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Emerging Therapy For Cancer

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Abstract: Globally, cancer is leading cause for death with the help of traditional treatment like surgery, radiation and chemotherapy gives limited effectiveness nowadays for advanced types of cancer. New advancements in cancer treatment are novel therapies that overcome resistance of conventional drugs and reduce side effects. This review includes therapies like immunotherapy, gene therapy, hormone therapy, epigenetic therapy, nanotechnology, hyperthermia, cold plasma therapy, photodynamic therapy, etc. Nanomedicine is also used to delivering drug directly to tumor with less toxicity and increase effectiveness of treatment. To develop a cancer vaccine is new approached for pharmaceutical industries to prevent and treat cancer by enhancing immune cell capacity to recognize and kill cancer cells.

Index Terms **- emerging, therapies, cancer, cancer therapy, oncology, immunotherapy, chemotherapy**

I. INTRODUCTION

Genetic or epigenetic changes in somatic cells cause cancer, a condition characterized by aberrant cell development that can spread to other body areas. [1] One of the most deadly illnesses in the world, and particularly in western nations, is cancer. The International Cancer Observatory estimates that 9.9 million individuals lost their lives to cancer in 2020. [2] In this day and age, is a significant issue? Researchers are always looking for additional elements that contribute to the carcinogenesis process. [3] Cancer is a disease characterized by aberrant cell proliferation that has the capacity to spread to other body parts. Is the world's leading causes of death? [4] In 2020, cancer will be responsible for about 10 million fatalities. Nearly 9.6 million individuals lost their lives to cancer in 2018. [5] Many people around the world suffer from cancer. [6] After cardiovascular illnesses, it is the second leading cause of death. Malignant transformation, cancer start, tumor growth, and prognosis are all significantly influenced by both genetic and epigenetic processes. [7] Women are more likely to develop cervical, thyroid, colorectal, and lung cancers than men, who are more likely to develop colorectal, lung, prostate, liver, and stomach cancers. [8] Cancer may arise from a variety of endogenous (like age or genetic predisposition), exogenous (like inherited mutations, hormones, and immune conditions), or acquired (like environment, lifestyle, nutrition, tobacco, diet, obesity, exposure to sun, radiation, chemicals, and infectious organisms) factors. [9] Malignant tumors continue to be the largest cause of death today, despite enormous advancements in screening and treatment options. The declining death rates from coronary heart disease and stroke help to explain some of the rising contribution of cancer to the global death rate. [10] In the United States, primary CNS tumors make up only 4% of all newly diagnosed cancer cases. [11] The World Health Organization reports that cancer killed 9.6 million people in 2018, making it the second worst cause of death worldwide. [12] Cancer accounts for 7.6 million deaths worldwide, or 13% of all fatalities. [13] Most startling discoveries and prevalent signs of a stem cell origin for cancer is heterogeneity. Numerous mixed tumors, such as breast, lung, and testicular malignancies, exhibit heterogeneity. We are aware that prostate and kidney malignancies exhibit heterogeneity in a variety of tumor

subtypes. [14] Epigenetics is the study of various heritable and reversible changes in gene expression patterns that are not caused by changes in the DNA sequence. [15] New approaches to managing the disease are being investigated as the global incidence of cancer keeps rising. As a multifactorial disease, cancer can develop as a result of a number of variables, including hormones, radiation exposure, and dietary and lifestyle choices. [16]. In addition to being the primary objectives for primary prevention, lifestyle behaviours like smoking, drinking, and eating habits are thought to be important contributing factors in the Etiology of cancer. [17] Globally, cancer represents a significant burden of disease. Worldwide, tens of millions of individuals receive a cancer diagnosis each year, and over half of those patients pass away as a result. Although there have been notable advancements in the treatment of cancer, the disease's incidence and fatality rates remain high. [18] Every year, head and neck cancer claims the lives of about 300,000 people and affects approximately 750,000 individuals. Although survival rates for aggressive locally advanced head and neck conditions have somewhat improved due to advancements in first-line surgical treatment, especially in affluent nations, these rates remain low. Patients and the healthcare system continue to face serious challenges due to recurrent and metastatic disease. [19] Ninety percent of liver cancer cases are hepatocellular carcinoma (HCC), the most common basic form of the disease. [20] It is regarded as the sixth most prevalent cancer globally, and its prevalence has been steadily rising. With a 5-year survival rate of only 3%, HCC is also the third most common cause of cancer-related death. [21] As a result, it is regarded globally as a serious public health issue. [22] In its broadest definition, cancer encompasses around 277 distinct forms of the disease. Different stages of cancer have been identified by scientists, suggesting that multiple gene alterations have a role in the Etiology of cancer. [23] In fact, cancer has become more common overall; by 2014, there were over 1,665,540 cases of cancer in the United States alone, and 585,720 of those cases resulted in death. [24] The colon and rectum, prostate, lung and bronchus, and bladder are the common type of cancer in humans. The breast, lung and bronchus, colon and rectum, uterine corpus, and thyroid are the areas in women where cancer is most common. According to this data, a significant percentage of cancers in men and women are caused by prostate and breast cancer, respectively. [25] Cancers of the brain and lymph nodes, respectively, and blood cancer account for the largest percentage of cancers in children. [26] To remove the majority of the tumor mass, chemotherapeutic drugs and ionizing radiation have been used in primary cancer treatments for decades. The incidence of tumor relapse is a serious issue that arises from the development of drug resistance mechanisms in a section of the tumor cell, even if many of these therapies have provided major benefits and some cures. [27] A well-established cornerstone of cancer treatment, immunotherapy has improved the prognosis for several patients with a wide range of solid and haematological cancers. [28] Numerous new treatments are being developed to treat cancer. Which encompass. James P. Allison's does the discovery of cytotoxic T-lymphocyte associated protein (CTLA-4) and Tasuku Honjo's does the discovery of programmed cell death protein 1 / programmed cell death protein ligand 1 (PD-1 / PD-L1) earned them the 2018 Nobel Prize in Physiology or Medicine, recognizing the significance of immunotherapy. [29] Following the significant progress in the creation and improvement of chemotherapies and radiotherapies, cancer immunotherapy offers a significant advancement in the treatment of cancer. [30] Chemicals have been used to treat cancer for many hundred years, but systemic chemotherapy was not used successfully and extensively until the 1940s. Chemotherapy for cancer refers to the use of cytotoxic chemicals, or substances that have the ability to kill cells, in an effort to either completely remove the tumor or at least lessen its burden, which may lessen the symptoms associated with the tumor and possibly extend its life. [31] the most popular treatments for severe and recurring gynaecological cancers are hormone therapy and chemotherapy, and prognosis and survival are significantly impacted by the patient's responsiveness to treatment. The severe side effects of chemotherapy have led to an increase in the use of hormone therapy in recent years. [32] Hormone therapy either prevents hormones from being produced or prevents hormones from enabling cancer cells to divide and proliferate. It does not, however, work against every canc. [33] Heat transfer to the tumor bulk is known as hyperthermia (HT). A treatment that raises the tumor mass's temperature between 41 and 45°C using external physical techniques is known as hyperthermia. Since the administered temperature in the complicated tumor mass cannot be maintained uniformly, the techniques aim to focus on the tumor cells by applying complementing heat. [34]In order to make up for the drawbacks of traditional cancer treatments, hyperthermia (HT) has started to show promise as a cancer

