Investigating Conventional and Personalized Approaches to Cancer Therapeutics

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Abstract

Cancer continues to be a destructive force in our society, with an estimated 1.9 million diagnosed cancer cases in 2024 (American Cancer Society, 2024). With its prevalence in society, it is increasingly important that we continue to advance the field of treatment to attempt to cure this terrible disease. This paper aims to better understand the different types of conventional and personalized therapies for cancer treatment and the pros and cons of each type of treatment. Specifically, the paper explores two types of conventional cancer therapeutics, chemotherapy and radiation therapy, and two types of personalized cancer therapeutics, immune checkpoint inhibitors and CAR-T. For each of the four treatments, the paper details how they combat cancer by describing the underlying biological mechanisms, situations in which they are implemented for therapy, and their benefits and limitations. Lastly, the paper touches on the use of all four of these cancer treatments in the use case of breast cancer, discussing integrative therapy in practice. The future of cancer treatment requires a thorough understanding of the underlying mechanisms of this disease to push innovation and find a cure. As research advances and technology evolves, we move closer to a world where effective and transformative treatment offers hope for every patient.

Introduction

The state of Cancer

Cancer has been around for millennia, with tumors being found in animals even before humans stepped foot on Earth (Hajdu, 2011), and it continues to be the leading cause of death worldwide (Twombly, 2005). Over the years, cancer has morphed and evolved in response to the ever-changing lifestyles of humans and advancements in medical treatment. Currently, the top three cancers with the highest mortality rates are lung and bronchus cancer, colorectal cancer, and pancreatic cancer (Siegel et al., 2024). In 2024 alone, around 2 million people will be diagnosed with cancer, and an estimated 611,720 people will die from it. Of these deaths, 125,070 are predicted to be from lung and bronchus cancer, 53,010 from colorectal cancer, and 51,750 from pancreatic cancer (Siegel et al., 2024). Thanks to improved early detection and treatment plans in the last few years, cancer deaths have steadily declined, with many cancers decreasing in mortality rate (Hashim et al., 2016). Despite this, there is still an increase in six of the most common cancers: pancreatic, melanoma, breast, kidney, prostate, and endometrial, with the leading cause being increased excess body weight (American Cancer Society, 2024). Therefore, it is essential to continue assessing the state of cancer and current advancements in treatments.

Biology Of Cancer

Cancer begins when a mutation in the body causes cells to proliferate rapidly without routine cell checks and signals (Bertram, 2000). This leads to rapid cell proliferation and a build-up of cells called tumors. These cancerous cells then attack normal healthy cells, allowing them to take

over the body, causing immense stress and harm (Weinberg, 1996). Cancerous cells typically contain mutations due to duplication and deletions in their chromosomal DNA, making them unstable (Richards, 2001). Normal cells that go through the cell cycle have checkpoints that ensure they have no harmful mutations (Kaufmann & Paules, 1996). However, since cancer cells do not have these standard checkpoints, harmful cells with mutations can continue proliferating instead of typically going to rest (Weinberg, 1996). Two significant regulators involved in regulating cell proliferation and suppressing growth are the RAS protein pathway and the TP53 gene - most commonly called the guardian of the genome (Guimaraes & Hainaut, 2002; Khosravi-Far & Der, 1994). The constant activation and inactivation of cyclin-dependent kinases (CDK), through the periodic creation and destruction of cyclins throughout the cell cycle, is the primary means of cell cycle regulation (D. G. Johnson & Walker, 1999). There are currently nine identified CDKs that each act as a checkpoint at various stages within the cell cycle (D. G. Johnson & Walker, 1999).

