

Oncogenes

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Introduction

For cells to function and grow properly, there is a sequence of deoxyribonucleic acid (DNA) found in genes that enables them to perform this function. Normal Genes contain codes that give cells instructions to make various proteins that have different body roles. These normal genes are called proto-oncogenes and are responsible for manufacturing proteins involved in cell growth, division, and differentiation. If mutation or change occurs in the proto-oncogene, the gene will be activated, which poses a problem because it can malfunction. The mutated gene is referred to as an oncogene. Once an oncogene develops, cells start growing out of control, and due to the uncontrollable growth of cells, Cancer creates. However, a proto-oncogene cannot cause Cancer unless a mutation occurs in it, turning the gene permanently into an oncogene. The errors or modifications that turn a proto-oncogene into an oncogene can be classified into at least three types: point mutation, which alters by inserting or deleting one or a few nucleotides this activating the proto-oncogene; gene amplification or duplication, which leads to the manufacturing of excess copies of the gene and chromosomal translocation or rearrangement whereby a gene is relocated to a new chromosomal site that leads to a fusion between a proto-oncogene and a second gene, in turn, producing a protein with oncogenic traits.

Research done by the American Cancer Society found out that most mutations that cause Cancer are acquired and not inherited. These mutations can result from radiation exposure and environmental toxins such as smoke and viral infections. Generally, Cancer is caused by a mutation in the proto-oncogenes and tumor suppressor genes, and DNA stability genes. Tumor suppressor genes are the normal genes that perform the function of slowing down cell division

repair the mistakes of DNA, and communicate to the cells when to die through apoptosis. Cancer can occur when the tumor suppressor cells do not work correctly, and cells to grow uncontrollably. Oncogenes result from proto-oncogenes' activation, hence causing Cancer, while tumor suppressor gene causes Cancer when they are inactivated. DNA stability causes Cancer when they lose their normal function during cell division and become mutated.

Oncogenes and Proto-oncogenes

Oncogenes

According to comprehensive toxicology 2010, an oncogene is a gene that encodes a protein capable of transforming cells in culture or inducing Cancer in animals. In simple terms, an oncogene is a gene whose expression causes cells to exhibit cancer cells' properties. The term oncogene was coined by R. Huebner and G. Todaro in 1969. The first oncogenes were discovered through the research of RNA tumor viruses. The oncogene was initially thought to be a viral gene that could transform normal cells into malignant cells in vertical transmission from a parent to its offspring.

Oncogene's Discovery began when Peyton Rous demonstrated that a filterable virus could cause fibrosarcomas in chickens in 1910. This virus was later named the Rous sarcoma virus (RSV). His theory was further investigated by Howard Temin and Harry Rubin in 1958 that developed various techniques for identifying the gene responsible for causing tumors. The first cellular proto-oncogene was discovered in 1976 by Michel Bishop and Dominique Stehelin. They found that the sarcoma gene was not viral in origin but rather integrated into a viral genome during transduction or rather arose from the preexisted cellular gene. This proved that it

was the normal cells that are activated and were thus the origin of the oncogenes of retroviruses. This formed the basis for the cellular origin of retroviral oncogenes. They called it the very.

However, vertically transmitted RNA tumor viruses do not transmit any oncogenes. They can only cause leukemia and mammary tumors in vivo when the hosts are psychologically and genetically susceptible. Retroviruses are oncogenic RNA viruses that contain reverse transcriptase. The Discovery of oncogenes laid a basic understanding of Cancer's molecular and genetic origin as studies of tumor viruses revealed that oncogenes could induce cell transformation.

The protein composition of oncogenesis responsible for regulating cell proliferation, growth, differentiation, control of the cell cycle, and apoptosis. This, in turn, produces product oncogenes of growth factors. It should be noted that the majority of human cancers are not caused by viruses and instead rise from other causes like radiation and the presence of chemical carcinogens. Some oncogenes that were discovered include the Avian myelocytomatosis virus (MYC), rat sarcoma virus (RAT), avian erythroblastosis virus (ERBBI), and Abelson murine leukemia virus (ABI). These viruses were named after the virus they were first discovered in.

