Editorial Commentary

Transverse colon cancer: a call for focused research in an understudied heterogenous disease

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Colorectal cancer is a heterogenous disease, with significant differences in embryologic origins, mutational profiles, histology, response to systemic therapy and prognosis based on primary tumor location within the colon (1). The distinction of right and left sided colorectal cancer has been created to reflect these differences, with right sided including the cecum, ascending colon and transverse colon, and left sided including the splenic flexure, descending colon, sigmoid colon and rectum. However, the precise categorization of the transverse colon remains controversial due to its derivation from both the midgut and hindgut, and demonstration of characteristics of both right and left sided cancers (2). This continuum of disease and the relative rarity of transverse colon cancer has historically lent investigators to include transverse colon cancer within right sided disease or exclude transverse colon cancer from trials. This has potentially limited our thorough understanding of transverse colon cancer and impedes treatment optimization for this patient population (3).

Transverse colon cancer represents an estimated 10% of all colon cancers, with estimates of right sided colorectal cancer ranging between 54% to 67% (4,5). However, those diagnosed under the age of 50 years, largely present with left sided disease, at an estimated 65% of total cases (6). Embryologically, the proximal 2/3 of the transverse colon originates from the midgut, while the distal 1/3 derives from the hindgut (4). This variation in origin results in differences in disease presentation, histology and mutational profile within the transverse colon itself. Right sided colorectal cancer, deriving from the midgut, often presents as mucinous adenocarcinomas or sessile serrated adenomas with flat morphology. In contrast, left sided colorectal cancer derives from the hindgut, exhibits typical adenocarcinomas, with polypoid morphology (7,8). Studies indicate that right sided colorectal cancer more often affects female patients and patients of older age, with left sided colorectal cancer having a male predominance and more frequently affects younger patients (8). Severity of disease at presentation and sites of metastasis also vary, with right sided colorectal cancer more often exhibiting higher T stage, poor differentiation, and peritoneal metastasis, whereas left sided colorectal cancer is more frequently found to have lower T stage, more differentiated tumors and more likely to develop pulmonary and hepatic metastasis (8-10). Due to the combined origins of transverse colon cancer, clear clinical and pathological features of these tumors have been difficult to define.

The multifarious presentation of colon cancer is preceded by its underlying mutational profiles. Right sided colorectal cancers exhibit higher rates of *BRAF*, *KRAS*, *PIK3CA*, *CTNNB1*, *SMAD4* mutations and microsatellite instability (MSI-H). Left sided colorectal cancers more often follow the chromosomal instability (CIN) pathway, observing mutations in *TP53*, *APC*, *KRAS*, *PIK3CA*, *SMAD4*, *NRAS*,

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FBXw7, *TCF7L2* along with *APC* deletions, *Wnt* and *RAS* pathway activation (1,2). Although commonly considered a right sided disease, transverse colon cancer has been found to have mutational clusters that vary relative to other right sided locations. In fact, when directly comparing mutational clusters of transverse colon cancer to right and left sided colorectal cancers, the transverse colon had statistically significant differences to right sided mutational clusters, but no statistically significant difference to left sided disease (2). In another analysis of a cohort of transverse colon cancer patients, transverse colon cancer had higher rates of *BRAF* mutations and MSI-H phenotype in line with right sided colorectal cancer, yet the majority of patients harbored *KRAS* wild type tumors, aligning with the left sided colorectal cancer mutational profile. When delineating these patients by the original tumor location within the transverse colon, the majority of the MSI-H phenotype were found originating in the proximal 2/3 of the transverse colon, and *KRAS* wild type tumors were found mostly in tumors originating from the distal 1/3 of the transverse colon. These findings were ultimately consistent with characteristics of the tumor's embryologic origin (4). Prognostically, right sided colorectal cancer has an inferior prognosis relative to left sided disease across age, gender, ethnicity and year of diagnosis (11). Transverse colon cancer appears to align with right sided colorectal cancer, demonstrating higher stages at presentation, less tumor differentiation, and poor prognosis, with later line treatment options agnostic to original primary tumor location (4,12).