Why Cancer is difficult to cure

Cancer contains many unique characteristics that make it extremely difficult to treat. One of the main reasons is that each tumor has its own set of genetic changes, known as cellular heterogeneity (Heppner, 1984; Rubin, 1990). This makes treating a tumor challenging, requiring multiple treatment plans or various drugs to combat all the different cells that make up the tumor (Campbell & Polyak, 2007). Another characteristic that makes cancer hard to treat is that cancerous cells contain ATP-binding cassette transport proteins, allowing them to flush out toxic agents from their intracellular cytoplasm to the extracellular space (Chakraborty & Rahman, 2012). This causes issues as it enables cancer cells to resist many chemotherapy drugs (Chakraborty & Rahman, 2012). This necessitates significantly more resources and extensive research to develop chemotherapy drugs that can evade detection, preventing cancer cells from expelling the medication. Lastly, the most significant reason why cancer is so difficult to cure is that cancer cells accumulate additional mutations throughout their lifespan and are, therefore, constantly evolving and adapting to various drugs (Wang et al., 2019). These accumulated mutations can enable cancer cells to build resistance against certain drugs, potentially leading to treatment immunity. Given cancer cells' incredibly high mutation rate, a particular chemotherapy drug may no longer be effective after just a few weeks (Martincorena & Campbell, 2015). In fact, drug resistance to cancer treatment is responsible for more than 90% of cancer-related deaths (Wang et al., 2019). Due to these challenges, a significant amount of time and resources have been poured into advancing treatment and diagnosis, particularly in personalized medicine. Consideration of a patient’s genetics and personalized treatment could greatly improve cancer outcomes. This review explores some conventional and personalized cancer therapeutics, assesses how these approaches differ, and evaluates the benefits and limitations of each approach.

Literature Review

Conventional Cancer Therapy

The simplest way to think of conventional cancer therapy is one size fits all. This means the same chemotherapy drugs are circulated through millions of genetically heterogeneous patients, leading to varied success. This is the premise of what traditional therapy for cancer entails.

Conventional cancer therapy is the first form of cancer treatment and has been around since the 1930s (Arruebo et al., 2011). Chemotherapy is one of the earliest created conventional therapies and has seen significant advancements throughout the decades (Arruebo et al., 2011). Because conventional therapy is not tailored to one's specific genetics, it remains the most basic and accessible form of cancer treatment. The cheaper price and increased accessibility make it still quite popular in today's age, even with personalized therapy on the rise. However, due to its one-size-fits-all basis, conventional therapies are more harmful to the body and less effective as they lack selectivity towards cancerous cells (Chidambaram et al., 2011).

The rise of genomics and personalized medicine

Sequencing the entire human genome once seemed impossible, but nowadays, genetic testing is widely accessible to the regular consumer. Over the years, as the technology has advanced and become cheaper to manufacture, the price of genetic testing has dropped significantly, with a whole genome test dropping in price to around $1,000 (Phillips et al., 2018). As of August 1st, 2017, there were 75,000 genetic tests on the market (Phillips et al., 2018). The demand for clinical genetic sequencing has grown exponentially over the past few years, with the market worth in 2020 being an estimated $7.7 billion (Phillips et al., 2018). Along with the increased accessibility to genetic testing, advancements in cancer prevention and treatment have also increased. This is due to genetic testing being able to detect specific mutations in a person's body that cause an increased chance of developing cancer, significantly improving early detection (Singh et al., 2023). In addition, advancements in genetic testing paved the way for large genetic databases, which scientists can use to study cancers across populations, map cancer pathways, and much more. Most notably, The Cancer Genome Atlas (TCGA) has molecularly identified over 20,000 primary cancers and matched healthy samples across 33 cancer types (National Cancer Institute, 2022). Additionally, TCGA has generated over 2.5 petabytes of proteomic, genomic, epigenomic, and transcriptomic data, all of which have significantly improved the ability to diagnose, treat, and prevent cancer (National Cancer Institute, 2022). Another major turning point for cancer treatment was the implementation of CRISPR-Cas9 for efficient and precise gene editing (Jinek et al., 2012). These genomic developments make the rise of personalized medicine possible.

Personalized Cancer Therapy

Given these advances in genomics, more customized treatment plans can be designed for cancer patients. Personalized cancer therapy is characterized as a treatment that is unique to oneself, meaning two patients never receive the same treatment. It is a therapy practice involving an individual's genetic profile to guide decisions about cancer diagnosis, treatment, and prevention (Sabatier et al., 2014). Unlike conventional cancer therapy, personalized cancer therapy takes into account a person's genetic makeup and disease history before a treatment plan is created (Verma, 2012). Personalized medicine is based on targeted therapy, making it essential to understand information on the altered pathways and components leading to cancer (Verma, 2012).

