

CASE REPORT

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Marked preoperative regression of biopsy-diagnosed microsatellite instability-high colon adenocarcinoma with a small residual traditional serrated adenoma: A case report

Kazuhito Minami, Hiroki Funakoshi, Yumi Oshiro,
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ABSTRACT

Introduction: Marked regression of colorectal cancer before surgery is extremely rare. Microsatellite instability-high (MSI-high) tumors are characterized by a high neoantigen burden and pronounced immunogenicity. Biopsy-associated tissue injury may induce local inflammatory and reparative responses; whether these responses can augment antitumor immunity in an immunogenic tumor microenvironment remains uncertain. We report a case of MSI-high colorectal adenocarcinoma that markedly regressed after biopsy, with no residual carcinoma identified in the resected specimen. The temporal and histopathologic findings raise the possibility of an interplay between biopsy-associated tissue injury and the intrinsic immunogenicity of an MSI-high tumor.

Case Report: Initial colonoscopy in a man in his 70s identified an ascending colon lesion consisting of a protruded component and a shallow depressed component.

Magnifying endoscopy demonstrated irregular vascular and surface patterns corresponding to Japan NBI Expert Team (JNET) type 2B, together with a type Vi pit pattern, raising suspicion of submucosal invasion. Biopsy specimens obtained from the lesion revealed moderately to well-differentiated adenocarcinoma. Polymerase chain reaction (PCR)-based MSI testing of the biopsy specimen demonstrated MSI-high status. Follow-up colonoscopy performed 36 days later showed regression of the protruded component. Laparoscopic ileocecal resection (LIR) with lymph node dissection was performed 77 days after the initial diagnosis. Macroscopically, only a small protruded lesion measuring approximately 3 mm remained at approximately the same longitudinal level as the tattoo but several centimeters away along the colonic wall. Histopathologic examination revealed a 3-mm traditional serrated adenoma (TSA) with low-grade dysplasia, accompanied by prominent submucosal fibrosis in the presumed tumor bed. No viable adenocarcinoma was identified despite extensive additional sectioning. Moderate lymphocytic infiltration accompanied by lymphoid follicle formation was observed within the presumed tumor bed.

Conclusion: Although the underlying mechanism cannot be definitively established, the interval endoscopic regression observed after biopsy and the absence of residual carcinoma in the resected specimen are consistent with a possible regression process. However, complete mechanical removal of the malignant component during biopsy or changes resulting from procedure-related tissue injury cannot be excluded. The MSI-high status and lymphocytic infiltration with lymphoid follicle formation provide a plausible immunologic context, but the contribution of an immune-mediated mechanism remains speculative.

Keywords: Biopsy, Colorectal cancer, MSI-high, Tumor regression

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INTRODUCTION

Marked regression of malignant tumors in the absence of anticancer treatment is rare, and well-documented cases in colorectal cancer are exceptional [1]. Microsatellite instability-high (MSI-high) colorectal cancers are characterized by a high tumor mutational burden and pronounced immunogenicity, often accompanied by tumor-infiltrating lymphocytes and prominent host immune responses [2, 3]. Immune checkpoint inhibitors have demonstrated substantial efficacy against advanced or unresectable MSI-high colorectal cancer; however, regression of clinically localized MSI-high colorectal cancer before definitive surgery has rarely been reported. Here, we describe a case of MSI-high colorectal adenocarcinoma that regressed after biopsy and before surgery, leaving only a residual serrated adenoma, together with stromal changes consistent with tissue injury and histologic evidence of a local immune response. This case report was prepared in accordance with the CARE (CAse REport) guidelines.

CASE REPORT

An asymptomatic man in his 70s had undergone annual surveillance colonoscopy, during which only small benign polyps had previously been detected, with no lesions requiring treatment. His medical history included hypertension and hyperlipidemia, for which he was receiving a fixed-dose combination of amlodipine and atorvastatin (Caduet). He had no history of autoimmune disease, including rheumatoid arthritis, and had never received corticosteroids, other immunomodulatory agents, or antitumor therapy. He also had no history of malignancy.