Proto-Oncogenes

Proto-oncogenes are also known as cellular oncogenes. Proto-oncogenes are usually expressed during growth, such as wound healing and regeneration of a damaged liver and cell mitosis. They are responsible for growth factors, growth factor receptors using tyrosine kinase activities, signal transduction to regulate proteins, and transcription factors. The combination of cellular proto-oncogenes with the genome of non-transforming retroviruses leads to the rise of

viral oncogenes. Other mechanisms such as point mutations and DNA rearrangement in the form of translocation and gene amplification can lead to Cancer causing oncogenes.

Once a proto-oncogene is activated, it turns into a tumor-inducing agent. Examples of proto-oncogenes include RAS, which is used for encoding intracellular transduction protein. If mutated, it can cause uncontrolled cell growth and has been associated with pancreatic Cancer, MYC associated with a type of Cancer called Burkitt's lymphoma. It occurs during chromosomal translocation and Cyclin D, which is activated due to mutation and is associated with the parathyroid gland's tumors. Translocation of pieces from chromosomes causes the BCR-ABL oncogene that causes myelogenous leukemia. However, it should be noted that proto-oncogenes do not cause Cancer unless a mutation occurs to turn them into oncogenes. The turning on permanently of a proto-oncogene is referred to as gain of function of transformation. Proto-oncogenes are necessary for the survival of the body.

The gain of function mutations can be made through point mutation, altering, inserting, or deleting one or a few nucleotides in a gene sequence, leading to a proto-oncogene activation. Extra copies of a gene through gene amplification can also lead to a function mutation in a cellular proto-oncogene. The last gain of function mutation in proto-oncogenes is chromosomal translocation, whereby a gene is relocated to a new chromosomal site. This relocation leads to a higher expression of cells.

Brief History of Cancer

Origin

Cancer occurs when cells in a part of the body begin to grow out of control. The origin of the word Cancer can be traced back to Hippocrates, a Greek physician between 460 and 370 BC

and is considered the father of medicine. He used the terms carcinomas and carcinoma, a Greek name for the crab, to describe non-ulcer forming and ulcer forming tumors. Celsus, a Roman physician, translated the word to Latin, and Galen referred to it as once a Greek word for swelling. The earliest evidence of Cancer can be found in human mummies' bones in ancient Egypt and ancient manuscripts. The oldest description of Cancer dates back to 3000BC, where an ancient Egyptian textbook on trauma surgery describes 8 cases of breast tumors removed through the process of cauterization using a fire drill. The book also stated that there's no treatment for the disease (DeVita and Chu, 2008).

Cancer in the sixteenth to the eighteenth century

The renaissance period saw scientists developing a more understanding of the human body using scientific methods to study diseases. Such scientific methods included autopsies used to study the relationship between a patient's illness and death (DeVita and Chu, 2008). Giovanni Morgagni is credited for using autopsies in 1761, and this laid the foundation for the study of Cancer or rather scientific oncology. Surgeon John hunter 1728 to 1793 described how some cancers could be operated on to cure them. Classic cancer operations such as radical mastectomy and anesthesia were developed a century later to help cancer treatment.

Cancer in the nineteenth century

The nineteenth-century provided a basis for the modern pathological study of Cancer through the use of the microscope to study diseased tissues. A correlation between microscopic pathology and illness was made by Rudolf Virchow, the founder of cellular pathology. This ensured that proper diagnosis could be made and also helped in cancer surgery.

Early Cancer causes theories.

Early physicians puzzled over the causes of Cancer and proposed some theories to explain the reasons. Such approaches included the humoral theory, nymph theory, blastema theory, chronic irritation theory, trauma theory, and infectious disease theory. The humoral theory's leading proponent was Hippocrates, who believed that the body had four humors or body fluids: blood, phlegm, bile, and black bile. If the four were balanced, a person was healthy, whereas the excess of black bile was thought to cause Cancer.

