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Case Report

Curative metastasectomy in a patient with colon cancer harboring *BRAF*^{V600E} mutation and mismatch repair deficiency – a case study

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Abstract

Introduction: Both the *BRAF*^{V600E} mutation and mismatch repair deficiency bear prognostic and predictive relevance in metastatic colorectal cancer, however, the impact of their simultaneous occurrence on prognosis remains under-investigated.

Aim: Although retrospective studies support localised therapies for oligometastatic CRC in the general population, data for tumours harbouring both alterations are scarce.

Case study: In 2013, a 66-year-old female underwent radical resection for pT3 N0 distal sigmoid adenocarcinoma. Five months later she was diagnosed with oligometastatic recurrence in the right ovary. In 2014, she underwent hysterectomy and bilateral adnexectomy. Histopathology of the resected material confirmed adenocarcinoma metastasis in the right ovary. As the radicality of the resection was uncertain, the patient received 12 cycles of FOLFIRI chemotherapy after which the treatment was suspended due to no evidence of the disease. The follow-up imaging showed no signs of recurrence. A retrospective testing of tumor samples from metastasectomy revealed a *BRAF*^{V600E} mutation and loss of MLH1 and PMS2 expression indicating dMMR.

Results and discussion: Recently emerging data may suggest a better prognosis in metastatic colorectal cancer with coexisting *BRAF*^{V600E} mutation and dMMR as compared to cases with either of these biomarkers. The literature on the outcomes of metastatic resection in populations with both alterations is scarce. The patient described here is an example of a favourable outcome, with over 10-year survival after local treatment of colorectal metastases with aforementioned molecular profile.

Conclusions: Metastasis resection should be further investigated in colorectal cancers harbouring high risk molecular alterations.

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1. INTRODUCTION

Colorectal cancer (CRC) is the third most common cancer worldwide; approximately two-thirds of patients present with advanced-stage disease.[1] The most common sites of metastases are the liver, lungs and non-regional lymph nodes. Typical mutations associated with CRC pathogenesis include *APC* (adenomatous polyposis coli), *TP53* (Tumour protein p53), *KRAS* (Kirsten rat sarcoma virus) and *BRAF* (B-type Raf kinase). The most common activating *BRAF* mutation in CRC is the *V600E* mutation, which is present in ~11.4% (range 3.14%–23.14%) of all tumours, although the prevalence is variable and notably lower in Poland (6.7%).[2,3]

BRAF^{V600E} mutation status is a strong negative prognostic factor as well as an important predictive factor.[2,4,5] Patients with *BRAF*-mutant CRC are more often female and older, and tumours are more frequently located in the proximal colon.[6–8] *BRAF*^{V600E} mutation is more frequent in CRCs with poor differentiation, as well as mucinous and mixed neuroendocrine histologies. The disease is usually more advanced at the diagnosis both in terms of clinical stage and tumour burden.[2,9,10]

Mismatch repair deficiency (dMMR) can be found in 10–15% of all CRCs although the prevalence in metastatic stage is only about 5%.[11] It is associated with the loss of function of, primarily, MLH1 (MutL homolog 1), MSH2 (MutS homolog 2) and MSH6 (MutS homolog 6) proteins, although other, less common mechanisms i.e. loss of function in MSH3 (MutS homolog 3), PMS1 (PMS1 Homolog 1), PMS2 (PMS1 Homolog 2), MUTYH (E. coli MutY homolog) are possible. The molecular consequence of dMMR is microsatellite instability (MSI) – the presence of characteristic insertion-deletion length alterations at short tandem repeat loci ("microsatellites") throughout the genome. The role of dMMR and MSI as a predictive and prognostic biomarkers is a subject of ongoing debate as the two biomarkers seem to convey similar but not identical biological and clinical implications.[12] MSI/dMMR status is more frequently found in poorly differentiated CRCs and in cancers originating from the proximal colon.[10,13–15] MSI/dMMR CRCs also are proven to have a higher incidence of peritoneal involvement.[10,16] Of note, a recently emerging biomarker sharing similar properties seems to be proofreading deficiency related to POLE (polymerase epsilon) and POLD1 (polymerase delta subunit 1) alterations.[17] There are several methods of MSI/MMR diagnostics with next-generation sequencing that are highly sensitive, while PCR-based assays and immunohistochemistry have been widely used complementary methods.[18]

Despite the less frequent occurrence of individual mutations, *BRAF*^{V600E} is oftentimes observed in MSI/dMMR tumours (34.6%–42%) compared with CRC lacking MSI/dMMR (6.8%–8.2%).[4,7,19] Individually *BRAF* mutation and MSI-H are usually associated with an unfavourable course of disease (with *BRAF* being a stronger negative prognostic). However, cancers harbouring both alterations simultaneously appear to have a more favourable prognosis as compared to the ones with only *BRAF*^{V600E} or only MSI-H.[4,7,9,20]