On Day 0, colonoscopy performed for surveillance of colorectal polyps identified a lesion in the ascending colon consisting of a protruded component and a shallow depressed component (Figure 1A). Magnifying narrow-

band imaging (NBI) demonstrated irregular vascular and surface patterns classified as JNET type 2B. Magnifying chromoendoscopy revealed a type Vi pit pattern, raising suspicion of submucosal invasion (Figure 1B) [4, 5]. Endoscopic ultrasonography showed irregularity and partial disruption of the submucosal layer without definite involvement of the muscularis propria (Figure 1C). A tattoo was placed at approximately the same longitudinal level of the colon as the lesion but several centimeters away from it in the circumferential direction for surgical localization (Figure 1D). Preoperative barium enema examination supported the endoscopic estimate of a lesion diameter of approximately 15 mm (Figure 1E).

Targeted biopsy using standard biopsy forceps yielded two tissue fragments measuring approximately 2–3 mm each. Although the exact proportion of the lesion sampled could not be calculated because of tissue fragmentation and the irregular three-dimensional configuration of the lesion, the biopsy specimens represented only a small fraction of the approximately 15-mm lesion. The biopsy specimen contained crowded, irregular glands with complex architecture and neoplastic cells showing nuclear stratification, loss of polarity, nuclear enlargement, prominent nucleoli, and hyperchromasia, accompanied by focal intraluminal necrotic debris. The original biopsy slides were independently re-reviewed by two experienced gastrointestinal pathologists, who confirmed the diagnosis of moderately to well-differentiated adenocarcinoma based on these cytological and architectural abnormalities according to Japanese histopathological criteria. However, because the biopsy specimen was superficial and did not permit assessment of stromal or submucosal invasion, the depth of carcinoma could not be determined histologically from the biopsy specimen alone. No serrated architecture was present in the background mucosa (Figure 1F). On the basis of the endoscopic and ultrasonographic findings suggesting submucosal invasion, surgical resection was planned.

Repeat colonoscopy performed on Day 36 for removal of other colorectal polyps demonstrated a clear change in the appearance of the lesion. The protruded component had partially regressed, although a shallow depressed area remained visible at the original lesion site (Figure 2B). As part of the preoperative evaluation, contrast-enhanced computed tomography was performed and revealed no lymph node or distant metastases. On Day 50, the lesion was clinically staged as cT1N0Mo. The serum carcinoembryonic antigen level was 2.2 ng/mL.

Laparoscopic ileocecal resection with lymph node dissection was performed on Day 77, with no intervening cancer-specific treatment administered between the initial biopsy (Day 0) and the operation. Macroscopic examination of the resected specimen revealed only a subtle elevated lesion measuring approximately 3 mm at the presumed original tumor site, located at roughly the same longitudinal level of the colon as the tattoo but several centimeters away from it (Figure 2C).

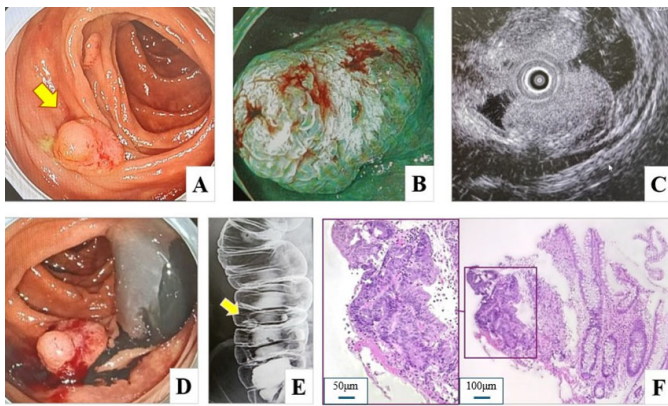


Figure 1: Pretreatment endoscopic, radiologic, and histopathologic findings. (A) White-light colonoscopy showing a protruded lesion with a shallow depressed component in the ascending colon (yellow arrow). (B) Magnifying narrow-band imaging (NBI) showing irregular surface and vascular patterns classified as Japan NBI Expert Team (JNET) type 2B, raising suspicion of submucosal invasion. (C) Endoscopic ultrasonography showing irregularity and focal disruption of the submucosal layer, with preservation of the muscularis propria, suggestive of submucosal invasion. (D) Endoscopic tattooing performed at approximately the same longitudinal level of the colon as the lesion but several centimeters away from it in the circumferential direction for surgical localization. (E) Pretreatment barium enema examination showing the corresponding lesion, which measured approximately 15 mm in diameter (yellow arrow). (F) Low-power view [hematoxylin and eosin staining (H&E), $\times 20$; scale bar, 100 μm] of the initial biopsy specimen showing crowded, irregularly shaped glands with complex and disorganized architecture, consistent with moderately to well-differentiated adenocarcinoma. The high-power inset on the left (H&E, $\times 100$; scale bar, 50 μm) demonstrates nuclear stratification, loss of polarity, nuclear enlargement, prominent nucleoli, and hyperchromasia. No serrated architecture was identified in the background mucosa.

