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(CASE REPORT)

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Metastatic Malignant Melanoma to the Descending and Sigmoid Colon, the Liver, and the Lung: Case Report and a Brief Review of the Literature

Nimra Khan [4], Natalia Berio-Perez [3], Oscar Ramos [3], Melissa Perez [5], Anthony Khalid Shams [3], Jessica Jahoda [1, 2] and Mohamed Aziz [1, 6, *]

[1] *Research Writing & Publication (RWP), LLC, NY, USA.*
[2] *Memorial Healthcare System, Pembroke Pines, FL, USA.*
[3] *American University of the Caribbean, AUC, St. Maarten.*
[4] *Ross University School of Medicine, Barbados.*
[5] *St. George's University School of Medicine, Grenada.*
[6] *Saint Vincent's Medical Center, New York City, NY, USA.*

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Abstract

Metastatic melanoma to the gastrointestinal tract is frequently detected at autopsy but remains underdiagnosed during life because of nonspecific symptoms such as anemia, weight loss, and altered bowel habits. Colonic involvement is rarer than small-bowel disease and can cause occult bleeding or iron-deficiency anemia.

We report a man with a history of ulcerated T3N0M0 cutaneous melanoma resected four years earlier who presented with fatigue, 15-lb weight loss, intermittent melena, and iron-deficiency anemia (hemoglobin 8.2 g/dL). Colonoscopy revealed two ulcerated pigmented masses in the descending and sigmoid colon. PET-CT demonstrated FDG-avid colonic lesions with mesenteric and retroperitoneal lymphadenopathy, pulmonary nodules, and hepatic metastases; brain MRI was initially negative. Histopathology showed pleomorphic epithelioid cells containing melanin pigment with strong S100, SOX10, HMB-45, and Melan-A expression and negative cytokeratin and CDX2, confirming metastatic melanoma. Molecular analysis identified a BRAF V600E mutation.

Combination nivolumab and ipilimumab produced a transient partial response, but the disease progressed within five months, followed by failure of BRAF/MEK-targeted therapy. Surgical resection was not feasible due to disseminated disease. The patient died eight months after diagnosis.

This case underscores the aggressive course of colonic metastatic melanoma and highlights the importance of early recognition, molecular profiling, and multidisciplinary management.

Keywords: Metastatic Melanoma; Sites of Metastatic Melanoma; Melanocytic Immunohistochemistry Markers; Combination Nivolumab and Ipilimumab; BRAF/NRAS Mutations

1. Introduction

Malignant melanoma is a highly aggressive cutaneous neoplasm with a notorious propensity for widespread systemic dissemination. While the most common sites of metastasis include the lungs, liver, and brain, the gastrointestinal (GI) tract is increasingly recognized as a site of involvement in advanced disease. [1] Post-mortem studies suggest that up to 60% of patients with terminal melanoma harbor GI metastases; however, these are clinically diagnosed in only 1%

* Corresponding author: Mohamed Aziz

to 5% of living patients due to their often asymptomatic or nonspecific presentation. [2] Within the GI tract, the small bowel is the most frequent site of involvement (51-71%), whereas colonic metastasis is significantly rarer, occurring in approximately 0.18% to 2.1% of cases. [3]

Patients typically present with occult bleeding, anemia, or altered bowel habits, often years after the primary tumor excision. [4] The diagnostic challenge lies in distinguishing metastatic lesions from primary colorectal malignancies, necessitating a high index of suspicion in patients with a history of melanoma. Advances in immunohistochemistry and molecular profiling, particularly for BRAF mutations, have revolutionized the management of these patients. This report describes a rare case of colonic metastatic melanoma presented with symptomatic anemia and weight loss, highlighting the critical role of multidisciplinary management and the evolving landscape of immunotherapy in treating extra-cutaneous disease.