treatment method. Because it has fewer side effects than traditional medicines and is reasonably priced given the significant therapeutic benefit it produces—especially when paired with other medications—HT is a viable choice. [35] Photodynamic therapy (PDT) has steadily gained popularity as a tumor treatment method since the late 1970s. Many nations, including the US, UK, France, Germany, and Japan, have approved it for use as a cancer treatment. [36] A less intrusive treatment option for a number of cancers and infectious disorders, such as bacterially infected wounds, acne vulgaris, human papillomavirus viral warts, and cutaneous leishmaniosis, is photodynamic therapy (PDT). [37] Surgery is a systemic treatment for breast cancer (BC), and radiation therapy is one of the most popular therapeutic techniques. Numerous studies have shown that RT is a promising adjuvant treatment for early breast cancer following conservative breast surgery. [38] The goal of radiotherapy is to kill the cancer cells while causing the minimum harm to healthy cells. Sometimes, to treat a particular type of cancer, chemotherapy and radiation therapy are combined. Combination therapy may be suggested if the cancer is not responding to one type of treatment, if the cancer cells cannot be surgically removed. [39] Delivering genetic material into target cells or tissue and expressing it with the goal of achieving a therapeutic impact is the goal of gene therapy. Its ability to be locally delivered allows it to deliver a high therapeutic dose without running the danger of systemic side effects, which gives it an edge over conventional medicines. Additionally, gene therapies can be cost-effective over time because they are typically one-time treatments. [40] A promising alternative in the fight against cancer is stem cell treatment, which encompasses all methods utilizing stem cells. [41]

History of cancer:

The Greek physician Hippocrates (460–370 BC), known as the "Father of Medicine," is credited with coining the term "cancer." Hippocrates referred to non-ulcer-forming and ulcer-forming tumors as carcinos and carcinomas, respectively. [42]

From 3000 BC to 1500 BC, the earliest evidence of human cancer can be found in the remains of Egyptian human mummies. A guy in his 50s who is now only known as M1 battled a protracted, excruciating, and progressing prostate cancer approximately 2250 years ago in Egypt. He must have had a throbbing pain in his lower back, which might have eventually forced him to stop fighting his illness after spreading to other regions of his body. [43] Tumor masses discovered in archaic human bones and petrified dinosaurs provide the earliest reliable proof of cancer in animals. A recent large-scale study that screened 10,000 specimens of dinosaur vertebrae for tumors using fluoroscopy and further evaluated abnormalities using computerized tomography (CT) may provide the strongest evidence of cancer in dinosaurs. [44]

The earliest known written account of human cancer was found in 19th-century Egyptian writings, including the papyri by Edwin Smith and George Ebbers that discuss surgical, pharmaceutical, and magical cures. They may have been inspired by writings from thousands of years ago and were composed between 1500 and 1600 BC. [45]. China, 250 BCE The Nei Ching, also known as The Yellow Emperor's Classic of Internal Medicine, provided the earliest clinical description of breast cancer, including its course, metastasis, mortality, and prognosis around ten years after diagnosis. [46] 100 AD A Greek physician from Italy named "Claudius Galen" (129–216 AD) surgically removed a few tumors. [46] Techniques for cancer surgery advanced in Germany in the 17th century, but the absence of anaesthesia and sterile conditions made surgery a dangerous option. [46] The German pathologist "Johannes Müller" proved in 1838 that cancer is composed of cells rather than lymph. [46] Within the United States, Fifteen doctors and business executives established the American Cancer Society in 1913 as the "American Society for the Control of Cancer" (ASCC) in New York City. Additionally, in Spain Madrid hosted the First World Cancer Congress in 1933. [46]

In 1963 in JAPAN Cancer research programs were established by the Ministry of Health and Welfare and the Ministry of Education, Science, and Culture. . [46] In 1970s Childhood leukaemia became one of the first cancers that could be cured by a combination of drugs. In the USA in 1994 the National Program of Cancer Registries (NPCR) is established. [46]

1. OBJECTIVES OF STUDY:

i. To Give a Summary of the Current Approaches to Cancer Treatment
ii. Investigating Novel and Creative Methods of Cancer Treatment
iii. To Assess Precision Medicine's Contribution to Cancer Treatment
iv. To Examine the Potential of Immunotherapy Developments
v. To Address Obstacles and Difficulties in the Clinical Application of New Therapies
vi. Examine Current Clinical Trials and Upcoming Opportunities
vii. Stress the Value of Multidisciplinary Methods in the Development of Cancer Therapies
viii. Estimate the Possible Effect of New Therapies on Cancer Survival Rates

2. Review of literature

2.1. Qiang Sun et al., he provide an overview on the main steps of the discovery of class sic immune-checkpoint blockade agents and summarise the most recent development of novel immunotherapeutic strategies, such as tumour antigens, bispecific antibodies and TCR-engineered T cells

2.2. Sonia Guedan at el, He provide an overview on the genetically engineered T cells are powerful new medicines, offering hope for curative responses in patients with cancer. Chimeric antigen receptor (CAR) T cells were recently approved by the US Food and Drug Admin istration and are poised to enter the practice of medicine for leukemia and lymphoma, demonstrating that engineered immune cells can serve as a powerful new class of cancer therapeutics. The emergence of synthetic biology approaches for cellular engineering provides a broadly expanded set of tools for programming immune cells for enhanced function. Advances in T cell engineering, genetic editing, the selection of optimal lymphocytes, and cell manufacturing have the potential to broaden T cell–based therapies and foe new applications beyond oncology, in infectious diseases, organ transplantation, and autoimmunity.

2.3. Kyusang Hwang et al., he first highlight the physiological and pathophysiological roles and signalling mechanisms of currently known and emerging therapeutic targets, including growth factors and their receptors as well as immune checkpoint proteins, in CRC. Additionally, we discuss the current status of monoclonal antibodies in clinical development and approved by US Food and Drug Administration for CRC therapy.

2.4. Marco Carlo Merlano et al., he provide the correlation between chemotherapy and the immune changes induced in the tumor microenvironment. Our ultimate aim is to pave the way for the identification of the best drugs or combinations, the doses, the schedules and the right sequences to use when chemotherapy is combined with immunotherapy.