Histopathologic examination of the 3-mm lesion revealed a traditional serrated adenoma (TSA) with low-grade dysplasia. A tissue defect, considered possibly artifactual, was present in its central portion (Figure 3A and B). The residual TSA was located at the site corresponding to the adenocarcinoma diagnosed in the initial biopsy. However, the available histologic findings did not establish whether the serrated lesion represented the precursor from which the carcinoma had arisen.

To investigate possible residual carcinoma, the presumed tumor bed was localized on the basis of the residual depressed area and surrounding stromal changes and was extensively sampled. Because precise localization of the original tumor bed was challenging owing to the marked regression, the mapped region and adjacent tissue were additionally sampled at gross intervals of approximately 2–4 mm, expanding the area examined. This procedure yielded five tissue blocks, encompassing an area of approximately 15 mm, corresponding to the estimated pretreatment extent of the lesion. From these blocks, four deeper levels were subsequently prepared and

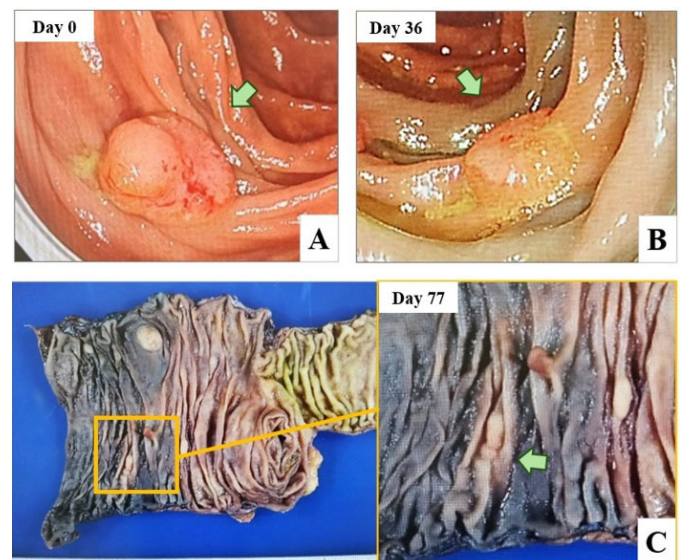


Figure 2: Interval endoscopic change before surgery and gross findings of the resected specimen. (A) Pretreatment colonoscopy showing the lesion in the ascending colon (green arrow). (B) Follow-up colonoscopy performed 36 days later showing partial regression and flattening of the protruded component, with persistence of the shallow depressed component (green arrow). (C) Gross view of the opened resected specimen showing no distinct residual tumor corresponding to the initial 15-mm lesion. Only a subtle elevated lesion measuring approximately 3 mm was identified at the presumed original tumor site (green arrow), at approximately the same longitudinal level as the tattoo but several centimeters away from it along the colonic wall.

examined histologically, with each level corresponding to approximately 100 μm deeper into the block. Although the mucosa at the tattoo site was inspected macroscopically, the entire tattoo-localized scar was not submitted as a separate histologic block. No residual adenocarcinoma was identified despite the expanded gross sampling and examination of deeper levels.

Prominent fibrosis, predominantly centered in the submucosa, was observed within the presumed tumor bed (Figure 3B). Moderate lymphocytic infiltration accompanied by lymphoid follicle formation was also present (Figure 3C). The fibrotic stromal area additionally contained a foreign-body giant-cell reaction and scar-like fibrosis of vascular walls (Figure 3D). According to the endoscopic procedure record and gross mapping of the resected specimen, the tattoo was located at approximately the same longitudinal level of the colon as the lesion but several centimeters away along the colonic wall. Therefore, the stromal and inflammatory changes within the presumed tumor bed did not anatomically correspond to the tattoo site. Although these changes may have reflected biopsy-related tissue injury and repair, their precise cause could not be determined. The pathologist also considered the possibility that mechanical or procedural factors had contributed to loss of the principal malignant component.

