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MINIREVIEWS

Radiotherapy treatment time delay evidence, part I: Update on cervical, anal, prostate, and head and neck cancers

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Abstract

Treatment delays during radiotherapy for head and neck cancer (HNC) are a well-established factor negatively affecting clinical outcomes, with similar trends observed in other cancers. In this first part of a two-part review, we assessed the impact of overall treatment time (OTT) prolongation on locoregional control (LRC) and survival (SV) in cervical cancer (CC), prostate cancer (PC), and anal cancer (AC), while updating evidence for HNC. A comprehensive literature search was performed in evidence-based databases, including MEDLINE, identifying studies evaluating the relationship between OTT prolongation and outcomes. Particular attention was paid to the strength of evidence, distinguishing univariate analysis from multivariate analysis (MV-An). For CC, 37 articles were identified, with 88.8% reporting a detrimental impact on LRC and/or SV, mostly supported by MV-An. In AC, 15 studies were found, with 33.3% showing negative impacts, although with weaker evidence. For PC, 12 articles were reviewed, with 66.6% demonstrating detrimental effects mainly on LRC or biochemical control, and occasional associations with cancer-specific SV. Recent studies in HNC reinforced prior findings. When available, radiobiological parameters and practical recommendations are provided. In conclusion, strong evidence confirms that prolonged OTT worsens outcomes in HNC and CC, with less consistent but relevant effects in PC and AC.

Key Words: Radiotherapy; Overall treatment time; Delays; Locoregional control; Survival; Cervix; Anal; Prostate; Head and neck cancer

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Core Tip: Prolongation of overall treatment time (OTT) during radiotherapy significantly impacts locoregional control and survival outcomes across several tumor types. This mini-review highlights strong evidence supporting the detrimental effect of OTT delays in head and neck and cervical cancers, with moderate evidence for prostate cancer and emerging concern in anal cancer. Recognizing the critical role of maintaining planned treatment schedules, we emphasize the need for proactive management of interruptions and suggest practical recommendations based on current evidence. Early identification and mitigation of treatment delays can substantially improve oncological outcomes across multiple malignancies.

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INTRODUCTION

The time factor has long been recognized as a critical component in radiation oncology[1]. In particular, the detrimental effects of overall treatment time (OTT) prolongation during radiotherapy (RT), and the necessity of implementing compensation strategies, have been increasingly emphasized over the past decades[2]. Accelerated tumor cell repopulation is widely regarded as the main biological mechanism underlying the adverse impact of extended OTT on locoregional control (LRC) and various survival (SV) outcomes across multiple tumor types[3].

Due to ethical and logistical constraints, randomized clinical trials specifically designed to address the consequences of OTT prolongation are rarely feasible, resulting in a limited availability of high-level evidence. Consequently, comprehensive literature reviews remain the most effective tool for synthesizing existing data and guiding clinical practice.

In 2015, we published an extensive review focused on head and neck cancer (HNC), a disease site for which the most robust evidence on OTT effects exists, analyzing 62 studies and confirming a significant negative impact of treatment delays on both LRC and SV[4].

This manuscript represents the first installment of a two-part review series. Here, we examine the clinical consequences of OTT prolongation in cervical cancer (CC), anal cancer (AC), and prostate cancer (PC). Additionally, we provide an updated analysis of the HNC literature by incorporating studies published since our original review.

Based on our findings, we propose practical, evidence-informed recommendations for mitigating treatment delays in each of the aforementioned tumor types. The second manuscript will expand this investigation to additional malignancies and present broader strategies for minimizing the negative effects of prolonged treatment duration.

SEARCH STRATEGY

An extensive bibliographic search was conducted to evaluate the potential relationship between OTT prolongation and LRC and/or SV outcomes in the four tumor types previously mentioned.

Studies were classified as "negative" if they demonstrated a statistically significant detrimental association between OTT prolongation and either LRC or SV (or both), even if the impact was limited to local or regional control, or to any SV endpoint. Conversely, studies showing no significant adverse association were categorized as "non-significant" (NS).