2.5. Xi-Wen Zhang et al., He comprehensively discuss recent innovations in CAR-T-cell engineering to improve clinical efficacy, as well as strategies to overcome the limitations of CAR-T-cell therapy in OC.

2.6. Aneta Kwiatkowska et al., he does the review focuses on the strategies used for gene delivery, including the most common and widely used vehicles (i.e., replicating and non-replicating viruses) as well as novel therapeutic approaches such as stem cell-mediated therapy and nanotechnologies used for gene delivery. We present an overview of these strategies, their targets, different advantages, and challenges for success. Finally, we discuss the potential of gene therapy-based strategies to effectively attack such a complex genetic target as GBM, alone or in combination with conventional therapy.

2.7. Jie Huang, Jian Jian Li et al., he does Cancer radiotherapy [RT] demonstrates the benefit of local control with fewer side-effects compared to chemotherapy. To further improve the overall efficacy of RT and RT-combined therapies, studies revealing the mechanisms involved in tumor response to radiation are required. Accumulating new evidence has demonstrated that the microenvironment of solid tumors, such as cancers in lung, breast, and liver, holds a highly heterogenic population of tumor cells, an array of stromal cells with different functions, as well as non-cellular components. The observed tumor responses to cancer radiotherapy

are pooled signals generated from the heterogenic cell populations such as the survival of radio resistant tumor cells and the infiltrated immune cells. Thus, further elucidation of key dynamics in irradiated Tumor microenvironment (ITME), including repopulation of cancer stem cells, metabolic shifting, and radiation-induced Tumor immunogenicity, will be necessary to significantly enhance the outcome of RT or RT-combined immunotherapy. This review summarizes the current experimental and clinical results of these three dynamics in ITME.

2.8. Kevin Dzobo et al., he revisit CSCs within the tumor microenvironment (TME) and present novel treatment strategies targeting CSCs. These promising strategies include targeting CSCs-specific properties using small molecule inhibitors, immunotherapy, microRNA mediated inhibitors, epigenetic methods as well as targeting CSCniche-microenvironmental factors and di erentiation. Lastly, we present recent clinical trials undertaken to try to turn the tide against cancer by targeting CSC-associated drug resistance and metastasis.

2.9. Monica Marzagalli et al., the mechanisms exploited by CSCs to overcome different therapeutic strategies, from chemo- and radiotherapies to targeted therapies and immunotherapies, shedding light on their plasticity as an insidious trait responsible for their adaptation/escape. Finally, novel CSC-specific approaches will be described, providing evidence of their preclinical and clinical applications.

2.10. Josep Tarragó-Celada et al., he review the recent advances in the field of metabolic adaptation of cancer metastasis, focusing our attention on colorectal cancer. In addition, we also review the current status of metabolic inhibitors for cancer treatment.

2.11. Heidie Frisco Cabanos et al., he provide the persisted state most closely corresponds to minimal residual disease from which relapse can occur if treatment is discontinued or if acquired drug resistance develops in response to continuous therapy. Thus, eliminating persisted cells will be crucial to improve outcomes for cancer patients. Using lung cancer targeted therapies as a primary paradigm, this review will give an overview of the characteristics of drug-tolerant persisted cells, mechanisms associated with drug tolerance, and potential therapeutic opportunities to target this persisted cell population in tumors.

3. METHODS OF THE TREATMENT:

3.1. TREATMENT OF CANCER:

Surgeons in antiquity were aware that cancer typically returned after surgery to eliminate it. Even now, many people believe that many cancers cannot be cured, so they may put off seeing a doctor when they are still in the early stages. Surgeons Bilroth, Handley, and Halsted performed cancer surgeries by excising the entire tumor together with lymph nodes once anesthesia was developed in 1846. [47] For both scientists and doctors, treating cancer remains a difficult endeavour. Nowadays, hormone therapy, biological therapy, chemotherapy, radiation, surgery, and targeted therapies are the most common methods used to treat cancer. [48].

3.2. CANCER THERAPIES:

3.2.1. Immunotherapy :-

Thanks to the impressive clinical outcomes of checkpoint inhibitors and chimeric antigen receptor (CAR) T cells, immunotherapy has emerged as the most recent advancement in cancer treatment. [49]. Immunotherapy targets cancer by using a patient's immune system, and it has produced innovative treatment strategies and unheard-of clinical results. Despite the effectiveness of immunotherapeutic methods in a range of clinical settings and cancer subtypes, obstacles still exist. [50] In order to mobilize the patient's immune system and improve the activation of the antitumor immune response, cancer immunotherapy aims to change the target from the tumor cells themselves. This aids in the immune cells' ability to identify, target, and ultimately destroy the tumor cells. [51] William B. Coley, dubbed the "Father of Immunotherapy," came up with the idea of using the immune system's power to prevent cancer in the late 1800s. Patients with incurable tumors responded well to Coley's studies in which live bacteria, S. pyogenic and S. marcescens (also known as Coley's toxin), were injected into them. [52] The discipline of cancer immunology entered the current era of cancer immunotherapy with Allison and Honjo's discovery of T-cell immune checkpoints CTLA-4 and PD-1, for

which they were granted the Nobel Prize. [53] With the aim of enhancing anti-tumor immunity while lowering off-target effects, cancer immunotherapy can stimulate and/or encourage the body's immune system to indirectly attack and kill tumor cells, in contrast to chemotherapy and other medications that kill tumor cells directly. [54] A lot of study has been done on cancer immunotherapy, which uses the body's immune system to destroy tumor cells. Cancer immunotherapy is emerging as a potent new cancer treatment strategy, as demonstrated by thousands of clinical trials. [55] The term "cancer immune-surveillance" describes the immune system's role in tracking the development of cancer. Studies have shown that the immune system is capable of identifying and getting rid of abnormal cancer cells that develop in the human body. [56] High specificity, minimal or no side effects with active immunization (although adverse effects may occur with adoptive cell transfer, or ACT), and a strong safety profile are some of the main advantages of cancer immunotherapy over conventional medicines. [57]