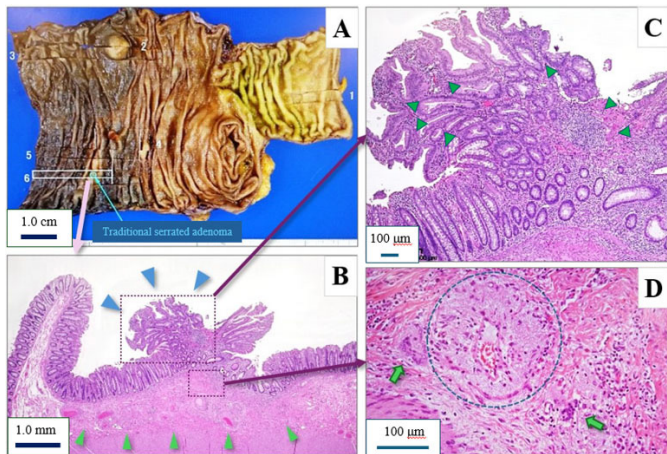


Figure 3: Macroscopic and histopathologic findings of the resected specimen. (A) A subtle elevated lesion (~3 mm) was identified at the presumed original tumor site, at approximately the same longitudinal level as the tattoo but several centimeters away along the colonic wall. Five tissue blocks were obtained at gross intervals of approximately 2–4 mm from the mapped region and adjacent tissue, encompassing an area of approximately 15 mm. Four additional deeper levels, each approximately 100 μ m apart, were examined from these blocks to assess for residual carcinoma. (B) Low-power view (H&E, $\times 20$; scale bar, 1 mm) showing a small elevated lesion corresponding to a traditional serrated adenoma with low-grade dysplasia (blue arrowheads) and fibrotic changes within the presumed tumor bed (green arrowheads). No residual carcinoma was identified. (C) Medium-power view (H&E, $\times 50$; scale bar, 100 μ m) showing lymphoid follicle formation and stromal lymphocytic infiltration (green arrowhead). (D) High-power view (H&E, $\times 100$; scale bar, 100 μ m) showing foreign-body giant cells (green arrows) and fibrosis of the vascular wall (within the green dashed circle).

All 19 retrieved lymph nodes were negative for metastasis. Overall, the histopathologic findings were consistent with a regression process, although its underlying mechanism could not be definitively established.

A PCR-based MSI assay was performed using DNA extracted from the pretreatment biopsy specimen, and the result was reported on Day 91. Testing was conducted by an external certified laboratory using a routine clinically validated assay. Although the clinical report did not provide details of the marker panel, individual unstable loci, tumor-cell content, quality-control procedures, or interpretation criteria, the tumor was reported as MSI-high.

The postoperative course was uneventful, and the patient was discharged on postoperative Day 7 (Day 84). At 2.5 years after surgery, he remains free of recurrence, and surveillance colonoscopy performed at 2 years showed no evidence of recurrent or metachronous neoplasia. A concise clinical timeline from the initial detection of the lesion through postoperative follow-up is presented in Table 1.

Table 1: Clinical timeline

Time point	Clinical event
Day 0	Surveillance colonoscopy identified an approximately 15-mm lesion in the ascending colon. Targeted biopsy demonstrated moderately to well-differentiated adenocarcinoma, and endoscopic tattooing was performed for localization.
Day 36	Follow-up colonoscopy showed partial regression and flattening of the protruded component, with persistence of the shallow depressed component.
Day 50	The lesion was clinically staged as cT1NoMo based on endoscopic and radiologic findings.
Day 77	Laparoscopic ileocecal resection with lymph node dissection was performed.
Day 84	The patient was discharged without postoperative complications.
Day 91	Polymerase chain reaction-based microsatellite instability testing of the pretreatment biopsy specimen was reported as MSI-high.
2 years after surgery	Surveillance colonoscopy showed no recurrent or metachronous neoplasia.
2.5 years after surgery	The patient remained free of recurrence.

This table summarizes the major clinical events from initial detection through postoperative follow-up.