There are many benefits to utilizing personalized medicine. For example, personalized medicine is optimal for obtaining the best medical outcomes due to the treatment being chosen depending

on what works best for the patient, the patient's genomic profile, or specific characteristics in cell or blood surface proteins (Sabatier et al., 2014). This allows the treatment to be much more tailored to oneself than conventional treatment, allowing for significant improvements; for example, we are now able to evaluate one's risk of cancer for improved prevention and can customize treatment based on one's cancer genetics, both of which can help solve many of the issues cellular heterogeneity brings along (Cheng & Zhan, 2017). With all these extra personalized modifications to treatment plans, personalized medicine has its fair share of downsides. The biggest one is the cost of personalized cancer treatments compared to conventional cancer treatments. Personalized cancer therapies are much more expensive compared to conventional therapies due to the extra time and resources it takes to modify treatment to one person's specific genetics (Jakka & Rossbach, 2013). With this background knowledge of conventional and personalized therapies, the paper will dive deeper into commonly implemented treatments for both categories.

Conventional Therapy

Chemotherapy

Chemotherapy is a standard cancer treatment that attacks rapidly proliferating cells (Kennedy et al., 1980). It was made after tests displayed mustard gas killing lymphatic tissue and bone marrow. These effects were later retested on mice using a derived form of mustard gas called nitrogen mustard. These later tests proved the initial findings and displayed regression of lymphoma tissues (Anand et al., 2022). Chemotherapy drugs have seen significant advancements since they were created, and now we have many different chemotherapy drugs that attack cancer cells in varying ways. One way is by damaging the cancer cells' DNA and RNA, preventing them from growing and dividing (Bashir, 2023). Specifically, the chemotherapy drug can be inserted into the DNA double helix to form a covalent bond. Then, it can hinder DNA replication, destroy DNA template formation, and block it from translation, transcription, and many other functions (Sun et al., 2021). Another way chemotherapy attacks cancer cells is by arresting cell division, specifically in mitosis (Sun et al., 2021). Mitosis is the part of the cell cycle in which cells divide and proliferate. Some chemotherapy drugs destroy the cytoplasmic microtubules during mitosis, halting cells that rapidly divide (Bingham, 1978). Since cancer cells rapidly divide, halting mitosis destroys many cancer cells actively attempting to multiply. There are many types of chemotherapy drugs consistently used throughout today's day and age. However, alkylating agents and antimetabolites are the most commonly used (Veazey, 2021). Alkylating agents attack DNA within cancer cells, preventing them from dividing. Since DNA is most sensitive to alkylation, inter or intrastrand cross-links of several nucleophilic centers occur, contributing to cytotoxicity (Connors, 1974). Antimetabolites resemble naturally occurring compounds that interfere with the S phase of the cell cycle, blocking the production of nucleic acids, inhibiting the growth of the cancer cell, and eventually starving it to death (Scagliotti & Selvaggi, 2016). However, these chemotherapy drugs, and all others, cannot distinguish between healthy and cancer cells, attacking any fast replicating cells in mitosis. Given the non-targeted approach and side effects of chemotherapy, the treatment is typically administered in cycles, with a period of treatment followed by rest, to allow the body to recover and replenish all necessary and healthy cells (Bingham, 1978).

The administration of chemotherapy is based on factors such as the phase of the patient's cancer, whether it is metastatic or in the early stages, and localized. Most of the time, chemotherapy is not used on its own. It is coupled with another form of cancer treatment, such as radiation or before surgery, to shrink the tumor (Bingham, 1978). However, chemotherapy is often used in metastatic cancer cases due to chemotherapies' ability to circulate through the bloodstream, reaching cancerous cells at varying distances across the body (Gabizon, 1995). Metastatic cancer is when the cancer has spread to different parts of the body, causing tumor growth in distant organs and evasion of immune surveillance (Ganesh & Massagué, 2021). In this case, chemotherapy is used to slow the spread of cancer cells and shrink tumors (Bingham, 1978). However, as mentioned before, chemotherapy can not differentiate between cancerous and healthy cells, leading to the death of many healthy cells and causing severe side effects. Chemotherapy remains an effective strategy, especially when considering specific stages of cancer and combinations of treatments.