Currently, the standard therapy for inoperable metastatic CRC is systemic treatment consisting of fluoropyrimidines, oxaliplatin, irinotecan, anti-EGFR, antiangiogenic antibodies and other agents. The prognosis is still unsatisfactory with the 5-year rate survival not exceeding 10%. Two subgroups of patients have a distinctively improved prognosis: patients presenting with resectable oligometastatic disease who undergo radical resection (~38% of 5-year survival and ~26% of 10-year survival) and patients with dMMR/MSI receiving immune checkpoint inhibitors (with >50% of patients treated with upfront pembrolizumab being alive and progression-free at 45 months of follow-up and >50% patients treated with ipilimumab + nivolumab in subsequent lines being alive and without progression at 51 months of follow-up) with recent trials (such as Checkmate 8HW) showing even more promising results.[3,4,7,18,21–24]

In contrast to overall good results after metastasectomy in oligometastatic CRCs, patients with either *BRAF*^{V600E} or MSI/dMMR tumours have poorer outcomes after metastatic surgery.[25] The Yaeger et al study indicated only 61% of patients with *BRAF*-mutant CRCs lived for 2 years after the surgery in comparison to 86% with wild type *BRAF*. Despite worse OS after surgical treatment in this group of patients it remains a treatment option for selected patients.[26–28] Data assessing the outcomes of metastatic resection in patients harbouring both *BRAF* mutation and MSI/dMMR are lacking.

2. AIM

The purpose of this publication is to present a case of a 66-year-old female patient with metastatic colorectal cancer harbouring both *BRAF^V600E^* and dMMR who achieved durable remission following radical metastasectomy.

3. CASE STUDY

A 66-year-old woman, with a history of nicotinism and coronary artery disease, demonstrated weakness, episodes of night sweats, and haematochezia. Iron deficiency without anemia was found in the laboratory studies. The colonoscopy showed an infiltration in the sigmoid colon. Biopsy confirmed adenocarcinoma.

The patient underwent an anterior rectal resection in October 2013 – 2 weeks after the diagnosis. The microscopic evaluation showed mucinous tubular adenocarcinoma pT3N0 (AJCC, 2010).

Five months later her CEA levels increased from 6.38 ng/mL to 9.88 ng/mL. A positron emission tomography (PET) using F-18 fluorodeoxyglucose (FDG) combined with computed tomography, revealed a tumorous mass in the pelvis, connected to the postoperative site and upper-right part of the uterus. The lesion had increased FDG uptake (Figure 1).

In June 2014, the patient underwent abdominoperineal resection of the rectum with hysterectomy and bilateral adnexectomy. Histopathological examination confirmed metastatic

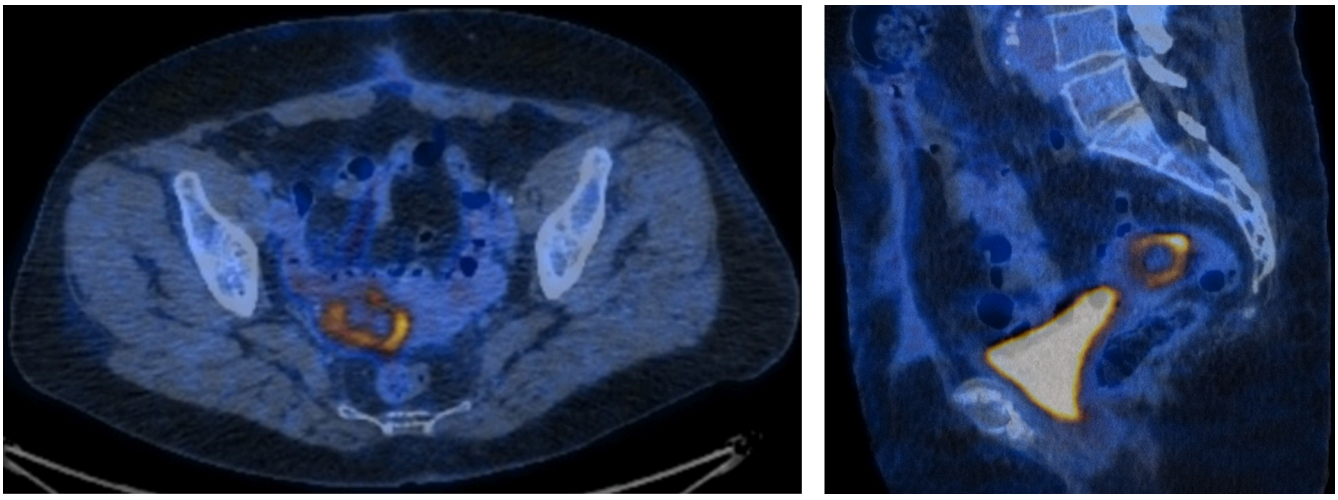

Figure 1. 18F-FDG PET/CT demonstrating a hypermetabolic pelvic lesion adjacent to the upper right uterus and right ovary (transverse and sagittal planes).

adenocarcinoma in the right ovary measuring 4 cm, and adenoma in the resected rectum with the radicality of the resection not assessable.

