

Unexpected favorable response of stage IV gastric adenocarcinoma to sintilimab combined with chemotherapy: A case report

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Abstract. Gastric cancer is the fifth most common and the third deadliest type of cancer worldwide. Standard treatment protocols for advanced, unresectable or recurrent cases usually involve combination regimens. These regimens typically consist of a fluoropyrimidine and a platinum agent, with the anti-HER2 monoclonal antibody trastuzumab added for HER2-positive cases in the first line of treatment. In second-line treatment, paclitaxel is used, sometimes in combination with ramucirumab. Despite these treatments, the median survival time for patients with advanced gastric cancer remains between 12 and 15 months, highlighting the need for innovative treatment options. Notably, immune checkpoint inhibitors have emerged as breakthrough treatments for various malignancies. These inhibitors allow immunotherapy to enhance traditional treatments such as surgery, chemotherapy, radiotherapy and targeted therapy. In this context, the present study describes a case of gastric adenocarcinoma that exhibited a favorable response to a combined regimen of chemotherapy and

immunotherapy, specifically using sintilimab, a programmed cell death protein-1 inhibitor. The patient with stage IV gastric cancer and peritoneal metastasis achieved a progression-free survival of >5 years. This case underscores the potential for integrating new immunotherapeutic agents into existing treatment frameworks to markedly extend patient survival and improve quality of life.

Introduction

Globally, gastric cancer is the fifth most common and the third deadliest type of cancer (1). In China, gastric cancer ranks as the second most prevalent type of cancer and the third leading cause of cancer-related deaths, representing a notable public health challenge (2,3). Standard treatment protocols for advanced, unresectable or recurrent gastric cancer typically include first-line therapy with a combination of a fluoropyrimidine and platinum agents. For HER2-positive cases, trastuzumab is added to this regimen. Second-line therapy often involves paclitaxel, which can be optionally combined with ramucirumab. Despite these treatment options, the median survival time for patients with advanced gastric cancer remains modest, at 12-15 months, highlighting the need for more effective therapies (4-8).

Immunotherapy, particularly the use of immune checkpoint inhibitors, has gained traction in the treatment of various types of cancer, including melanoma, non-small cell lung cancer and renal cell carcinoma, and is now considered alongside traditional methods, such as surgery, chemotherapy, radiotherapy and targeted therapy (9,10). Evidence from early- and late-phase clinical trials has shown that targeting immune checkpoint proteins, such as programmed cell death protein-1

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(PD-1)/programmed death-ligand 1 (PD-L1), can markedly improve overall survival for patients with advanced, metastatic or recurrent gastric cancer (11-13).

Sintilimab, which is a PD-1 inhibitor, has demonstrated promise in clinical trials (12,14,15). In a phase Ib study, 20 patients with locally advanced or metastatic gastric or gastroesophageal junction adenocarcinoma received 200 mg sintilimab intravenously, in combination with oxaliplatin and capecitabine every 3 weeks for up to six cycles (21 days per cycle). After this initial treatment, sintilimab was administered as maintenance therapy every 3 weeks for up to 24 months, or until disease progression, unacceptable toxicity or withdrawal of consent occurred. This treatment regimen resulted in a median progression-free survival time of 7.5 months, an objective response rate of 85% and a disease control rate of 100% (12). The present study describes a case of stage IV gastric adenocarcinoma that was effectively managed with sintilimab in combination with chemotherapy.

Case report

A 67-year-old man was admitted to Tongji Hospital of Tongji Medical College (Wuhan, China) in November 2018 due to melena, weakness and heart palpitations. The patient had no prior history of malignancy. Routine blood tests revealed microcytic hypochromic anemia with a hemoglobin level of 64 g/l (reference range, 130-175 g/l). Iron metabolism tests confirmed iron-deficient anemia and a fecal occult blood test returned positive results. Tumor marker tests indicated an elevated carcinoembryonic antigen (CEA) level at 12.15 ng/ml (reference range, ≤ 5.0 ng/ml). The clinicopathological characteristics of the patient are summarized in Table I.