Currently, treatment of resectable transverse colon cancer does not vary from standard fluoropyrimidine based adjuvant therapy, however, stage IV disease follows the treatment paradigm of right sided colon cancers (13). Cytotoxic chemotherapy with the addition of vascular endothelial growth factor (VEGF) monoclonal antibody bevacizumab has shown to improve overall survival (OS) and remains the standard targeted agent in this setting (14,15). Targeting epidermal growth factor receptor (EGFR) signaling was examined by pivotal first line therapy trials such as CRYSTAL, which confirmed the utility of the addition of EGFR inhibitors-based therapy in *RAS* wild type tumors to cytotoxic chemotherapy, in addition to FIRE-3 which suggested OS benefit of the EGFR in combination with FOLFIRI in *RAS* wild type patients over the use of bevacizumab. However, neither trial stratified based on sidedness of the primary tumor, until retrospective analysis of both trials indicated superior objective response rate, progression-free survival (PFS) and OS with the addition of cetuximab to chemotherapy for left sided colorectal cancer, compared to minimal efficacy seen in right sided wild type tumors (16-18). CALG/SWOG 80405 was one of the first trials to suggest significantly worse survival for *KRAS* wild type right sided colorectal cancer treated with cetuximab plus chemotherapy relative to left sided disease (19). Most recently, the PARADIGM trial prospectively found that the efficacy of anti-EGFR therapy derives from *RAS* wild type left sided colorectal cancer, with right sided disease showing no significant difference in OS between anti-EGFR therapy or bevacizumab in addition to chemotherapy (20).

Given the lack of stratification for transverse colon cancer in the above landmark trials, and the paucity of specific transverse colon cancer outcomes or dedicated transverse colon cancer therapy investigations, Solar Vasconcelos *et al.* sought to examine the efficacity of anti-EGFR therapeutic agents in the treatment of transverse colon adenocarcinoma via a retrospective pooled population analysis of two prospective randomized Canadian Cancer Trials Group/Australian Gastro-Intestinal Trials Group (CTG/AGITG) trials. CCTG/AGITG CO.17 and CO.20 both enrolled patients with heavily pre-treated metastatic colorectal cancer, that had adequate performance status and had no previous exposure to anti-EGFR therapy (21). CO.17 randomized EGFR positive patients to cetuximab plus supportive care (SC) or SC alone. Notably, the trial did not require prospective *RAS/BRAF* testing, however, *KRAS/NRAS* exons 2-4 and *BRAF* V600E genotyping was performed in later correlative analyses and was utilized by Solar Vasconcelos *et al.* in their patient selection. CCTG/AGITG CO.20 randomized the same patient population to receive cetuximab plus brivanib alaninate (a multi-targeted tyrosine kinase inhibitor) versus cetuximab plus placebo, with *KRAS* wild type testing performed after trial initiation, resulting in exclusion of *KRAS* exon 2 mutated patients (22-25).

Authors of this analysis pooled data from patients with confirmed *KRAS/NRAS/BRAF* wild type tumors from both arms of CO.17 and *KRAS* wild type from the single agent cetuximab arm in CO.20. Analyses were then performed based on the location of the primary tumor. Right sided tumors were defined as originating from the cecum distally to the hepatic flexure and left sided tumors originating at the splenic flexure distally to the rectum. The remaining tumors were considered in the transverse colon. A total of 553 patients were included, with most patients diagnosed with left sided primary tumors (75.9%). Transverse colon primary tumors were only found in 32 patients (5.8%), with the remaining considered right sided primary tumors

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(18.35%). The overwhelming majority (82.6%) received cetuximab, while 17.4% received SC alone. Outcomes including PFS and OS were examined for each primary tumor location comparing patients treated with cetuximab *vs.* SC alone. Survival outcomes were also analyzed for all patients treated with cetuximab based on primary tumor location, using patients with a transverse colon primary tumor as a reference. Additionally, using primary tumor location of the rectum as a reference, authors analyzed the outcomes of nine distinct areas of the colon (cecum, ascending colon, hepatic flexure, transverse colon, splenic flexure, descending colon, sigmoid, rectosigmoid junction, rectum). Multivariate analysis controlled for primary tumor location, treatment, and multiple baseline characteristics including stage at presentation, histologic tumor grade, lymph node involvement, tumor T-stage, metastatic or recurrent disease at presentation, time from initial diagnosis to randomization, number of previous systemic chemotherapies, number of organs with metastatic disease, presence of lung or liver metastases, basic demographic information (age, sex, performance status) and laboratory values.

