Phase II study of helical tomotherapy for oligometastatic colorectal cancer

B. Engels1, H. Everaert2, T. Gevaert1, M. Duchateau1, B. Neyns3, A. Sermeus4, K. Tournel1, D. Verellen1, G. Storme1 & M. De Ridder1*

Departments of 1Radiation Oncology; 2Nuclear Medicine; 3Medical Oncology; 4Gastroenterology, UZ Brussel, Vrije Universiteit Brussel, Brussels, Belgium

Received 15 April 2010; accepted 26 May 2010

Background: To evaluate the efficacy and toxicity of helical tomotherapy in the treatment of oligometastatic colorectal cancer (CRC) patients who were not amenable for metastasectomy and/or (further) systemic treatment.

Patients and methods: CRC patients with five or less metastases were enrolled. No limitations concerning dimension or localization of the metastases were imposed. Patients were treated with intensity-modulated and image-guided radiotherapy using helical tomotherapy, delivering a total dose of 40 Gy in fractions of 4 Gy. Positron emission tomography–computed tomography (PET-CT) was carried out at baseline and 3 months after the initiation of radiotherapy to evaluate the metabolic response rate according to PET Response Criteria in Solid Tumors (PERCIST) version 1.0. Side-effects were scored using National Cancer Institute Common Terminology Criteria for Adverse Events (NCI–CTC AE) version 3.0.

Results: Twenty-three patients were enrolled. A total of 52 metastases were treated. One patient (4%) experienced grade 3 vomiting; two patients (9%) grade 2 diarrhea and dysphagia, respectively. Twenty-two patients were evaluated by post-treatment PET-CT. Five (23%) and seven patients (32%) achieved a complete and partial metabolic response, respectively, resulting in an overall metabolic response rate of 55%. The actuarial 1-year local control, progression-free survival, and overall survival were 54%, 25% and 86%, respectively.

Conclusion: The use of helical tomotherapy in oligometastatic CRC patients resulted in a promising metabolic response rate of 55%.

Key words: helical tomotherapy, metastatic colorectal cancer, oligometastases, SBRT

Introduction

More than half of the patients undergoing resection of colorectal cancer (CRC) have recurrence of this disease. CRC tend to develop metastases limited in number and location. This supports the hypothesis of oligometastatic disease, a clinical disease state between locoregionally confined and widely spread metastatic disease [1]. For these patients, complete metastasectomy is the only curative treatment option. Surgical series of patients with metastatic colorectal cancer (mCRC) undergoing resection of liver metastases demonstrated actuarial 5-year survival rates of 25%–40%, in contrast to a 5-year survival rate of <5% for mCRC patients receiving chemotherapy only [2–6]. Recent data emerge for the use of perioperative chemotherapy to decrease the relapse rate in patients undergoing metastasectomy. Nordlinger et al. [7] reported a 3-year progression-free survival (PFS) of 35% for patients with limited liver metastases undergoing perioperative FOLFOX4 chemotherapy. This supports the premise that aggressive local therapy of oligometastases may result in prolonged life. However, only 20% of patients with liver-confined mCRC are candidates for potentially curative surgery [3–6, 8, 9]. Indeed, limitations imposed by localization, multifocal character or comorbidity exclude many patients from metastasectomy. Nonsurgical local ablative alternatives include cryosurgery ablation (CSA), laser-induced interstitial thermotherapy, radiofrequency ablation (RFA) and stereotactic body radiotherapy (SBRT), the latter delivering tumoricidal doses of radiation in a minimal number of fractions to small targets by the combination of high conformal radiotherapy (RT) and image-guided radiotherapy (IGRT) [10–13]. Phase I/II studies assessing the SBRT approach for eradication of metastases reported long-term local control (LC) rates of 60%–95%, with a limited toxicity to the surrounding healthy tissues [14–19]. These studies enrolled patients with rather small metastases at distance from hollow viscous organs, allowing the delivery of ablative radiation doses. In the present study, we explored prospectively the efficacy and toxicity of helical tomotherapy, a relatively new technology combining rotational intensity-modulated radiotherapy (IMRT) and IGRT by megavoltage (MV) computed tomography (CT) scanning, in...
patients with oligometastatic CRC who were not amenable for metastasectomy and/or (further) systemic therapy. A moderately hypofractionation schedule, 10 daily fractions of 4 Gy, was used with the aim of achieving a high response rate and a broad applicability, including patients with large metastases and critically located lesions. The primary objective was to evaluate the complete metabolic response (CMR) rate 3 months after initiation of RT by carrying out whole-body 18F-fluorodeoxyglucose (FDG)–positron emission tomography (PET) at baseline and at evaluation. Secondary end points were toxicity, LC and PFS.