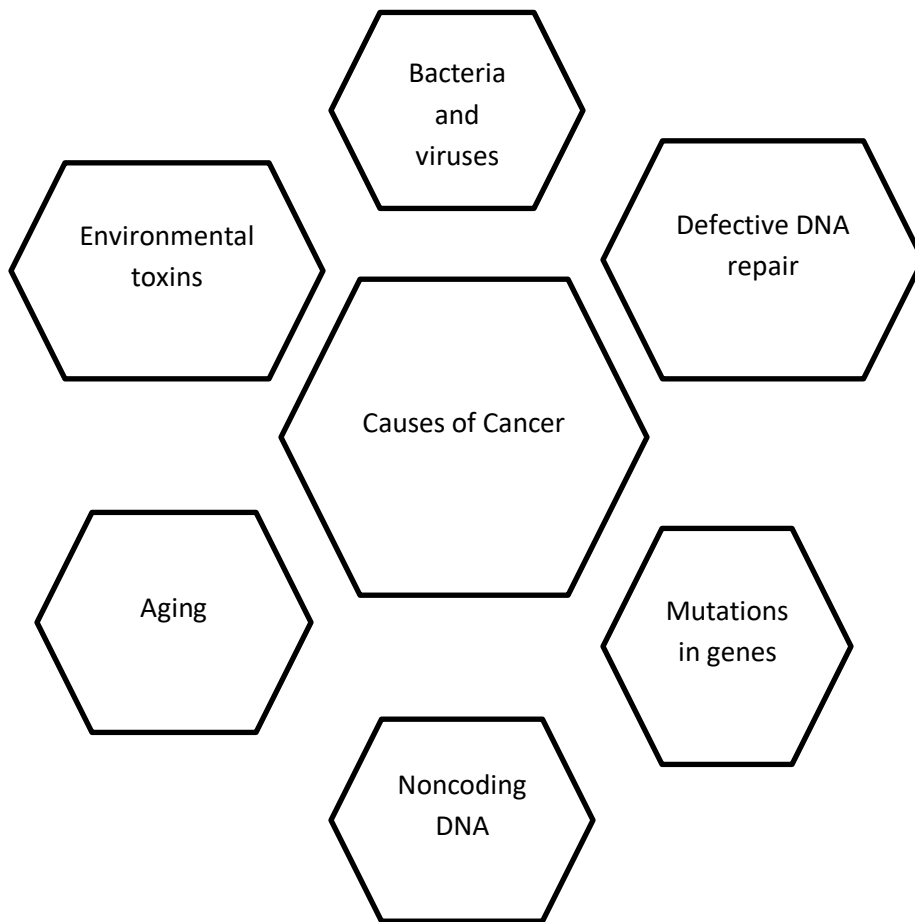
According to DeVita and Chu (2008), the lymph theory argued that Cancer was composed of fermenting and degenerating lymph, which varied in density, acidity, and alkalinity. The blastema theory proposed by German pathologist Johannes Muller believed that cancer cells did not come from normal cells but developed from budding elements between the normal tissues. On the other hand, chronic irritation theory thought that Cancer developed through the spread of malignant cells. Trauma theory believed that specific injuries caused Cancer. Other people thought that Cancer was contagious through the infectious disease theory.

Modern knowledge about the causes of Cancer

Viral and chemical carcinogens: carcinogens are substances that are believed to cause Cancer in humans. They are found in tobacco, benzene, hydrocarbons, and radiation (DeVita and Chu, 2008). Viruses that can cause Cancer in humans include infections with hepatitis B, can cause Cancer of the liver, herpes viruses lead to nasopharyngeal Cancer. People who have HIV are vulnerable to developing Cancer, especially Cancer of the skin called Kaposi sarcoma. Human papillomaviruses are also responsible for causing cancers of the cervix, vagina, vulva, and penis. Research by the world health organization's international agency for research on Cancer identified several chemicals, physical and biological carcinogens responsible for causing Cancer.

Oncogenes and tumor suppressor genes were discovered to cause Cancer in the 1970s. There is also Cancer caused by heredity though it makes a small percentage as compared to Cancer that develops in a person's lifetime. More research is being done on the causes of Cancer and how it can be treated.

