Adaptive Treatment of Metastatic Prostate Cancer Using Generative Artificial Intelligence

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ABSTRACT: Despite the expanding therapeutic options available to cancer patients, therapeutic resistance, disease recurrence, and metastasis persist as hallmark challenges in the treatment of cancer. The rise to prominence of generative artificial intelligence (GenAI) in many realms of human activities is compelling the consideration of its capabilities as a potential lever to advance the development of effective cancer treatments. This article presents a hypothetical case study on the application of generative pre-trained transformers (GPTs) to the treatment of metastatic prostate cancer (mPC). The case explores the design of GPT-supported adaptive intermittent therapy for mPC. Testosterone and prostate-specific antigen (PSA) are assumed to be repeatedly monitored while treatment may involve a combination of androgen deprivation therapy (ADT), androgen receptor-signalling inhibitors (ARSI), chemotherapy, and radiotherapy. The analysis covers various questions relevant to the configuration, training, and inferencing of GPTs for the case of mPC treatment with a particular attention to risk mitigation regarding the hallucination problem and its implications to clinical integration of GenAI technologies. The case study provides elements of an actionable pathway to the realization of GenAI-assisted adaptive treatment of metastatic prostate cancer. As such, the study is expected to help facilitate the design of clinical trials of GenAI-supported cancer treatments.

KEYWORDS: Generative artificial intelligence, cancer treatment, prostate cancer, adaptive therapy, intermittent androgen deprivation therapy, deep learning

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Introduction

Sustained world-wide efforts in basic and translational cancer research are expanding the repertoire of treatment options available to cancer patients. Notwithstanding the great strides this expanding cancer armamentarium is enabling towards improving patient outcomes, the emergence of therapeutic resistance is often inevitable for most cancer patients[1] and remains the ultimate barrier to achieving cancer cure.[2] This persisting reality of cancer treatments is perpetuated by the inability of standard of care (SOC) to effectively and fully consider the adaptive complexity of cancer[3] and its underlying eco-evolutionary,[4-6] genetic,[7-11] and immunological dimensions.[12-16] Indeed, the effective control of cancer as a time-varying nonlinear dynamical system cannot be achieved with predefined fixed schedules of therapeutic interventions.[17,18] Combination therapies have long been accepted as appropriate strategies to optimize treatment outcome, delay the onset of resistance, and reduce the risk of minimal residual disease.[19,20] On the other hand, adaptive therapy has been championed as an intuitively sound approach to address the evolutionary dynamics underlying resistance.[17,21-25] Adaptive combination therapy would further expand the horizon of possible improvements in treatment outcomes.[26,27] However, despite the tens of thousands of clinical trials for combination therapy that are currently registered in ClinicalTrials.org, it is not clear how to safely and effectively combine multiple drugs. Furthermore, significant advances are needed to address the feasibility, accuracy, and reliability of continuous disease monitoring and treatment response predictions to support adaptive therapy. Challenges to the development of more effective cancer treatments may however be more amenable to clinically viable resolutions by unlocking the potential of the growing big multimodal data being collected about cancer and the meteoric rise of data-driven generative artificial intelligence (GenAI).[28] Indeed, GenAI potential utility in oncology is garnishing increasing attention.[29,30] Notable explorations of GenAI applications to cancer care are many, including the use of large language models (LLM) such as ChatGPT as an assistant that can be queried about cancer by patients and health care practitioners,[31-36] extraction of clinical information from medical reports,[37-39] and clinical decision support for diagnosis and treatment recommendations.[40-44] Many of these studies explore the performance of pre-trained LLMs, such as ChatGPT, and often compare it against benchmarks and human medical expertise.[42,45-47] One particularly promising oncological application of GenAI is radiotherapy (RT) treatment planning, where dedicated LLM-enhanced algorithms have been developed to automatically delineate tumour volume targeted by radiotherapy.[44,48] Given the pathway that has already been chartered towards maturity and clinical adoption of ML/AI (machine learning/artificial intelligence) assisted diagnosis of cancer,[49] GenAI-augmented cancer diagnosis algorithms are expected to attract heightened research interests.[50-52] On the other hand, assessment of LLMs used for treatment recommendations revealed that they are still