We searched evidence-based databases using a comprehensive strategy that combined free-text terms and Medical Subject Headings in the title/abstract fields. The following search terms were employed: (1) Tumoral repopulation; (2) Radiotherapy delays; (3) Treatment interruptions; (4) Overall treatment time; (5) Time factor; (6) Compensation maneuvers; and (7) Time relationship. In addition to screening original full-text articles, we performed reference list reviews of selected publications to identify additional relevant studies.

Given the predominance of retrospective data in the available literature, particular attention was paid to study quality. We classified studies based on whether their conclusions were supported by univariate analysis (UV-An) or multivariate An (MV-An). A study was classified as MV-An if MV-An was explicitly stated by the authors or could be clearly inferred from the methodology or results; otherwise, it was categorized as UV-An.

Several studies also attempted to quantify the additional daily dose required to offset LRC loss resulting from OTT prolongation. Despite methodological heterogeneity, we grouped these dose-compensation estimates under the term "k factor". Similarly, when studies estimated the time point at which accelerated tumor repopulation begins after RT initiation, we referred to this parameter as "Tk".

RESULTS BY CANCER TYPE

Result of HNC

Our previous review[4] encompassed 62 studies, with the larynx being the most frequently reported anatomical site (26/ 62, 41.9%), followed by studies involving mixed head and neck subsites (24/62, 38.7%). In the current update, we identified 8 additional studies [5-12], increasing the total to 70. Among these, mixed-site reports predominated (6 of 8).

Of the newly included studies, three of six demonstrated a statistically significant negative impact of OTT prolongation on LRC[5,7,8], while one showed significance in UV-An but not in MV-An[11]. Regarding SV outcomes, four of seven studies reported a significant negative association-all based on MV-An[7,8,10,12]-and one study approached significance in MV-An (P = 0.052)[11].

Most studies showing a negative effect involved patients treated with curative intent. Among three studies that included patients receiving adjuvant RT, only one reported a negative impact [8]. Additionally, studies showing adverse OTT effects often involved patients receiving chemotherapy (QT)-either neoadjuvant or concomitant [7,8,10,12]suggesting that QT does not fully compensate for the detrimental consequences of treatment prolongation, as previously

The negative impact of prolonged OTT was typically observed with delays exceeding 2 days or when total treatment duration surpassed 45-49 days, particularly in stage II-IV disease [7,8,10], though early-stage disease may also be affected

One study specifically evaluated the day of the week on which RT was initiated in nasopharyngeal cancer and found no difference in SV, including for Friday starts, provided OTT was not significantly prolonged[12].

Result of CC

Among 37 studies[13-49] assessing OTT prolongation during radical RT for CC, 32 (88.8%) reported a significant negative impact on LRC and/or SV[13-25,27-33,35-37,39-43,46-49], while 5 were NS[26,34,38,44,45]. The detrimental effect was more pronounced in stage IIB-IV disease[14,31,37], with variable significance in stage IB-IIA[32,38], and limited evidence for stage IA[33].

Most studies with negative findings conducted MV-An. Lin et al [24], in a cohort of 2594 patients, identified OTT as an independent predictor of worse cancer-specific SV (CSS) and overall SV (OS).

Several studies estimated daily LRC loss of 0.7%-1.6% per day of delay[27,28,30]. Chen et al[25] reported 5-year LRC rates of 88% vs 67% for OTT \leq 56 vs > 56 days in patients treated with chemoradiotherapy (CRT) (P = 0.04). Huang et al [49] demonstrated a drop in tumor control probability from 100% (at 38 days) to 33% (at 54 days).

While some studies (Shaverdian et al[15] and Tergas et al[20]) suggested that OTT up to 10-12 weeks may not significantly affect SV when concurrent CRT is used, others (Hong et al[43]) observed a loss in OS when OTT exceeded 64 days, indicating that QT may only mitigate repopulation if delays are limited to < 7 days.

Two studies reported a Tk value of 19 days [33,49], and a potential doubling time (Tpot) of 4-5 days has been described, supporting strict OTT thresholds between 56-64 days[32].

The most widely accepted recommendation based on 11 studies is to complete external beam RT (EBRT) plus brachytherapy (BT) within 8 weeks (56 days)[15-22,25,42], or within a maximum of 9 weeks (63 days) when using low-dose-rate (LDR) BT[33]. Tanderup et al[21] proposed completing OTT within 7 weeks, with total doses ≥ 85 Gy for achieving > 94% LRC in small tumors.