**methods**

**patient population**

Patients eligible for this study were CRC patients with five or less metastases, showing increased metabolism on 18F-FDG–PET. Additionally, patients were required to be >18 years and were considered inoperable by the localization, number or dimension of the metastases, medically unfit to undergo resection or refusing surgery. Patients who did not receive previous chemotherapy for metastatic disease had to be medically unfit to undergo systemic treatment or refusing chemotherapy. Patients who received previous chemotherapy were included if progressive disease was present or if cumulative toxicity limited further systemic treatment. The primary tumor had to be radically resected, with the histological proof of a colorectal adenocarcinoma. Patients should have a World Health Organisation performance status of zero to two, no Child B or C liver cirrhosis and a functional liver volume of >1000 cc in case of liver metastases and a lung diffusion capacity for carbon monoxide (DLCO) of >50% in case of lung metastases. No limitations were imposed concerning the localization or dimension of the metastases. No upper age limit was defined. Patients with an active second primary tumor were excluded. An informed consent was obtained for the study protocol. The protocol was reviewed and approved by the ethical committee and registered (inter)nationally (Eudract 2008-008300-40; NCT00807313).

**pretreatment evaluation**

Pretreatment evaluation included a complete medical history and physical examination. All patients underwent a pretreatment 18F-FDG–PET and CT using a dedicated PET-CT camera (Gemini TF; Philips Medical Systems, Cleveland, OH) and laboratory tests, including a full blood count, C-reactive protein, carcinoembryonic antigen (CEA) and carbohydrate antigen (CA) 19.9. Assessment of the Child-Pugh parameters and liver enzymes were carried out in all patients with liver metastases. Pulmonary function tests (PFTs), including total lung capacity (TLC), DLCO and 1 s forced expiratory volume (FEV1), were carried out before the treatment for patients with lung and/or mediastinal lymph node metastases.

**radiotherapy technique**

RT was carried out using the TomoTherapy Hi Art II System (TomoTherapy Inc., Madison, WI), which fully integrates IGRT by means of megavoltage computed tomography (MV-CT) scanning and IMRT by means of dynamic rotational therapy [20–23]. The CT and 18F-FDG–PET images were co-registered to delineate the gross tumor volume (GTV). The GTV was defined as the visible gross tumor mass on CT, fully encompassing the 18F-FDG–PET-positive volume. The planning target volume (PTV) was defined as the GTV with a nonuniform margin of 10, 10 and 12 mm for the anteroposterior, laterolateral and craniocaudal direction, respectively. Treatment planning was carried out on the TomoTherapy TPS (TomoTherapy Inc., Madison, WI). The planning goals were to deliver at least 38.0 Gy (95% of 40 Gy) to 95% of the PTV, while keeping the maximum dose (Dmax) to the PTV below 42.0 Gy (105% of 40 Gy). In case of lung irradiation, the lung volume receiving >20 Gy (V20) was kept below 20%. In patients with liver metastases, the liver volume receiving >22 Gy (V22) and 30 Gy (V30) was kept to <50% and 30%, respectively. A Dmax of 36.0 Gy (90% of the prescribed dose) was set to the spinal cord, small bowel and stomach if the overlap volume between PTV and small bowel/stomach was 25 cc (Figure 1).

The treatment was delivered daily in 10 fractions, excluding weekends. Before each treatment session, patients underwent scanning using the integrated MV-CT scan modality and were repositioned after co-registration of these images with the planning kilovoltage CT scan.

**toxicity monitoring**

Toxicity was evaluated and scored according to the National Cancer Institute Common Terminology Criteria for Adverse Events (NCI–CTC AE) version 3.0, with toxicity occurring within 3 months after the initiation of RT classified as acute toxicity. Laboratory tests were repeated at the first follow-up visit 3 months after initiation of RT, including assessment of the Child-Pugh parameters and liver enzymes for patients with liver metastases. Radiation-induced liver disease (RILD) was defined as either anicteric elevation of alkaline phosphatase level of at least twofold and nonmalignant ascites (classic RILD) or elevated transaminases of at least fivefold the upper limit of normal or of pretreatment level (nonclassic RILD) [24, 25]. PFTs were repeated in patients with lung and/or mediastinal lymph node metastases, respectively. Patients were contacted and/or invited for follow-up 4-monthly during the first year, 6-monthly thereafter.