Figure1. Causes of Cancer



How Oncogenes and Tumor Suppressor Genes can lead to Cancer

Oncogenes and tumor suppressor genes have been attributed to be the significant causes of Cancer. This is because Cancer is caused by genetic changes such as the activation of proto-oncogenes or inactivation of tumor suppressor genes, which can lead to progression in tumorigenesis. For example, gene amplification in N-*myc* is associated with the passage of human neuroblastoma. Therefore, it can be said that Cancer is caused by defective tumor suppressor genes (TSGs) and hyperactive oncogenes (OCGs) because they are responsible for cell proliferation and apoptosis through generic development like deletions. Oncogenes and tumor suppressor genes lead to Cancer through the loss of function or gain of function. (Weinberg, 1994).

How oncogenes lead to Cancer

Oncogenes lead to Cancer from the activation of proto-oncogenes. For example, prostate cancer is caused by the fusion of the TMPR₅₅₂ gene and other two transcription factors such as ETV₁ that increase proliferation and inhibit the death of cells, which leads them to transform into cancer cells. To understand how oncogenes lead to Cancer, the categories of oncogene factors will be discussed. This includes:

- Growth factors
- Growth factor receptors
- Protein kinases
- Signal transducers
- Nuclear oncogenes
- Transcription factors

Growth factors

This leads to activation of oncogenes, for instance, in peptide derived growth factor. PDGF stimulates the division of fibroblasts during the wound healing process. It has a *thesis* proto-oncogene that codes for the growth of platelets (Weinberg, 1994). If a simian sarcoma gene is illegally activated during this process, the oncogene action will lead to uncontrolled cell growth, leading to Cancer.

Growth factor receptors

This constitutes the *erbB* oncogene, and it's used for coding the epidermal growth factor receptors. The receptors play an essential role in regulating cell growth, especially in the hemopoietic system. Cancer occurs when a normal receptor gene is pathologically activated by structural change, leading to continuous cell division.

Protein kinases

The *Src* is an example of an oncogene that belongs to the protein kinase category. If oncogenic activities occur in the genes, a high activity will take place in the plasma membrane. High activity automatically translates to more expression of cells, which causes Cancer.

Signal transducers

The *ras* oncogene is used in transducing the signal from the membrane receptor to the cell interior. If point mutation occurs, then the proteins will be impaired and thus hinder their normal function.

Nuclear oncogenes

Examples of nuclear oncogenes are *my* and *my* and are responsible for coding for DNA phosphoproteins. This can stimulate DNA replication and activate cells, leading to them not proliferating by preventing terminal differentiation.

Transcription factors

Transcription oncogenes can contribute to cancer development due to constitutional activation and structural changes that prevent them from following regulatory signals.

Tumor suppressor genes

Tumor suppressor genes act as negative growth regulators. They usually modulate proliferation by alienating the biochemical functions of proto-oncogenes and responding to unchecked growth signals. Tumor suppressor genes cause Cancer when a recessive gene is formed due to inactivation of both copies of tumor-suppressing genes, thus losing the function of suppressing malignant transformation. Examples of tumor suppressor genes include; BRCA1, BRCA2, and p53 (Weinberg, 1994).

Often, a tumor suppressor gene is lost through somatic homozygosity whereby one chromosome of a pair is lost, and deletion occurs in the remaining chromosome. Thus the chromosome with deletion replicates, leading to the formation of a tumor. Failure to respond to restrictive growth signals also leads to Cancer when inactive tumor suppressor genes fail to restrict cell division, thus allowing cells to escape the extracellular anti-proliferative signals.

Inability to apoptosis: cell death originates from either the cell membrane or the mitochondria. If the cells are deficient, there will be reduced apoptosis levels since the tumor suppressor gene *p53* is not enough, and it acts as a modulator of oncogene-induced apoptosis.

According to Weinberg (1994), DNA tumor viruses activate cell replication of the cell machinery by blocking key tumor suppressors' action. A good example is the papillomavirus causes human warts and is also known to cause uterine and cervix carcinomas. To cause Cancer, this virus integrates specific viral replication genes into a host chromosome.