2. Case Presentation

2.1. Clinical Presentation

The patient initially presented with a three-month history of progressive fatigue, unintentional 15-pound weight loss, and intermittent dark stools. He reported altered bowel habits with increasing constipation alternating with diarrhea. Initially, he attributed the symptoms to dietary changes and age and managed them conservatively with over-the-counter fiber supplements. However, worsening fatigue prompted his wife to insist on a medical evaluation. His primary care physician noted pallor and ordered basic laboratory tests, which revealed significant anemia (hemoglobin 8.2 g/dL). Given his melanoma history and concerning gastrointestinal symptoms with iron-deficiency anemia, urgent gastroenterology referral and colonoscopy were arranged for suspected gastrointestinal malignancy.

2.2. Physical Examination, Laboratory Findings, and History

Physical examination revealed a cachectic-appearing male with conjunctival pallor. Vital signs were stable. Abdominal examination showed mild left lower quadrant tenderness without masses or organomegaly. No peripheral lymphadenopathy was detected. Skin examination revealed a well-healed scar on the upper right side without satellite lesions. Laboratory results: Hemoglobin 8.2 g/dL (MCV 72 FL), ferritin 12 ng/mL, LDH 890 U/L (elevated), normal liver enzymes, CEA normal. Personal history: T3N0M0 (2.8mm Breslow thickness, ulcerated) melanoma excised from the right upper back four years prior; local recurrence treated with wide re-excision two years later. Completed adjuvant interferon therapy. Family history: Father, colon cancer at age 68.

2.3. Radiographic Studies, Findings, and Differential Diagnosis

Colonoscopy identified two polypoid masses in the descending colon (4.5 cm) and sigmoid colon (3.2 cm), both with central ulceration and dark pigmentation. The mucosa appeared intact between lesions. Biopsies are obtained from both lesions. CT chest/abdomen/pelvis with contrast: Demonstrated circumferential wall thickening of the descending and sigmoid colon corresponding to colonoscopy findings. Multiple enlarged mesenteric and retroperitoneal lymph nodes (largest 2.3 cm). Small pulmonary nodules bilaterally (largest 8 mm, right lower lobe). Liver showed two hypodense lesions (1.2 cm and 0.9 cm) with peripheral enhancement, no evidence of bowel obstruction. Old surgical changes were noted in the subcutaneous tissues of the right upper back. PET-CT: Demonstrated FDG-avid colonic lesions (SUV max 12.4 and 10.8), hypermetabolic mesenteric and retroperitoneal lymphadenopathy, pulmonary nodules with mild uptake, and hepatic lesions showing moderate FDG activity (SUV max 6.2).

Differential diagnosis included metastatic melanoma (primary consideration given history and pigmented lesions), primary colorectal adenocarcinoma with melanosis coli, gastrointestinal stromal tumor (GIST), synchronous primary malignancies, and metastatic disease from another primary source.

2.4. Multidisciplinary Tumor Board Discussion and Management Consensus

The multidisciplinary tumor board included medical oncology, surgical oncology, gastroenterology, radiology, and pathology. Discussion centered on confirming the diagnosis through tissue analysis and determining optimal systemic therapy versus surgical intervention. Given the pigmented appearance and melanoma history, metastatic melanoma was strongly suspected. The consensus was to proceed with comprehensive immunohistochemical analysis of colonic biopsies and molecular profiling for BRAF/NRAS mutations. Surgery was deferred pending pathologic confirmation and completion of staging. The team recommended initiating systemic therapy as first-line treatment. A brain MRI was ordered to complete staging before treatment initiation.

