to see a doctor if one appears. Tumor may be benign (not cancer) or malignant (cancer).Tumor can appear almost anywhere inthe body (2).

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How it is formed: -

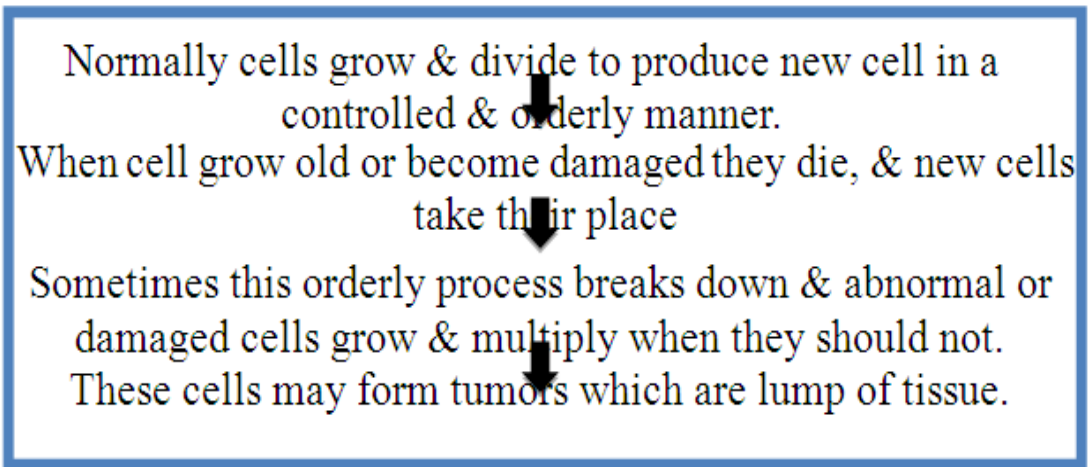

Figure 1: - How tumor formed.

Types of tumor: - There are three main types of Puma -

1. Benign tumor
2. Premalignant tumor
3. Malignant tumor

1. Benign tumor: - These are non cancerous. They either cannot spread all grow. If they do, they do it very diminished speed. If removed surgically, they usually, do not return. Most benign tumors are not harmful and they are unlikely to affect other parts of the body. However, they can cause pain or other problems, if they press against nose or blood vessels. They can also trigger the over production of hormones, as in endocrine system (3).

Example of benign tumor:

a) Adenomas: - Adenomas usually develop in glandular epithelial tissue booster the blind dealer epithelial tissue is the thin membrane that covers glance, and other structures in the body. Usually the add enormous occurs in the police in the colon, as fibro adenomas whichis the common form of benign breast tumors, hepatic add enormous, which occurs on the liver (4).

b) Fibroids: - Fibroids, or fibromas, are the benign tumors that can grow on the fibrous or connective tissue of any organ. Examples: Angiofibroma, which can appear as small as red bumps on the face. Dermatofibroma mass, which appears on the skin, often on the lower leg (5).

c) Hemangiomas: - Hemangiomas are the benign tumors that form when blood vessels grow excessively. They can appear as red as strawberry marks on the skin, or they can develop inside the body. They are often present at birth and disappeared during childhood. Hemangiomas do not usually need treatment but laser surgery and other options are available if they do not go away (6).

d) Lipomas: - Lipomas, are a form of soft tissue tumors and consist of Fat cells. They can appear at any stage, but often, Effect people from 40 to 60 years of age, according to American Academy of orthopedic surgeons. Most lipomas are small, painless, rubbery and soft to the touch and are usually movable by surgery (7).

2. Premalignant tumor: - In this tumor, the sales are not yet cancerous, but they have the potential to become malignant. Although that Tumor is not cancerous, but, it does need close monitoring, in case it changes.

Examples of pre malignant tumors:-

a) Actinic keratosis: - This keratosis is also known as solar keratosis. The growth of this tumour involves patches of crusty commerce Kelly and thick skin. It is more likely to affect fair skinned people. And also the sun exposure increases therisk to this keratosis. Sometimes, acting nick keratosis will transform into squamous cell carcinoma, so doctors usually recommend treating it (8).

b) Cervical dysplasia: - In cervical dysplasia, as change occurs in the cell that line the cervix. Usually, this dysplasia is identified using “pap smear test” of the cells, done by the doctor. Although the cells are not cancerous, but they may become malignant after 10 to 30 years,

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resulting in cervical dysplasia. A surgeon may remove the cells using freezing techniques or by taking a cone of tissuefrom the cervix (9).

c) **Metaplasia of the lung: -** The growth of this metaplasia occurs in the bronchi, the tubes that carry air into the lung.The lining of the bronchi contains glandular cells. In some people, including smokers, these can change and become squamous cells or the metaplasia of the cancer (10).

d) **Leukoplakia: -** Lego play gear causes take white patches to form in the mouth. Anyone with this typeof patch should see a doctor, if it does not go away within two weeks. They should also monitor the patches for change and quit smoking or chewing tobaccoif relevant. If a doctor believes that the patches could become cancerous, they may use a laser surgical scalpel to remove them (11).