![Figure 1.](image-url) Pretreatment 18F-fluorodeoxyglucose–positron emission tomography–computed tomography (PET-CT) (left) and dose distribution of a plan (right) of a patient with a liver metastasis and a perigastric lymph node metastasis. Surgical clips in the liver remind to a previous partial hepatectomy and segmentectomy. Note the overlap of the planning target volume (PTV) with the stomach (right), with a maximum of 36.0 Gy (90% of 40.0 Gy) to the intersection volume between PTV and stomach (white arrow).
response evaluation
The primary objective, CMR rate, was evaluated by comparing the PET-CT at baseline with the PET-CT carried out 3 months after initiation of RT. The 18FDG uptake in the lesion(s) was assessed semiquantitatively by measuring the maximal standardized uptake value (SUVmax) within a three-dimensional ellipsoidal region of interest covering the target. Corrections were made for the administered dose, body weight and decay. No correction was carried out for glycemia, lean body mass or body surface. Metabolic response was assessed according to the PET Response Criteria in Solid Tumors (PERCIST) version 1.0 [26]. Briefly, a CMR in a patient was defined by a complete resolution of 18FDG uptake within all lesion(s), so that it is less than mean liver activity and indistinguishable from surrounding background blood-pool levels, and with no new 18FDG-avid lesions in pattern typical of CRC. For a partial metabolic response (PMR), a reduction in SUVmax for patients with one lesion or a reduction of the sum of the SUVmax data for patients with more than one lesion of minimum 30% was required, without the appearance of new 18FDG-avid lesions. Obvious progression of any lesion (>30% increase in SUVmax) or new 18FDG-avid lesions negate a partial response and indicate a progressive metabolic disease (PMD). Stable metabolic disease (SMD) is defined as not CMR, PMR or PMD.

During follow-up, the Response Evaluation Criteria in Solid Tumors (RECIST) were used to evaluate response. A local recurrence was defined as the regrowth of tumor within or at the periphery of the irradiated volume. The appearance of new lesions was considered as distant recurrence.

statistics
A Richard Simon two-stage optimal design was carried out to obtain the sample size. Aiming at an overall acceptable and unacceptuble CMR probability of 30% and 10%, respectively, with an α and β value of 0.10, the sample size for first and second stage were 1/12 and 5/35. Actuarial LC, PFS and overall survival (OS) rates were estimated by Kaplan–Meier analysis; log-rank testing was used to evaluate the association between metabolic response and treatment outcome. PFTs and laboratory tests were evaluated by t tests.

results
patient characteristics
Between July 2008 and December 2009, 23 oligometastatic CRC patients with a total number of 52 metastases were enrolled. Patient characteristics are given in Table 1. All patients were considered inoperable by the number, dimension or localization of the metastases (n = 20), medically unfit for surgical resection (n = 2) or refusing surgery (n = 1). Sixteen patients (70%) received previously one or more line of chemotherapy for treatment of metastatic disease, of which all had evidence of progression (n = 11) or cumulative toxicity (n = 5), limiting further continuation of the same systemic treatment regimen. Seven patients (30%) received no previous chemotherapy for treatment of metastatic disease, four patients were considered medically unfit to undergo systemic therapy and three patients refused chemotherapy. Ten patients (43%) received previous local treatment of metastases: liver resection (n = 6), lung resection (n = 1), SBRT (n = 2) and RFA (n = 2). The median number of metastases was 2, with a median GTV of 22 cc (range, 2–274 cc). The majority of the patients presented with metastases located in the liver (n = 7), lung (n = 7) and lymph nodes (n = 10), the latter located thoracic (n = 3) or abdominal/pelvic (n = 7). Nine patients (39%) presented with metastases in more than one organ. In 14 patients (61%), the GTV was located in the vicinity of organs at risk (OARs), such as small bowel, stomach or esophagus.

toxicity
Treatment discontinuation due to acute toxicity was required for one patient experiencing acute grade 3 vomiting after having received 28 Gy (seven fractions) to a solitary bone metastases located in the D10 vertebral body. As no medication could be identified in this patient to interact with radiation and as the dose to the nearby stomach was limited strongly (<1 Gy/fraction), radiation hypersensitivity may explain the excessive toxicity in this patient. No other grade ≥3 adverse events occurred. Two patients (9%) experienced grade 2 dysphagia and diarrhoea, respectively. In the patients who were irradiated for lung and/or mediastinal lymph node metastases (n = 8), the recorded average V20, mean lung dose and average decline in FEV1, DLCO and TLC was 7.5% ± 5.2%, 6.2 ± 4.0 Gy, 2.5% ± 8.4%, 7.6% ± 10.9% and 0.4% ± 16.3%, respectively. Grade 1 pneumonitis (asymptomatic, radiographic findings only) and grade 1 changes in FEV1, DLCO and TLC (90%–75% of