A gastroscopy revealed a giant ulcer affecting the lesser curvature of the gastric body, the angular notch, the gastric antrum and the pylorus (Fig. 1A-C). Histopathological analysis of a biopsy sample from the gastric lesion confirmed the diagnosis of gastric adenocarcinoma (Fig. 2A and B). The biopsy sample was fixed in 10% neutral buffered formalin at room temperature for 24 h, embedded in paraffin and sectioned at a thickness of 4 μ m. The sections were stained with Harris hematoxylin for 5 min and aqueous eosin for 2 min at room temperature, and examined using a Nikon Eclipse 80i light microscope (Nikon Corporation).

The immunohistochemical findings showed positivity for pan-cytokeratin (PCK), E-cadherin, MutS homolog (MSH)2, MSH6, p53 (mutated type) and Ki-67 (70% of labeled cells in the hot-spots), but negativity for MutL homolog 1 (MLH1), post-meiotic segregation increased 2 (PMS2) and HER2. For immunohistochemical analysis, paraffin-embedded tissue sections (4 μ m) from the gastric lesion biopsy sample were used. Endogenous peroxidase activity was blocked with 3% hydrogen peroxide for 5 min at 25-28°C. The sections were then incubated with the following primary antibodies for 40 min at 25-28°C: PCK (ready-to-use; cat. no. 760-2135; Roche Diagnostics), E-cadherin (ready-to-use; cat. no. ZM-0092; Beijing Zhongshan Jinqiao Biotechnology Co., Ltd.), p53 (ready-to-use; cat. no. GA616; Dako; Agilent Technologies, Inc.), Ki-67 (1:800; cat. no. RMA-0731; Fuzhou Maixin Biotechnology Development Co., Ltd.), MLH1 (ready-to-use;

cat. no. ZM-0152; Beijing Zhongshan Jinqiao Biotechnology Co., Ltd.), PMS2 (ready-to-use; cat. no. ZA-0542; Beijing Zhongshan Jinqiao Biotechnology Co., Ltd.), MSH2 (ready-to-use; cat. no. ZA-0702; Beijing Zhongshan Jinqiao Biotechnology Co., Ltd.), MSH6 (ready-to-use; cat. no. ZM-0367; Beijing Zhongshan Jinqiao Biotechnology Co., Ltd.) and HER2 (ready-to-use; cat. no. 790-7167; Roche Diagnostics). The sections were subsequently incubated with a ready-to-use HRP-conjugated secondary antibody (cat. no. SD3209; Mirus Medical LLC) for 30 min at 25-28°C. The stained sections were examined using a Nikon Eclipse 80i light microscope (Nikon Corporation). Epstein-Barr virus-encoded RNA (EBER) was negative, as determined by chromogenic *in situ* hybridization (CISH) (Fig. 2C-L). For EBER CISH, paraffin-embedded tissue sections (4 μ m) were analyzed using the BOND EBER Probe (cat. no. PB0589) and BOND Polymer Refine Detection system (cat. no. DS9800; both Leica Biosystems). The slides were deparaffinized and rehydrated using BOND Dewax Solution (cat. no. AR9222; Leica Biosystems). The sections were digested with proteinase K (cat. no. RE7160-K; Leica Biosystems) for 10 min at 37°C, followed by three washes with BOND Wash Solution (cat. no. AR9590-CN; Leica Biosystems). The ready-to-use fluorescein-conjugated oligonucleotide BOND EBER Probe (cat. no. PB0589; Leica Biosystems) was hybridized at 37°C for 2 h on the Bond-III system. Endogenous peroxidase activity was blocked with 3% H₂O₂ for 5 min at 26°C. The slides were incubated with BOND Ready-to-Use Anti-Fluorescein Antibody (cat. no. AR0222; Leica Biosystems) for 15 min at 26°C. The ready-to-use post-primary and polymer reagents from the BOND Polymer Refine Detection system (cat. no. DS9800; Leica Biosystems) were each applied for 8 min at 26°C. The signal was visualized using DAB for 10 min at room temperature. The slides were then washed with BOND Wash Solution followed by deionized water and counterstained with hematoxylin for 25 sec at 26°C. The stained sections were examined using a Nikon Eclipse 80i light microscope (Nikon Corporation). The absence of MLH1 and PMS2 indicated deficient mismatch repair (dMMR) and a microsatellite instability-high (MSI-H) status.

