Advances in Personalised Medicine for Cancer Treatment in Patients with Metastatic Disease: A Comparative Review of Recent Innovations

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ABSTRACT

OBJECTIVE

This study aims to review recent advancements in personalised medicine in metastatic cancer treatment. It compared the efficacy of different personalised medicines, immunotherapies, and non-invasive diagnostic approaches across different cancer types.

METHODS

A comparative review was conducted using a systematic search of peer-reviewed literature. Clinical trials published between 2014 and 2024 were included. Data sources included PubMed, MEDLINE, and Scopus were searched. Studies focusing on personalised medicine, targeted therapies, immunotherapies, and companion diagnostics were selected based on relevance and quality. The review synthesises evidence on treatment efficacy, survival rates, and biomarkers, comparing outcomes across different cancer subtypes.

FINDINGS

The review identified that targeted therapies, such as epidermal growth factor receptor (EGFR) and B-Raf proto-oncogene (BRAF) inhibitors, improve treatment outcomes by addressing specific genetic alterations in cancer cells. Immunotherapies, including chimeric antigen receptor T (CAR-T) cell therapy and immune checkpoint inhibitors (ICIs), showed promising results in hematologic malignancies. However, their effectiveness in solid tumours remains variable. Liquid biopsies also emerged as a valuable non-invasive diagnostic tool that enables the real-time monitoring of tumour progress and improves patient management.

CONCLUSION

Personalised medicine is transforming metastatic cancer treatment by offering tailored therapy based on genetic and molecular tumour profiles. However, challenges such as drug resistance, high costs, and ethical concerns persist. Future research should focus on overcoming these barriers to optimise personalised cancer care.

Keywords: Metastatic cancer, Personalised medicine, Targeted therapies, Immunotherapy, Liquid biopsy

Introduction

Metastatic cancer is a clinical stage when cancer spreads to other parts of the body from its origin. It is the most challenging stage in the treatment and prognosis of cancer. Poor survival outcomes and higher treatment costs associated with metastatic cancer make it one of the major global health concerns.[1–5] In 2022, over 18 million people were living with a history of cancer in the United States (US). This number is expected to increase to over 22.1 million by 2030.[6–8] The increase in the ageing population is the main contributor to the increase in the cancer incident rate over the past two decades.[2] The incident rates of metastatic cancer have rapidly increased between 2016 and 2018 in the United Kingdom (UK).[8] It is estimated that more than 623,000 individuals are currently living with metastatic cancer in the US. This number is estimated to increase to 700,000 by 2025.[9]

Metastatic disease has a poor survival rate, around 5 years, which varies depending on the type of cancer.[6] Metastatic lung and breast cancers are identified to have further low outcomes. Metastatic lung cancer has only 9% 5-year survival rate, while metastatic breast cancer has 30% 5-year survival rate in women and 19% in men.[4] Despite the advancement in the medical field and treatment options, the incidence of metastatic disease remains high. The median survival time is predicted to be only 10 months, and the disease affects 53 out of every 100,000 people.[5–7]

Radiation therapy, chemotherapy, immunotherapy, and surgery are common treatment options for metastatic cancer. The treatment plan often utilised a combination approach to address the complex and resistant nature of the disease.[7] In recent practice, personalised medicine, such as targeted therapies and immunotherapies, has gained sufficient medical attention. These treatment approaches are adapted to a patient's genetic and molecular tumour profile and have shown promise in improving patient outcomes.[10] Personalised medicine is an emerging concept based on offering therapies to the individual characteristics of each patient; therefore, it is also known as precision medicine.[11]

Personalised medicine is designed to increase treatment efficacy and avoid the harmful side effects of traditional treatments. Traditional treatments, such as chemotherapy, have limited efficacy due to their nonspecific nature.[11,12] Personalised medications use patient-specific data, such as immunologic markers and genomic information, to target tumours. CAR T-cell therapy and monoclonal antibodies are targeted approaches that have shown promising outcomes by improving the patient's immune system to fight against cancer.[13]

The concept of personalised medicine has gained more popularity in metastatic cancer treatment as treatment becomes challenging. The high degree of inter-patient and intra-tumour heterogeneity among patients with metastatic cancer suggests that cancer in each patient is genetically and biologically unique. Therefore, the standardised treatment approaches become less effective due to this patient variability.[12,13] Personalised medicine and precision therapies utilise advanced approaches, including companion

diagnostics and biomarker-driven therapies. Crizotinib and Olaparib are the personalised treatment approaches based on discovering gene mutations, the *ALK* gene in non-small-cell lung cancer (NSCLC) and the *BRCA* gene in ovarian cancer. These approaches can improve survival rates and quality of life for patients with metastatic cancer.[14–16]

This review compared the recent innovations and advancements in personalised medicine for metastatic cancer treatment. This review summarised key developments in personalised medicine to offer a better understanding of their efficacy across different cancer types and patient populations. This review identifies the most effective treatment option, helping clinicians refine their therapeutic strategies and adapt them to individual patient needs.