Table 1. Patient characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Distribution</th>
<th>No. of patients</th>
<th>%</th>
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<td>Sex</td>
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<td>61</td>
</tr>
<tr>
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<td>Median 64</td>
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<tr>
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<td>Range 60–90</td>
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<td>31</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>7</td>
<td>31</td>
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<td>10</td>
<td>43</td>
</tr>
<tr>
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<td>6</td>
<td>26</td>
</tr>
<tr>
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<td>5</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Gross tumor volume (cc)</td>
<td>Median 22</td>
<td>Range 2–274</td>
<td></td>
</tr>
<tr>
<td>Number of involved sites</td>
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<td>61</td>
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<td></td>
<td>3</td>
<td>1</td>
<td>4</td>
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<tr>
<td>Localization</td>
<td>Liver</td>
<td>7</td>
<td>31</td>
</tr>
<tr>
<td></td>
<td>Lymph node</td>
<td>10</td>
<td>43</td>
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<tr>
<td></td>
<td>Lung</td>
<td>7</td>
<td>31</td>
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<tr>
<td></td>
<td>Soft tissue</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Bone</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Peritoneum</td>
<td>2</td>
<td>9</td>
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<td>Follow-up (months)</td>
<td>Median 12</td>
<td>Range 3–18</td>
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</tr>
</tbody>
</table>

364 | Engels et al. | Volume 22 | No. 2 | February 2011
baseline value) were observed in 4, 1, 2 and 2 patients, respectively. No grade ≥2 lung toxicity occurred. No cases of classic or nonclassic RILD were observed in the seven patients irradiated for liver metastases. No differences in Child-Pugh score were observed at 3 months after RT as compared at time of inclusion. No violation of the liver dose-volume constraints ($V_{22}$ and $V_{50}$) occurred. Grade 2 elevation of the liver enzymes ($>2.5–5.0 \times$ upper limit of normal) was observed in two patients, only for gamma-glutamyl transferase. No grade ≥3 level elevation of the liver enzymes was recorded. At a median follow-up of 12 months (range, 3–18 months), no patients experienced chronic toxic effects.

**response evaluation**

All 23 patients underwent PET-CT at baseline, 1 patient failed to receive a post-treatment PET-CT. For the whole-patient group, mean $SUV_{max}$ ($\pm$ SD) at baseline was 7.7 ± 5.4 (range, 1.9–25.4), post-treatment 5.3 ± 2.6 (range, 1.2–12.4). Metabolic response rates are listed in Table 2. Five patients (23%) achieved a CMR, seven patients (32%) achieved a PMR, resulting in an overall metabolic response rate of 55%. The mean fractional change in $SUV_{max}$ at first analysis as compared to baseline was $-64.1\% \pm 14.8\%$ and $-45.2 \pm 15.0\%$ for complete and partial metabolic responders, respectively. SMD was seen in three patients (13%). Seven patients (32%) presented with metabolic progression, of which two patients with isolated infield metabolic progression. Monitoring of the tumor markers at baseline showed mean values of 44.6 ± 89.4 μg/l and 69.9 ± 216.9 U/ml for CEA and CA 19.9, respectively, versus 50.2 ± 126.3 μg/l and 51.9 ± 136.1 U/ml for CEA and CA 19.9 at post-treatment analysis ($P = 0.79$). Baseline elevated CEA (>5 μg/l) and CA 19.9 levels (>37 U/ml) were recorded in only 53% and 11% of the patients, respectively. Among patients with elevated tumor markers at baseline, no significant differences were observed in the fractional change of CEA and CA 19.9 between responders and nonresponders.