An abdominal contrast CT scan showed uneven thickening of the gastric body and antrum wall, uneven enhancement, a blurry serous membrane and enlarged adjacent lymph nodes, staging the cancer as T4N2 according to the eighth edition of the American Joint Committee on Cancer TNM staging system (16) (Fig. 3A). Laparoscopic exploration revealed metastasis to the greater omentum and pancreatic head, leading to a final staging of T4N2M1.

Following a multidisciplinary evaluation based on imaging, endoscopy, laparoscopic exploration and histopathology, and with the informed consent of the patient, treatment was initiated with one cycle of an S-1 plus oxaliplatin (SOX) regimen [oxaliplatin (130 mg/m²), intravenous (i.v.) administration, day 1; S-1 (60 mg) twice a day, oral administration, days 1-14] in combination with sintilimab (200 mg) via i.v. administration every 3 weeks. After 3 weeks, the patient returned and the tumor marker test indicated a decrease in CEA to 5.03 ng/ml. However, the patient experienced vomiting and

Table I. Clinicopathological characteristics of the patient.

Characteristic	Details
Age, years	67
Sex	Male
Ethnicity	Chinese
Initial presentation	Melena, weakness, palpitations, iron-deficiency anemia
Initial hemoglobin level, g/l	64
Serum CEA level, ng/ml	12.15
Histological diagnosis	Gastric adenocarcinoma
Primary tumor location	Lesser curvature of the gastric body, angular notch, gastric antrum and pylorus
Clinical stage	cT4N2
Final stage after laparoscopy	T4N2M1 (greater omentum and pancreatic head metastases)
HER2 status	Negative
MMR/MSI status	dMMR/MSI-H (loss of MLH1 and PMS2 expression)
EBER status	Negative
First-line treatment	SOX plus sintilimab
Subsequent treatment	FOLFOX7 plus sintilimab, followed by albumin-bound paclitaxel plus sintilimab, then sintilimab monotherapy maintenance until June 2022
Best response	Complete response (RECIST v1.1)
Progression-free survival	>5 years
Current status	Alive without disease progression at last follow-up in July 2026

CEA, carcinoembryonic antigen; dMMR, deficient mismatch repair; MSI-H, microsatellite instability-high; MLH1, MutL homolog 1; PMS2, postmeiotic segregation increased 2; EBER, Epstein-Barr virus-encoded RNA; SOX, S-1 plus oxaliplatin; FOLFOX7, folinic acid, fluorouracil and oxaliplatin; RECIST, Response Evaluation Criteria in Solid Tumors.

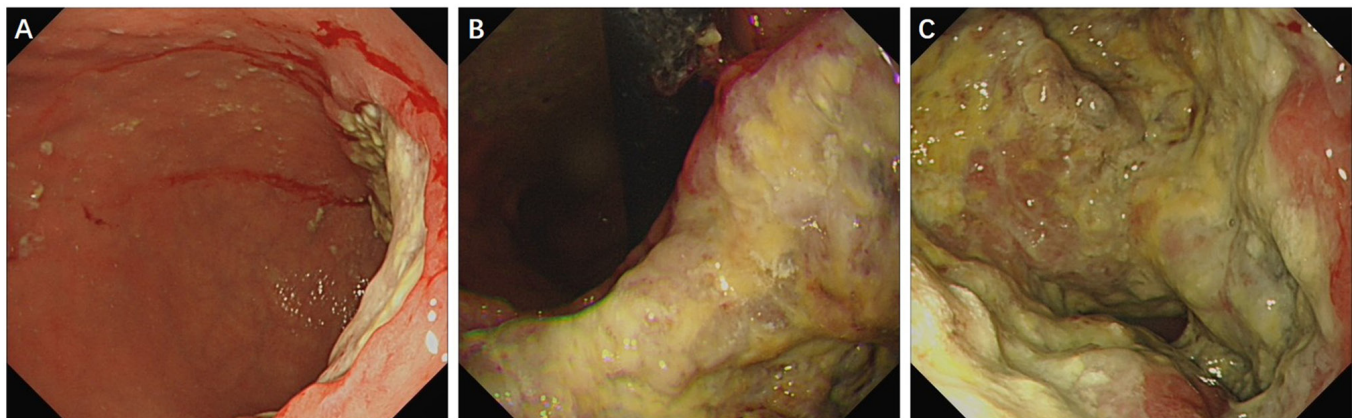


Figure 1. Gastroscopic images obtained in November 2018 showing a giant ulcer involving the (A) lesser curvature of the gastric body, (B) angular notch, (C) gastric antrum and pylorus.