DISCUSSION

Spontaneous regression (SR) of colorectal cancer is exceptionally rare, and its underlying mechanisms remain incompletely understood [1]. We report a case of an ascending colon lesion diagnosed as moderately to well-differentiated adenocarcinoma on biopsy that subsequently showed regression of its protruded component on repeat colonoscopy and no residual carcinoma in the resected specimen. Notably, PCR-based testing of the pretreatment biopsy specimen demonstrated MSI-high status.

In the present case, regression of the protruded component was documented endoscopically 36 days after biopsy, and laparoscopic ileocecal resection with lymph node dissection was performed 77 days after biopsy. Histological examination of the resected specimen revealed no residual carcinoma. The combination of histologically confirmed adenocarcinoma at presentation, interval endoscopic changes consistent with regression, absence of residual carcinoma despite extensive sectioning of the presumed tumor bed, reliable lesion localization using an endoscopic tattoo, and absence of recurrence during 2.5 years of follow-up supports the possibility that tumor regression occurred. Nevertheless, definitive SR cannot be established because complete removal of the malignant component by biopsy or regression secondary to biopsy-induced tissue injury cannot be excluded.

Microsatellite instability-high status is noteworthy because mismatch repair-deficient colorectal cancers frequently exhibit an immunogenic tumor microenvironment characterized by tumor-infiltrating lymphocytes and other host immune responses [2, 3]. Because MSI testing was performed on the pretreatment biopsy specimen containing histologically confirmed adenocarcinoma, the regressed malignant component was likely MSI-high. Diagnostic biopsy may have caused local tissue injury and the release of tumor antigens and damage-associated molecular signals, potentially augmenting a pre-existing antitumor immune response within this immunogenic microenvironment [6]. However, this proposed mechanism cannot be established retrospectively.

Histological examination of the presumed tumor bed revealed stromal fibrosis, a foreign-body giant cell reaction, vascular wall fibrosis, lymphoid follicles, and lymphocytic infiltration. These changes were localized to the presumed tumor bed and were spatially distinct from the tattoo site, which was located several centimeters away. Although these findings may represent biopsy-induced injury and subsequent tissue repair, the initial biopsy consisted of only two conventional forceps-biopsy specimens obtained from limited portions of the protruded and depressed components. Given the limited sampling volume relative to the initial lesion, mechanical

removal alone may not fully explain the complete absence of residual carcinoma. Taken together with the MSI-high status, the histological findings raise the possibility that biopsy-associated tissue injury occurred within an immunologically active tumor microenvironment and may have contributed to regression. Nevertheless, an immune-mediated mechanism remains speculative and cannot be distinguished conclusively from conventional biopsy-induced injury and repair.

Accurate identification of the original tumor site is another important consideration in suspected SR. In the present case, an endoscopic tattoo placed near the lesion during the initial colonoscopy served as a consistent landmark during follow-up colonoscopy and surgery, thereby minimizing the possibility of wrong-site resection. Because the residual 3-mm lesion was markedly discordant in size with the initial lesion, extensive additional sections were prepared from the presumed tumor bed; however, no residual carcinoma was identified. All retrieved lymph nodes were negative for metastasis, and no recurrence was detected during 2.5 years of surveillance, including colonoscopy performed 2 years after surgery.

The clinicopathological features of the present case and four previously reported MSI-high or mismatch repair (MMR)-deficient colorectal cancers showing regression after biopsy are summarized in Table 2 [6, 7].

Table 2: Comparison of the present case with previously reported MSI-high or mismatch repair-deficient colorectal cancers showing regression after biopsy