**follow-up**

At a median follow-up of 12 months (range, 3–18 months), five patients (22%) are in remission in all irradiated areas without evidence of distant recurrence. Three patients died, two because of progressive metastatic disease and one because of noncancer-related sudden death. With a median time to progression of 5 months (range, 3–9 months), 18 patients (78%) developed progressive disease, of which 5 patients (22%) with isolated local progression, 6 patients (26%) with synchronous local and distant progression and 7 patients (30%) with distant recurrence only. Among these 18 patients, 4 underwent an additional course of RT (2 patients because of distant relapse, 1 for local relapse and 1 patient for local and distant relapse), in 11 patients, systemic chemotherapy was (re)initiated, and 3 patients received best supportive care. We report a 1-year actuarial LC, PFS and OS of 54% [95% confidence interval (CI) 20%–66%], 25% (95% CI 4%–36%) and 86% (95% CI 61%–93%), respectively. Log-rank testing revealed that patients with a metabolic response (CMR + PMR) at 3 months after RT exhibited statistically significant superior LC ($P < 0.01$) and PFS ($P < 0.01$) compared with metabolic nonresponders (SMD + PMD), who progressed all within 6 months follow-up (Figure 2). Of the five patients with a CMR at 3 months, four patients are still in complete remission with a median follow-up of 13 months (range, 3–18 months). A statistically significant correlation of the post-treatment outcome (LC, PFS or OS) with factors such as number of metastatic lesions, number of involved sites, number of previous lines of chemotherapy or baseline CEA level was not found.

**discussion**

Despite the improvements in systemic treatment during the last decade in mCRC, there is no chance for long-term cure for mCRC patients treated with chemotherapy only. Complete metastasectomy remains the only curative treatment option for oligometastatic CRC patients. Fong et al. and Scheele et al. reported a 5-year OS of 30% and 40%, respectively, for patients undergoing curative resection of colorectal liver metastases [3–6]. However, as many patients are not amenable to undergo surgery, nonsurgical local ablative therapies are often considered as valid alternatives. The use of SBRT has expanded in the last decade since it allows to optimize the balance between tumor control probability and normal tissue complication probability. Several studies reported on SBRT of CRC metastases [19, 27–32]. Hamamoto et al. [27] reported 1- and 2-year LC rates of 48% and 25%, respectively, for lung metastases treated with SBRT of 48 Gy in four fractions. Although this dose schedule is successfully used for eradication of early-stage lung cancer in Japan, the LC rates for CRC lung metastases are disappointing. In a study by Hoyer et al. [28] on SBRT delivering three fractions of 15 Gy to CRC metastases, of which 88% primarily located lung or liver and in 6% in more than one site, a 2-year actuarial patient based LC of 63% was reported. Other authors evaluating the SBRT approach for CRC metastases report LC rates of 53%–100% [19, 29, 32]. The 1-year LC rate of 54% in the present study is disappointing compared with the durable remissions reported in most series of SBRT, where often a biological equivalent dose of >100 Gy is delivered in a minimal number of fractions to small volumes. However, one should notice the carefully selection of metastases on the base of localization and dimension in most of these SBRT series. For those patients, toxicity is not anymore a dose-limiting issue since the introduction and availability of SBRT [30, 31]. In a considerable number of patients, however, metastases are localized in the proximity of OARs. Hence,

**Table 2.** Metabolic response rate 3 months after start of radiotherapy by $^{18}$F-fluorodeoxyglucose–positron emission tomography (PET) ($n = 23$ patients)

<table>
<thead>
<tr>
<th>Metabolic status</th>
<th>No. of patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete metabolic response</td>
<td>5</td>
<td>23</td>
</tr>
<tr>
<td>Partial metabolic response</td>
<td>7</td>
<td>32</td>
</tr>
<tr>
<td>Stable metabolic disease</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>Progressive metabolic disease</td>
<td>7</td>
<td>32</td>
</tr>
<tr>
<td>No PET</td>
<td>1</td>
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</table>
overlap between PTV and OARs in these patients would make the delivery of cytotoxic doses to the tumor impossible with regard to normal tissue toxicity. Patients of the current study had a variety of treatment sites, with no limitations imposed on the dimension of the metastases. Fourteen patients (61%) presented with metastases located next to OARs, such as small bowel, stomach or esophagus. The use of a moderately hypofractioned regimen in conjunction with helical tomotherapy, which combines rotational IMRT and IGRT by means of daily MV-CT imaging, resulted in a safe toxicity profile in this setting, with acute grade 2 and 3 toxicity recorded in 9% and 4% of the patients, respectively, without any chronic radiation toxic effects recorded until yet. Hence, the combination of dose sculpting by IMRT with image guidance techniques is mandatory for these indications [23, 33].