eating obstruction, indicating a pyloric obstruction, making the SOX regimen, including the S-1, inappropriate for the condition. Therefore, S-1 was replaced with a 5-fluorouracil injection, and the treatment regimen was switched to one cycle of the traditional folinic acid, fluorouracil and oxaliplatin (FOLFOX7) regimen [oxaliplatin (130 mg/m²), i.v. administration, day 1; 5-fluorouracil (400 mg/m²), i.v. bolus, day 1, followed by continuous i.v. infusion of 5-fluorouracil (2,400 mg/m²) over 46 h beginning on day 1; leucovorin (400 mg/m²), i.v. administration, day 1] combined with sintilimab (200 mg)

administered intravenously every 3 weeks. After one cycle of FOLFOX7 plus sintilimab, chemotherapy was interrupted due to grade 2 hematological toxicity [platelet count, 55x10⁹/l (reference range, 125-350x10⁹/l); white blood cell count, 2.47x10⁹/l (reference range, 3.5-9.5x10⁹/l); neutrophil count, 1.4x10⁹/l (reference range, 1.8-6.3x10⁹/l)], from which the patient recovered after hematopoietic supportive therapy.

Due to worsening eating obstruction, the patient was transferred to the Department of General Surgery at Tongji Hospital and subsequently underwent palliative gastrectomy.