The EBRT-BT interval should ideally be < 7-10 days[17,18,30], though not all authors concur[19].

Prolonged OTT (> 56 days) has also been associated with increased toxicity, including grade 3-4 proctitis[16,19].

While most data focus on squamous cell carcinoma, evidence on adenocarcinoma remains limited[24,44].

Result of AC

Among 15 identified studies[50-64], only 5 reported a significant negative effect of OTT prolongation on LRC and/or SV [50-54], while the remaining 10 were NS[55-64].

Five studies employed split-course schedules [50-52,62,63], with only two demonstrating a negative impact [52,62].

In continuous RT using conventional fractionation, evidence remains limited, with small sample sizes and a lack of robust MV-An data. Negative impacts on LRC have been suggested when OTT exceeded 40-41 days for doses of 45-50 Gy, and 53 days (or delays > 8 days) for higher doses (up to 60 Gy), particularly in T3-T4 tumors[51-53].

A post hoc analysis of the ACT II trial (CRT, 50.4 Gy, 1.8 Gy/fraction, 28 fractions) found that progression-free SV worsened when OTT exceeded 42 days, based on UV-An[54].

Short treatment interruptions (< 7 days), especially in T1-T2 tumors, may be mitigated by QT[51]. Interestingly, one retrospective study (abstract only) reported improved LRC and OS with short QT-related interruptions (< 6 days)[57].

Result of PC

Of 12 studies on PC[65-76], 8 demonstrated a negative effect of OTT prolongation[65-72], while 4 were NS[73-76]. All data pertain to conventional fractionation; the impact in hypofractionation (HypoFx) remains uncertain but should not be overlooked[65,66].

OTT prolongation primarily affected LRC and biochemical (BQ) control, with occasional associations with CSS[67], mainly in MV-An studies.

Earlier studies suggested that high doses (> 72-74 Gy) might offset OTT prolongation[67]; however, more recent findings indicate that even doses of 76-80 Gy are vulnerable to delays > 3-4 days [65].

An OTT exceeding 8-9 weeks (or > 52-58 days) was generally associated with worse outcomes [69].

Delays of > 5-7 days were more harmful in patients not receiving concurrent hormone therapy, although this effect is less clear when hormones are used[66].

The detrimental impact appeared more pronounced in low-risk/Low histologic grade tumors and less consistent in intermediate risk/grade, with minimal or no effect in high risk/grade groups [65,69,70].

Reported Tk values: (1) By T stage: Mean Tk 34 days (30 days in T1c, 35 days in T2 and 69 days in T3[68]; and (2) By risk group: Mean Tk 31 days (33 days in low risk, 35 days in intermediate and 37 in high risk)[71].

Reported k-factors included: (1) 0.24 Gy/day[69]; (2) 0.52 Gy/day[71]; and (3) 0.21 Gy/day[72].

DISCUSSION AND COMPENSATION TIPS BY CANCER TYPE

As stated in our previous work[4], this remains the largest and most comprehensive bibliographic review to date addressing the negative impact of OTT prolongation on outcomes for patients with HNC, CC, AC, and PC treated with RT. Our review highlights not only the quantity but also the quality of available evidence, with particular attention to LRC and SV endpoints.

Table 1 summarizes practical recommendations for mitigating treatment delays across these tumor types. In general, we endorse the principle advocated by the Royal College of Radiologists (United Kingdom)[2]: Treatments should adhere to the As Short As Reasonably Achievable principle [77].

HNC

Combining findings from our previous review[4] with updated data, we confirm a strong and statistically significant association between OTT delays and reduced LRC (53/64 studies, 82.8%) and SV (19/28 studies, 67.8%), with most studies incorporating MV-An. When excluding studies based on accelerated fractionation, these numbers improve to 50/ 58 (86.2%) for LRC and 18/21 (85.7%) for SV. The better outcomes may reflect excess RT-related mortality reported in some accelerated regimens[4].

Delays lead to an average LRC loss of 1.2% per day, translating into 12%-14% per week, and typically require dose escalation of 0.6-0.8 Gy/day to compensate. Accelerated tumor repopulation generally begins after a lag phase of 3-4 weeks[4].