no match for human medical experts.[53] Most of these explorations were undertaken in the context of standard of care, where traditional fixed schedules of treatments are used. For the case of adaptive cancer therapy, OncoGPT, a transformer-based model, was proposed for the prediction of treatment response.[28] The training of OncoGPT is assumed to place patients on a phenotypic space, in accord with their phenotypic similarity. This provides a basis for the plausibility of one-step-ahead predictions of treatment response, by generalizing from the treatment response trajectories of patients who underwent similar treatments.[28] Notwithstanding GenAI potential to revolutionize cancer care,[30,52,54] the use of LLMs faces challenges that are typical to ML/AI systems, including the lack of explainability, opacity, ethical concerns, and the need for large training datasets.[55-57] In addition, LLMs have an intrinsic risk for hallucinations,[58] which in the context of oncology means that they may yield incorrect or clinically implausible outputs such as nonsensical treatment recommendations.

To highlight some concrete aspects of the application of generative artificial intelligence to advance the ongoing progress in adaptive therapy for prostate cancer,[22,24,26,27,59-62] metastatic castrate-sensitive prostate cancer (mCSPC) is selected as a study case for the use of OncoGPT.[28] The proposed analysis spans GenAI model training and inferencing as well as mitigation strategies for the hallucination problem and its implications to clinical validation and integration. The analysis is prefaced with a short overview section on adaptive mCSPC therapy. This serves as a baseline for the proposed GenAI-assisted adaptive therapy of mCSPC, detailed in the subsequent sections. Insights are shared in the discussion section about the potential, challenges, and limitations of the proposed GenAI-assisted approach to the treatment of mCSPC.

Adaptive Therapy of Metastatic Castrate-Sensitive Prostate Cancer

Depending on factors such as disease volume and side effects, mCSPC may be treated with androgen deprivation therapy (ADT), combination of ADT and androgen receptor-signalling inhibitors (ARSI), or triplet therapy that includes ADT, ARSI, and docetaxel or radiotherapy.[63-66] Each one of these treatment modalities may be appropriate to select for a specific class of mCSPC patients based on risk stratification. Ultimately, treatment response will, for all patients, be time-varying and nonlinear, accompanied with an often-inevitable emergence of drug resistance due to the adaptive complexity of cancer and its underlying eco-evolutionary, genetic, and immunological dimensions.[3-16] Adaptive therapy has been argued to be the most sensible approach an oncologist would take to stay one step ahead of cancer's adaptation and resistance by changing the timing and treatment doses based on continuously monitored biomarkers.[17,22,24,67] Adaptive therapy was explored for metastatic prostate cancer, where treatment response is assumed to be determined by the competition between 3 types of cancer cells: (1) androgen receptor positive (AR+), CYP17+ testosterone-producing cells (TP), (2) AR+, CYP17− cells that require androgens (T+), and (3) AR−, CYP17− androgen independent cells (T-).[24,25,68] Based on this model, monitored prostate-specific antigen (PSA) and testosterone serum levels are used to guide the timing of intermittent ADT therapy in an adaptive androgen deprivation trial for mCSPC.[24] Depending on monitored testosterone and PSA levels, luteinizing hormone–releasing hormone (LHRH) antagonist, new hormonal agent (NHA) such as abiraterone, enzalutamide, or apalutamide, or LHRH + NHA were used to target a 50% PSA reduction, after which all treatments are intermittently stopped before they are resumed on PSA or radiographic progression.[24] It is presumed that the monitoring of PSA and testosterone provides sufficient information about the fractions of TP, T−, and T+ cells in the tumour to support the evolutionary-based adaptive treatment strategy described above. This model is aligned with the notion that the cell is the correct level of abstraction to consider in understanding function,[69,70] and it effectively integrates relevant genetic information (eg, expressions of AR and CYP17 and their relevant signalling pathways) into observable phenotypes, in a way akin to coarse-graining abstraction of knowledge.[71] The effectiveness of this adaptive treatment approach may be further enhanced by the use of GenAI models trained to learn patient treatment-response maps that would support treatment adaption through the provision of treatment response predictions.