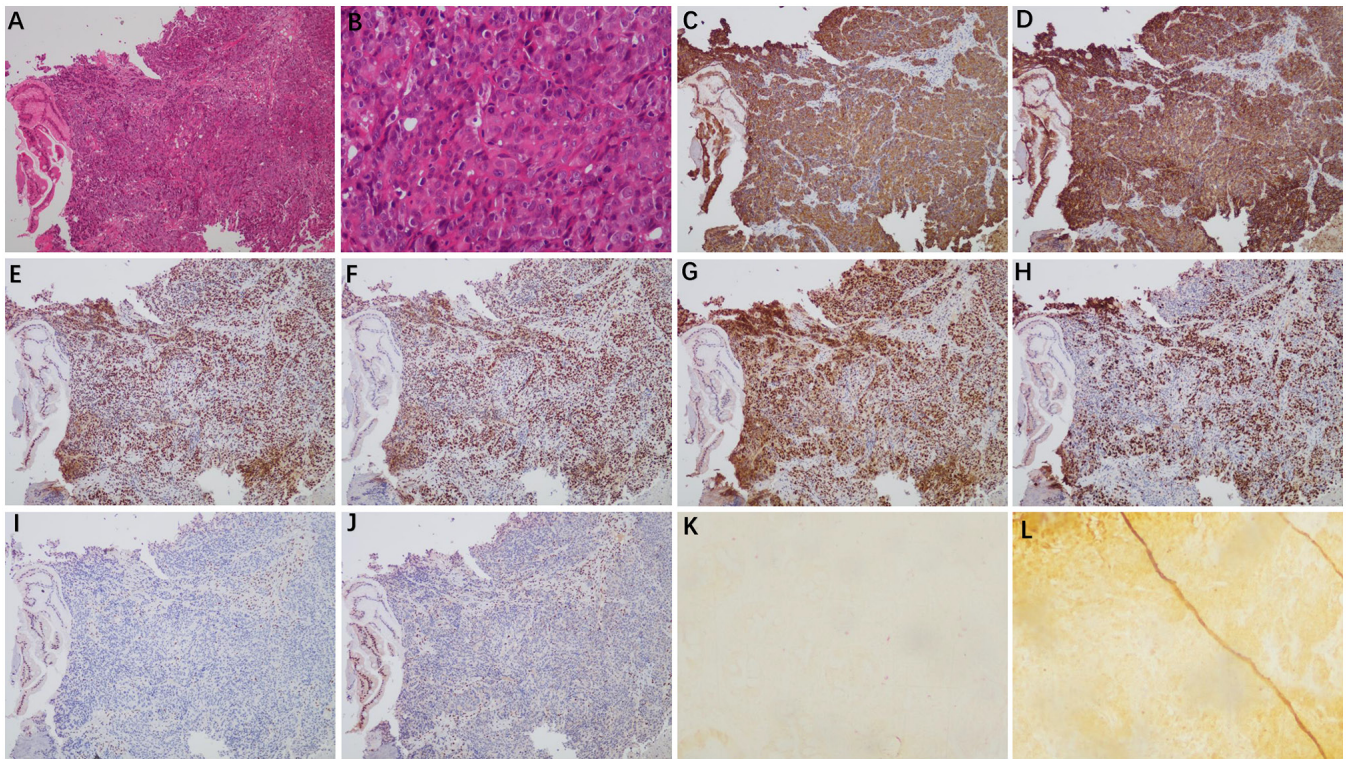


Figure 2. Histopathological and immunohistochemical images of the biopsy sample from the gastric lesion, which were obtained in November 2018, confirmed the diagnosis of gastric adenocarcinoma. Hematoxylin and eosin staining: (A) Magnification, x100 and (B) magnification, x400. Biopsy sample was positive for (C) pan-cytokeratin, (D) E-cadherin, (E) MSH2, (F) MSH6, (G) p53 (mutated type) and (H) Ki-67 (70% of labeled cells in the hot-spots), and negative for (I) MutL homolog 1, (J) postmeiotic segregation increased 2, (K) HER2 and (L) Epstein-Barr virus-encoded RNA chromogenic *in situ* hybridization. Panels C-L: Magnification, x100. MSH, MutS homolog.

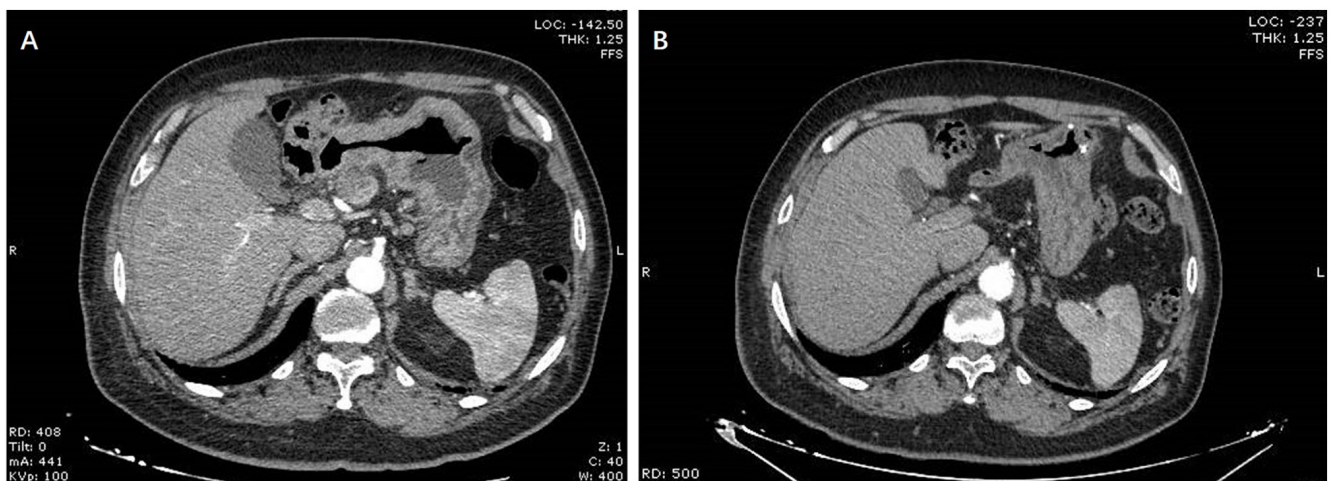


Figure 3. Contrast-enhanced abdominal CT images before and after treatment. (A) Baseline contrast-enhanced abdominal CT image obtained in November 2018 showing uneven thickening of the gastric body and antrum wall, uneven enhancement, blurry serous membrane, and increased and enlarged adjacent lymph nodes. (B) Follow-up contrast-enhanced abdominal CT image obtained in July 2019 demonstrating an improvement in the thickening of the gastric wall and marked regression of previously enlarged regional lymph nodes, with only residual sub-centimeter lymph nodes (<5 mm in short-axis diameter), considered reactive rather than metastatic.

for gastric outlet obstruction. Post-surgery, the symptoms of vomiting and eating obstruction were markedly improved. After 3 weeks, the patient received sintilimab (200 mg) via i.v. administration every 3 weeks for the third time.