Methods

Study Design

A comparative review methodology was adopted in this study, focusing on recent innovations in personalised medicine for metastatic cancer treatment. The study analyses current literature and available clinical trials to evaluate the impact of emerging treatment strategies.

Personalised medicine approaches were studied, including immunotherapy, targeted therapies, and companion diagnostics. We compared their clinical outcomes across different cancer types. This study evaluated the advancements and challenges in personalised cancer treatment by synthesising evidence from peer-reviewed sources.

Search Strategy

A systematic search was conducted across various data sources, including PubMed, MEDLINE, and Scopus. The search strategy was employed to gather updated and relevant data on personalised medicine for metastatic cancer. The search was limited to publications from the past 10 years, from 2014 to 2024. Studies published only in English were included to ensure accessibility and relevance. Boolean operators were used to refine the search. The terms such as "personalised medicine" AND "metastatic cancer" AND ("targeted therapies" OR "immunotherapy" OR "companion diagnostics") AND ("vemurafenib" OR "erlotinib" OR "gefitinib" OR "cetuximab" OR "trastuzumab" OR "pembrolizumab") were used. Inclusion criteria were applied to select studies involving meta-analyses and clinical trials. It helped to provide a foundation for evaluating the efficacy and impact of these innovations on clinical outcomes.

Study Selection

The retrieved studies were selected based on their relevance to the objective of this review. Only peer-reviewed articles were selected, considering the quality of research and the extent of innovation reported. The selection process involved screening the abstracts and titles to ensure their relevance to the topic. The selected studies were then reviewed to assess their relevance to the research objective and evaluated based on inclusion criteria. Studies that appeared to meet these criteria were further evaluated by reading the full text. The focus was placed on assessing the quality of the research based on the methodology adopted, study design, sample size, and data on treatment efficacy and safety.

Data Extraction and Synthesis

Data were systematically extracted from the selected studies meeting the inclusion criteria. The key data points included the approach to personalised medicine, cancer type, clinical outcomes, survival rates, biomarkers, and patient subgroups. Two reviewers were independently involved in the data extraction to ensure accuracy and minimise selection bias. Data synthesis involved the systematic recording of information on the effectiveness of specific treatments.

Extracted data were compiled and synthesised based on the identified trends, treatment efficacy was compared, and personalised treatments' impact on patients' clinical outcomes was assessed. This synthesis aimed to provide a clear overview of the current advancements and innovations in personalised medicine for metastatic cancer.

Comparative Analysis of Innovations in Personalised Medicine

Genomic and Targeted Therapies

Recent studies[15–18] have identified that genomic alterations and instabilities are the main factors that influence tumorigenesis and metastasis. Therefore, clinicians focus on personalised treatment approaches that address these genomic complexities. Targeted therapies inhibit tumour metastasis by targeting specific genetic mutations or proteins in cancer cell growth. Hence, they offer a personalised approach to cancer treatment.[16]

Epidermal Growth Factor Receptor (EGFR) Inhibitors

EGFR is a transmembrane glycoprotein involved in cell signalling pathways. It regulates cell proliferation, cell survival, and metastasis. It is an important receptor tyrosine kinase family member and involves normal cell growth and development.[15–18] EGFR inhibitors have been identified as a significant class of targeted treatments in recent studies. Several EGFR receptors have been identified, including HER1 (ErbB1), HER2 (ErbB2), HER3 (ErbB3), and HER4 (ErbB4).[15] These receptors are important in RAS/RAF/MEK and PI3K/AKT/mTOR signalling pathways.[16] Epidermal growth factor and transforming growth factor act as ligands for EFGR activation.[17] Its activation can lead to receptor dimerisation, which affects intracellular signalling. It also promotes cell proliferation and inhibits apoptosis.

EGFR was first discovered in the early 2000s, and several studies have established target mutations and overexpression of EGFR in various cancers.[15,16] Specifically, NSCLC, colorectal cancer, and head and neck cancers are reported to have overexpression.