2.5. Pathology (Microscopic Description, Differential Diagnosis, IHC, and Molecular Studies)

Sections from both colonic biopsies revealed diffuse infiltration of the lamina propria and submucosa by sheets of pleomorphic epithelioid cells with abundant cytoplasm. Many cells contained prominent brown-black melanin pigment. Nuclei were large, irregular, and hyperchromatic with prominent nucleoli. Mitotic figures were frequent (>15/10 HPF). The overlying colonic mucosa showed focal ulceration but no dysplasia. No glandular architecture or features of adenocarcinoma were identified. (Figure 1 A, B, C, D) Pathology differential diagnosis included Metastatic melanoma (primary consideration, Primary mucosal melanoma of the colon (extremely rare), Poorly differentiated carcinoma with melanin-like pigment, Melanotic neuroendocrine tumor. Immunohistochemistry (IHC) Results were reported as follows: S100: Strongly positive (diffuse), SOX10: Strongly positive (nuclear), Melan-A: Positive, HMB-45: Positive, MART-1: Positive, Cytokeratin (AE1/AE3): Negative, CDX2: Negative, Chromogranin: Negative, Synaptophysin: Negative. Next-generation sequencing panel revealed BRAF V600E mutation (allele frequency 42%). No NRAS, KIT, or NTRK mutations detected. Tumor mutational burden was 8 mutations/megabase. PD-L1 expression by IHC showed a tumor proportion score of 15%.

Additional biopsy was obtained from the liver, which showed identical features as the masses in the colon. Final diagnosis was metastatic melanoma involving the colon, and other identified sites, consistent with metastasis from the patient's previously resected cutaneous melanoma.

2.6. Second Tumor Board: Treatment Decision and Outcome

Brain MRI was negative for intracranial metastases. The tumor board reconvened after pathology confirmation. Given the BRAF V600E mutation, the team discussed targeted therapy (BRAF/MEK inhibitors) versus immunotherapy. Given the 4-year disease-free interval and the potential for durable response, combination immunotherapy with nivolumab and ipilimumab was recommended as first-line treatment. Surgical resection was deferred unless complications arose or after achieving disease control. The patient consented and began treatment with nivolumab 1 mg/kg plus ipilimumab 3 mg/kg every three weeks for four doses, followed by nivolumab maintenance.

2.7. Outcome, Follow-up, and Patient Status After Treatment: Initial Response (Weeks 0-16)

The patient tolerated induction immunotherapy with grade 2 diarrhea and fatigue managed supportively. Restaging CT at 12 weeks demonstrated a partial response with 45% reduction in colonic lesions, decreased lymphadenopathy, and stable pulmonary nodules. Hepatic lesions showed near-complete resolution. LDH normalized to 245 U/L. Hemoglobin improved to 11.8 g/dL. He transitioned to nivolumab maintenance monotherapy.

2.8. Unfavorable Course (Months 5-8)

At five months, surveillance imaging revealed disease progression with new liver metastases, enlarging pulmonary nodules, and a new 1.4 cm left adrenal lesion. Colonic lesions remained stable. The patient developed worsening fatigue and confusion. Brain MRI demonstrated three new parenchymal metastases (0.8 cm, 1.2 cm, and 1.5 cm). Despite stereotactic radiosurgery to brain lesions and switching to combination BRAF/MEK inhibitor therapy (dabrafenib/trametinib), the disease continued progressing. He ultimately transitioned to hospice care and died eight months after initial diagnosis.