After a further 3 weeks, during a subsequent hospital visit for further combined treatment with albumin-bound paclitaxel and sintilimab, CEA levels showed a decrease to the normal

range at 3.86 ng/ml. Subsequently, treatment was resumed with albumin-bound paclitaxel (130 mg/m², i.v. administration, days 1 and 8) combined with sintilimab (200 mg) administered intravenously every 3 weeks for three cycles. After completion of this combination therapy, cytotoxic chemotherapy was discontinued, and the patient continued with sintilimab (200 mg) monotherapy administered intravenously every 3 weeks.

In July 2019, the patient was admitted for an efficacy assessment. The tumor marker test showed that CEA remained in the normal range (4.06 ng/ml). An abdominal contrast CT scan indicated improvement of the thickening of the gastric wall and marked regression of the previously enlarged regional lymph nodes, with only residual sub-centimeter lymph nodes (<5 mm in short-axis diameter) (Fig. 3B). Gastroscopy exhibited marked improvement of the giant ulcer, which had healed, leaving only a scar, and the pylorus was no longer visible (Fig. 4A-C). Furthermore, histopathological analysis of the rough mucosa of the angular notch exhibited chronic inflammation without malignant characteristics (Fig. 4D-F).

Thus, the overall evaluation of the condition of the patient was a complete response according to Response Evaluation Criteria in Solid Tumors version 1.1 (17). The patient was initially diagnosed with gastric cancer in November 2018, which served as the baseline imaging time-point. After that, imaging examinations were performed at predefined follow-up time points in February 2019, April 2019 and July 2019, followed by annual surveillance thereafter. The patient continued receiving sintilimab (200 mg) monotherapy via i.v. administration every 3 weeks as maintenance until June 2022.

As of the last follow-up in July 2026, the patient experienced a progression-free survival time of >5 years. During treatment with sintilimab combined with chemotherapy, the patient was monitored for treatment-related adverse events; notably, no immune-related adverse events were observed and no grade ≥ 3 chemotherapy-related adverse events were documented. All adverse events were evaluated according to Common Terminology Criteria for Adverse Events version 5.0 (18). At present, the patient is still alive, attends annual examinations, including gastroscopy and abdominal contrast CT scans, and has regular telephone follow-ups.

Discussion

First-line chemotherapy for advanced or metastatic HER2-negative gastric or gastroesophageal junction adenocarcinoma typically achieves a median overall survival time of <1 year (13). In this context, immune checkpoint blockade targeting the PD-1/PD-L1 pathway has markedly reshaped the treatment landscape of advanced gastric cancer by restoring antitumor immune responses and overcoming immune evasion (13,19,20), leading to the development of multiple anti-PD-1/PD-L1 agents that have subsequently been evaluated in clinical trials and real-world settings (10,21-24). The CheckMate-649 study established nivolumab, an anti-PD-1 monoclonal antibody, combined with chemotherapy as an effective and safe first-line treatment strategy for patients with advanced gastric or gastroesophageal junction adenocarcinoma, demonstrating marked improvements in overall survival and progression-free survival compared with chemotherapy alone (13). This finding is corroborated by data showing that nivolumab plus chemotherapy can notably enhance survival and response rates compared with chemotherapy alone, particularly in Chinese patients (25). Further evidence of the efficacy of PD-1 inhibitors has been provided by Wang *et al* (26); this previous study observed that sintilimab-based regimens offer substantial disease control

and tolerability in real-world settings for treating advanced gastric and gastroesophageal junction adenocarcinoma, and the treatment was shown to be more effective when administered in the first- or second-line setting. Moreover, the ORIENT-16 study revealed that sintilimab combined with first-line chemotherapy markedly improves overall survival in patients with unresectable locally advanced or metastatic gastric or gastroesophageal junction adenocarcinoma compared with chemotherapy alone (15).