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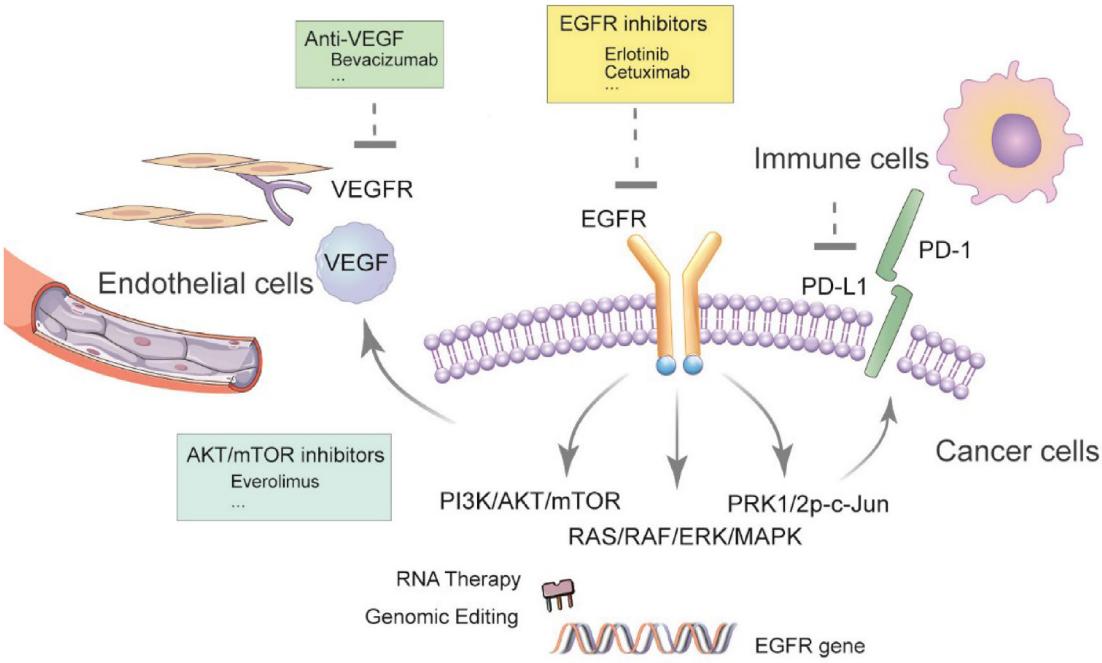

Fig 1 | Molecular targets and therapeutic strategies in cancer treatment

Source: Vennepureddy et al.[17]

Note: This figure highlights key molecular targets in cancer cells and the corresponding therapies. Anti-VEGF agents like Bevacizumab inhibit VEGF signalling to reduce angiogenesis in tumours. EGFR inhibitors such as Erlotinib and Cetuximab block the EGFRs. It prevents downstream signalling pathways like PI3K/AKT/mTOR and RAS/RAF/ERK/MAPK. These pathways are involved in tumour cell proliferation and survival. AKT/mTOR inhibitors (e.g., Everolimus) target cancer growth via the PI3K/AKT/mTOR pathway. ICIs (e.g., PD-1/PD-L1 inhibitors) stimulate immune cells to attack cancer cells by preventing the PD-1/PD-L1 interaction. RNA therapy and genomic editing represent future precision medicine approaches targeting the EGFR gene.

Kelly et al.,[18] have studied the various EGFR inhibitors, including Erlotinib, Gefitinib, and Osimertinib. They discussed that these inhibitors are particularly effective in patients with specific EGFR mutations. These mutations are identifiable through genetic testing (Figure 1).

Studies[13,14] have also reported that EGFR can be a critical oncogenic driver in 20–40% of NSCLC cases. In these cases, mutations such as exon 19 deletions and exon 21 L858R were commonly observed. However, some studies[14] also reported that EFGR can influence treatment resistance in cases with mutations like *T790M*. Advanced therapies such as third-generation TKIs like Osimertinib are recommended due to their demonstrated efficacy, particularly in managing brain metastases.

Ayati et al.,[13] reported that a combination of Capmatinib and Gefitinib has promising efficacy, particularly in patients with MET-dysregulated NSCLC. In phase II of their trial, the overall response rate (ORR) was 29%, suggesting better outcomes in patients with higher MET gene copy numbers. These results reinforce the potential of targeting EGFR and MET pathways in overcoming acquired resistance to EGFR-TKIs. In a study conducted by Diociaiuti et al.,[14] the efficacy of Cetuximab, another EGFR inhibitor, was discussed. In this study, Cetuximab was observed to impact treating metastatic cutaneous squamous cell carcinoma (SCC) in patients with dystrophic epidermolysis bullosa. Their results showed varying responses based on EGFR expression levels. These results suggest that an early administration of such inhibitors can improve survival and quality of life among patients with advanced or inoperable SCC.