The primary objective was to evaluate the CMR rate by comparing 18FDG–PET at baseline with 18FDG–PET 3 months after start of RT. Based on a Richard Simon two-stage optimal design aiming at an overall acceptable and unacceptable CMR probability of 30% and 10%, respectively, a sample size for second stage of 5/35 was calculated. After having enrolled 23 patients, a CMR was documented in 5 patients, resulting in a CMR rate of 23%. A promising overall metabolic response rate (CMR + PMR) of 55% at 3 months was observed. This is superior to the response rates achieved with second- and third-line systemic treatment regimens in mCRC. In this setting, objective response rates are within the range of 9%–37%, even with the combination of 5-fluorouracil based chemotherapy and epidermal growth factor receptor- or vascular endothelial growth factor-directed antibodies [34–36]. As 70% of the patients received previously one of more line chemotherapy in the current trial, all with progressive disease or cumulative toxicity limiting further continuation of systemic treatment, and as all patients were not amenable for surgery, this represents a group with poor prognosis. This is reflected in the follow-up, with 78% of the patients showing relapse. However, among the patients with relapse, one should notice the five patients (22%) developing local recurrence without evidence of distant progression. Taking into account these facts, the use of helical tomotherapy resulting in an encouraging response rate may play a substantial role in the multidisciplinary treatment of oligometastatic CRC. Moreover, as 10 fractions of 4 Gy resulted in a limited toxicity, we should explore more effective treatment schedules to further improve the tumor control probability. To do so, we are investigating the efficacy and toxicity of helical tomotherapy delivering 50 Gy in daily fractions of 5 Gy.

Molecular imaging by 18FDG–PET has emerged as a pivotal diagnostic tool in the evolving management of mCRC. Several studies described the improved diagnostic accuracy of 18FDG–PET imaging over anatomical imaging in mCRC, especially by its capability to detect unsuspected extrahepatic disease [37]. Furthermore, it proved to have a positive impact on management decision in mCRC patients, with an unexplained rise of serum CEA and CA 19.9 level after primary curative treatment and in patients with colorectal liver metastases suitable for curative resection [9, 38–41]. Beyond staging, there is growing interest in its role for evaluation and prediction of treatment effectiveness in mCRC. 18FDG–PET plays a central role in detecting tumor recurrence after CSA or RFA at an earlier stage as compared with CT information [42–44]. Langenhoff et al. [43] demonstrated a positive and negative predictive value of 80% and 100%, respectively, for the detection of local recurrence by 18FDG–PET 3 weeks after RFA in patients with unresectable liver metastases. Concerning chemotherapy response monitoring in mCRC by 18FDG–PET, several studies indicated its potential value in the early prediction of therapy outcome [45–49]. In a prospective study by de Geus-Oei et al. [46] where mCRC patients underwent 18FDG–PET at 2 and 6 months after start of chemotherapy, a significant correlation between changes in glucose metabolism...
and treatment outcome was observed. These findings are in line with other reports with small series of patients [47–49]. Noteworthy is the importance of a correct timing of 18F-FDG–PET, as a marked increase in 18F-FDG uptake occurring at 1–2 weeks after initiation of chemotherapy, so called flare phenomenon, may mimic false progression of metastases that will respond later on [49]. In contrast to these reports, Byström et al. [50] found no significant correlation between metabolic response and treatment outcome in mCRC patients undergoing first-line chemotherapy. Concerning the data of the present study, a metabolic response 3 months after initiation of RT was found to be predictive for outcome in terms of time to progression or local failure, with a superior LC (P < 0.01) and PFS (P < 0.01) for metabolic responders (CMR + PMR) compared with metabolic nonresponders (SMD + PMD). At a short median follow-up, all metabolic nonresponders (n = 10) developed progressive disease, whereas only one of the complete metabolic responders (n = 5) presented with evidence of disease progression on imaging at last follow-up. To our knowledge, these findings for the first time indicate a potential role for 18F-FDG–PET in the prediction of RT response in the oligometastatic setting of CRC. Hence, 18F-FDG–PET should be offered complementary to anatomical imaging for patients with mCRC undergoing SBRT.

conclusions

Ten fractions of 4 Gy by helical tomotherapy for the treatment of oligometastatic CRC is a feasible approach, resulting in a promising metabolic response rate of 55%, with a limited toxicity profile. We found a potential role for 18F-FDG–PET in the prediction of RT response in oligometastatic CRC.

funding

Foundation against Cancer, foundation of public interest (219.2008); Belgian Government (Nationaal Kankerplan).

disclosure

None of the authors declare conflicts of interest.

references