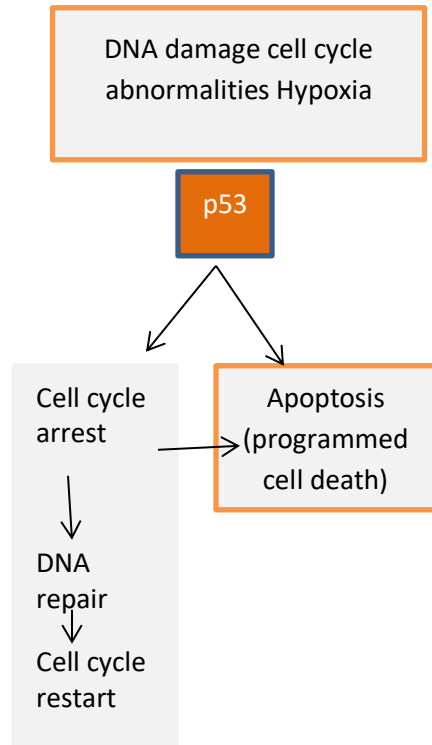
Tumors are usually named depending on their cells of origin, for example:

- Sarcomas refer to cancer abnormalities that occur in the muscle, bone, fats, and connective tissues.
- Carcinomas are used to refer to Cancer that arises from epithelial cells.
- Leukemia is a tumor that originates from cancerous white blood cells.
- Lymphoma is used to denote the malignancy of cells from the bone marrow.
- Myelomas, on the other hand, refer to cancers of white blood cells that synthesize antibodies.

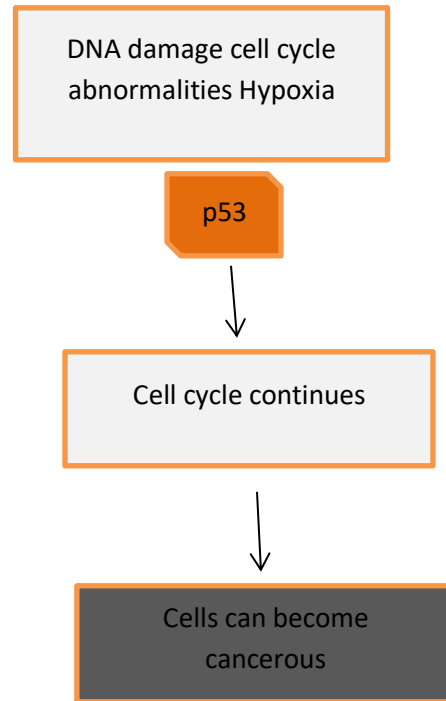
Figure 2: tumor suppressor genes

Normal p53	Mutated p53
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When cellular damage occurs, p53 arrests the cell cycle until the damage is repaired. If damage cannot be repaired, apoptosis occurs.



Mutated p53 does not arrest the cell cycle. The damaged section continues to divide, which may result in Cancer.



Modes of Activation

Some modes of activation of oncogenes include Gene amplification, translocation, and point mutations in coding sequences.

Gene amplification

Gene amplification is the increase in the copy number in a specific chromosomal region. It can also be referred to as the expansion in the copy of a gene's number within a cell's genome. This is usually due to overexpression of the amplified genes, and this is one of the characteristics of Cancer. Amplification can be in the form of defects in DNA replication. This defective replication leads to the rise of karyotypic abnormalities referred to as double minute chromosomes (Thorpe et al., 2015). Gene amplification is used in the average growth and survival of cells, but when activated, proto-oncogenes' amplification leads to tumor progression. The amplification of proto-oncogenes contributes to the development of a malignant phenotype. An example of an amplified proto-oncogene is the ERBB2, which results in ovarian and breast cancers. Gene amplifications help medical practitioners to understand how genetically unstable tumor cells can be due to the alteration of a genome,

Chromosomal translocation

According to Thorpe et al. (2015), chromosomal translation usually occurs when a chromosome segment is transferred to a new site in the same chromosome. This generates cells with odd pairing. Some translocations activate oncogenes that are responsible for malignant transformation. The rearrangement of chromosomes can result in the fusion of a proto-oncogene to regulatory regions, leading to increased expression resulting in an oncogene. An example is chronic myelogenous leukemia that results from the fusion of a protein called the Philadelphia

chromosome. This chromosome comes about through the chromosome's translocation containing an ABL gene on chromosome 9 and another chromosome containing the BCR gene called chromosome 22. The fusion product of ABL-BCR is the Philadelphia chromosome. These oncogenic fusion genes cause cancers such as sarcomas, prostate cancer, and hematological cancers.