3. Malignant tumors: - Malignant tumors are the most cancerous one they develop when cells grow uncontrollably. If the cell continues to grow and spread the disease, it can become life threatening. Since malignant tumors, can grow quickly and spread to other parts of the body this process of spreading is known as metastasis. Different types of malignant tumors originate in different types of cells (12).

Examples of malignant tumors include: -

a) **Carcinoma: -** These are the tumors formed from epithelial cells, which are present in the skin and thetissue that covers or lines the body organs. Carcinoma scan occur in the stomach, prostate, pancreas, long, liver, Cologne, or breast. They are the commonest type of malignant tumors (13).

b) **Sarcoma: -** These tumors usually begin in connective tissue, Such as cartilage, bones, fat andnerves. They originate in the cells outside the bone marrow.Most sarcomas are malignant.

c) **Germ cell tumor: -** These tumors develop in the cell that produces sperms eggs. They usually occur in the goodnight that is ovaries or testicles, but they may also appear in the brain, abdomen or chest.

d) **Blastoma: -** These tumors form from embryonic tissue or developing cells. Usually, the blastomas are much more common in children then in adults. They canlead to tumors in the brain, eye, or nervous system (14).

Cancer its beginning: -

The problem of the nature and possible treatment of malignant disease are studied in many laboratories and results are published in different journals. Cancer is characterized by the proliferation of cells that have been managed to invade Control, endogenous control mechanism. The cancer I grouped according to their organ or tissue of their origin, but, increasingly also based on molecule are characteristic of the respective cancer cells. Do you do the rapid advancements in technology, since the last decade, it is now possible to explain molecular markup of the respectivecells (15).

In the long run, the goal is to offer every cancer patient therapeutic regime or that is tailored to his individual. Cancer develops when body normal control mechanism stops working the old cells do not die an instead growth out of control forming knew abnormal cells. This extra cell may form a mass of tissue called a tumor. Cancer can start almost anywhere in the body, which is made up of trillions of cells. Normally, human cells grow and multiply through a process called cell division, to form new cells as the body needs them. Then cells grow old or become damaged, they die and new cells take their place. Cancer is the beginning of this malfunctioning (16).

Types of cancer in men and women: - Although, men and women have different anatomies, they share some similarities and types of cancer they develop. Colorectal cancer and lung cancer for example, are common cancer developed in both men and women. The most common cancer differs in each gender. Prostate cancer in breast cancer is the mostcommon in men and woman.

Table 1: - Types of cancer in men and women with Percentage.

S. No.	Types of cancer in men	Percentage (%)	Types of Cancer in Women	Percentage (%)
1	Mouth	10%	Mouth	6%
2	Trachea	13%	Thorax	20%
3	Lungs	9%	Esophagus	5%
4	Esophagus	7%	Stomach	3%
5	Stomach	7%	Digestive organ	7%

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6	Digestive organ	10%	Uterus	5%
7	Urine and Reproductive organ	11%	Cervix Canal	24%
8	Leukemia and Lymphoma	12%	Leukemia and Lymphoma	5%
9	Other	21%	Other	25%

Table 2: - Chemicals that trigger the growth of cancer.

S. No.	Name of cancer causing chemical	Affected Parts
1.	Bitumin (3, 4 benzopyrene)	Skin, Lungs
2.	Smoke	Skin, Lungs
3.	Mustard gas	Lungs
4.	Cigarette smoke (N-Nitrisodimethylene)	Lungs
5.	Cadmium oxide	Prostate gland
6.	Afatoxin	Liver
7.	BES (Diethyl stilbestrol)	Vagina
8.	Vinyl chloride	Liver
9.	Asbestos	Pleural membrane

Genetics of Cancer: -

Chromosomes, the thread like structures which contain genes. The 46 chromosomes are divided inpair of two in sets of 23. Genes and DNA of each cell in our body researchers estimate that each cell contains 30000 different genes. Within each cell genes are located on the chromosomes. The cell division process is dependent on a tightly control sequence of events. These events are dependent on the proper level of transcription and translation of certain genes. Of the 30,000 or so genes that currently exist in human genome, there is a small subset that seems to be particularly important in the progression, development and prevention of cancer. These genes have been found to be either malfunctioning or none functioning in many different kinds of cancer. These genes that have been identified to date have been categorized into two broad categories, depending on their normal functioning in the cell (17).