Baseline characteristic analysis revealed that relative to patients with right or left side primary tumors, patients with transverse colon primary tumors had more poorly differentiated histologic grade (43.3%), more metachronous disease and peritoneal/retroperitoneal disease at presentation (50% and 21.9% respectively), had less liver or lung metastasis at presentation (21.9%), and were more often allocated to SC as primary treatment (31.2%).

When evaluating the efficacy of cetuximab within each primary tumor location, patients with transverse and right sided colon cancer did not see a statistically significant benefit in PFS or OS relative to SC alone, whereas left sided tumors saw improvement in both PFS and OS [hazard ratio (HR) 0.37, 0.51 respectively]. The use of cetuximab yielded similar minimal response rates in right sided and transverse tumors, no statistically significant difference in disease control rates (DCR) in right sided or transverse colon tumors relative to SC, and a lack of objective responses in transverse tumors. This was unlike left sided tumors, which exhibited a 10.4% objective response rate and significant improvement in DCR to 60.7% *vs.* 13.5% with SC alone. Outcomes were similar when examining only patients randomized to cetuximab, indicating no statistically significant difference in DCR, PFS or OS between right sided tumors and transverse tumors, but significantly inferior outcomes of patients with transverse colon tumors relative to left sided tumors.

As mentioned above, the authors analyzed the efficacy of cetuximab based on nine subsites within the colon, including: cecum, ascending colon, hepatic flexure, transverse colon, splenic flexure, descending colon, sigmoid colon, rectosigmoid junction, and rectum. Using the rectum as the reference group, multivariate analysis indicated patients with primary transverse colon malignancy had a significantly worse PFS and OS when treated with cetuximab relative to the rectum. Finally, the authors evaluated the prognostic impact of the tumor origination in the transverse colon, assessed by evaluating the PFS and OS of patients receiving SC alone, with the transverse colon as the reference. No statistically significant differences in PFS and OS were observed between the transverse colon and right and left sided primary tumors, however, numerically, left sided cancers had improved PFS and OS (HR 0.75 and 0.54 respectively) (*Table 1*) (21).

Overall, the authors' results reinforce the general perception that the transverse colon exhibits treatment responses similar to that of right sided colon cancers, with poor response to EGFR inhibitors, and similar prognostic features. This analysis is an important contribution to the paucity of literature addressing the treatment of transverse colon cancer, especially in the treatment refractory setting, and further clarifies the biological behavior of the disease. However, the authors recognize the limitations of their analysis. The study was a retrospective pooled analysis, with a very small patient population of transverse colon cancer, limiting the statistical power and causal relationships that can be determined. Additionally, CCTG/AGITG CO.20 likely included *RAS* and *BRAF* mutant colon cancers as patients were only screened for *KRAS* exon 2. Though the authors do note that if there were a high percentage of these cases included, there would have been a decreased ability to find statistical significance between the included cohorts. Further, although these results align with first line treatment studies of anti-EGFR therapy in combination with cytotoxic chemotherapy, indicating benefit only in left sided colorectal cancer, this pooled analysis was limited to treatment in the refractory setting. Therefore, it is difficult to draw clear conclusions of the efficacy of these interventions for transverse colon cancer in the front-line setting. This highlights the lack of data for the optimal treatment of transverse colon cancer in the first line setting and necessitates the evaluation of therapeutic agents for this subset of colon cancer.