Total days of OTT delay seems to be what really matters; gap position and the number of missed consecutive treatment days seem to have no prognostic significance, except, perhaps, in longer extensions (≥ 1 week); and prolongations ≥ 3 days or \geq 45-49 total days should be discouraged regardless of their timing[4].

The detrimental effect of OTT prolongation is most pronounced in radical-intent treatment and advanced-stage disease but remains evident across all disease stages [4,8,10]. Tumor differentiation may also influence sensitivity to delays: Welland moderately-differentiated tumors are more vulnerable than poorly differentiated ones, and mainly depending on the primary tumor (not for lymph node metastases). It's has been suggested as biological mechanism to explain this observation that the tumor needs to have a functional mechanism capable of regeneration, which most likely happens in well-differentiated tumors, while poor-differentiated ones would lost the ability for repopulation [4,5].

Although QT is frequently used, current evidence suggests it does not consistently neutralize the negative effects of OTT prolongation [4,10].

Although the number of studies is smaller than in HNC, the trend is similar: 86.1% of studies using MV-An report a significant negative impact of OTT prolongation on LRC and/or SV. Mid-to-advanced stages (IIB-IV)[14] are more sensitive to delays, though early-stage disease is also affected[31-33,37,38].

Reported data[27,28,30,32,33] include: (1) LRC losses of 0.7%-1.6% per day; (2) Tpot of 4-5 days; and (3) Tk around 19 days[49].

In contrast to HNC, concurrent QT appears to offer partial protection against OTT prolongations of up to one week [15, 20,43].

Best practices (12 studies) include completing EBRT and BT within 8-9 weeks, with an EBRT-BT gap ≤ 7-10 days[15-22, 25,30,33,42].

Most data focus on squamous cell carcinoma, and while evidence for adenocarcinoma is limited, the potential impact of delays should not be overlooked.

AC highlights the importance of comprehensive literature reviews. While guidelines such as those from the Royal College of Radiologists[2] classify AC among tumors most sensitive to OTT prolongation (based on a few reports from the literature), our broader review shows that only 5 of 15 studies (33.3%) demonstrated a significant negative effect.

Notably, of five studies using split-course schedules-which typically amplify delay-related risks-only two showed detrimental effects.

Table 1 Resume and	basic recommenda	ations to deal with delavs

Localization (number of articles negative/published)	LRC and/or SV lost	RT intention	Stages	Recommendations
Head and neck cancer (59/70, 84.3%)	Yes, both	Radical and adjuvant	All	Avoid/compensate if delays \geq 3-5 days (3, preferable; 5, obligatory), especially if G1 (or even G-2) histological grade. Do so for every pathological stage and RT intention (especially in II-IV stages and radical intention) and regardless if QT is used
Cervix cancer (32/37, 88.8%)	Yes, both	Radical	≥ II-B, and maybe in I-B/II-A, at least	Complete EBRT + BT in < 8-9 weeks (8, preferable), although if chemoradiotherapy the limit could be up to 9-10 weeks. Timing EBRT/BT: Keep on \leq 10 days (7, if possible). Pelvic adjuvant EBRT: For precaution, in treatments of 45-50 Gy in 5-5.5 weeks, avoid/compensate delays > 5-7 days
Anal cancer (5/15, 33.3%)	LRC: Probable, yes. SV: No, except, maybe, CSS and free- progresion survival	Radical	Likely more important in T3-4 and more dubious in T1-2	In continuous RT schemes at usual doses of 45-60 Gy, avoid/compensate OTT > 40-42 days for lower doses and OTT > 53 days (or delays > 8 days) for higher doses. QT could inhibit repopulation if delays < 7 days, especially in T1 and T2
Prostate cancer (8/12, 66.6%)	LRC: Yes, and biochemical control, too. SV: No, except, maybe, CSS	Radical	Likely more important in localized stages/Low risk groups, more dubious in interme- diates and less likely in advanced/high risk	Avoid/compensate especially in localized stages/Low risk groups, more dubious in intermediates. Avoid/compensate if delays \geq 5-7 days in patients without concurrent hormonotherapy. Normofx: Avoid/compensate if OTT > 8-9 weeks (> 52-58 days). MHipofx (about 2.5 Gy/day): Avoid/compensate on precaution delays > 4-7 days, especially if concur at least one of the first two points. MHipofx (around 3 Gy/day): Avoid/compensate on precaution delays \geq 7-10 days, especially if concur at least one of the first two points. Adjuvant RT (Normofx and MHipofx, 1.8-3 Gy/day): Avoid/compensate on precaution delays \geq 7-10 days, especially if concur at least one of the first two points

BT: Brachytherapy; CSS: Cancer-specific survival; EBRT: External beam radiotherapy; LRC: Locoregional control; MHipofx: Moderate hipofraccionation; Normofx: Normofraccionation; OTT: Overall treatment time; QT: Chemotherapy; RT: Radiotherapy; SV: Survival.