One of the significant benefits of chemotherapy is its ability to be coupled with various other cancer treatments. It is excellent at reducing cancer spread and shrinking tumors, making the job of other cancer therapies, such as radiation and surgery, much easier. However, the major downside to chemotherapy is its toxicity to the human body due to it killing off many healthy cells, leading to a low quality of life. Therefore, chemotherapy patients may lose their hair as a side effect of the treatment because rapidly dividing hair follicles are also attacked by chemotherapy drugs (Bingham, 1978). In addition to hair follicles, bone marrow and digestive system cells replicate quickly and are regularly attacked by chemotherapy drugs (Bingham, 1978). The type and severity of side effects vary depending on the type of chemotherapy drug and the dosage; however, the most common side effects are fatigue, vomiting, mouth sores, hair loss, nausea, anemia, easy bruising or bleeding, increased risk of infection, changes in appetite and taste, and neuropathy (Bingham, 1978). A significant side effect that arises with the use of chemotherapy is decreased kidney function due to many chemotherapy drugs being filtered out through the kidney, potentially leading to nephrotoxicity (rapid deterioration of kidney function) later in life (van den Boogaard et al., 2022). Ultimately, chemotherapy is effective at slowing down the spread of metastatic cancer and shrinking tumors, making the job of combining therapies easier.

Radiation Therapy

Radiation therapy has been a staple of cancer treatment for decades now, with the invention of the X-ray being traced back to 1895 (Huh & Kim, 2020). Similar to its chemotherapy counterpart, radiation therapy falls under the one-size-fits-all category, as the radiation delivered is not unique to one's genetic makeup. Nowadays, radiation therapy is much more advanced and effective than it once was due to its ability to focus more effectively and deliver radiation to a tumor (Bortfeld & Jeraj, 2011). Radiation therapy utilizes ionizing radiation due to its ability to form ions and deposit energy into the cells of the tissues it targets. This large amount of deposited energy is enough to kill cancer cells or at least cause genetic changes that can result in cancer cell death (Baskar et al., 2012). The high-energy output of the radiation damages the DNA and RNA of cancer cells, preventing them from dividing further (Baskar et al., 2012). More specifically, the radiation attempts to cause DNA double-strand breaks; even a single

double-strand break is enough to kill an entire cell or disturb its genomic integrity (Baskar et al., 2012).

The central premise behind radiation delivery is that radio waves are aimed at a select area in the body that contains either a tumor or cancerous cells. There are two different ways in which radiation can be delivered to the location of the cancer. The first is through external beam radiation, administered from outside the body by aiming high-energy rays, either photons, protons, or particle radiation, at the tumor's location. The second way is through internal radiation, which is delivered from inside the body through radioactive sources sealed in either catheters or seeds directly placed into the cancer site. Internal radiation tends to be less commonly used due to its short-range effects (Baskar et al., 2012).

Radiation therapy can be administered in the hope of curing cancer or, more commonly, used in combination with other treatments. In most use cases, radiation therapy is combined with surgery to either shrink the tumor pre-surgery or destroy any remaining tumor cells after surgery. Typically, radiation therapy is used when a patient has a solid malignant tumor, with around 50% of patients with solid malignant tumors receiving radiation therapy.

Radiation therapy provides many benefits but also side effects. Similar to chemotherapy, radiation therapy is unable to distinguish between healthy cells and cancerous cells, leading to the death of normal cells as well. This has led to radiation therapy's main goal to maximize the dosage while minimizing exposure to normal cells (Baskar et al., 2012). Unfortunately, radiation therapy comes with some severe early and late toxicity, potentially affecting the long-term health of patients (Bentzen, 2006). Late toxicity effects can include neural damage, atrophy, radiation-induced fibrosis, a range of growth-related and endocrine effects, and vascular damage, all capable of leaving a significant impact on patients' physical and social functioning (Bentzen, 2006). However, these side effects are not guaranteed, as one of the many benefits that arise with utilizing radiation therapy is its ability to achieve symptom control in a variety of clinical situations. In patients with metastatic cancer that is causing pain, significant organ compromise, or bleeding, radiation therapy is sometimes able to provide symptom relief (Eifel, 2017). Overall, radiation therapy remains an effective treatment to reduce the size of localized tumors and relieve potential negative symptoms.

Personalized Therapy

CAR-T

Due to the genetic complexity of different patients and their specific cancer types, treatments that go beyond a one-size-fits-all method are necessary. This is the central premise behind CAR-T. It was made with the idea that tailoring treatment to one's specific cancer DNA mutations would be more effective. Everyone receiving CAR-T has a different and unique treatment; two people never receive the same one.