Point mutations

Point mutations usually occur when transformation occurs in DNA or RNA cell material whereby a single nucleotide is added, deleted, or altered. The cell sequence of these nucleotides is responsible for encoding the information for carrying out cell processes. Point mutations can occur through substitution whereby a pair of nucleotides is substituted with another, insertion and deletion whereby in insertion an extra pair of nucleotides is added to the base sequence while in deletion, base pair is deleted from the arrangement (Weinberg, 1994). An example of where point mutation takes place is the RAS family. For instance, Ras proteins usually contain two states; the inactive form bound to the guanine nucleotide and the active form, which is generally inactivated by activating proteins. Ras mutations interfere with the process and produce functional oncoproteins that trigger signaling pathways for continuous cell growth. Point mutations cause pancreatic Cancer, and activation of point mutations in the epidermal growth factors leads to lung cancer.

Types of Oncogenes

The types of oncogenes include; growth factors, growth factor receptors, signal transduction proteins, non-receptor protein kinases, transcription factors, cell cycle control proteins, and apoptosis regulators.

Growth factors

There are different types of oncogenes, such as growth factors or mitogens responsible for cell growth, proliferation, and differentiation and are in the form of proteins. The proteins can induce abnormal proliferation in cells, thus leading to an abnormal oncogene. An example is the proto-oncogene SIS, which results in the overproduction of the platelet-derived growth factor when they are mutated. Platelet-derived growth factor is present in many cancers, particularly in bone cancer or osteosarcoma.

Growth factor receptors

Another type is the receptor tyrosine kinases such as epidermal growth factor receptor (EGFR), usually found in lung cancers, platelet-derived growth receptor, vascular endothelial growth factor receptor, and human epidermal growth receptor. Another example is the HER2 oncogene that increases the number of HER2 proteins rapidly on breast cancer cells' surface.

Signal transduction proteins

These oncogenes affect the proteins involved in transmitting signals from the receptor of a cell to the nucleus. Examples of such oncogenes include the RAS family, which comprises KRAS, HRAS, and NRAS. Another example is BRAF melanoma.

Non-Receptor protein kinases

They are used for carrying signals from the receptor of a cell to the nucleus. These oncogenes come about when the proteins produced by this gene of a tyrosine kinase are continually produced, which leads to continual cell growth and division. An example is the Bcr-Abl that is involved in causing chronic myelogenous leukemia.

Transcription factors

These are proteins that regulate the movement and progression of cells during the cell cycle. An example is the Myc gene that is excessively active in some cancers such as lymphomas and leukemia.

Regulators of apoptosis

Oncogenes sometimes produce oncoproteins that reduce programmed cell death, leading to a prolonged life cycle of cells. For example, the BCL-2 oncogene has a protein associated with the cell membrane that hinders apoptosis.

Cell cycle control proteins

These are proteins that affect the cell cycle in different ways. For example, Cyclin D1 and Cyclin E1 work towards progressing through precise phases of the cell cycle.

Roles of Heredity and Acquired Mutation

Hereditary gene mutations can be called germline, while acquired mutations can be referred to as somatic. Somatic mutations are the mutations that are developed in cancer formation, meaning they are not present at birth. They cannot be transmitted from a parent to her offspring and are only present in cells affected by Cancer. On the other hand, heredity mutations are inherited from either parent, and they increase the chances of a person to develop Cancer (Jass et al., 1994).

Heredity/ germline mutations

Germline mutations are rare, and they usually occur in a sperm or egg cell and are passed directly from a parent to a child during conception. This means that as the embryo develops into a baby, the mutation that already occurred in the initial sperm or egg cell will be copied into every cell in the body. This causes a type of Cancer called Inherited Cancer, and it accounts for 5 to 20% of all cancers.