This suggests AC may be less sensitive to OTT prolongation compared to HNC or CC. However, a 33% rate of negative studies still justifies clinical caution.

Similar to CC, QT appears to mitigate the impact of short interruptions (< 7 days)[51]. No data were identified regarding the adjuvant setting.

PC

Traditionally considered a slow-growing tumor with low sensitivity to OTT delays, PC nonetheless showed a 66% rate of studies reporting negative effects, primarily on LRC and BQ control, rather than SV.

All reviewed data are based on conventional fractionation regimens (up to 80 Gy). Delays of 4-7 days were associated with worse outcomes, and on-current hormone therapy may offer a protective effect, (similar to QT in CC and AC)[65,66, 69]. The negative effect of OTT prolongation was most pronounced in low-risk and intermediate-risk patients, with little or no impact in high-risk groups-similar to patterns observed in HNC[65,69,70]. However, no biological mechanism to explain this has been suggested by the authors and we found the explanation given in HNC possible for PS, too.

While no studies have specifically assessed Hypofx regimens, the reported k-factor of 0.52 Gy/day [71] implies that even in moderate Hypofx, OTT delays of 4-7 days (equating to 2.6 Gy loss, which is in the range of these schemes) may be clinically significant and should be avoided.

Evidence from adjuvant settings is lacking, but delays exceeding 7 days should generally be prevented.

Limitations and future directions

Our review is subject to inherent limitations, primarily due to its reliance on retrospective data. Approximately 60% of the literature cited was published before/equal 2010, limiting our ability to evaluate emerging factors such as targeted therapies, immunotherapy, tumor genetics/mutations, biomarker expression, advances in imaging (e.g., positron emission tomography-computed tomography), or artificial intelligence (AI) and machine learning in prognostic modeling. These variables were not addressed even in the more recent studies in our review.

Finally, optimizing management of RT delays requires interdisciplinary collaboration. It is the responsibility of radiation oncologists to communicate the clinical significance of OTT prolongation to colleagues in medical oncology, pathology, radiology, and other specialties. Enhancing mutual understanding is crucial to improving treatment efficiency and outcomes across the cancer care continuum.

CONCLUSION

This comprehensive review confirms a substantial detrimental effect of OTT prolongation on outcomes in HNC and CCthe two tumor types with the most robust and consistent evidence. Over 80% of studies, primarily incorporating MV-An, demonstrate that OTT prolongation negatively affects both LRC and SV. The principal clinical recommendation is clear: Avoid OTT prolongations whenever possible, and when delays are unavoidable, implement appropriate compensatory measures (e.g., dose escalation or treatment acceleration).

For AC, although the level of evidence is less consistent-only 33% of studies reported negative outcomes, many of which relied on UV-An-OTT minimization remains advisable given the potential clinical impact.

PC occupies an intermediate position: 66% of reviewed studies still reported a negative effect of OTT prolongation, particularly on LRC and BQ. This is especially relevant in conventional fractionation schedules, although it must be recommended in moderate Hipofx schemes, too.

An important observation is the differential effect of concurrent systemic therapies: (1) In HNC, concurrent QT does not appear to fully mitigate the adverse impact of delays; (2) In CC and AC, concurrent QT may offer some protective effect, particularly for short interruptions; and (3) In PC, concurrent hormonal therapy appears to provide a buffering effect, reducing the harm of brief delays.

Future studies should expand this analysis to additional tumor types where preliminary evidence exists, and explore, if possible, the role of emerging therapeutic modalities, biomarkers, and advanced analytics (e.g., AI, radiomics) in predicting and managing the impact of OTT prolongation.

FOOTNOTES

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