The problem at hand is cancer's ability to evade T-cell surveillance. This is because T-cell receptors only become activated when in contact with the histocompatibility complex (MHC1). As many cancer cells downregulate the expression of MHC1 molecules to avoid immune

surveillance, cancer cells are seen as invisible to T-cell receptor-mediated recognition (Abreu et al., 2020). T-cells typically recognize foreign antigens in the body and activate an immune response to attack them (Krogsgaard & Davis, 2005). However, since cancer can hide from the T-cell's surveillance, the T-cells are oblivious to the fact that cancer is in the body, leading to no immune response activation.

CAR-T begins with extracting blood from the patient and filtering for one's T lymphocytes (T-cells). These cells play a central role in regulating our immune system by providing an immune response against foreign pathogens (Khan & Ghazanfar, 2018). Once the T-cells are retrieved from the patient, they go to a lab where they are genetically engineered to express chimeric antigen receptors (CARs). CARs are highly personalized because they are made with unique receptors designed to identify and target a specific tumor antigen on the surface of the patient's cancer cells (Mohanty et al., 2019). Once the T-cells develop CARs, they are replicated and inserted back into the cancer patient. The new T-cells can now detect harmful cancerous cells, attach to specific antigens on the cancer cell's surface, and kill it. The reason for CARs is due to cancer's unique ability to subvert the immune system, preventing T-cells from identifying them and halting the immune response necessary to attack cancer. (Mohanty et al., 2019). There are many different ways that DNA can be administered into cells. The most traditional method is recombinant viruses (Mohanty et al., 2019). This method entails using a reverse transcriptase that promotes the integration of artificial genes into the host genome. To create a recombinant virus, coding sequences are substituted by a gene of interest, such as a gene coding for the creation of chimeric antigen receptors. Recombinant viruses are great at gene therapy due to their innate ability to disturb the sections of genes, resulting in neoplastic transformation (Mohanty et al., 2019).

CAR-T may seem like a flawless alternative to chemotherapy and radiation therapy; however, it is not perfect and comes with its fair share of severe side effects. For example, cytokine release syndrome, tumor lysis syndrome, neurological toxicity, anaphylaxis, and B cell aplasia. Cytokine release syndrome is a severe side effect that can lead to extreme discomfort and lower quality of life due to it potentially causing throat tightness, dizziness, hypotension/hypertension, dyspnea, flushing, fever, chest and back pain, gastrointestinal symptoms, and some cases even death (Norton & Broyles, 2017). Continued research and advancements are continuing to be made to minimize the severe potential side effects of CAR-T. On the flip side, CAR-T has many benefits; one significant benefit is the T-cells ability to remain stable for several years in the patient's body, allowing them to continue targeting cancer cells in case of a relapse (Mohanty et al., 2019). Another significant benefit is its decreased toxicity in the patient's body as it can decipher between healthy and cancerous cells, thus being safe and nonlethal to host cells and keeping the patient healthy (Mohanty et al., 2019).

CAR-T is excellent in combination with other treatments, such as chemotherapy and radiation, as it is best used when some cancer cells are still left to attack after previous treatment (American Cancer Society, 2022). CAR-T therapy is particularly effective and recommended for patients with Leukemia, as this strand of cancer has a very common antigen known as CD19 (Moffitt Cancer Center, 2024). Overall, CAR-T is a great and promising new cancer therapy that genetically engineers one's T-cells to improve their ability to recognize and attack cancerous cells. CAR-T continues to be advanced and researched, attempting to address the limited

efficacy against solid tumors and minimizing the potentially life-threatening side effects (Sterner & Sterner, 2021).