Another study by Folprecht et al.,[15] has discussed the combined effect of FOLFOXIRI with anti-EGFR MoAbs on metastasis. Their results suggest that this combined approach has a higher ORR of 87.3% in metastatic colorectal cancer (mCRC) patients. However, they also reported some challenges, including similar mPFS and mOS compared to control due to surgical resections, tumour heterogeneity, and potential gastrointestinal toxicity. Studies[13–15] have also discussed novel approaches of combining EGFR and ICIs to address the above challenges. External quality control and more sensitive testing methods are also recommended to ensure accurate molecular characterisation in determining treatment efficacy.

B-Raf proto-oncogene (BRAF) Mutations and Targeted Therapies

BRAF gene encodes a protein kinase involved in the MAPK signalling pathway, essential for cell growth and differentiation.[16,17] Molecular studies[16] have shown that mutations in *BRAF*, particularly the *V600E* mutation, are most common in melanoma and can cause oncogenic signalling. *BRAF* has been linked to melanoma progression and influences angiogenesis by activating VEGF secretion. Therefore, it promotes tumour growth and metastasis through

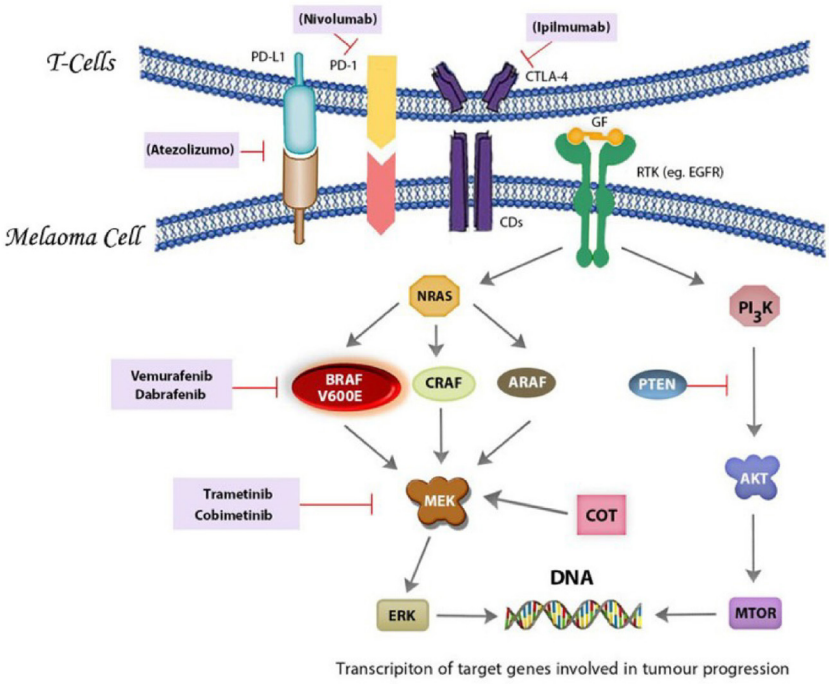

Fig 2 | Signalling pathways and therapeutic targets in melanoma treatment

Source: Alqathama et al.[16]

Note: This figure illustrates key signalling pathways involved in melanoma progression. The BRAF V600E mutation activates the MAPK/ERK pathway. It promotes tumour growth. Inhibitors like Vemurafenib and Dabrafenib target this mutation. Trametinib and cobimetinib inhibit MEK. They downstream BRAF to prevent tumour cell proliferation. Immunotherapies such as Nivolumab (PD-1 inhibitor), Atezolizumab (PD-L1 inhibitor), and Ipilimumab (CTLA-4 inhibitor) enhance T-cell responses against melanoma cells. Additional pathways like PI3K/AKT further influence tumour progression and are potential therapeutic targets.

enhanced blood vessel formation and pro-angiogenic signalling. *BRAF* inhibitors target this mutated kinase and inhibit its activity to disrupt tumour growth and progression (Figure 2).

Alqathama[16] studied the repressive action of *BRAF* inhibitors and discussed that they work by blocking the aberrant *BRAF* kinase activity, interrupting the *MAPK* signalling pathway that drives tumour growth. Recent advancements in *BRAF* inhibitors include third-generation pan-RAF inhibitors, LY3009120 and TAK-580. These inhibitors address the RAF dimerisation issues and prevent paradoxical ERK activation by inhibiting monomeric and dimeric RAF complexes.