GenAI-Supported Adaptive Androgen Deprivation of mCSPC

The application of GenAI to the prediction of treatment response was explored in the context of a theoretical framework for adaptive cancer therapy.[28] The framework views cancer treatment as a problem of controlling cancer as a time-varying nonlinear system whose states are observable through repeated monitoring of treatment response biomarkers covering the genetic, immunological, and eco-evolutionary causal dimensions underlying cancer dynamics. To leverage the learning capabilities of transformers[72] in adaptive therapy, treatments are represented by a sequence of therapeutic actions (controls) $u_0, u_1, \ldots, u_i$, where $u_i \in U$ is the control applied at the discrete instant of time identified by the non-negative integer i. U is a finite set of therapeutic actions defined through the consideration of available drugs, and the quantization of drug doses and time intervals between consecutive therapeutic actions. Likewise, treatment response is also framed to take discrete states $s_0, s_1, \ldots, s_i$, where s_i is the treatment response to the control u_i and $s_i \in S$, with S being a defined finite set of treatment responses, obtained through the quantization of biomarker signals of treatment response.

Considering the critical importance of clinical feasibility of future GenAI applications to cancer treatment, the Phase 1b Adaptive ADT trial[24] is taken as a baseline for the setup of this

case study. In particular, the objective of the case study is to demonstrate, at least theoretically, how OncoGPT[28] can be used to support adaptive ADT for the treatment of mCSPC. Although there are no limits on the number and type of biomarkers that can be used to define treatment response, for the purpose of anchoring this case study with respect to the mentioned clinical trial, PSA and testosterone are the only response biomarkers required to be monitored in addition to RECIST (response evaluation criteria in solid tumours)[73] assessments. Likewise, LHRH agonist/antagonist drugs and NHA are assumed to be the main types of drugs under consideration. To define the set of possible treatment responses (disease states), serum PSA is quantized into 30 possible discrete levels. The PSA range from 0 to 4 ng/mL is quantized into 20 distinct levels each representing a 0.2 ng/mL wide interval. For example, level 1 represents the range (0-0.2 ng/mL) while level 2 represents the range (0.2-0.4 ng/mL) and so on. The range (4-10 ng/mL) is divided into 5 equally wide intervals representing levels 21 to 25, respectively, while the PSA range (10-110 ng/mL) is divided into 4 equally wide intervals representing levels 26 to 29, respectively. PSA values that are higher than 110 ng/mL are represented by level 30. This quantization scheme attempts to achieve a monitoring resolution that recognizes PSA ranges that are clinically relevant for PSA screening.[74] Testosterone may be quantized into 20 discrete levels representing the intervals between the following possible testosterone values in nmol/L: 0, 0.1, 0.2, 0.3, 0.4, 0.5, 0.7, 0.9, 1.1, 1.3, 1.5, 1.7, 2, 5, 10, 15, 20, 25, 30, and 35 nmol/L. Here too, the quantization scheme is intended to provide a monitoring resolution that reflects clinical practice regarding salient ranges of testosterone levels for patients undergoing ADT.[75] Considering the 4 possible RECIST disease states leads to 2400 ($30 \times 20 \times 4$) possible treatment responses. Advances in the analysis of tumour circulating DNA (ctDNA) using liquid biopsy (LB) are expected to enable the use of additional biomarkers of treatment response,[76,77] further enhancing the resolution of treatment response monitoring. For instance, abundance of ctDNA, AR amplification, and alterations of PTEN, RB1, P53, and DNA damage repair (DDR) genes, including BRCA1/2, ATM, and CDK12, may be monitored using LB to serve as biomarkers of mPC treatment response.[77-81] In addition, cytokines such as interleukin IL-6, IL-5, IL-8, IL-10, IL-23, tumour necrosis factor (TNF)-α, and interferon (INF)-γ may also be monitored using LB[82] as biomarkers of the immune dimension of treatment response. Given the critical involvement of AR amplification in mCRPC,[83] the prognostic nature of ctDNA detection,[84] and the role of IL-8 in promoting the infiltration of myeloid-derived suppressor cells (MDSCs),[85] it is also possible to include the abundance of ctDNA, the amplification of AR, and IL-8 level as additional LB monitored biomarkers of mCSPC treatment response. Assuming 3 quantization levels that could be assigned to each one of these potential biomarkers (ie, Low, Medium, and High), this would bring the size of the set of disease states to 21 600.