- **Check point inhibitors**

An unparalleled breakthrough in cancer immunotherapy has been achieved with the recent FDA approval of immune checkpoint inhibitors and T cells expressing chimeric antigen receptors (CAR T) for cancer therapy. Without developments in the field of cancer immunology, cancer immunotherapy would not have achieved this significant milestone. [58] T-cell tolerance and activation are both involved in immune checkpoints. They are crucial for maintaining immune homeostasis and self-tolerance under typical physiological conditions. Tumor antigens may immunely evade detection due to immune-inhibiting signals from tumors. . [59] A member of the CD28 family of receptors, CTLA-4 is constitutively expressed on regulatory T (T reg) cells, a specific subset of CD4+ T cells with the ability to halt T cell responses, and is produced on the cell surface of conventional T cells by antigen activation. [60] Immune checkpoints are inhibitory proteins/receptors that are typically found on the surface of effector immune cells, T cells, or NK cells. Their ligands are transmembrane proteins that are expressed on B and T lymphocytes, antigen-presenting cells (APCs), tumor cells, macrophages, or other nonlymphoid tissues. [61] Immune checkpoint inhibitors (ICIs) are now the gold standard treatment in the first-line context for various tumors, including non-small cell lung cancer (NSCLC) (1-3) and BRAF wild-type (WT) melanoma. [62] Checkpoint receptors, including cytotoxic T-lymphocyte-associated protein 4 (CTLA-4), programmed cell death protein 1 (PD-1), lymphocyte-activation gene 3 protein (LAG-3), T cell immunoglobulin and mucin 3 domain (TIM-3), T cell immunoreceptor with Ig and ITIM domains (TIGIT), and others, are immunosuppressive molecules that inhibit immune effector cell activation. [63] In cancer, these chemicals are thought to cause immunological fatigue of effector cells and downregulation of the antitumor response. [64]

- **Adoptive cell therapies:-**

- **Cart- T cell therapy:**

Chimeric antigen receptors (CARs) are artificial cell surface receptors made up of several domains from costimulatory molecules, an intracellular hinge-containing domain, an external single-chain variable fragment, and a transmembrane region. [65] CAR-T cell treatment uses autologous T cells that have been modified to identify and destroy cancerous cells. [66] Over the past 25 years, CAR T-cell transplantation technology has seen a significant evolution. [67] The monoclonal antibody's single-chain variable region (scFv) and the T-cell co-receptor signaling region combine to generate a CAR-T cell. By building distinct monoclonal antibodies that substitute the complementarity defining areas of the T-cell receptors (TCR) for various tumor cell antigens, we can successfully get beyond the MHC restriction. By specifically identifying antigens unique to tumors, the scFv in CAR activates T cells, which then release cytokines to destroy tumor cells. [68] Target tumor antigens, such as CD19, can be precisely recognized and bound by CAR T-cells, which are genetically engineered T-cells that express a chimeric-antigen receptor. The tumor cells are then cytotoxically eliminated through performing/granzyme-induced apoptosis. [69]. Because CAR-T cells have the benefits of immunotherapy, they might be the best approach. Regardless of the underlying oncogenic driver mutations, CAR-T cells can be employed to suppress resistant cancer cells following treatment. Furthermore, unless the tumor antigen changes, CAR-T cells may be able to stop tumor recurrence if they are maintained for a few years. [70]

- **Monoclonal antibodies :-**

George Kohler and Cesar Milstein discovered the technology to produce monoclonal antibodies (mAb) in the middle of the 1970s. [71] Behring and Shibasaburo initially identified antibodies as a neutralizing agent in blood in 1890 while studying animal forms of diphtheria. [72] Innate antitumor immunity can be boosted by tumor-targeting monoclonal antibodies (mAbs). Despite the fact that immunotherapy using monoclonal antibodies has improved cancer patients' chances of survival, a sizable percentage of patients do not react to

treatment. Both acquired and primary resistance to ICIs are partially to blame for this phenomena. [73] Clones of a particular B cell produce monoclonal antibodies (mAbs), which are antibodies that bind to particular regions of an antigen, sometimes referred to as an epitope. Schwaber is discovered the initial techniques for producing the monoclonal antibodies using human-mouse hybrid cells in the 1973. Köhler and Milstein then employed these techniques to create the human-derived hybridomas that have been a mainstay in the large-scale manufacture of therapeutic antibodies ever since. [72] Technology based on monoclonal antibodies [mAbs] is a crucial tool for finding new therapeutic targets and creating new therapeutic agents, such as medications based on antibodies. Pancreatic cancer has not yet received approval for any of the 45 monoclonal antibodies (mAbs) that have been approved to date for the treatment of individuals with a variety of cancers. [74] The majority of anti-humoral antibodies are able to identify antigens on the tumor cells' surface. Others, instead of targeting the tumor cells directly, target metalloprotease secretion and tumor cell invasion, or identify surface antigens that obstruct neo-vascularization or movement. [75]. Antibody fragments can be made more cheaply and still have the same targeting specificity as complete monoclonal antibodies. Moreover, compared to monoclonal antibodies with a greater molecular weight of 150 kDa, these fragments are smaller and more readily infiltrate tissues and tumors more quickly and thoroughly. [76]

- **Oncolytic virus therapy :-**

Oncolytic virus therapy has recently been identified as a viable new treatment option for cancer patients. An oncolytic virus is a genetically created or naturally occurring virus that may selectively reproduce and kill cancer cells while causing no harm to healthy organs. [77] These "oncolytic" viruses (OVs) kills the cancer cells without affecting the normal cells. Some viruses, such as myxoma virus or reovirus, show innate predilection for tumor cells while remaining non-pathogenic in healthy humans. Other OVs, including as adenovirus, herpes simplex virus type-1 (HSV-1), and vesicular stomatitis virus (VSV), have been genetically altered to serve as vectors for anti-tumor immune responses. [78] Historically, two important achievements sparked interest in oncolytic virotherapy. Martuza et al. demonstrated that utilizing genetically modified HSV, nude mice with intracranial U87 gliomas survived longer. Second, in 2015, the FDA approved the talimogene laherparepvec (T-VEC) HSV, which expresses granulocyte-macrophage colony-stimulating factor (GM-CSF), for the treatment of unrespectable advanced melanoma. [79] Oncolytic viruses help antigen-presenting cells (APCs) travel to lymph nodes, where they activate cytotoxic CD8+ T lymphocytes (CTLs) and attract them to the infection site, which kills tumor cells. [80] Oncolytic virus (OV) therapy is a treatment that uses the virus to infect the tumor and then use its cytopathic effects to eliminate the tumor. In addition to its cytopathic effects, OV is likely to be helpful for metastatic cancers that are not directly infected by the virus because it activates systemic tumor immunity. [81].