Despite these advances, responses to immune checkpoint inhibitors remain heterogeneous among patients with gastric cancer, highlighting the importance of identifying predictive biomarkers that can guide treatment selection (20). Molecular biomarkers have emerged as critical determinants of treatment response in gastric cancer (20). Among these, MSI resulting from dMMR represents a key predictive biomarker for immunotherapy response (27-29). HER2 status, PD-L1 expression and claudin 18.2 expression have also been recognized as clinically relevant biomarkers for treatment stratification (20). HER2 remains an established therapeutic target enabling HER2-directed treatment approaches (30); HER2 upregulation is associated with a better pathological response and has been shown to independently predict improved overall survival in advanced gastric cancer treated with neoadjuvant or conversion chemotherapy (31). In addition, PD-L1 expression provides an important rationale for immune checkpoint blockade (32). Recently, claudin 18.2 has emerged as a promising predictive and therapeutic target with growing clinical relevance (33-35), reflecting the evolving landscape of precision medicine in gastric cancer. Together, these biomarkers illustrate the transition from histology-based treatment strategies toward molecularly guided therapeutic decision-making.

Among these biomarkers, MSI-H/dMMR status has emerged as one of the most robust predictors of response to immune checkpoint inhibition (27-29). The MMR system serves a critical role in maintaining genomic stability by correcting replication-associated DNA errors, and its dysfunction leads to MSI and the accumulation of numerous somatic mutations (36). This hypermutated phenotype is understood to generate a high burden of tumor neoantigens, thereby enhancing tumor immunogenicity and increasing sensitivity to immune checkpoint blockade (28). Consistent with this biological rationale, tumor mutational burden, which reflects the overall number of somatic mutations per coding area within the tumor genome, has also been proposed as a complementary predictive biomarker associated with the response to immunotherapy (37,38). Clinically, the predictive value of MMR status for pembrolizumab-based PD-1 blockade was first demonstrated in the KEYNOTE-016 study, which showed notable responses in both colorectal and non-colorectal MSI-H/dMMR cancer, and provided early evidence for the tumor-agnostic activity of immune checkpoint inhibition (27). Subsequent studies, including KEYNOTE-158, further validated these findings by demonstrating durable responses to PD-1 blockade across a broad spectrum of MSI-H/dMMR solid tumors (28,29). Collectively, these data support the concept of biomarker-driven, tumor-agnostic immunotherapy and led to the landmark Food and Drug Administration approval of