The second step in defining the GenAI model is the discretization of therapeutic control. Relugolix[86] and abiraterone with prednisone[87] will be the example for LHRH and NHA drugs under consideration, respectively. Given the Food and Drug Administration (FDA)-recommended dosage of relugolix,[88] the dosage of this LHRH antagonist will be discretized into 7 different levels separated by an interval of 60 mg, where doses of 0 and 360 mg are mapped to levels 1 and 7, respectively. Likewise, given FDA abiraterone dosage recommendations,[89] the dosage of this NHA drug is discretized into 5 levels separated by an interval of 250 mg and were 0 and 1000 mg corresponding to levels 1 and 5, respectively. The dosage of prednisone will be tied to that of abiraterone, where 0, 1.25, 2.5, 3.75, and 5 mg would be administered for dosage levels 1, 2, 3, 4, and 5 of abiraterone, respectively. As a result of the above discretization of LHRH antagonist and NHA drugs, the total number of possible controls is 35 (7×5). Notice that no-treatment is represented by a zero value of drug doses.

Although therapeutic control is assumed to be applied daily through the administration of ADT + NHA drugs, the monitoring of PSA and testosterone and the assessment of imaging progression may be done once every few weeks. Blood tests, MRI (magnetic resonance imaging), and CT (computed tomography) routinely used in clinical setting would be the main methods used for biomarker monitoring. However, advances in LB and disease monitoring instrumentation, including portable devices, are expected to enable a more frequent sampling of treatment response. Meanwhile, the discrepancy between the frequency of drug administration and that of disease monitoring can be bridged through the use of estimated treatment responses regularly calibrated with the monitored treatment response as an injection of ground truth as illustrated in Figure 1.

The ground truth injection consists in using the recently monitored treatment response, ie, s_{i-1} to adjust the estimated treatment response sequence $\hat{s}_{i-N},\ldots\hat{s}_{i-2}\hat{s}_{i-1}$ prior to providing it as an input to OncoGPT. $N>0$ is the length of the treatment response history under consideration. One simple approach to implement this ground truth injection is to augment the elements of the estimated response sequence by $\vec{v} \triangleq s_{i-1} - \hat{s}_{i-1}$. Note that $\vec{v}$ is a vector whose components are equal to the differences between the corresponding components of treatment response and its estimate. The adjustment moves the sequence of estimated states in the direction of $\vec{v}$ towards the ground truth (ie, the last monitored treatment response) with a displacement equal to the magnitude of $\vec{v}$. The magnitude of the adjustment may also be modulated using a forgetting function to attenuate the impact of new monitored treatment responses on past, temporally distant estimations of treatment response. The adaptive therapeutic strategy of the