- **Cancer vaccines:-**

A variety of strategies are used in cancer vaccination in a trouble to produce, enhance, or skew (or a blend of these) antitumor impunity. Multitudinous strategies include direct excrescence revision or the delivery of excrescence antigens, constantly in confluence with antigen- presenting cells (APCs) or other vulnerable modulators, to achieve this thing. Crucially, small patch signaling pathway impediments and conventional cancer treatments like radiation, chemotherapy, and surgery and ablation can have similar goods to cancer vaccines. [82] By the Centers for Disease Control and Prevention. Like vaccines against contagious conditions, cancer vaccines are new remedial munitions in the fight against excrescences. They're made to increase a person's vulnerable system's capability to identify and respond to particular antigens set up in cancer cells.[83] Due to ongoing changes in the excrescence medium (TME), a lack of knowledge about the vaccine's optimal factors for producing strong vulnerable responses against tone- one-antigens, and testing in delicate clinical settings, the maturity of cancer vaccines have n't yet produced the stylish vulnerable responses to completely annihilate cancers.[84] Cancer vaccination is challenging due to several hurdles, and two review papers in this Special Issue give sapience into these immunological and specialized challenges.[85] Cancer vaccines are another fashion to stimulate the vulnerable system.[86] Cancer vaccination, also known as cancer immunization or cancer immunotherapy, is a treatment fashion that stimulates the vulnerable system to fete and fight cancer cells.[91] Cancer vaccines are divided into the four types on the base of their medication ways these are nucleic acid- grounded vaccinations, cell- grounded vaccines, contagion- grounded vaccines, and peptide vaccines.[87] Cancer vaccines are principally characterized as precautionary or remedial. Preventative vaccines are used to cover against unborn ails, whereas remedial vaccines are used to treat those who have a being complaint. [88]

- **Immune system modulators**

Immune modulation in cancer refers to a group of treatments that try to use a case's vulnerable system to regulate excrescences, stabilize them, and perhaps annihilate the complaint. Immune checkpoint- blocking

antibodies are a new class of remedial medicines that modify T- cell pathways and have the eventuality to extinguish an anticancer vulnerable response. Ipilimumab, the first FDA- approved vulnerable checkpoint antibody certified for the treatment of metastatic carcinoma (MM), inhibits cytotoxic T- lymphocyte antigen 4 (CTLA- 4).[89] T- cell transfer treatment is another way to regulate the vulnerable system. This treatment crops vulnerable cells from excrescence napkins. These cells are sorted to see which are the most effective against cancer cells. [90] Recent studies have concentrated on the creation of small patch immunomodulatory, which have the eventuality to overcome the limitations of antibodies. [92].

3.2.2. Chemotherapy:-

Grounded on their origin, chemotherapy medicines can be distributed into two types. They may be of synthetic origin or factory- deduced (uprooted from shops).[93] Chemotherapeutic specifics contain genotoxic chemicals that help DNA conflation during proliferation, and chemotherapy and radiation remedy primarily cause DNA damage to cancer cells. On the other hand, cancer cells can repair damaged DNA by barring lesions caused by chemotherapy, which increases cancer cell survival and resistance to anticancer treatment. [94] Chemotherapy damages gesture pathway activation by causing cell stress. In these circumstances, cytoplasmic proteins like chromatin associated high- mobility group box 1 (HMGB- 1) and purine metabolites like adenosine triphosphate (ATP) are released into the extracellular space, while apoptotic cancer cells translocate endoplasmic reticulum (ER) - related chaperon proteins like calreticulin, heat shock protein [HSP] - 70, and HSP- 90 to the cell membrane face. [95]. One of the traditional styles of treating cancer is chemotherapy. The original attempts were accepted in May 1942 by Yale School of Medicine experimenters Goodman and Gilman, who set up that edging in a case with advanced on-Hodgkin's carcinoma with nitrogen mustard significantly reduced the excrescence's size. [96]. although it's infrequently used to treat BC, chemotherapy may be advised in some circumstances. [97] Chemotherapy is a possibility for certain molecular subtypes of bone cancer, which are generally distributed into several groups. Triadic-negative BC is regarded as one of the most aggressive molecular subtypes, and compared to the others, it has an advanced rate of treatment response. still, these individualities continue to have a dismal overall survival rate indeed with adjuvant treatment.[98] New approaches and molecular prognostic labels are demanded to ameliorate the case's prognostic because chemotherapy is generally employed for triadic-negative, seditious, and advanced- stage BC.[99] The foundation of treatment continues to be the use of chemical specifics to stop the growth of excrescences. But as long as chemotherapy specifics are administered, cancers appear to be growing more resistant, which ultimately reduces the effectiveness of the treatments' killing effect. (One hundred) The current standard of care for first- line chemotherapy in advanced cases is combination chemotherapy with gemcitabine and cisplatin.

In more advanced cases, the gemcitabine and platinum complex combination chemotherapy showed good efficacy. The United Kingdom published a randomized phase II exploration (ABC- 01) that compared gemcitabine and cisplatin as a fellow against gemcitabine alone.[101] Eurhythmic chemotherapy treatment (mCHT) refers to the habitual administration of low boluses chemotherapy that can sustain dragged , and active tube situations of medicines, producing favorable tolerability and it's a new promising remedial approach in solid and in hematologic excrescences.[102]

3.2.3. Radiation therapy:-

Radiation therapy is an effective cancer treatment method that is also the most cost-efficient single modality treatment. Radiation therapy employs high doses of radiation to kill or halt tumor growth. [103] Radiotherapy remains one of the most common cancer treatment modalities worldwide. In the United States, around 60% of patients receive curative radiation, despite breakthroughs in other treatments over the past 127 years. This research demonstrates that radiotherapy has shown to be an effective tool in oncological treatment. [104] Principles of radiation therapy: Radiation is a physical agent used to eliminate cancer cells. The radiation utilized is known as ionizing radiation because it produces ions [electrically charged particles] and deposits energy in the cells of the tissues it passes through. This deposited energy can either harm cancer cells or create genetic alterations that lead to cancer cell demise. [105]

➢ **TYPES OF RADIATION THERAPY:**

i. External beam radiotherapy (EBRT)
ii. Stereotactic body radiotherapy (SBRT)
iii. Stereotactic radiosurgery (SRS)
iv. Low-dose radiation (LDR)
v. High-dose radiation (HDR)
vi. Intensity modulated radiotherapy (IMRT)
vii. Image-guided radiotherapy (IGRT)
viii. Cone beam computer tomography (CBCT)

➢ **FUTURE PERSPECTIVES:**

Professionals hope that combining radiation therapy with MRI will improve the effectiveness of IGRT. Because MRI is known for its superiority in imaging soft tissues compared to CT, it may allow doctors to better target the tumor. [106]

3.2.4. Gene therapy:

Gene therapy it is the "introduction, removal, or changes in the content of a humans genetic coding with the goal of treating a disease," according to the American Society of Gene and Cell Therapy. [107]. Delivering genetic material to the target is the goal of gene therapy. One of the first to show a preliminary proof-of-concept for virus-mediated gene transfer was Ogres et al. He demonstrated how viruses can be used to introduce foreign genetic material into cells of interest. [108]. Combining new gene therapy techniques with currently available cytotoxic chemotherapeutic medications and radiation therapy allows cells or tissue to express themselves with the goal of achieving a therapeutic effect in a disease where new therapeutic innovations are desperately needed. Gives patients with advanced-stage liver tumors hope for improved prognoses. [109] the main goal of gene therapy, both past and present, is to use the anticancer effects of pro-inflammatory proteins—more especially, inflammatory cytokines. [110] Excellent results obtained using viral-mediated cryotherapy to treat disorders caused by genetic deficits, a clear area of interest for gene therapy-based techniques, demonstrate the effectiveness of gene therapy employing viral vectors. [111]. an alternative to using viral vectors, nonviral gene therapy offers the chance to get beyond some of the problems, like the favoured integration sites that most viruses have. [112] the absence of a highly effective and safe gene delivery mechanism continues to be one of the barriers to clinical success in gene therapy. As the main challenges of gene therapy have long been identified, an ideal vector for gene therapy should specifically target the tumor cells allowing:

i. increased transfection efficiency;
ii. Decreased off-target transfection; and
iii. decreased nontoxicity. [113].