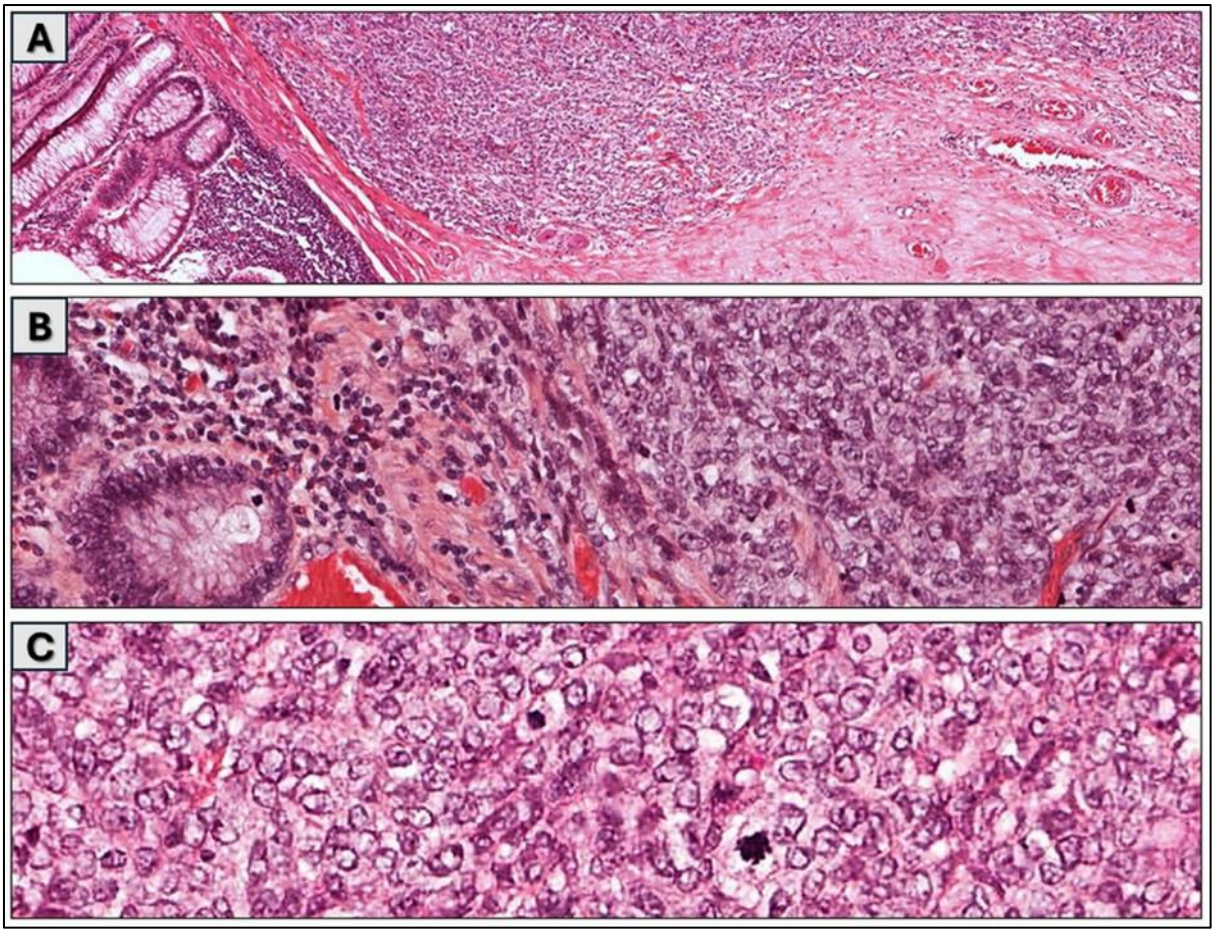

Figure 1 Microscopic examination of metastatic melanoma to the colon

- 1A: Low power view showing diffuse infiltration of the colonic lamina propria and submucosa by solid sheets of metastatic melanoma (H&E Stain X20).
- 1B: Intermediate power view showing no glandular architecture or features of adenocarcinoma. The overlying colonic mucosa shows no evidence of dysplasia (H&E Stain X40).
- 1C: High power view showing pleomorphic epithelioid cells with abundant cytoplasm. Nuclei are large, irregular, and hyperchromatic with prominent nucleoli. Mitotic figures are frequent (H&E Stain X60)

3. Discussion

3.1. Background (History, Epidemiology, Risk factors, and WHO Classification)

Melanoma is a malignancy that arises from melanocytes, which are present in the skin, uvea, and mucosal membranes such as those in the nasopharynx, genital region, or oral cavity. [5] Risk factors include age over 50 years, light skin, male sex, exposure to ultraviolet (UV) rays, and a family or personal history of melanoma. [6] The disease often presents aggressively and is prone to metastasize to sites including the lungs, liver, lymph nodes, and brain. [7]

Evidence of metastatic melanoma has been identified in skeletal remains from approximately 2,400 years ago, indicating the disease has existed for centuries. [8] Metastasis to the colon is uncommon, with symptomatic involvement reported in only 0.18% to 2.1% of cases. However, gastrointestinal (GI) involvement is found in up to 60% of autopsies performed on patients who died from advanced melanoma. The small bowel is the most frequent site of GI metastasis, while the stomach is the least common. [9] Prognosis declines significantly with metastasis; the five-year survival rate drops from 98.2% in localized disease to approximately 30% in advanced stages. [10]