- Genes whose protein products stimulate or enhance the division and viability of cells. This first category also includes genes that contribute to tumor growth by inhibiting cell death.
- Genes whose protein products can directly or indirectly prevent cell division or lead to cell death.

The most commonly mutated gene with cancer is p53 Or TP53. More than 50% of cancers involved a missing or damaged P 5More than 50% of cancers involve a missing or damaged p53 gene. Most p53 gene mutations are acquired. Germline p53 mutations are rare, but patients who carry them are at a higher risk of developing many different types of cancer.

Oncogenes: -

The normal versions of genes in the first group are called proto-oncogenes. Many of these genes are responsible for providing the positive signals that lead to cell division. Some proto-oncogenes work to regulate cell death. The muted or otherwise damaged versions of these genes are called oncogenes which can cause a cell to divide in an unregulated manner. This growth can occur in the absence of normal pro growth signals such as those provided by growth factors. A key feature of oncogene activity is that a signal altered copy leads to unregulated growth. Different cancer types tend to depend on a limited number of driver oncogene mutations. These mutations are the main changes that make the cancer progress (18).

Numerous genes have been identified as proto-oncogenes. Many of these genes are responsible for providing the positive signals that lead to cell division. Some proto-oncogenes work to regulate cell death. And their absence/defectiveness can lead to the unregulated cell division. The proto- oncogene that have been identified so far have many different functions in cell. Despite the differences in their normal roles, these genes all contribute to unregulated cell division if they are present in oncogenic form that is mutant form. The proteins often retain some of their capabilities but are no longer sensitive to the controls that regulate the normal form of protein (19).

Different cancer types tend to depend on a limited number of ‘driver’ oncogene mutations.

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These mutations are the main changes that make the cancer progress. All cancers have lots of additional changes, the so called passenger mutations that may contribute to the cancer, but are not the maingenes.

Table 3: - Selected oncogenes that have been associated with numerous cancer types are described in moredetail below.

S. No.	Oncogenes	Function	Types of Cancer Caused	Ref.
1.	ABL1	Promotes cell growth through tyrose kinase activity	Chronic myelogenicleukemia.	(20)
2.	AFF4/MLLT11	Fusion affects the AFF4 transcription factor (methyltranferase)	Acute leukemia	(21)
3.	AKT2	Encodes a protein- serine / threonine kinase	Ovarian Cancer	(22)
4.	ALK	Encodes a receptor tyrosinekinase	Lymphoma	(23)
5.	ALK/NPM	Translocation creates fusion protein with nucleophosmin (npm)	Large cell lymphomas	(24)
6.	AXL	Encodes a receptor tyrosinekinase	Hematopoi cancer	(25)
7.	BCL-2, 3,6	Block Apoptosis	B-cell lymphomas leukemia	(26)
8.	BCR/ABL	New protein created by fusion if bcr and abl triggers unregulated cell growth	Chronic myelogenic and acute lymphotic leukemia	(27)
9.	CSF1R	Tyrosine kinase	Sarcoma	(28)
10.	DEK/NUP214	New protein created by fusion	Acute myelogenic leukemia	(29)
11.	EGFR	Cell surface receptor that triggered cell growth through tyrosine kinaset (11;19)	Myeloid leukemia	(30)
12.	ERBB2	Cell Surface receptors that triggers cell growth through tyrosine kinase activity, also known as capital HER2	Breast, Salivary gland and ovarian carcinomas	(31)
13.	ETS1	Transcription factor	Lymphomas	(32)
14.	EESR1/FLI1	Fusion protein created by the (11;22)	Ewing Sarcomas	(33)
15.	FES	Tyrosine kinase	Sarcoma	(34)
16.	FOS	Transcription factor for API	Osteosarcoma	(35)
17.	FGF4	Encodes fibroblast growthfactor; aka HST1	Breast ame squamous carcinomas	(36)
18.	GNAS (GSP)	Membrane associated G protein	Thyroid carcinoma	(37)