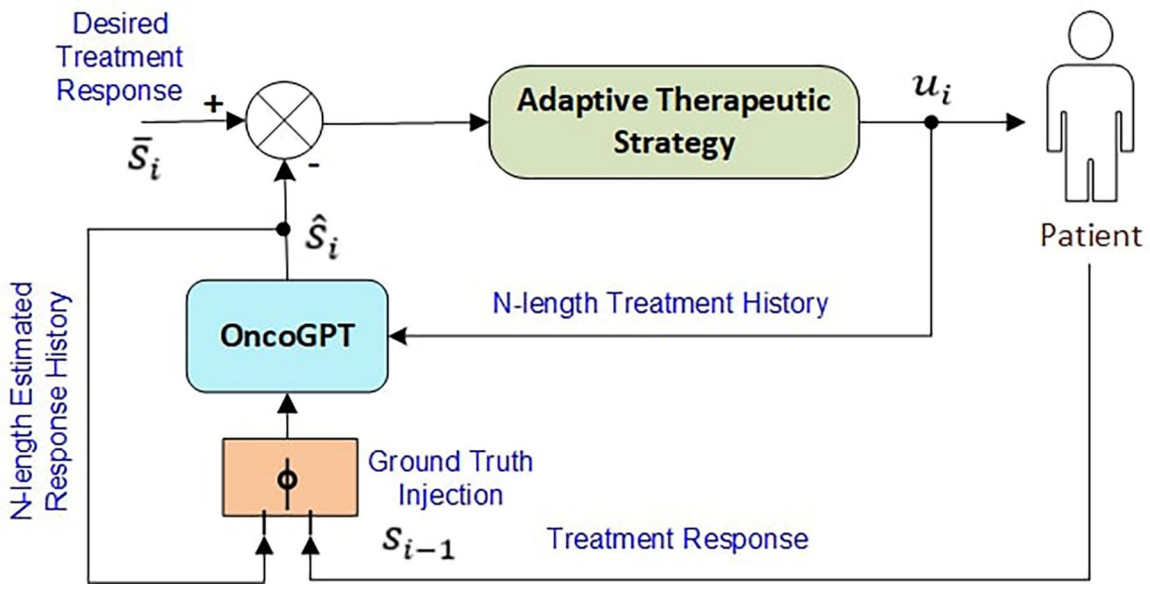

Figure 1. GenAI-supported adaptive androgen deprivation therapy for metastatic sensitive prostate cancer.

proposed GenAI-assisted treatment of mPC may be selected from the many possible adaptive control approaches proposed in the litterature,[18] based on their robustness and the feasibility of their clinical integration. Adaptive PID (proportional-integral-derivative) control embody time-proven, widely used real-world control strategy[90-93] and would hence be an adequate fit for the real-world clinical context. Planning the trajectory of desired treatment response used as the daily setpoint $\bar{s}_i$ for an adaptive controller would necessarily be informed by expert clinical knowledge about the dynamics of treatment response. Given a desired treatment duration of M days, an initial disease state s_0, and a target final disease state $\bar{s}_M$, the desired treatment response $\bar{s}_{i+1}$ at time $i+1$ may be set to the disease state closest, in Euclidean distance, to $\hat{s}_i + \frac{1}{M-i}\left(\bar{s}_M - \hat{s}_i\right)$. In other words, since $\bar{s}_M$ and $\hat{s}_i$ are vectors, the next desired treatment response is set to be one step worth $\frac{1}{M-i}\left(\bar{s}_M - \hat{s}_i\right)$ closer towards the final desired response. The planning of the desired treatment response trajectory may be improved through the consideration of treatment response dynamics and planning strategies available from other application domains.[94]

OncoGPT Training on mCSPC Treatment Response Data

OncoGPT uses the original encoder-decoder transformer architecture,[72] with the sequences of controls (treatments) and disease states (treatment responses) as inputs to the encoder and decoder, respectively. The encoder and decoder are constructed using deep neural networks, ie, neural networks with large number of hidden layers.[95] Both controls and disease states as well as their respective positions within their respective sequences are represented by embeddings, which are vectors of values. Embeddings are ultimately multiplied by weights to be learned through training using a dataset consisting of pairs $d \triangleq \left(\{u_i\}_{i=0}^{N-1}, \{s_i\}_{i=0}^{N-1}\right)$ of control sequences and corresponding sequences of treatment response. The sequence length N is the duration of one patient-treatment cycle, which would be chosen to account for the longitudinal causal dependencies between treatments and responses. N may be set to 256 days representing a 9-month-long patient-treatment cycle as a clinically plausible cycle duration. The training consists in adjusting the weights so as to minimize a so-called loss function which represent a measure of how far is the output of the transformer from the desired treatment response expected for a treatment provided as input. The weights are adjusted by minimizing the loss function using optimizers such as gradient descent,[96] and the backpropagation algorithm,[97] which backpropagates the error between the actual and desired output to the hidden layers of the network to adjust their weights. These weights represent the actual learning achieved by the transformer.