Case	Location	Initial diagnosis	MSI/MMR status	Interval change and timing	Final pathology	Key histological features related to regression	Reference
Present case	Ascending colon	Moderately to well-differentiated adenocarcinoma; protruded lesion with a depressed component	MSI-high, determined by PCR assay of pretreatment biopsy specimen	Regression of the protruded component on repeat colonoscopy at Day 36; surgery performed 77 days after biopsy	3-mm traditional serrated adenoma with low-grade dysplasia; no residual carcinoma	Stromal fibrosis, lymphoid follicles, lymphocytic infiltration, foreign-body giant cell reaction, and vascular wall fibrosis	Present case
Yokota et al., Patient 1; 76/F	Transverse colon	10-mm 0-Is+Iic lesion; moderately differentiated tubular adenocarcinoma	dMMR; loss of MLH1 and PMS2 expression	Complete regression after biopsy; surgery performed 52 days after biopsy	No residual carcinoma; fibrotic scar and mucin lake	Fibrotic scar and mucin lake	[6]
Yokota et al., Patient 2; 64/F	Cecum	15-mm 0-Is lesion; well-differentiated tubular adenocarcinoma	dMMR with loss of MSH2 and MSH6 expression	Complete regression confirmed in the resected specimen; surgery performed 85 days after biopsy	No residual carcinoma; fibrotic scar and foreign-body giant cells	Fibrotic scar and foreign-body giant cell reaction	[6]
Yokota et al., Patient 3; 64/M	Transverse colon	20-mm type 2 lesion; moderately differentiated tubular adenocarcinoma	dMMR with loss of MLH1 and PMS2 expression	Complete regression confirmed in the resected specimen; surgery performed 42 days after biopsy	No residual carcinoma; granulation tissue and foreign-body giant cells	Granulation tissue and foreign-body giant cell reaction	[6]
Karakuchi et al.; 78/M	Transverse colon	30 × 30-mm type 2 lesion; poorly differentiated adenocarcinoma	dMMR with loss of MLH1 and PMS2 expression, suggestive of MSI-high status	At resection approximately 2 months after the initial colonoscopy, the 30-mm tumor had been replaced by a 10-mm ulcer containing an 8.5-mm polypoid lesion	No residual carcinoma	Tumor-infiltrating lymphocytes in the biopsy specimen; granulation tissue, submucosal fibrosis, and epithelial sloughing in the resected lesion	[7]

Abbreviations: dMMR: deficient mismatch repair; F: female; M: male; MMR: mismatch repair; MSI-high: microsatellite instability-high; PCR: polymerase chain reaction.

These five cases comprised the three patients reported by Yokota et al., the patient reported by Karakuchi et al., and the present patient. All five lesions were located in the proximal colon, and no residual carcinoma was identified in the subsequently resected specimens. The interval between biopsy and surgery ranged from 42 to 85 days; in the present case, surgery was performed on Day 77. The regressed sites exhibited varying combinations of fibrotic, inflammatory, and reparative changes, including fibrotic scarring, granulation tissue, lymphocytic infiltration, and foreign-body giant cell reactions. These shared clinicopathological features support the biological plausibility of an association between biopsy-related tissue injury, an immunogenic tumor microenvironment, and tumor regression. However, the small number of cases and differences in pathological and molecular assessment preclude definitive conclusions regarding the underlying mechanism.

Histological examination of the resected specimen in the present case identified only a 3-mm TSA with low-grade dysplasia. This finding raises the possibility that the initial carcinoma arose through the serrated neoplasia pathway, which is associated with a substantial proportion of sporadic MSI-high colorectal cancers [8]. However, because no residual carcinoma was available for pathological or molecular comparison, a direct relationship between the residual TSA with low-grade dysplasia and the original adenocarcinoma cannot be established.

Further molecular characterization, including MMR protein immunohistochemistry, MLH1 promoter methylation analysis, and BRAF V600E testing, would have helped clarify the biological basis of MSI-high status in this case [9, 10]. Although the patient had neither a family history suggestive of Lynch syndrome nor a history of multiple primary malignancies, germline testing was not performed; therefore, Lynch syndrome could not be definitively excluded. These additional molecular analyses could not be performed because they were not clinically available in this case under the applicable institutional and reimbursement conditions.

Clinically, this case illustrates that apparent regression of a malignant colorectal lesion after biopsy does not exclude residual microscopic disease. Because such disease may not be detectable endoscopically and the natural history of this phenomenon remains uncertain, standard oncological management should not be withheld solely on the basis of apparent regression.

Careful pathological examination of the resected specimen, including extensive evaluation of the presumed tumor bed, remains essential. Accumulation of well-documented cases with standardized pathological assessment and molecular characterization is needed to further clarify the potential interplay among tissue injury, antitumor immunity, and tumor regression in MSI-high colorectal cancer.

CONCLUSION

We report a case of early-stage MSI-high colorectal adenocarcinoma that showed marked post-biopsy morphological changes, with no residual carcinoma identified in the resected specimen. Histological examination of the presumed tumor bed revealed lymphocytic infiltration with lymphoid follicle formation and stromal changes consistent with tissue injury and repair. These findings are consistent with a possible regression process; however, complete mechanical removal of the malignant component during biopsy or changes resulting from procedure-related tissue injury cannot be excluded. Although the MSI-high status provides a plausible immunologic background, the contribution of local antitumor immunity remains speculative.