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19.	GLI1	Transcription factor	Glioblastoma	(38)
20.	HER/NEU	Overexpression of signaling kinase due to gene amplification	Breast and cervicalcarcinomas	(39)
21.	K-SAM	Fibroblast growth factor receptor	Stomach carcinomas	(40)
22.	LCK	Tyrosine kinase	T-cell lymphoma	(41)
23.	LMO1, LMO2	Transcription factor	T-cell lymphoma	(42)
24.	LYK1	Transcription factor	Acute T-cell lymphomas	(43)
25.	MAS1	Angiotensin receptor	Mammary carcinoma	(44)
26.	MDM2	Encodes a protein that inhibits and leads to the degradation of p53	Sarcomas	(45)
27.	MOS	Serine/threonine kinase	Lung cancer	(46)
28.	MYB	Transcription factor	Colon carcinoma andleukemia	(47)
29.	NEU	Tyrosine kinase, also calledERBB2 or HER	Glioblastoma and squamous cell carcinoma	(48)
30.	NOTCH1 (TAN1)	Altered form of notch		(49)
31.	PRAD1	Encodes cycling D1. Involved in cell cycle regulation	Breast and squamous cell carcinomas	(50)
32.	PBX1/E2A	Fusion protein formed via text(1:19) translocation, transcription factor	Acute pre B leukemia	(51)
33.	PAX-5	Transcription factor	Lympho-plasmacytoid cell lymphoma	(52)
34.	RAF1	Serine/threonine kinase	Many cancer types	(53)
35.	RARA/PML	Fusion protein caused by the(15:17) translocation, Retinoic acid receptor	Acute premyelocyticleukemia	(54)
36.	SRC	Tyrosine kinase	Sarcoma	(55)
37.	SET/CAN	Fusion protein formed by rearrangement of chromosome	Protein localization	(56)
38.	TIAM1	Guanine nucleotide exchange factor	T-lymphomas	(57)
39.	TSC2	GTP-ase activator	Renal and breast tumors	(58)
40.	TAL1, TAL2	Transcription factor	Acute T-cell leukemia	(59)

Tumor Suppressors:

Tumor suppressor gene is a gene that regulates a cell during a cell division and replication if the cell grows uncontrollably it will result in cancer. When a tumor suppressor gene is mutated, it results in a loss or reduction in its function. Suppressors function in many key cellular processes including the regulation of transcription, DNA repair and cell to cell communication.

The loss offunction of these genes leads to abnormal cellular behavior. Tumor suppressors can be preferred as breaking power to the cell. When both copies of a tumor suppressor gene are functioning, the cells can stop dividing. A single defective tumors suppressor will still leave the cell with one functioning copy.

The cells with a single defective version of a tumor suppressor can still control their cell

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division. When this gene is mutated, it results in a loss or reduction in its function. In combination with other genetic mutations, this could allow the cell to grow abnormally (60). When the second copy in the cell is lost, the cell loses the ability to prevent division. Tumor suppressor genes can be categorized into the following category:

- Caretaker genes ensures stability of genome via DNA repair and subsequently whenmutated allow mutations to accumulate.
- Gatekeeper genes directly regulate cell growth by either inhibiting cell cycle progression or inducing apoptosis.
- Landscaper genes regulate growth by contributing to the surrounding environment, when mutated can cause an environment that promotes unregulated proliferations.

Tumor Suppressors and the "2-hit hypothesis": -

Tumor Suppressor genes Tend to be recessive. We can understand this from the given example: Tumor suppressor genes BRCA1/ BRCA2 genes, otherwise known as breast cancer genes. People who have a mutation in one of these genes have an increased risk of developing breast cancer among the other cancers. However, not everyone with the gene develop breast cancer.

The first copy of these genes is mutated at birth, but it's not until another mutation occurs after birth, the mutation can be acquired mutation or somatic mutation, that abnormal repair proteins are madethat increase the risk of cancer (61).

This recessive nature is what refers to in the "2-hit hypothesis" of cancer. The first copy, as in the example above, the inherited copy of defective gene is the first hit, and a later mutation in the other copy of the gene later in life is the second hit. Together the two mutated copies of the tumors suppressor gene are unable to create effective proteins to repair the damage.

Tumors suppressor genes were first identified among children with retinoblastoma. In retinoblastoma, and contrast too many tumors suppressor genes, that tumor that is inherited is dominant and therefore allow cancers to develop in young children if one parent carries the mutated gene, then 50% of their children inherit the gene and be at risk for retinoblastoma (62).