Although most GI melanomas are asymptomatic, they may present with nonspecific symptoms. Metastatic melanoma to the colon is classified as stage IV (advanced or metastatic disease) according to the Tumor, Node, Metastasis (TNM)

system, and is assigned the International Classification of Diseases (ICD)-11 code 2D85, which denotes malignant neoplasm metastasis in the large intestine. [11]

3.2. Pathogenesis, Pathophysiology

Metastatic melanoma involving the colon originates from malignant transformation of melanocytes, neural crest–derived cells normally located in the skin, eyes, and mucosal surfaces. [12] Approximately 50% of cutaneous melanomas harbor the BRAF V600E mutation, which activates the MAPK signaling pathway, promoting uncontrolled cellular proliferation, survival, and metastatic potential. [13,14] Additional molecular alterations, including TERT promoter mutations and loss of tumor suppressor function in CDKN2A and PTEN, further contribute to tumor progression and invasion. [15]

Dissemination of the gastrointestinal tract occurs primarily via hematogenous spread. Within the digestive system, the small bowel is the most common site of involvement, followed by the colon. [1] Proposed mechanisms facilitating gastrointestinal homing include activation of the CCR9/CCL25 chemokine axis and increased tissue factor expression, which enhance vascular adhesion and extravasation. [16,17] The interval between primary melanoma and detection of colonic metastasis typically ranges from 48 to 63 months. [1,18]

Clinically, mucosal and submucosal infiltration leads to ulceration and bleeding, often presenting as iron-deficiency anemia and altered bowel habits. [18,19] Histopathology characteristically demonstrates pleomorphic epithelioid cells containing melanin pigment with immunohistochemical positivity for S100, SOX10, Melan-A, and HMB-45. [20] Elevated serum lactate dehydrogenase correlates with tumor burden and disease extent. [1]

3.3. Comparative Analysis of Our Case with the Existing Literature. (Clinical, Radiology, Pathology, Lab, Diagnosis, Management, Outcome)

The clinical presentation of colonic metastatic melanoma often mimics primary colorectal adenocarcinoma, characterized by fatigue, weight loss, and iron-deficiency anemia, as seen in our patient. However, the radiographic and endoscopic features of melanoma are distinct. Endoscopically, metastatic melanoma typically presents as multiple polypoid, ulcerated, or pigmented masses, whereas primary adenocarcinoma usually appears as a solitary, circumferential, or fungating lesion [21] Radiologically, CT and PET-CT are essential for staging melanoma metastases, which are frequently hypermetabolic (high SUV max) and may involve multiple non-contiguous segments of the bowel, a pattern less common in primary GI cancers. [22]

Pathologically, the diagnosis relies on immunohistochemical (IHC) markers. Unlike primary adenocarcinoma, which is typically CDX2- and cytokeratin-positive, metastatic melanoma expresses S100, SOX10, HMB-45, and Melan-A. [23] The presence of melanin pigment, though characteristic, is not universal, underscoring the need for IHC in amelanotic variants. Management has shifted from primarily surgical palliation to a precision-oncology approach. Historically, the 5-year survival for metastatic GI melanoma was less than 10%. [23] In the current era, combination immunotherapy (e.g., nivolumab and ipilimumab) and targeted therapies for BRAF V600E mutations have improved median overall survival to nearly 72 months in selected cohorts. [24]

While surgical resection remains indicated for complications like obstruction or perforation, systemic therapy is now the cornerstone of management for oligometastatic disease. This case demonstrates that even with a significant initial partial response to dual-agent immunotherapy, the biological volatility of melanoma can lead to rapid systemic progression, including to the central nervous system. This highlights the need for continuous, high-frequency surveillance and a flexible treatment strategy that can pivot between immunotherapy and targeted BRAF/MEK inhibition as the clinical situation evolves.