BRAF inhibitors, including vemurafenib and dabrafenib, have been repeatedly reported[17] to show a higher clinical efficacy. Vemurafenib is a selective BRAF inhibitor which has demonstrated a 90% tumour regression rate in various clinical trials. Dabrafenib also exhibits high clinical response rates when combined with MEK inhibitors.[16] Other third-generation inhibitors like LY3009120 and TAK-580 are also reported in recent clinical trials to have promising outcomes in overcoming resistance and targeting a broader range of RAF mutations.[15,16]

The reviewed studies highlighted that *BRAF* inhibitors positively impact tumour prognosis and can substantially improve patient outcomes. Their observed efficacy includes extended progression-free survival (PFS) and reduced tumour burden.[16] According to Alqathama et al.,[17] combination therapies involving BRAF and MEK inhibitors have significantly reduced mortality risk and tumour progression. However, resistance is identified as the main challenge in adapting these combined approaches, as some patients reported developing secondary resistance mechanisms that limit the long-term efficacy of these treatments. Therefore, the current focus of the ongoing research is to enhance the efficacy of these therapies and address resistance through novel inhibitors and combination approaches.

Immunotherapies: Advances, Comparison, and Outcomes

Immunotherapy is another novel cancer treatment approach that harnesses the body's immune system to target and eradicate cancer cells. Studies have reported various strategies for immunotherapy, including chimeric antigen receptor T (CAR-T) cell therapy, checkpoint inhibitors, and personalised vaccines; each has unique modes of action.[18–28] Their effectiveness varies depending on the type of tumour and stage of the disease. Moreover, patient-specific factors also influence the outcomes of these therapies.[20–22] We compared these immunotherapies, focusing on tumour regression rates, success in metastatic cases, and safety profiles (Table 1).

CAR-T Cell Therapy

CAR-T cell therapy is a novel approach that genetically modifies a patient's T-cells to express a receptor that targets tumour antigens.[19–21] CAR-T cell therapy has shown significant efficacy in haematologic malignancies, including acute lymphoblastic leukaemia and certain types of non-Hodgkin lymphoma.[20] CAR-T cells are engineered to recognise and attack tumour cells expressing surface markers, such as *CD19* in B-cell malignancies.[21–25] Qi et al.,[20] studied the impact of CAR-T cell therapy on two different metastatic pancreatic cancer patients. The patients were treated with Claudin 18.2 (CLDN 18.2) CAR-T cell therapy after standard treatments failed. The use of CAR-T cell therapy showed promising results. They reported that one patient achieved a partial response with significant tumour shrinkage, while the other achieved a complete response with sustained tumour control.

Wang et al.,[24] also studied the impact of CAR T cells on tumour prognosis. They reported that a phase I trial of CD133-directed CAR T cells (CART-133) in patients with advanced CD133-positive malignancies demonstrated feasibility and manageable toxicity. Of the 23 patients treated, 3 achieved partial remission and 14 achieved stable disease. In contrast, a 3-month disease control rate of 65.2% and a median PFS of 5 months were also observed.

In another study by Frieling et al.,[27] γδ-enriched CAR-T cell therapy positively impacted tumour regression and improved survival rate. They used CAR-T cell therapy to target prostate stem cell antigen combined