Table 4: - Some of the tumor suppressors with the type of cancer and defectiveness caused, is given below: -

S.No.	Tumor Suppressors	Function	Type of Cancer	Ref.
1.	APC	Controls the function of specific transcription factors which are involved in tumerigenesis, and development and homeostasis of some cell types including epithelial and lymphoid cells. APC has also been implicated in cell proliferation and other cellular activities such as migration, and adhesion.	Familial adenomatousand non-inherited colorectal carcinoma	(63)
2.	BRCA1, BRAC2	DNA damage and repair	Inherited breast cancers, ovariancancers	(64)
3.	CDKN2A	Gene locus that encodes the tumor suppressors p16 and p14ARF	Brain Tumors, melanomas	(65)
4.	DCC	Netrin-1 receptor. Regulation of cell proliferation and apoptosis of intestinal epithelium	Colorectal carcinoma	(66)

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5.	DPC4	Transcription factor involved in development; Implicates in metastasis and tumor invasiveness	Colorectal tumors, pancreatic neoplasia	(67)
6.	NF1	RAS GTPase activating protein (RAS-GAP)	Neurofibromatosistype 1	(68)
7.	NF2	ERM Protein; plasma membrane by assembling protein complexes and linking them to actin	Neurofibromatosistype 2	(69)
8.	TP53 (p53)	Encodes a transcription factor for p21, a protein that arrests the cell cycle in G1 phase. p53 integrates signals related to cell size, DNA integrity and chromosome replication	Bladder, breasts, colorectal, esophageal, liver, lung, prostate, and ovarian carcinoma, brain tumors, lymphomas and Leukemia	(70)
9.	RB1	Binds to, and inhibits, the E2F transcription factor. Halts cell cycle progression	Retinoblastoma, sarcomas, bladder, prostate and lung carcinomas	(71)
10.	WT1	Transcription. Essential role in development	Wilms tumors (pediatric kidney cancer)	(72)

Treatments of cancer: -

1) SURGERY: - Surgery is an option for most cancers other than blood cancers, with specialized cancer surgeonsattempting to remove all or most of assault table. It is an especially effective treatment for early stages of cancer that has not spread to other parts of the body**.** As said by Marta Batus, MD, a medical oncologist at Rush while, "it depends on the size of thetumor and other factors, many patients with stage one cancers do not need any other treatment except for surgery and surgery can play a role in cancer treatment even when a tumor has spread beyond its original size (73). Another oncologist Busts says, "our option for treating cancer even at later stages have grown, and surgery is a big part of that. The role of surgery has expanded, and it is very encouraging."

Depending on the cancer and the stage, minimally invasive surgery may be an option. For example, thoracic surgeon at Rush often use video assisted thrills kosko pick surgery we eight years, a minimally invasive procedure, to remove early stage lung cancer tumors. VATS use smaller incisions than open surgery and typically offers patients less pain, shorter hospital stays after surgery and fewer complications (74).

2) IMMUNOTHERAPY: - Immunotherapy, a relatively newer type of cancer treatment, uses medications to Rev up the patient's own immune system to fight cancer. A minute therapy treatments can work across different cancer types a may be effective in treating even the most advanced and hard to treat cancers. Researchers continue to look into potential of immunotherapy but if several effective FDA approved drugs are not commonly used to treat certain cancers. Immunotherapy has definitely opened up more options for a lot of patients, and it is now the frontline treatment for certain patients, better says (75). Patients don't lose their hair. They don't have nausea or vomiting. Most of the time they experience minimal side effects, if any some patients, though, may experience the side effects, depending on the drug administered the type of cancer being treated. Immunotherapy administered through IV infusion. An immunotherapy gives some patients with late stage cancers are treatment option they previously didn't have, sometimes allowing them to live longer than they would have been expected to leave otherwise. For example, immunotherapy has redefined how doctors are treating Melanoma, the most dangerous and deadly form of skin cancer. Five years ago, overall survival for a patient diagnosed with metastatic Melanoma cancer that has spread to other parts of the body wasabout nine months. Today, thanks to combination of immune

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therapies the majority of patients with metastatic Melanoma are alive and doing well at least for one year later and many are living many years beyond that (76).