Response to mCSPC treatment is defined based on the 3 required biomarkers, namely, PSA, testosterone, and RECIST, augmented with IL-8 level, AR amplification, and ctDNA abundance as additional biomarkers. The discretization of these biomarkers leads to 21 600 possible disease states. On the other hand, therapeutic control based on the combination of LHRH antagonist (relugolix) and NHA (abiraterone) drugs is discretized into 35 possible controls, which include one no-treatment control. The numbers of possible controls and disease states for the specific application of OncoGPT to prostate cancer is far below the size of the vocabularies supported by LLMs such as CroissantLLM,[98] suggesting that the required size of the training dataset would be relatively modest. Given the inherent privacy challenges to the collection and curation of clinical data, the study case will explore the implementation and deployment of OncoGPT in the confine of a single health care organization. For example, assuming the average number of prostate cancer patients treated yearly in a major cancer centre to be 15 000, one may estimate that the records for at least

150 000 treatment cycles accumulated over a decade span would be available to train/retrain OncoGPT at any point in time. Treatment data size that could be curated to train OncoGPT for prostate cancer may reach millions of records through data sharing agreements under the umbrellas of consortiums such as the Prostate Cancer Clinical Trial Consortium (PCCTC). In addition, synthetic training data may be generated using clinically parameterized mathematical models of tumour dynamics such as the Lotka-Voltera model.[18] However, an even more promising approach to the curation of training data would be the use of patient-derived xenografts (PDXs), which are known to yield an accurate replication of treatment response.[99]

Given the different types of ADT drugs that have been used in prostate cancer treatment (eg, degarelix, abarelix, relugolix, leuprorelin/leuprolide, goserelin, and triptorelin), the curation of treatment data would involve discretizing the dosage of these drugs into levels as illustrated earlier for the examples of relugolix and abiraterone. Furthermore, as radiotherapy and chemotherapy (eg, docetaxel) may also be used in addition to ADT and NHA,[63] their dosage need to be also discretized into appropriate levels as part of the curation of treatment data. On the other hand, data for treatment response biomarkers are expected to be sparse and incomplete in treatment records being collected over long periods of time and across multiple institutions. In such cases, other recorded clinical and genetic treatment response variables may be used to estimate missing biomarker data towards the curation of a reasonably complete dataset for the training of OncoGPT.