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Use of artificial intelligence tools

During the preparation of this manuscript, ChatGPT by OpenAI was used only for English language editing and improvement of readability. No artificial intelligence tool was used to generate clinical data, pathological data, endoscopic images, radiological images, figures, tables, references, or scientific conclusions. All clinical information, interpretations, references, and final manuscript content were reviewed and verified by the authors, who take full responsibility for the integrity and accuracy of the work.

Author Contributions

Kazuhito Minami – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Final approval of the version to be published, Agree to be 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

Hiroki Funakoshi – Acquisition of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be 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

Yumi Oshiro – Acquisition of data, Analysis of data, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be 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

Yoshiaki Nozaka – Acquisition of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be 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

Shinjiro Egashira – Acquisition of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be 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

Guarantor of Submission

The corresponding author is the guarantor of submission.

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None.

Consent Statement

Written informed consent for publication of clinical details and images was obtained from the patient. According to the institutional policy of our hospital, single anonymized case reports are classified as exempt from formal ethics committee review; therefore, additional Institutional Review Board (IRB) approval was not required.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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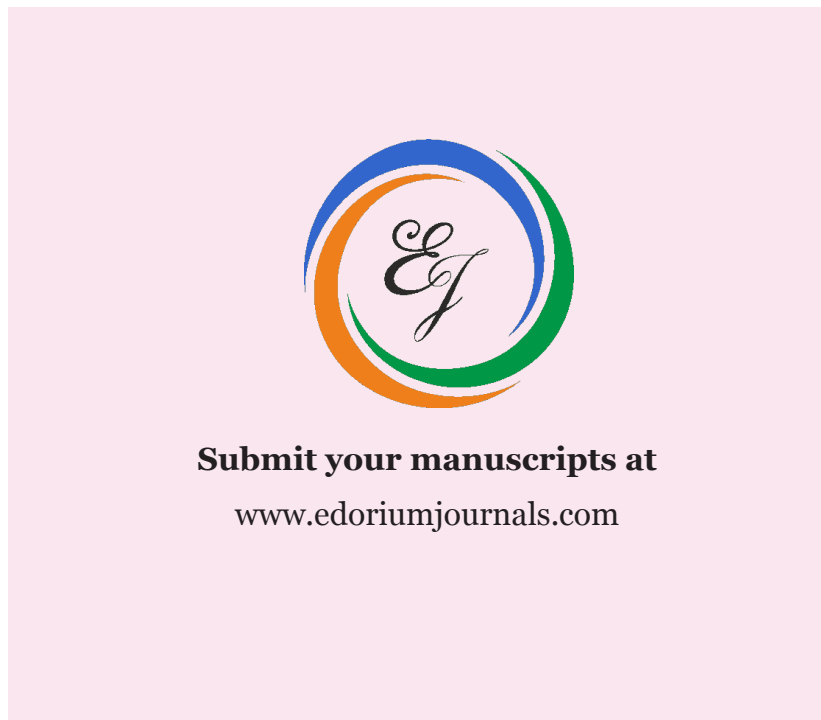
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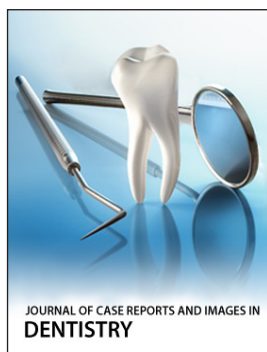
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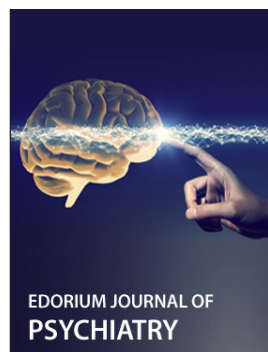
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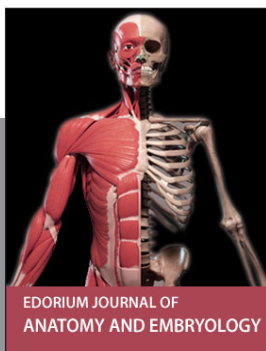
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