Acquired mutations

Acquired mutations account for almost 80% of all cancers. They mostly occur from damaged genes and cells during a person's lifetime (Jass et al., 1994). Let's take the example of a breast .cell; if it divides many times, it can form a tumor, an abnormal mass. Cancer that develops due to acquired mutations is known as sporadic Cancer. It should be clear that acquired mutations are not found in every cell of the body and are not transmissible from a parent to a child. Some factors that cause acquired mutations include tobacco, radiation, viruses, and age.

So how do mutations relate to Cancer? In most instances, a single transformation does not cause Cancer, but it occurs from multiple mutations over a long period. This can explain why Cancer affects older adults as they have had many opportunities for the building up of modifications. The genes linked to Cancer include tumor suppressor genes, which perform the function of limiting cell growth by monitoring cell division, repairing mismatched DNA, and controlling cell death. If a tumor suppressor gene mutates, cells will grow uncontrollably, leading to the formation of tumors.

Heredity mutations in the tumor suppressor genes of BRCA1 or BRCA2 genes increase the risk of a woman developing hereditary breast and ovarian cancers. In contrast, these genes increase a man's risk of developing pancreatic Cancer and melanoma in both. P53 or TP53 genes are mostly acquired while germline p53 mutations are rare, but it should be noted that patients who carry them are more vulnerable to developing different types of cancers (Jass et al., 1994).

Oncogenes turn healthy cells into cancerous cells. However, these gene mutations are not inherited. Oncogenes such as HER2, which is a specialized protein used for controlling the growth and spread of Cancer has been known to be in cancer cells, for instance, in ovarian and breast cancer cells. Another oncogene is the RAS family involved in the manufacture of proteins responsible for cell communication pathways, growth, and cell death.

According to Jass et al. (1994), other mutations can be in the form of DNA repair genes, whereby if a person has an error in a DNA repair gene. If these remain uncorrected, they may turn into mutations that may eventually lead to Cancer, especially the transformations in oncogenes and tumor repressing genes. Mutations that occur in DNA repair genes can be both be

inherited or acquired. An example of the inherited repair gene is lynched syndrome. Gene mutations occur through average growth, whereby an error may occur when cells divide.

Types of Cancer Treatment

There are different types of cancer treatment, and each is unique in the kind of Cancer, locality of the tumor, and progression stage. According to the world health organization, Cancer treatment is a series of interventions aimed at curing the disease and prolonging a patient's life while improving the patient's quality of life. Clinically, Cancer can be defined as a large group of malignant diseases (Miller et al., 2019).

Characteristics of Cancer

- Abnormal cell proliferation caused by either genetic or cellular translocations
- Uncontrollable growth of cells and division that leads to the formation of tumors and interference to the tissues that is in proximity to the tumor cells
- The ability to metastasize or spread to other areas and form secondary tumors
- Another characteristic of cancer cells is that they are capable of involving any tissue in the body and
- They evade apoptosis

Cancer treatment types include:

- Surgery
- Radiation therapy
- Chemotherapy
- Hormone therapy

- Targeted therapy
- Psychosocial support

Surgery

Surgery is a procedure in which the tumor is removed from the body. Surgery is usually preferred because it causes minimum damage to the surrounding tissues compared to other treatment methods such as chemotherapy. Surgery also ensures that the tumor can be safely removed without risking tissue damage (Miller et al., 2019). Surgery can be either open or minimally invasive depending on factors such as a patient's preference whereby the patients pick an option that they feel is good for them, the part of the body where the surgery is to be performed, and the size of the tumor to be removed.

In open surgery, a large cut is made to remove a tumor with other healthy tissues along with some lymph nodes. On the other hand, minimally invasive surgery is done by making small cuts instead of massive cuts. Then, with a camera and laparoscope, the tumor is removed with a small amount of healthy tissue. Surgery can be used to obtain specimens for cytopathology, and it can also be used as a palliative measure to get rid of or lessen intolerable symptoms.