Due to uncertain resection margins and a high risk of recurrence, the patient was informed about palliative chemotherapy options and started on the FOLFIRI regimen (irinotecan 180 mg/m^2; L-folic acid 100 mg/m^2; bolus of 5-fluorouracil 400 mg/m^2; continuous 46-h infusion of 5-fluorouracil 600 mg/m^2) 2 months after the last operation. After 12 well-tolerated cycles, treatment was suspended considering no evidence of disease on imaging.

4. RESULTS

As molecular testing became standard of care, archived metastasectomy samples were retrospectively analyzed. The tests revealed an activating *BRAF*^{V600E} mutation (polymerase chain reaction and Sanger sequencing methods) and loss of *MLH1* and *PMS2* with *MSH2* and *MSH6* intact (on immunohistochemistry) consistent with dMMR.

After the treatment was concluded, the patient was actively followed up for >10 years, without any signs of recurrence.

5. DISCUSSION

This showcased case report illustrates an example of *BRAF*^{V600E} mutant dMMR CRC manifesting excellent response to primary tumour resection, followed by metastasectomy combined with chemotherapy with FOLFIRI regimen.

The impact of MSI/dMMR on prognosis appears to differ between metastatic and non-metastatic patients. In M0 disease, MSI/dMMR does not appear to significantly reduce overall survival.[29] Some studies proved MSI/dMMR status to be a positive prognostic factor in stage II and III CRC.[30,31] In contrast, MSI/dMMR patients with advanced CRC have shorter OS and progression-free survival (PFS) independent of somatic mutation status, and treatment used (with exception of immunotherapy). Despite the above results, recent studies may suggest that the coexistence of MSI/dMMR and *BRAF*^{V600E} mutation in metastatic CRCs results in a less aggressive course of disease simultaneously with longer PFS and OS.[4,7,9]

Ovarian metastases are uncommon, occurring in approximately 3–5% of women with colorectal cancer with a higher prevalence in younger patients[32,33] and some cases presenting with significant tumor burden.[34] Clinically, CRC ovarian metastases frequently coexist with peritoneal dissemination and are difficult to stage accurately because cross-sectional imaging tends to underestimate small-volume disease. Diffusion-weighted magnetic resonance imaging and FDG-PET offer higher accuracy for mapping disease burden and operability.[35] Differential diagnosis of mucinous ovarian tumors relies on immunohistochemistry: a CK7–/CK20+ and CDX2+ (often SATB2+) phenotype with PAX8 negativity supports colorectal origin, whereas primary ovarian mucinous neoplasms are typically CK7+ and PAX8+.[36]

The oligometastatic CRCs are currently advised to be treated with radical resection, if the surgery is technically and medically feasible. In retrospective studies, patients with either MSI/dMMR or *BRAF*^{V600E} mutated cancers, undergoing local treatment of metastases had worse outcomes as compared to the rest of CRC patients[27] however, outcomes still exceed those typically achieved with systemic therapy alone.[26,28] Ovarian metastases show relative resistance to systemic chemotherapy compared with extra-ovarian sites and there is data supporting the prognostic impact of ovarian metastasectomy.[37,38]

Typical chemotherapy regimens for advanced *BRAF*-mutated CRC include 5-fluorouracil or capecitabine combined

with oxaliplatin, irinotecan, or both, with optional bevacizumab. Current options also include immunotherapy and *BRAF*-targeted therapy.[18] The described patient received the FOLFIRI regimen, due to an uncertainty on the radicality of the resection, very high risk of recurrence as judged by the treating physician and constraint on bevacizumab reimbursement at the time.

By the end of the 10-year active follow-up in the reported patient, there was no sign of disease recurrence. It is worth noting that patients with previously resected oligometastases have a potential for late recurrences – therefore a longer, 10-year, follow-up is required.[39]

The *BRAF* protein is also a therapeutic target. In the exemplary BEACON study $BRAF^{V600E}$ advanced CRC patients were randomized into 3 groups: encorafenib with binimetynib and cetuximab vs encorafenib with cetuximab vs chemotherapy with cetuximab. The study showed a significant advantage of both triple-agent and double-agent anti-*BRAF* therapy compared to the control group, manifested by prolonged OS and PFS.[40] Given similar effectiveness in both active arms – only the doublet was approved by the regulatory authorities. Recent BREAKWATER study data suggest encouraging first-line activity of encorafenib + cetuximab + chemotherapy, with response durability and early OS comparable to non-mutant CRC.

6. CONCLUSION

(1) This case showcases a satisfactory result of surgical treatment of $BRAF^{V600E}$ mutant and MSI-H metastatic CRC.
(2) The treatment consisting of metastasectomy and chemotherapy yielded a durable response ongoing beyond 10 years, consistent with potential cure.
(3) Given the single-case nature of this report and potential selection and reporting biases, these observations should be regarded as hypothesis-generating rather than definitive. Management decisions should be individualized and, where feasible, evaluated within prospective studies.

Informed consent

Informed consent was obtained from participant included in the study.

Ethics approval

Article has been conducted according to the principles stated in the Declaration of Helsinki.

The patient consented to the publication of the case study.

All authors have read and agreed to the published version of the manuscript.

Conflict of interest

None declared.

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Author Contributions

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Data interpretation: NO, MR-S, PM, AS-S, PW, PMP
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