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Table 1 | Summary table

Type of Therapy		Key Findings			Study Reported
		Efficacy	Challenges	Patient Outcomes	
Targeted therapies	EGFR inhibitors	Erlotinib and Osimertinib improve response rates and survival in NSCLC patients with specific EGFR mutations (e.g., exon 19/21).	Resistance due to T790M mutation is common with Osimertinib (3rd-gen TKI).	Enhanced survival and PFS in NSCLC. Resistance mechanisms are a barrier to long-term efficacy.	[13–18]
	BRAF inhibitors	Vemurafenib and Dabrafenib effectively treat melanoma with BRAF V600E mutations, improving tumour regression and PFS.	Combination with MEK inhibitors reduces resistance, but secondary resistance remains a challenge in many cases.	Improved tumour regression and extended PFS in melanoma patients; secondary resistance reduces the long-term efficacy.	[16,17]
Immunotherapies	CAR-T cell therapy	CAR-T cell therapy is highly effective in haematologic malignancies. It can achieve remission in ALL and B-cell cancers.	There is limited efficacy in solid tumours due to poor cell migration and an immunosuppressive tumour microenvironment.	Complete and partial remissions in blood cancers but minimal success in solid tumours like melanoma.	[19–25]
	ICIs	ICIs (e.g., PD-1, PD-L1 inhibitors) show modest responses in metastatic breast cancer and better outcomes in PD-L1-positive tumours.	Unpredictable toxicity patterns and delayed onset of responses. Pseudo-progression and hyper-progression can complicate treatment assessments.	Improved survival rates in patients with breast cancer and melanoma. Success is greater when combined with chemotherapy or targeted therapies.	[26–29]
Combination therapies	BRAF + MEK inhibitors	Combined BRAF and MEK inhibitors reduce tumour progression in melanoma patients with BRAF mutations.	Resistance mechanisms still limit long-term efficacy and can cause secondary progression despite initial positive responses.	Enhanced clinical efficacy in melanoma, improving PFS. Secondary resistance remains a challenge to long- term survival.	[16,17]
	EGFR + MET inhibitors	EGFR and MET inhibitors (e.g., Capmatinib + Gefitinib) show promising results in overcoming acquired resistance in NSCLC patients with MET dysregulation.	Challenges include toxicity and managing adverse effects due to combining agents. Poor treatment adherence.	Promising results in MET-dysregulated NSCLC patients, improved response rates, and a potential pathway to overcoming acquired resistance to EGFR inhibitors.	[13–15]
	EGFR + MoAbs	EGFR inhibitors combined with MoAbs (e.g., Cetuximab) show success in mCRC patients, significantly increasing ORRs.	Treatment-related gastrointestinal toxicity and surgical resection challenges. It can limit OS despite improved ORR.	Higher ORR in mCRC patients. Although OS may remain like controls due to tumour heterogeneity and other complications.	[15,16]
Non-invasive Diagnostics	Liquid biopsies (ctDNA-based)	Liquid biopsies allow for non-invasive, real-time monitoring of tumour evolution. Low treatment resistance makes them valuable in personalised cancer care.	Sensitivity and specificity issues may limit their effectiveness in early-stage cancers or low-burden diseases. It requires improvements in biomarker isolation.	Easier sampling and real-time monitoring reduce patient discomfort, with the potential for early disease detection and personalised treatment adjustments.	[31–33]

Note: EGFR: epidermal growth factor receptor; NSCLC: non-small cell lung cancer; BRAF: B-Raf proto-oncogene; CAR-T: chimeric antigen receptor T-cell; ICIs: immune checkpoint inhibitors: and ctDNA: circulating tumour DNA.

with zoledronate in a murine model of bone metastatic castrate-resistant prostate cancer. This therapy was found to have enhanced antitumor efficacy and reduce cancer-associated bone disease.

Simon and Uslu[23] studied the applications of CAR-T cell therapy in melanoma. They reported that CAR-T cells have remarkable success in treating haematological malignancies. However, their efficacy in solid tumours like melanoma has been less impressive and non-effective. They also highlighted various challenges during CAR-T cell therapy, including cell migration to tumour sites, the development of the immunosuppressive tumour microenvironment, and the difficulty in identifying safe target antigens. These challenges suggest the need for novel approaches to overcome. Stern et al.,[22] studied the challenges of using CAR-T cell therapy in solid tumours. While CAR-T cells have successfully treated haematological cancers, their efficacy in solid tumours remains limited.

ICIs

ICIs block proteins that prevent immune cells from attacking tumour cells.[26,28] This action enhances the body's ability to combat tumours and inhibit growth. ICIs target proteins such as PD-1 and PD-L1, which tumours use to evade immune surveillance.[26] Several studies[26,28,29] have discussed the efficacy of ICIs in metastatic cancers. Zou et al.,[28] reported that ICIs demonstrate a modest overall response in metastatic breast cancer. However, patients with PD-L1-positive tumours and those receiving first-line therapy have higher efficacy. They also suggested predictive factors for better response included high tumour-infiltrating lymphocytes and $CD8^+$ T-cell levels, while liver metastasis was associated with poorer outcomes.

Heeke et al.,[26] reviewed the application of checkpoint inhibitors in metastatic triple-negative breast cancer. They focused on FDA-approved therapies such as atezolizumab and pembrolizumab. Outcomes

revealed promising activity with these inhibitors, particularly in combination with chemotherapy. Their results showed enhanced antitumor effects and provided insights into predictive biomarkers for future treatment strategies. Vosoughi et al.,[29] evaluated survival outcomes for patients with melanoma brain metastasis while using checkpoint inhibitors and targeted therapies. They found a notable improvement in overall survival (OS), specifically with anti-PD-1 therapy. They also identified factors influencing survival outcomes, such as treatment modality and number of brain lesions.