Immune Checkpoint Inhibitor

Immune checkpoint inhibitors are a form of personalized cancer treatment that leverages the patient's immune system, similar to CAR-T. Immune checkpoint inhibitors work by genetically modifying T-cells to prevent cancer cells from turning off T-cell's immune response, while CAR-T modifies T-cells to seek out and target cancer cells. Usually, when foreign agents are noticed in the body, an immune response will activate, attacking potentially harmful agents. For the T-cells to not kill off healthy cells, they have a surface receptor that other cells can attach to and deactivate the T-cell. This unique ability makes T-cells great at fending off harmful cells while keeping their host body healthy (West, 2015). However, cancer cells can manipulate this ability by binding to the T-cell receptor, pretending it is a healthy cell, and deactivating it, preventing it from getting killed (West, 2015). Immune checkpoint inhibitors prevent cancer cells from deactivating T-cells by genetically engineering one's T-cells and removing the inhibitory signals of T-cell activation, enabling tumor-reactive T-cells to overcome regulatory mechanisms and activate a deadly response to cancerous cells and tumors (Wei et al., 2018). There are varying ways in which immune checkpoint inhibitors are delivered into the body. The most common way is through transdermal microneedle patches loaded with anti-programmed death-1 antibody and anti-CTLA4. Both were developed to facilitate the release of immune checkpoint inhibitors into the side of the tumor or cancer (Han et al., 2020).

Cancer utilizes various pathways to shut off the T-cell immune system response (Kiyotani et al., 2021). This means that each patient's cancer could use different pathways to shut off T-cells. This leads to immune checkpoint inhibitors needing to be personalized for each patient, enabling them to block the specific pathway the patient's cancer is using. For immune checkpoint inhibitors to work, oncologists must first determine the specific pathway the cancer is using to shut off the T-cell. After this determination, scientists genetically modify the T-cells to inhibit this particular pathway (Sharpe & Mount, 2015). The most common pathways cancer uses to deactivate T-cells are program death 1 (PD1) and cytotoxic T lymphocyte-associated antigen 4 (CLTA-4) (Kiyotani et al., 2021). Understanding the cancer patient's genetic profiles allows for more informed decisions about which immune checkpoint inhibitor to utilize.

Immune checkpoint inhibitors are great at increasing the efficiency and ability of T-cells, but it comes with some heavy downsides. For example, conventional chemotherapy drugs can shrink a tumor within a few weeks; immune checkpoint inhibitors can take several months to work (West, 2015). In addition, due to immune checkpoint inhibitors removing T-cell's inhibitory signals, in 10% of patients, this makes them unable to differentiate between healthy and cancerous cells, leading to an imbalance of the immune system and the death of many healthy cells (West, 2015). However, on the flip side, the side effects often due to immune checkpoint inhibitors are mild, including diarrhea, rash, difficulty breathing, eye irritation, jaundice, and a change in energy level. In most cases, patients on immune checkpoint inhibitors experience excellent quality of life with minimal symptoms (D. B. Johnson et al., 2017). Immune checkpoint inhibitors come with one notable advantage over other cancer treatments. Enhancing the cell-mediated immune response against cancer cells generates a long-term memory lymphocyte

population that patrols the body and attacks the growth of new cancer cells or tumors (Lee et al., 2016). This is highly beneficial as it sustains the therapeutic effects, allowing the body to continue attacking cancer cells in case of a relapse (Lee et al., 2016). Recently, researchers found that the use of immune checkpoint inhibitors led to a significant increase in survival for patients with metastatic melanoma and non-small-cell lung cancer, cancers where many conventional cancer therapies have failed (Lee et al., 2016). Ultimately, immune checkpoint inhibitors are considered one of the best cancer treatments for low body toxicity and long-term therapeutic sustainability. However, its difficulty with large tumors poses a challenge to utilizing this treatment. Therefore, many cancer cases still recommend another treatment strategy in addition to immune checkpoint inhibitors.

Integrative Cancer Treatment in Practice

Given the unique characteristics of cancer, cancer treatment often entails an integrative approach. Breast cancer is one example that has benefited from the unison of conventional and personalized medicine. For example, surgery is frequently used in combination with chemotherapy to remove the tumor or sometimes even the entire breast before treating the cancer cells. When utilizing chemotherapy in breast cancer, it can be used preoperatively and postoperatively (Trayes & Cokenakes, 2021). Preoperatively, chemotherapy is used to either shrink the tumor or stop/slow the spread of cancer cells to other parts of the body; if the cancer does not contain estrogen, progesterone, or ERBB2 receptors, chemotherapy is the only form of cancer treatment that will be effective as hormone therapy is unsuited (Trayes & Cokenakes, 2021). If used postoperatively, chemotherapy is used to kill any remaining cancer cells that could still linger after the tumor is removed, decreasing the chance of relapse.