By addressing the underlying cause of the condition—the genetic level—gene therapy is transforming the treatment of a number of diseases, such as melanoma, pancreatic cancer, retinal dystrophy, spinal muscular atrophy, polyneuropathy, hereditary transthyretin-mediated amyloidosis, nasopharyngeal cancer, bacillus Calmette Guérin (BCG)-unresponsive non-muscle invasive bladder cancer, nasopharyngeal cancer, bacillus Calmette Guérin (BCG)-unresponsive non-muscle invasive bladder cancer, haemophilia B, aromatic L-amino acid decarboxylase deficiency, multiple myeloma, cerebral adrenoleukodystrophy, lymphoma, ADA-SCID, large B cell lymphoma, acute lymphoblastic leukemia, beta thalassemia, and metachromatic leukodystrophy. [114].

➢ **Types of Gene Therapy:**

i. Ex-Vivo Gene Therapy: This type of gene therapy involves genetically modifying cells that are not within the body.
ii. In-Vivo Gene Therapy: In in vivo gene therapy, therapeutic nucleic acids are administered directly to the patient's body by a non-viral or viral delivery method. [115]

3.2.5. Hormone Therapy:-

It is still unclear how menopausal hormone therapy (MHT) affects breast cancer survivors. MHT has been reluctantly prescribed by doctors and taken by patients due to concerns about its oncological safety, especially in patients with hormone receptor-positive malignancies. [116] it has been demonstrated in recent years that hormone therapy is an exceptional cancer treatment choice. Medications that lower hormone levels or block their biological function are used to treat gynaecological cancers. This stops or slows the growth of the cancer. The way that hormone therapy works is by stopping the growth of cancer cells by hormones. [117] in the early years of menopause, hormone treatment (HT) is commonly used to reduce osteoporosis and subsequent fractures in women as well as to relieve climacteric symptoms. Among women without cancer, using HT is linked to a higher risk of ovarian, endometrial, or breast cancer. [118]. All patients with hormone receptor-positive breast neoplasms must get hormonal therapy. Tamoxifen is the only active adjuvant hormonal treatment used before and beyond menopause. [119]. although many different hormonal therapies have been studied, natural and synthetic progestational drugs are the most often employed therapeutic hormones. An antioestrogen that has been shown to improve survival and recurrence in hormone-sensitive breast cancer is tamoxifen. [120]

3.2.6. Epigenetic therapy:

The inheritable phenotype brought about by variations in gene expression without alterations to the DNA sequence is the subject of epigenetics. Therefore, the inheritable but reversible alterations linked to dysregulation of gene expression that show up as a pre-malignant phenotype while the genomic sequence remains unchanged are the focus of cancer epigenetics. [121] the molecular characteristics that control gene expression without changing the DNA sequence itself are referred to as epigenetics. Along with changes in the genetic landscape, the development, growth, resistance to treatment, and progression of cancer also involve changes in the epigenetic landscape. [122] DNA and histone proteins, two macromolecules that are physically and functionally entangled in chromatin, are the starting point for epigenetics. The nucleosome, the fundamental chromatin unit, is made up of 146-bp repeating DNA segments encircled by an octamer of histone proteins. [123] Regulation of gene transcription, genomic stability, and preservation of normal cell growth, development, and differentiation are examples of epigenetic mechanisms. The most significant feature of epigenetic modifications is that they do not rewire genomic information or affect the genetic material. [124] Combining epigenetic drugs with traditional therapy or cutting-edge anticancer treatments could provide an alternative to traditional chemotherapy and enhance therapeutic outcomes. By chemo sensitizing and immune-stimulating cancer cells, epigenetic medications can prime cancer cells for chemotherapy. They can also overcome acquired therapeutic resistance or work in concert with other anticancer treatments. Epigenetic medications and chemotherapy can work in concert to re-sensitize tumor cells that are resistant to both chemotherapy and radiation. [125] By locally controlling the chromatin's accessibility to transcriptional factors and its capacity to form active transcriptional higher order chromatin organization, epigenetics describes DNA methylation, histone modifications, and the dynamic binding of variable proteins that shape the chromatin compaction status and, consequently, determine the gene expression. [126]Although epigenetic medications have a lot of promise to improve patient outcomes, global transcriptional effects must also be taken into account. Drug-induced epigenetic changes may produce an abnormal pattern of gene expression, resulting in a worldwide transcriptional change that will cause cancer and extreme genomic instability. [127] Numerous cellular processes, such as chromatin remodelling, imprinting, X chromosome inactivation, and carcinogenesis, have been demonstrated to be significantly impacted by both genetic and epigenetic changes. [128]

- **Epigenetic Modifications**:

The transmissible alterations in chromatin structure and gene expression that take place without a physical alteration in the DNA sequence are referred to as epigenetic modifications. Changes in chromatin structure, the methylation status of DNA segments, chemical changes in histone chromosomal proteins (acetylation, methylation, ADP-ribosylation, ubiquitination, and phosphorylation), and the control of non-coding RNAs (ncRNAs) are all examples of epigenetic modifications. Long non-coding RNAs (lncRNAs) that are involved in chromatin remodelling, transcriptional and post-transcriptional regulation, microRNAs (miRNAs) that mediate the post-transcriptional regulation of genes, the availability of a selection of transcription factors, and the alteration of the functional properties of proteins following the final stage of translation are among the altered ncRNAs. [129]

- **Mechanisms of Epigenetic therapy:**

i. DNA Methylation
ii. Histone Modification
iii. Non-Coding RNA [127]

3.2.7. Nanotechnology therapy:

Nano biotechnologies like nanoparticles (NPs) have been used for effective drug delivery to mitigate poor solubility and stability, prevent degradation by proteases, improve drug distribution, and enhance drug resistance in order to overcome the low bioavailability of anticancer agents, including natural compounds. [130]. the primary benefit of using nanoparticles (NPs) is that they have a better pharmacokinetic (PK) profile than small-molecule medications. Small medications spread throughout the body, creating a very large distribution volume (Vd) that necessitates high dosages to reach the target site's therapeutic drug concentration, which might have harmful side effects. [131]