We greatly appreciate the authors' efforts to advance the care of this understudied patient population. Yet, we believe there is still significant work that needs to be done

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Table 1 Clinical outcomes based on primary tumor location and intervention

Intervention	Transverse colon (n=32)	Left sided colon (n=420)	Right sided colon (n=101)
Cetuximab *vs.* SC	DCR: 27.3% *vs.* 20.0%; OR: 1.50 (95% CI: 0.25–9.18; P=0.66)	DCR: 60.7% *vs.* 13.5%; OR: 9.88 (95% CI: 4.91–19.90; P<0.0001)	DCR: 33.7% *vs.* 16.7%; OR: 2.54 (95% CI: 0.52–12.3; P=0.25)
	mPFS: 1.8 *vs.* 1.3 months; HR: 0.57 (95% CI: 0.26–1.28; P=0.16)	mPFS: 3.8 *vs.* 1.8 months; HR: 0.37 (95% CI: 0.28–0.48; P<0.0001)	mPFS: 1.9 *vs.* 1.9 months; HR: 0.7 (95% CI: 0.38–1.3, P=0.25)
	mOS: 5.9 *vs.* 2.1 months; HR: 0.63 (95% CI: 0.28–1.42; P=0.26)	mOS: 9.7 *vs.* 4.9 months; HR: 0.51 (95% CI: 0.39–0.68; P<0.0001)	mOS: 5.6 *vs.* 4.7 months; HR: 0.68 (95% CI: 0.35–1.32; P=0.25)
ORR with cetuximab	ORR: 0% (95% CI: 0–15.4%)	ORR: 10.4% (95% CI: 7.4–14.1%)	ORR: 3.4% (95% CI: 0.7–9.5%)
Sided colon *vs.* transverse colon: cetuximab alone	N/A	DCR: 60.7% *vs.* 27.3%; OR: 4.12 (95% CI: 1.57–10.78; P=0.004)	DCR: 33.7% *vs.* 27.3%; OR: 1.36 (95% CI: 0.48–3.82; P=0.58)
		mPFS: 3.8 *vs.* 1.8 months; HR: 0.49 (95% CI: 0.31–0.76; P=0.001)	mPFS: 1.9 *vs.* 1.8 months; HR: 0.78 (95% CI: 0.49–1.26; P=0.31)
		mOS: 9.7 *vs.* 5.9 months; HR; 0.42 (95% CI: 0.27–0.67; P=0.0002)	mOS: 5.6 *vs.* 5.9 months; HR: 0.82 (95% CI: 0.5–1.34; P=0.43)
Sided colon *vs.* transverse colon: SC alone	N/A	mPFS: 1.8 *vs.* 1.3 months; HR: 0.80 (95% CI: 0.4–1.60; P=0.52)	mPFS: 1.9 *vs.* 1.3 months; HR: 0.80 (95% CI: 0.33–1.93; P=0.62)
		mOS: 4.9 *vs.* 2.1 months; HR: 0.56 (95% CI: 0.28–1.14; P=0.11)	mOS: 4.7 *vs.* 2.1 months; HR: 0.70 (95% CI: 0.28–1.74; P=0.45)

SC, supportive care; DCR, disease control rate; OR, odds ratio; 95% CI, 95% confidence interval; mPFS, median progression-free survival; mOS, median overall survival; HR, hazard ratio; N/A, not applicable.

to optimize the care for patients with transverse colon cancer. Tumor characteristics and treatment response based on the right and left colon cancer models does not remain consistent amongst studies. For example, one very large observational study did observe that age, tumor grade and histological subtype had a linear correlation with distance from the ileocecal valve, consistent with the right and left colon cancer model, however, more aggressive phenotypes were observed in cecal and splenic flexure tumors, with less aggressive characteristics seen with ascending and descending colon (10). This underscores that this predetermined cut off right and left sided colorectal cancer does not fully encompass the fundamental complexity of colon cancer development.

Moreover, biologic behavior and efficacy of treatment may likely vary between locations in the transverse colon alone due to the differences in embryologic origin and associated mutational features. There is a significant need for clinical trials to include stratification based on tumor location within the colon, including the specific location of tumors within the transverse colon, so that differences in therapeutic responses can be ascertained between primary tumor origins. Due to the relative rarity of transverse colon cancer, the analysis of real-world data could prove crucial to our understanding of this disease. Ultimately, to properly inform our patients of their optimal treatment options and prognosis, it is imperative for future clinical trials to not only include patients with transverse colon cancer, but also have dedicated therapeutic trials for transverse colon cancer. We believe that grouping transverse colon cancer solely within right sided disease may prove to be suboptimal, and that judicious categorization based on embryologic origin, mutational profile and tumor characteristics is imperative to attain ideal outcomes.

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Footnote

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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