Studies[28,29] have also reported several challenges in the use of ICIs, including unpredictable toxicity patterns, delayed onset of adverse events, and difficulties in assessing true efficacy due to phenomena like pseudo-progression and hyper-progression. Clinical trials often struggle with suboptimal endpoint selection, and many trials focus on similar agents. Despite these challenges, ICIs are considered a significant opportunity for advancement in cancer treatment as they improve the immune system, leading to long-term responses and durable remissions. However, studies have also highlighted that innovations in trail designs and personalised dosing strategies are needed to establish a safety profile of ICIs. The discovery of new biomarkers could also help enhance the efficacy and safety of these promising therapies.

Vosoughi et al.,[29] conducted a retrospective study to assess survival outcomes in patients with melanoma brain metastasis. Their trial used checkpoint inhibitors and targeted therapies and reported several challenges. The unpredictability and delayed onset of adverse effects and the complexity of assessing true treatment response due to phenomena like pseudo-progression and hyperprogression were discussed as main challenges. Their findings also suggest that ICIs have the potential to offer durable responses and extended survival rates. They emphasised the promise of personalised treatment approaches to enhance patient outcomes and broaden the applicability of ICIs.

Liquid Biopsies and Non-invasive Diagnostics

Liquid biopsies are a revolutionary advancement in non-invasive cancer diagnosis, particularly those focusing on circulating tumour DNA (ctDNA). They offer numerous advantages over traditional tissue biopsy methods.[31–33] As Marrugo-Ramírez et al.,[31] highlighted, conventional tissue biopsies involve invasive procedures to extract solid tumour samples. While they offer detailed genetic and biomarker information, they bring risks, discomfort, and logistical challenges. These biopsies are often costly, time-consuming, and may not be feasible for certain tumours in inaccessible locations.

De Miguel et al.,[32] reviewed the emerging role of liquid biopsy and suggested it as a non-invasive alternative to traditional tissue biopsy techniques. Their study discussed that ctDNA, circulating tumour cells (CTCs), and other tumour-derived materials in blood plasma can offer sufficient genetic information. Liquid biopsy has several advantages, including reduced risk, lower cost, quicker turnaround time, and addressing the challenges of tissue heterogeneity and sample accessibility.

Lone et al.,[33] reviewed the transformative potential of liquid biopsies in clinical oncology. They discussed the non-invasive techniques that isolate tumour-derived entities, such as CTCs, ctDNA, and tumour extracellular vesicles, from body fluids. They found several advantages of liquid biopsies over traditional invasive biopsies. These advantages include ease of tumour sampling, continuous monitoring, personalised treatment, and screening for therapeutic resistance. They also discussed their growing role in understanding tumour characteristics and progression.

The novel ctDNA-based liquid biopsies analyse genetic material released from tumours into the bloodstream through a simple blood draw. According to Marrugo-Ramírez et al.,[31] this technique can eliminate the need for surgical intervention, reduce patient discomfort, and can be performed repeatedly. It provides a dynamic view of tumour evolution and treatment response.

Liquid biopsies are particularly useful for monitoring metastasis and understanding tumour dynamics, which are crucial for personalised treatment strategies.

However, studies have also highlighted several limitations in the use of liquid biopsies. The sensitivity and specificity of liquid biopsy can vary, and it may not detect all genetic alterations, especially in early-stage cancers or low-burden disease.[31–33] Improvements in biomarker isolation technologies are recommended to enhance their clinical utility. Enhanced purification methods for ctDNA are expected to enhance further the clinical use of liquid biopsies, which have the potential for earlier disease detection and more personalised treatment options.[31]

Results and Discussion

Efficacy of Personalised Approaches in Clinical Settings

Our review highlighted the significant advancement in personalised therapies for metastatic cancer. It is indicated that targeted therapies, including EGFR and BRAF inhibitors, have shown improved treatment outcomes by addressing the tumour genetic alterations. EGFR inhibitors, such as Erlotinib and Osimertinib, have been notably discussed in recent literature to show good efficacy in metastasis patients with specific EGFR mutations.[13–16] These inhibitors have demonstrated better response rates and prolonged survival in NSCLC and other malignancies. Similarly, BRAF inhibitors have shown substantial benefits in melanoma. They demonstrated tumour regression and extended PFS in patients with BRAF V600E mutations.[14,15] Immunotherapies, including CAR-T cell therapy and ICIs, have also shown promising outcomes. These therapies are effective, particularly in haematologic malignancies, although their effectiveness in solid tumours remains variable.[17–22]

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Impact on Patient Survival and Quality of Life

Comparative data on OS and PFS across different personalised therapies revealed that these approaches substantially improve patient outcomes. Some studies have demonstrated enhanced OS and PFS with EGFR inhibitors in NSCLC and BRAF inhibitors in melanoma.[13–18] The combined approach of targeted therapies, such as ICIs or other targeted agents, has been discussed to improve patient outcomes further.