- **What are nanomaterials?**

The recommendations state that 50% of particles should have one exterior dimension that is between 1 and 100 nm. In certain situations, materials including fullerenes, graphene lakes, and carbon nanotubes that are smaller than 1 nanometer are regarded as nanomaterials, and 1% to 50% of particles with a size between 1 and 100 nm may be permitted. [132]. recently, the multidisciplinary topic of nanotechnology has become one of the most promising areas for cancer treatment. The use of nanotechnology for drug delivery and in vitro diagnostics has increased dramatically in recent years. Only a few parts of this technology—albeit analytical ones—are being put together in order to win the fight against cancer. [133]. numerous recent studies have shown that sensors and nanoparticles offer a great deal of promise for enhancing cancer diagnostics and boosting tumor detection sensitivity [13]. The identification of mutations and methylation patterns has reportedly been employed as a marker for the diagnosis of cancer. [134]. the size of nanoparticles is the 1–100 nm. Drug delivery nanoparticles can vary in size, shape, and content. Additionally, they could suffer from drug loading capacity, drug and particle stability, drug release rates, and the ability to deliver drugs precisely. [135].

- **TYPES OF NANOTECHNOLOGY:**

i. Carbon Quantum Dots
ii. Gold Nanoparticles
iii. Iron Oxide Nanoparticles
iv. Lipid Nanoparticles
v. Polymeric Nanoparticles
vi. Silica Nanoparticles [136]

3.2.8. Stem cell transplant therapy:

Typically referred to as tumor starting cells, cancer stem cells are the most aggressive cell type found within the tumor mass. CSCs are distinguished by their capacity to self-renew and preserve their undifferentiated condition, which, when combined with their high potential for differentiation, enables both the maintenance of a stem cell pool and the production of a diverse progeny of differentiated tumor cells. [137] The aggressiveness of some solid cancers is believed to be caused, at least in part, by the growth of cancer stem cells (CSC), since efforts to create therapeutic strategies that target CSCs have increased over the past few years. One of these is YAP1, a transcriptional regulator and the primary e sector of the Hippo pathway, which typically controls differentiation and cellular proliferation but also encourages treatment resistance and malignant phenotypes in a variety of malignancies. [138] although they make up fewer than 2% of all cancers, cancer stem cells, also known as tumor-initiating cells (TICs), are a subtype of tumor cells. Numerous investigations have demonstrated the tight relationship between CSCs and the genesis, development, metastasis, recurrence, and therapeutic resistance of tumors, which may be the cause of therapy failure. CSCs have the same capacity for self-renewal and multipotential differentiation as stem cells. [139] Both initial tumor heterogeneity and metastases to other tissues and organs can be replicated by CSCs. Numerous research have shown that CSCs can resist traditional treatments like chemotherapy and radiation, which makes them accountable for the emergence of therapy resistance. [140]

- **Concept of Cancer Stem Cells [CSCs]:**

Self-renewal and differentiation are two traits that cancer stem cells (CSCs) share with normal stem cells. Initially demonstrated by injecting AML cells into SCID mice via xenotransplant, CSCs are also known as tumor-initiating cells (T-ICs) or cancer stem-like cells. The experiments showed that expression of a particular CSC marker (CD34+CD38) could stimulate the production of a large number of colony-forming progenitors. Lung, pancreatic, breast, prostate, colon, glioma, and liver cancer are among the cancers from which several CSCs have been isolated. [141]

- **Type of Stem Cells for Cancer Treatment:**

i. Pluripotent Stem Cells (PSCs)
ii. Adult Stem Cells (ASCs)
iii. Cancer Stem Cells (CSCs)

- **Mechanisms of Action of Stem Cells in Cancer:**

i. Homing to Bone Marrow
ii. Tumor-Tropic E etc.
iii. Paracrine Factor Secretion and Di erentiation Capacity
iv. Signalling in CSCs **[142]**

3.2.9. Hyperthermia therapy:-

The anti-cancer treatment known as hyperthermia (HT), which involves applying heat (between 39 and 45◦C) to stop the growth of tumors, is frequently used in conjunction with radiotherapy and chemotherapy. It can be difficult to direct heat toward tumors rather than healthy tissues, though. Oncology research has shown a great deal of interest in hyperthermia (HT), one of the recently suggested non-invasive cancer treatments. [143]. In order to make up for the drawbacks of traditional cancer treatments, hyperthermia (HT) has started to show promise as a cancer treatment method. Because it has fewer side effects than traditional medicines and is reasonably priced given the significant therapeutic benefit it produces—especially when paired with other medications—HT is a viable choice. [144] in clinical cancer treatment, hyperthermia (HT), or exposing malignant tissues to temperatures above normal, is becoming more and more common. A variety of HT-based techniques have been created to accomplish different clinical objectives. We pay special attention to temperatures between 39 and 43 °C, or mild HT, in this review. [145] Cell type, temperature, and time all had an impact on how HT suppressed cell growth. After 6 hours of HT treatment, apoptosis was seen, which was finally followed by secondary necrosis. [146]

3.2.10. Photodynamic Therapy:-

Future therapeutic uses for photodynamic therapy (PDT), a non-invasive cancer treatment approach, seem promising. [147] to differentiate photo oxidation from the sensitization process in photography, Hermann von Tappeiner first used the word "photodynamic" in 1904. [148] the destruction of cancer cells by light irradiation with eosin-coated tumors was first documented in 1903. Following that, a variety of hematoporphyrin derivatives were used in photodynamic treatment [(PDT). [147] PDT has become a viable alternative cancer treatment because of its high cancer selectivity, which could result in less harm to healthy tissue and more effectiveness at the tumor location. [149] A photosensitizing agent is applied to the tumor site, and light energy at a certain wavelength is then used to activate the agent. When light energy and oxygen are combined, a photodynamic reaction occurs that is both cytotoxic and vascular toxic. Normal tissue in the patient's body is not significantly harmed by PDT. PDT selectivity is not perfect, though, and normal tissue may sustain some harm. [150] PDT is a unique form of light therapy that relies on the interaction of three primary components: molecular oxygen, a light source, and a photosensitizer (PS). Depending on the target location, the photosensitizer's absorption spectrum, and the necessary light dose, the three primary light source types utilized in PDT are lasers, light-emitting diodes, and lamps. [151] Photosensitizers (PS) are chemicals used in cancer treatment that transfer light energy to modify molecular structures near their position when illuminated. When enough mutations occur in a cancer cell, they can become irreparable, resulting in cell death. The majority of clinically utilized PS have a tetra-pyrrole structure and are derived from porphyrins, chlorines, or dyes. [152]

