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Pancreas Tumor Control and Tolerance Doses: Insights from Dose Response Models

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Abstract
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Abstract

Objective: We investigated maximizing prescription dose for tumor control while respecting dose constraints in established co-operative group protocol for the treatment of Pancreas Cancer with SBRT. In five fractions, the Alliance A021501 protocol constrains 1cc, 3cc, and 9cc of duodenum, stomach, and bowel each to 33Gy, 20Gy, and 15Gy, respectively. These are compared to recent tumor control probability (TCP) and normal tissue complication probability (NTCP) models to assess the effect on TCP when meeting various NTCP limits.

Methods: A typical head of pancreas case study on the CyberKnife platform was used to compare TCP and NTCP tradeoffs from recently published dose response models. The planning target volume (PTV) was created with a 3mm expansion customized to zero expansion adjacent to the duodenum. The tumor vessel interface (TVI) was created as a 3mm expansion of vessels that overlapped the PTV. The initial goal was a 35Gy/5 fraction plan, but failed to meet the Alliance A021501 protocol dose constraints. Therefore a series of treatment plans were created for three alternate prescription levels: 25Gy, 30Gy, and 35Gy, each in five fractions, to approximately the 60% isodose line. The plans were optimized to maximize PTV and TVI coverage subject to one main duodenum constraint for each plan: the 35Gy plan was designed to comply with the D1cc=33Gy limit, the 30Gy plan was designed to comply with the D3cc=20Gy limit, and the 25Gy plan was designed to comply with the D9cc=15Gy limit.

Local control was estimated using the model in figure 1 of the High Dose per Fraction, Hypofractionated Treatment Effects in the Clinic (HyTEC) Pancreas TCP paper, Mahadevan et al (IJROBP 2021 May 1;110(1):206-216). Dose tolerance was estimated using the models in figures A1 and A4 of Goldsmith et al (SRO 2016 Apr;26(2):149-56), which represent treatment with and without the usage of live respiratory tracking, respectively. Additional details of the TCP and NTCP models and assumptions/parameters are provided in the two cited references.

Results: The estimated 1-year local control for 25, 30, and 35Gy from the HyTEC Pancreas TCP model would be 63%, 73%, and 80%, respectively, if the patient remains unresectable. The estimated risk associated with the D3cc and D9cc achieved levels in all three plans were less than 1% with live tracking and about 25% without live tracking. For this patient, when constraining to the D3cc limit, the D1cc limit was automatically met, and when constraining to the D9cc limit all the others were automatically met, thus for this patient they were consecutively more conservative in order of volume effect.

Of the three, the D1cc values had the most influence on model-based dose tolerance predictions. The 25Gy plan had duodenal D1cc=22.9Gy, which had estimated risk of 2% with live tracking and 25% without live tracking. The 35Gy plan had duodenal D1cc=26.7Gy which had estimated risk of 3% with live tracking and 30% without live tracking.

Conclusion: For systems with the best live tracking accuracy, the D3cc=20Gy and D9cc=15Gy duodenal constraints might be more conservative than necessary. Improved tumor control might be achievable with dose escalation for pancreas tumors. Many other factors specific to each individual patient may need to be considered clinically, beyond the scope of dose response models. The current dose response models are based on few patients and short follow-up, so there is a need for much more long-term data on larger prospective datasets to refine these estimates, taking into account many of the factors which can affect outcomes.