Discussion

Predictions of treatment response based on curated treatment datasets are predicated on the assumption that these data embody the phenotypic diversity of the patient population. Furthermore, the tuning of OncoGPT into instances personalized to individual patients or groups of patients will require the curation of additional datasets covering patients that share some degree of phenotypic similarity which may be defined based on genomic, immunological, and eco-evolutionary biomarkers.[28] The collection and curation of quality treatment datasets in sufficient quantity to meet the needs of OncoGPT training face numerous challenges related to privacy, consent, and data ownership. These may likely be overcome within the respective confines of clinical and research institutions where compliance with protocols and quality control standards of data collection, curation, and use can be maintained and verifiably assured. Like any other deep-learning-based AI model, OncoGPT has limitations regarding transparency, bias, generalizability across health care communities, and performance drift.[100] In this respect, assuming the existence of data sharing frameworks among multiple cancer centres that enable the access to quality data in sufficient quantity to train OncoGPT, its deployment should be contingent on the local curation of training data to validate and regularly tune it for the target population.[100] Beyond these data-related challenges lies the fundamental question of how to mitigate hallucination, which is an unwelcome and inevitable feature of LLMs.[101] Among the many techniques of hallucination mitigation that have been proposed,[102] fine-tuning OncoGPT using a loss function specific to treatment response predictions and a high-quality treatment dataset may be most appropriate as this would be task-specific. However, irrespective of the mitigation method being used, it is critical to have an estimation of the effect of hallucination on the accuracy and reliability of treatment response predictions. Another equally important question is how potential metrics of accuracy and reliability would be used in the clinical validation of GenAI-supported cancer therapy. The accuracy of disease state predictions may be defined using the Euclidian distance between observed and predicted disease states given by $MSE=\frac{1}{N}\sum_{i=1}^{N}\|s_i-\hat{s}_i\|$ for a disease state trajectory $\{s_i\}_{i=1}^{N}$ corresponding to one treatment cycle, and where $\|\ \|$ is the Euclidean norm. Disease state trajectories reflect the dynamics of disease progression being controlled through therapy, supporting hence the clinical pertinence of this accuracy metric. In particular, E would quantify the extent to which OncoGPT have learned the dynamics of treatment response for the phenotypic classes of mCSPC patients on which it was trained. Phenotypic classification of training datasets may be carried out based on the status of genetic alterations affecting the PI3K and androgen receptor pathways, and DNA damage repair, given their clinical relevance to the treatment of mPC patients.[77] As treatment response predictions are generated within the context of an adaptive closed-loop involving the patient, assessing the predictive performance of OncoGPT would be normally carried out as part of the analysis of clinical trial results. The prediction accuracy of OncoGPT may be defined as $A=\frac{1}{M}\sum_i A_i$, $A_i=\frac{1}{K}\sum_j MSE_{ij}$, where M is the number of patients in the clinical trial, K is the number of treatment cycles per patient while MSE_{ij} is the prediction error for the jth treatment cycle of the ith patient. The reliability of treatment response prediction would also need to be assessed as an essential step towards clinical validation. Given the definition of reliability for measurements,[103] the reliability of treatment response predictions may be defined as the fraction of variance of disease state predictions that can be ascribed to the true variance of treatment response, namely: $R=\frac{\sigma^{(s)}}{\sigma^{(\hat{s})}}$, where $\sigma^{(s)}$, $\sigma^{(\hat{s})}$ are the variances of monitored and predicted treatment responses across all patients, respectively. As disease states are multi-dimensional variables, the variances $\sigma^{(s)}$ and $\sigma^{(\hat{s})}$ may be computed as the means $\frac{1}{L}\sum_i \sigma_i^{(s)}$ and $\frac{1}{L}\sum_i \sigma_i^{(\hat{s})}$ of variances for the L components of monitored

and predicted treatment responses, respectively. Accuracy and reliability metrics as defined above would be instrumental in improving trust in the performance of OncoGPT's support for adaptive therapy to improve treatment outcomes. On the other hand, the guidance issued by the FDA on the clinical evaluation of software as a medical device (SaMD)[104] provides an appropriate 3-pronged framework (ie, valid clinical association, analytical validation, clinical validation) for undertaking the clinical validation of GenAI-supported adaptive therapy systems. In particular, the metrics of accuracy and reliability defined above would be used to establish analytical validity, ie, that the predicted treatment response is what would be technically expected. Ultimately, clinical trials would be needed to establish the validity of clinical association between predicted treatment response and disease state as well as to clinically validate the performance of GenAI-assisted adaptive therapy with respect to clinical end-points such as time to progression, disease free survival, and overall survival.

Conclusions

GenAI-assisted adaptive androgen deprivation therapy is explored for the treatment of metastatic prostate cancer. OncoGPT is integrated in the treatment loop to predict treatment response. Both treatment and treatment response are discretized to yield finite sets of disease states and therapeutic controls to serve as the vocabularies underlying the sequences of controls and disease states used as inputs and outputs of OncoGPT, respectively. Disease states and therapeutic controls are defined based on biomarkers and drugs typically used in the treatment of mPC, respectively. The study case addresses the various steps and issues related formulation, training, integration, and assessment of OncoGPT-tailored application to adaptive androgen deprivation therapy for mPC. These include training data curation, model fine-tuning, and the exploration of treatment response accuracy and reliability as critical instrument in mitigating the risk of hallucination and improving trust in the use of GenAI to assist in improving cancer treatment outcomes.

Author Contributions

The author is the sole contributor to the article.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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