However, certain challenges, such as drug resistance and treatment-related adverse effects, continue to impact the efficacy and patient quality of life.[19–22]

Impact of Personalised Medicine on Metastatic Cancer Treatment Outcomes

Our review highlights the transformative impact of personalised medicine on metastatic cancer treatment. We identified a trend of increased use of targeted therapies and immunotherapies in cancer treatment, which has led to improved treatment outcomes and patient-specific approaches. Personalised medicine has significantly changed treatment practices by offering personalised therapeutic options based on genetic and molecular tumour profiles. These modified approaches showed enhanced efficacy and better patient management.[2–8]

Challenges Identified

Our review identifies several challenges in the application of personalised therapies. Drug resistance remains a significant challenge, with mutations like *T790M* requiring more advanced therapies.[7–12] Cost barriers to novel therapies also pose difficulties and limit their widespread application. Ethical concerns regarding using advanced genetic testing and the equitable distribution of personalised treatments are also crucial considerations. Ongoing debates in personalised cancer care focus on concerns about overtreatment, as personalised approaches may sometimes lead to excessive or unnecessary interventions.[14–18] Accessibility to advanced therapies remains a challenge, with disparities in treatment availability across different regions.

The generalisability of findings from clinical trials to broader patient populations is also a subject of concern. These challenges emphasise the need for continued research and innovation to address limitations and improve therapeutic strategies.[22,23]

Future Directions in Personalised Medicine

The future of personalised medicine in cancer treatment holds immense potential. Advancements in genomic profiling, artificial intelligence, and non-invasive diagnostics have driven this field forward. Technologies like liquid biopsies are expected to improve real-time monitoring of tumour evolution. It can allow for more dynamic treatment adjustments based on ctDNA and other biomarkers. Future research should aim to address resistance mechanisms in solid tumours. Moreover, clinical trials examining the combination of targeted medicines and immunotherapies such as CAR-T cells can also help establish treatment efficacy. Innovations in AI-driven diagnostics can potentially enhance patient-specific treatment recommendations by analysing vast datasets of genetic mutations and treatment outcomes. Personalised medicine will likely expand to more cancer types as these technologies evolve and can offer precision-targeted treatments and improved survival rates.

Conclusion

Personalised medicine has advanced the management of metastatic cancer by customising therapies to individual genetic and molecular profiles. These advancements have led to improved treatment outcomes and enhanced patient care. Targeted therapies, such as EGFR, BRAF inhibitors, and innovative immunotherapies, have shown promising outcomes. They showed extended survival and improved quality of life for patients with metastatic disease. Despite these advancements, several challenges were identified. They include drug resistance, high costs, and ethical considerations. Future research should focus on developing cost-effective solutions and improving diagnostic precision. Advancements in biomarker discovery and combination therapies could help optimise personalised medicine and expand its benefits to more patients.

Abbreviations

Abbreviation	full form
EGFR	Epidermal Growth Factor Receptor
NSCLC	Non-Small Cell Lung Cancer
HER1/ERBB1	Human Epidermal Growth Factor Receptor 1
HER2/ERBB2	Human Epidermal Growth Factor Receptor 2
HER3/ERBB3	Human Epidermal Growth Factor Receptor 3
HER4/ERBB4	Human Epidermal Growth Factor Receptor 4
CAR-T	Chimeric Antigen Receptor T-Cell Therapy
CD19	Cluster of Differentiation 19
PD-1	Programmed Death-1
PD-L1	Programmed Death-Ligand 1
BRAF	B-Raf Proto-Oncogene
VEGF	Vascular Endothelial Growth Factor
PFS	Progression-Free Survival
ORR	Overall Response Rate
CT041	CAR T Cell Therapy for Claudin 18.2-positive
TKIS	Tyrosine Kinase Inhibitors
MEK	Mitogen-Activated Protein Kinase Kinase
MCRC	Metastatic Colorectal Cancer
MOABS	Monoclonal Antibodies
mCRPC	Metastatic Castrate-Resistant Prostate Cancer
PSCA	Prostate Stem Cell Antigen
ZOL	Zeledronate

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