Similar to chemotherapy, when radiation is used to treat breast cancer, it is used in combination with surgery. In most cases, radiation is used when a patient is receiving a lumpectomy: a slightly invasive way of removing cancerous cells in the breast. The role of radiation is to shrink the tumor and remove some cancer cells in the area of the cancer for the lumpectomy surgery to be less invasive and easier (Trayes & Cokenakes, 2021). The combination of a lumpectomy with radiation is considered a breast-conserving treatment, allowing the patient to maintain their breasts. Radiation is not typically used when the cancer in the breast is so invasive to the point where a mastectomy (complete removal of the breast) must occur (Trayes & Cokenakes, 2021).

Conventional therapies have shown to be relatively effective in the fight against breast cancer; however, the rise of personalized medicine has opened the door to an entirely new world of breast cancer treatment. CAR-T and immune checkpoint inhibitors have revolutionized the way we treat breast cancer—especially those with the HER2 mutation. The HER2 mutation is a common mutation in breast cancer patients that allows for the modification of T-cells to recognize TAAs (tumor-associated antigens). In recent clinical trials, HER2 targeting CAR-T treatment has been a game-changer in inhibiting the growth of tumors and metastasis (Yang et al., 2022). In the case of immune checkpoint inhibitors, their development and advancement have also been keen in the fight against breast cancer. This is due to most breast cancers expressing co-inhibitory molecules that can suppress T-cells (Bedognetti et al., 2016). Specifically, immune checkpoint inhibitors have been a game-changer in the application of triple-negative breast cancer (Gaynor et al., 2022). Recently, it was discovered that around 20%

of triple-negative breast cancers contain PD-L1, a ligand that suppresses T-cells (Gaynor et al., 2022). This means that by treating a patient who has triple-negative breast cancer with immune checkpoint inhibitors, the PD-L1 on the surface of the cancer cells will be unable to suppress the T-cell's immune response, and the cancer cell will be attacked (Gaynor et al., 2022). Currently, a combination of atezolizumab (immune checkpoint inhibitor) and nab-paclitaxel (chemotherapy drug) has been used to treat breast cancer patients (Gaynor et al., 2022). Ultimately, there are many ways in which breast cancer can be treated, all depending on the severity and type of breast cancer each patient has. There is no right or best treatment, leading to the use of a combination of treatments. The rise of personalized medicine has advanced the field of cancer and the way we treat and create treatment plans for patients, but an integrative approach is likely to be the key.

Conclusion

In conclusion, the path to curing cancer is not linear, and many factors must be taken into account when discussing cancer treatment. However, every day, scientists are creating more effective and novel treatments. The recent advancement from conventional therapy to personalized medicine has ushered us into a new day and age where cancer treatment is highly effective and tailored to one's genetics. All the moving parts that make cancer a complex disease to cure, such as heterogeneity and its ability to mutate and adapt to treatment continuously, are challenges that personalized medicine is pushing us to resolve. However, the creation of personalized medicine has yet to ultimately move us away from using traditional treatments such as chemotherapy and radiation. These treatments continue to be heavily involved in treating many different cancers and are still effective. Now, with the discussion of all the new and novel therapies we have today, it is crucial to understand the future of cancer research and where we are headed as a society. Today, a new and exciting piece of technology is being researched in the hopes of being able to detect cancer in its very early stages. This technology is called liquid biopsies; they have been around for some time but have yet to contain the sensitivity required to successfully identify early-stage cancer (Connal et al., 2023). This review paper is not a comprehensive evaluation of all possible cancer treatments, but serves as a good foundation for understanding the current state of oncology. Other treatments include hormone therapy, stem cell transplant, and photodynamic therapy. It is also essential to understand that methods to improve detection and prevention, such as liquid biopsy, exist as well (National Cancer Institute, 2017). In addition, advancements in detection and diagnosis are vital to improving the quality of life in patients, decreasing the mortality rate, and decreasing its prevalence in society. Ultimately, this paper is merely a preview into the world of cancer research, and there are still hundreds of conventional and personalized cancer treatments out there that were not touched on. With cancer’s devastating impact on human health, now more than ever, it is essential to understand the complexities of cancer and the current approaches available to help tackle this ever-changing problem in the future.

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