4. What Have We Learned from This Case?

This case provides several critical clinical insights into the management of late-stage melanoma. First, it reinforces the "red flag" of iron-deficiency anemia in any patient with a history of cutaneous melanoma, regardless of the disease-free interval. Our patient’s four-year interval underscores that melanoma can remain dormant and recur in atypical sites long after the primary treatment. Clinicians must recognize that GI symptoms in these patients should prompt immediate endoscopic and radiographic evaluation rather than conservative management.

Diagnostically, the case highlights the necessity of comprehensive molecular profiling. The identification of the BRAF V600E mutation not only confirmed the diagnosis but also provided a secondary therapeutic target when immunotherapy eventually failed. A key lesson is the unpredictable nature of the treatment response. At the same time, the patient initially showed a robust 45% reduction in tumor burden; the rapid development of intracranial and adrenal metastases illustrates the high biological volatility of metastatic melanoma. This suggests that even with a good initial response to immunotherapy, frequent and comprehensive surveillance (including brain MRI) is mandatory.

Furthermore, the case reinforces that while immunotherapy has transformed the prognosis of metastatic melanoma, it is not curative for all, and the transition to palliative care remains a necessary consideration in the face of rapid progression. Ultimately, this case challenges the traditional view of GI metastasis as a purely terminal event, suggesting that a multidisciplinary approach can provide meaningful, albeit sometimes temporary, disease control.

5. Conclusion

We present this case to emphasize the rare but clinically significant occurrence of colonic metastasis from cutaneous melanoma. This report adds value to the medical community by illustrating the entire clinical trajectory, from the initial presentation of nonspecific GI symptoms to the implementation of modern dual-agent immunotherapy and the eventual management of treatment resistance. The rationale for reporting this case is twofold: to increase clinical awareness of the GI tract as a site for late melanoma recurrence and to document the real-world challenges of managing BRAF-mutated metastatic disease in the immunotherapy era. While therapeutic advancements have significantly extended survival, this case serves as a sobering reminder of the aggressive nature of melanoma and the limitations of current treatments. It underscores the importance of a high index of suspicion, the utility of IHC in definitive diagnosis, and the need for personalized, multidisciplinary care to optimize outcomes for patients with extra-cutaneous metastatic spread.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Statement of informed consent

This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified prior to analysis. No patient contact or intervention was involved, and informed consent was not required in accordance with institutional policies.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

References

[1] Yee EJ, Thielen ON, Truong R, et al. Metastatic Melanoma to the Small Bowel and Colon: A Systematic Review of the Global Experience and Institutional Cohort Analysis Detailing a Rare Clinical Entity. J Surg Oncol. 2024;131(4):671-684.

[2] Tan SBT, Lamparelli M. A Case Report of Metastatic Melanoma in the Transverse Colon. Cureus. 2025;17(3):e80659.

[3] Patel JK, Didolkar MS, Pickren JW, Moore GE. Metastatic cancer to the gastrointestinal tract. A study of 435 cases. Arch Surg. 1978;113(3):261-264. (Updated with recent incidence data from Yee et al., 2024).

[4] Liang KV, Sanderson SO, Nowakowski GS, Arora AS. Metastatic malignant melanoma of the gastrointestinal tract. Mayo Clin Proc. 2006;81(4):511-516.

[5] Heistein JB, Acharya U, Mukkamalla SKR. Malignant melanoma. U.S. National Library of Medicine; 2024. StatPearls. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470409/

[6] Serrao EM, Costa AM, Ferreira S, McMorran V, Cargill E, Hough C, Shaw AS, O'Carrigan B, Parkinson CA, Corrie PG, Sadler TJ. The different faces of metastatic melanoma in the gastrointestinal tract. Insights into Imaging. 2022 Oct 4;13(1):161.

[7] Drevelegkas K, Bangeas P, Paramythiotis D, Andrews T, Zakeri S. Metastatic spread of cutaneous melanoma to the sigmoid colon: a rare but important consideration. Hippokratia. 2017 Oct;21(4):194.

[8] Rebecca VW, Sondak VK, Smalley KS. A brief history of melanoma: from mummies to mutations. Melanoma research. 2012 Apr 1;22(2):114-22.