3) TARGETED THERAPIES: - Oncologists use targeted therapies, also known as precision medicine, to Taylor medications for each individual patient and cancer first stop a tumour or blood samples tested to identify a genetic profile. That allows clinicians to administer medications that target the gene that are causing the cancer. These could be five or six gene processes that turn a cancer on or off, birds says. With genetictesting, we can find out which medicines to use and which ones not to use. Medications, delivered in pill or Ivy form, either destroy cancer cells or stop the cancer from continuing to grow. Like immunotherapy, targeted therapies can be used at any stage that is as a first treatment, to keep the cancer from coming back or if the cancer returns (77).

For instance come up patients with breast cancer typically tested to see if they can carry the each year to gene, which can play a role in causing breast cancers cells to grow. If a patient tests positive for a cheer too, oncologist address user meditation or a combination of medications developed to target the gene, such as trastuzumab or pertuzumab. These medications help stop the growth of cancer cells, often without harming healthy cells. These days, a cancer diagnosis doesn't have to mean a chemo and the nausea vomiting and hairloss most people think of (78).

4) ACTIVE SURVEILLANCE: - Active surveillance also called watchful waiting may be all that's needed for certain types of cancers. Doctors may recommend this approach if the cancer isn't at early stage and is growing slowly or not at all. For example, doctors offer recommend active surveillance for prostate cancer, which tends to grow very slowly. Doctors monitor patient's prostate specific antigen PSA with blood tests an monitor symptoms. If or when symptoms worsen or test so the cancer is growing more rapidly, they then begin discussing additional treatments. Often, patients who are receiving active surveillance have no symptoms and go on living their lives as usual, beta's says. Surveillance may also be an option for patients who want to break from treatment side effects or for those who have exhausted all other treatment options (79).

5) SUPPORTIVE CARE: - Supportive cancer care can be effective complement to standard treatment, helping to minimize the physical and emotional stress of cancer treatment. For example, psychotherapy and massage therapy can help ease patient's anxiety as they cope with a diagnosis, acupuncture can be beneficial for pain relief, and nutrition counselling can keep a patient from losing too much weight during treatment and keeping the body as healthy as possible (80). Types of integrative medicine include the following: -

- Aromatherapy
- Fitness classes
- Hypnosis
- Mindfulness medication

Immune system after treatment: - Most cancer patients know that chemotherapy weakens their immune systems, quitting them at riskfor viral and bacterial infection. A month or 100 days after chemo ends full, however, most people assume their immune system has returned to normal. Now, new research suggests that effects of chemotherapy can compromise part of immune system for up to nine months after the treatment, leaving patients vulnerable to infection. The small observation study conducted at UK University of Leeds and Leeds teaching hospital NHtrust also demonstrated that smoking. This study has demonstrated that the adaptive immune system is altered following chemotherapyfor at least nine months post therapy, as written by the authors, published in breast Search journalblue stuff we were surprised that the impact of chemotherapy is so long lived (81).

Researchers looked at the immune system of breast cancer patients measuring their levels of lymphocytes that is white blood cells that work together to fight viral and bacterial infections. Before up to nine months, after receiving chemotherapy probably, the level of lymphocytes, whichinclude various types of natural killer cells, T cells and be sales, which dropped significantly after chemo, but the impact was shorter. Nine months later, most of the immune cells were up and running at pre chemo levels. When it comes to certain types of NK, T&B cells, however the researchers found that chemo had a long term effect. After nine months, B cells which is the most important for creating antibodies and CD4 plus T cells which are also known as helper T cells had only artificial recovery, revealing Only 69% of pre chemo levels respectively, potentially leaving patients vulnerable. In smokers, recovery was substantially and significantly impaired. The researchers wrote reaching only 51% of pre chemo levels after nine months (82).

Doctor Steve program: - Infectious disease

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researchers at Fred hutch men Cancer Research center and director of infection control at Seattle cancer care alliance said the study results were not totally surprising. The immune system is a host defense system it. It comprises much biological structure ranging from individual white blood cells to entire organs as well as many complex biological processes. The immune system responds to foreign antigens and cancer cells by activating specific and non specific immune responses knowledge of this influence of stress on immune system and cytokine response is evolving (83).

II. CONCLUSION

Cancer develops from the changes occurring in proto-oncogenes and tumor suppressor cells. The abnormality leads to rapid division of cells causing the formation of mass (lumps) of cells known as tumor in the body. The abnormality usually leads to spreading of tumor cells to various parts of body. Micro RNA is the RNA formed via various functioning of cell. They play an important role in gene regulating functioning. The most common treatment is surgery, which involves removal of tumor cells. Another method of treatment includes immunotherapy, active surveillance of patients. Usually there is weakness in immune system after the treatment of cancer which takes a period of time to recover.

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