Radiation therapy

This is a type of treatment whereby energy is directed at a specific target in the body. Radiation therapy can be done after surgery to prevent the recurrence of a tumor. It can be used together with chemotherapy because using only radiation can cause damage to the bone marrow. It is therefore used together with chemotherapy in the process of chemoradiation and can also be combined with a radioisotope to be administered as radioimmunotherapy. From Miller et al. (2019), ionizing radiation is used to charge electrically upon incidence of biological bodies, thus

killing cancer cells or altering them. The criterion behind radiation is that damaged DNA cells are unable to replicate, hence stopping cell division, causing cells to die.

Chemotherapy

Chemotherapy is a form of systematic therapy whereby drugs are distributed throughout the body by the bloodstream. Chemotherapy is usually used in combination with other cancer treatment methods. Chemotherapy usually works by having a tumoricidal effect on the hormone-sensitive by reducing or blocking the hormone's source (Miller et al., 2019). Chemotherapy stops tumor progression by killing the ability of cells to divide, thus leading to apoptosis. Since chemotherapy affects normal cells, it can sometimes lead to side effects such as hair loss. Some drugs used in chemotherapy include alkylating agents used to treat leukemia and several cancers of the breast, ovary, and lungs. Others have antimetabolites used to treat cancers of the ovary and intestinal tract, anthracyclines, mitotic inhibitors used to treat Cancer of the lungs, myelomas, lymphoma, and hormone chemotherapeutic drugs.

Hormone therapy

Hormone therapy is used to slow down cancers that use hormones to grow, such as breast and prostate cancers. This treatment is effective in treating Cancer without any cytotoxicity. Some hormone therapy drugs include corticosteroids, which give relief from nausea and vomiting after chemotherapy. They can also be used before chemotherapy to mitigate hypersensitivity during treatment (Miller et al., 2019).

Targeted therapy

These are a systematic treatment that works by modifying the immune defenses of the patient. They are usually specific and only target single receptors on the surface of tumor cells or the section's enzymes. However, this treatment may cause tumor regression and stimulate hematopoiesis.

Psychosocial support

Psychosocial support should be offered to cancer patients to provide the patients and their families' emotional support and more information on Cancer, communicating to patients sensitively, referring patients to peer groups such as cancer survivor groups. These enable patients to experience less anxiety and depression and can live a productive life after treatment.

Other cancer treatment strategies include antiangiogenesis inhibitors, whereby chemical inhibitors are used to cut off blood supply to the tumor cells. Examples include thalidomide, bevacizumab, and cilengitide, and these inhibitors are usually used with chemotherapeutic drugs to make them more efficient. According to Miller et al. (2019), different classes of inhibitors such as the selective androgen receptor modulators work by block binding the site for testosterone, hence slowing down testosterone growth modulated cancers angiogenesis inhibitors that prevent the growth of tumor cells.

Another treatment is the use of stem cells in cancer treatment. Stem cells are undifferentiated cells found in the bone marrow, and they can differentiate into any type of body cell. These cells can be used to replace abnormal ones.

Conclusion

Cancer is a fast-growing disease in the world. It is the second killer disease in America. Considering the severe effects of Cancer, it is better to learn more about the condition to understand its works. Through this, the general population can know how they can prevent it, measures to take in case one develops it, and the available treatment options. Through the study of Cancer, it has led to the development of molecular techniques such as gene identification, and this, in turn, has helped in better comprehension of genetic pathways that are responsible for tumor proliferation (Miller et al., 2019). The study of oncogenes has led to better diagnosis and prognosis of different types of tumors. In the course of carcinogenesis, genetic abnormalities have been characterized well, leading to better patient management. Understanding oncogenes and proto-oncogenes can lead to the development of therapeutic interventions in the future that will be able to reverse the significant biological interventions resulting from molecular mutation. Early detection programs should also be put in place so that treatment can be started earlier, and palliative care should be administered to patients whose Cancer has progressed. Early detection programs have been put in place in the United States of America; there are screening methods for cervical Cancer, mammography for breast cancer, and a fecal occult blood test for colon cancer. This will help to manage and cure different types of Cancer.

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