[9] Tan SB, Lamparelli M. A Case Report of Metastatic Melanoma in the Transverse Colon. Cureus. 2025 Mar 16;17(3).

[10] Adla A, Tirumalareddy RR, Snell P. Metastatic Melanoma to the Stomach: A Rare Case of Tumor Burden Beyond the Skin. Cureus. 2025 Jul 9;17(7):e87636.

[11] Kohoutova D, Worku D, Aziz H, Teare J, Weir J, Larkin J. Malignant melanoma of the [12] gastrointestinal tract: symptoms, diagnosis, and current treatment options. Cells. 2021 Feb 5;10(2):327.

[12] Castro-Pérez E, Singh M, Sadangi S, Mela-Sánchez C, Setaluri V. Connecting the dots: melanoma cell of origin, tumor cell plasticity, trans-differentiation, and drug resistance. Pigment cell & melanoma research. 2023 Sep;36(5):330-47.

[13] Bharti J, Gogu P, Pandey SK, Verma A, Yadav JP, Singh AK, Kumar P, Dwivedi AR, Pathak P. BRAF V600E in cancer: Exploring structural complexities, mutation profiles, and pathway dysregulation. Experimental Cell Research. 2025 Mar 1;446(1):114440.

[14] Castellani G, Buccarelli M, Arasi MB, Rossi S, Pisanu ME, Bellenghi M, Lintas C, Tabolacci C. BRAF mutations in melanoma: biological aspects, therapeutic implications, and circulating biomarkers. Cancers. 2023 Aug 8;15(16):4026.

[15] Schadendorf D, Van Akkooi AC, Berking C, Griewank KG, Gutzmer R, Hauschild A, Stang A, Roesch A, Ugurel S. Melanoma. The Lancet. 2018 Sep 15;392(10151):971-84.

[16] Amersi FF, Terando AM, Goto Y, Scolyer RA, Thompson JF, Tran AN, Faries MB, Morton DL, Hoon DS. Activation of CCR9/CCL25 in cutaneous melanoma mediates preferential metastasis to the small intestine. Clinical cancer research. 2008 Feb 1;14(3):638-45.

[17] Mueller BM, Reisfeld RA, Edgington TS, Ruf W. Expression of tissue factor by melanoma cells promotes efficient hematogenous metastasis. Proceedings of the National Academy of Sciences. 1992 Dec 15;89(24):11832-6.

[18] Park JS, Ng KS, Saw RP, Thompson JF, Young CJ. Metastatic melanoma to the colon, rectum, and anus: a 50-year experience. Annals of Surgical Oncology. 2018 Aug;25(8):2178-83.

[19] La Selva D, Kozarek RA, Dorer RK, Rocha FG, Gluck M. Primary and metastatic melanoma of the GI tract: clinical presentation, endoscopic findings, and patient outcomes. Surgical endoscopy. 2020 Oct;34(10):4456-62.

[20] Cazzato G, Battista S, Ingravallo G, Castelvetere M, Sassano A, Cassano M, Macciomei MC, Parente P. Gastro-intestinal metastasis from Melanoma: Clinico-pathological clues. Pathology-Research and Practice. 2023 Jan 1;241:154259.

[21] Schuchter LM, Green R, Fraker D. Primary and metastatic diseases in malignant melanoma of the gastrointestinal tract. Curr Opin Oncol. 2000;12(2):181-185.

[22] Bender GN, Maglinte DD, McLarney JH, et al. Malignant melanoma: patterns of metastasis to the small bowel, sigmoid colon, and rectum. AJR Am J Roentgenol. 2001;176(6):1441-1445.

[23] Ordóñez NG. Value of melanocytic markers in the diagnosis of tumors of the gastrointestinal tract. Adv Anat Pathol. 2014;21(2):117-133.

[24] Wolchok JD, Chiarion-Sileni V, Gonzalez R, et al. Long-Term Outcomes with Nivolumab Plus Ipilimumab or Nivolumab Alone Versus Ipilimumab in Patients with Advanced Melanoma. J Clin Oncol. 2022;40(2):127-137.