3.2.11. Targeting metabolism as an emerging therapy in cancer:

Cancer metabolic reprogramming is widely recognized as an important feature of cancer, with cancer researchers and doctors paying increasing attention to this phenomenon. Even when exposed to oxygen, cancer cells boosted their glucose absorption and lactate generation. Later, numerous metabolic changes in other pathways were discovered in cancer cells. [153] the study of the metabolic properties of tumors and their relationship to cancer growth is a new subject in cancer research. Metabolic plasticity in tumors is not only influenced by the glycolytic phenotype (as explained by Warburg), but mitochondrial energy reprogramming has recently been identified as a tumour feature to meet the high demand for energy and biomolecule precursors for rapid cancer cell proliferation. [154]

3.2.12. Targeted therapy:

Over the last few decades, we've witnessed the emergence and evolution of molecularly targeted cancer medicines. Advances in our understanding of somatic genetic changes or "driver" mutations in oncogenic kinases, in particular, have led to the development of successful medicines that target specific oncogenic signalling pathways, resulting in substantial therapeutic responses. [155] in cancer, constant activation of VEGFR2 promotes the development of new blood vessels, aiding disease progression and metastasis. Tumor cells produce galectins that attach to T-cell glycoprotein receptors such as CD45 and CD71. [156]

- **Targeted Therapies Agents:**

1) Tyrosine Kinase Inhibitors
a) Sorafenib:-
b) Lenvatinib:
c) Cabozantinib:
d) Regorafenib:
2) VEGF Inhibitors:
a) Bevacizumab:
b) Ramucirumab:
3) Other Targeted Agents:
a) Tyrosine Kinase Inhibitors:
b) Non-Tyrosine Kinase Inhibitors Targeting the Extracellular Space or Membrane
c) Agents Targeting the Intracellular Space: [157]

3.2.13. Cold plasma therapy:

In 1879, William Crookes developed the principles of plasma science by ionizing gas in an electrical discharge tube using a voltage coil. The ionized gas was dubbed radiant matter. Irvin Langmuir coined the word plasma nearly fifty years later, in 1927. [158] Plasma is commonly referred to as the fourth state of matter and is considered the most reactive. UV radiation, charged particles, reactive oxygen species (ROS), reactive nitrogen species (RNS), and electrical fields all work together to produce therapeutic effects in a single treatment modality. Cold atmospheric plasma (CAP) works selectively on cancer cells, destroying them more effectively than non-neoplastic cells. [159] CAP has a wide range of applications in medicine, including wound healing, microbial sterilization, biofilm inactivation, and cancer therapy. CAP is an ionized gas made up of reactive chemicals like reactive oxygen species (ROS) and reactive nitrogen species (RNS), and it is designed to operate at atmospheric pressure and around room temperature. [160]The effect of CAP was studied in several adherent human cancer cell lines, including melanoma (A375), extra hepatic bile duct carcinoma (TFK-1), osteosarcoma (MNNG-HOS), colon carcinoma (WiDr), prostate cancer (PC3andLnCap), urinary bladder grade 3 carcinoma (HT-1376), oesophageal adenocarcinoma (OE19), hormonal receptor positive breast cancer (MCF7), triple-negative breast cancer (HCC1806), lung cancer (H1299), and endometrial cancer (ECC-1). A human fibroblast cell line (HFF-1) was also investigated. [161] the use of cold physical plasma to specifically target cancer cells has piqued researchers' interest in recent years. Physical plasma's impact on tongue cancer and breast cancer cell lines revealed a clear dose-dependent reduction in cell viability in all cell lines tested. Physical plasma affects a variety of cancer cell types, including brain, lung, blood, cervical, melanoma, breast, colorectal, head and neck, hepatocellular, prostate, ovarian, osteosarcoma, pancreatic, bladder, gastric, thyroid, uterine, and epidermal cancer cells. [162]

The potential selectivity of CAP for cancer cells over their non-malignant counterparts has increased interest in CAP as a novel cancer treatment. CAP may impact various intracellular signal transduction pathways, determining the destiny of the cell and perhaps triggering cell death. [163]

4. Result and discussion:

1. Immunotherapy:

This therapy works by blocking checkpoint inhibitors. This therapy uses monoclonal antibodies, cancer vaccine, and oncolytic virus therapy to treat cancer.

Discussion:

The various advanced treatments against immune therapy have been arised. Not all patients respond to this therapy due to different in their immune system capability. Immune related disorder like cytokine release syndrome [CRS] can complicate this treatment, to address this researcher's focus on biomarkers to predict which patient benefit the most with combining to other therapies.

2. Targeted Therapy:

This therapy helps in treatment of cancer with specific genetic mutations such as HER2-positive breast cancer and EGFR mutations in lung cancer. Drugs in this therapy act by blocking signalling pathway essential for tumor growth. Recent advancements in this therapy are multi targeted therapy. Also researchers work on therapies that targeted tumor micro environmental factors such as immune suppressive cells within tumors.

3. Discussion:

This therapy are more specific and have less side effect compared to traditional chemotherapy. The main problem in this therapy lies development of resistance. Recently researchers works on to identifying new molecular sites for targeting and to decrease resistance factor.

4. Gene Therapy:

In this therapy particularly CRISPR-Cas9 directly modify cancer cell DNA. It is also to enhance immune cells such as T-cells to recognize cancer cell and fight against it.

Discussion:

Researchers work on to discovered novel form of this treatment. CRISPR could become key tool in personalized cancer treatment.

5. Epigenetic therapy:

This therapy seeks to modify gene regulation without altering gene sequence. This therapy reverse abnormal activity in gene of cancer cells. This therapy helps in treating cancer like leukemia and breast cancer.

Discussion:

Researchers try to discovered new drugs which controls gene activity in cancer cells without altering DNA.

6. Radiopharmaceuticals:

This therapy involves radioactive isotopes like Radium-223 which directly act on targeted cancer cells. In prostate cancer with bone metastases show efficacy in reducing pain and improve survival rate of patients.

Discussion: The main problem in this therapy is to manage radiation exposure to other healthy tissues and their side effects.

5. Conclusion:

Traditional methods like surgery, chemotherapy, radiation have been avoided due to their side effects and decrease in effectiveness due to their resistance. Nowadays emerging therapies like immunotherapy, gene therapy, cold plasma therapy, and targeted therapy offering new hopes for patients by directly acting on molecular site of cancer cell.

Immunotherapy show remarkable success in treating melanoma and lung cancer by increasing body's immune system to fight against cancer cells. Similarly, targeted therapy also show significant change in cancer treatment by targeting at specific site of genetic mutations and site of cancer like breast cancer and leukaemia where gene therapy directly not treating cancer cells but helps in correcting genetic mutations that leads to cancer.

In conclusion, during emerging of various cancer therapies are still in developmental stages, they represent fundamental change in oncology, despite challenges such as resistance, less toxic, cost effective. With continued research and innovation these new emerging therapies have capability to improve survival rate and quality of life for cancer patients offering strength to fight against this disease.

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