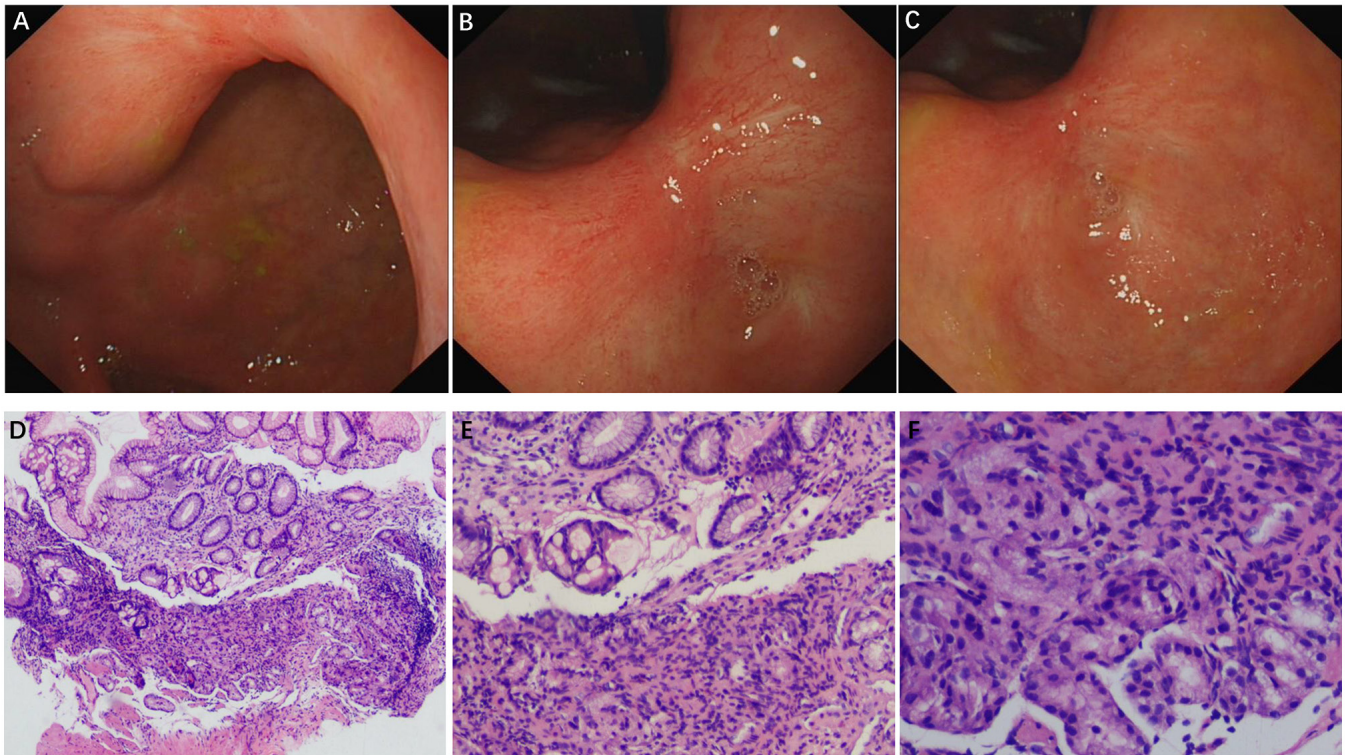


Figure 4. Gastroscopic images obtained in July 2019 indicated that the giant ulcer exhibited marked improvement and had healed, as only a scar could be seen in the (A) gastric body, (B) angular notch and (C) gastric antrum. (C) Closure of the pyloric channel was also observed at its expected anatomical location, likely due to tumor regression and post-treatment fibrotic remodeling. (D-F) Histopathological findings taken from the rough mucosa of the angular notch during follow-up showed chronic inflammation without malignant characteristics. Hematoxylin and eosin staining: (D) Magnification, x40; (E) magnification, x100; and (F) magnification, x200.

pembrolizumab in 2017 for the treatment of MSI-H/dMMR solid tumors, representing a major milestone in precision oncology (39,40).

In gastric cancer, the clinical relevance of MSI-H status has been evaluated in multiple clinical studies. Exploratory analyses from KEYNOTE-059, KEYNOTE-061 and KEYNOTE-062 studies suggested that MSI-H tumors are associated with enhanced clinical benefit from pembrolizumab-based therapy across different lines of treatment in advanced gastric and gastroesophageal junction cancer (41-44). Consistently, a meta-analysis by Pietrantonio *et al* (45) further demonstrated that MSI-H status is markedly associated with higher response rates and improved survival outcomes compared with microsatellite stable tumors in patients receiving anti-PD-1 therapy for advanced gastric cancer.

Sintilimab, a novel engineered PD-1 inhibitor, has demonstrated favorable pharmacological properties, including high binding affinity and durable receptor occupancy, compared with established PD-1 inhibitors, such as nivolumab and pembrolizumab (46). Its favorable cost-effectiveness profile may further support its long-term clinical application in appropriately selected patients (47). In the present case, the tumor was confirmed as HER2-negative and MSI-H/dMMR, providing a strong biological rationale for the durable and marked response observed with sintilimab plus chemotherapy. Although PD-L1 expression and claudin 18.2 status were not available at the time of diagnosis, their emerging roles as predictive biomarkers reflect

the evolving landscape of precision oncology in gastric cancer (20). The patient achieved sustained progression-free survival >5 years, highlighting the potential for long-term disease control with PD-1 blockade-based therapy in selected molecularly defined subgroups of advanced gastric cancer with peritoneal metastases. This case underscores the importance of biomarker-driven treatment strategies to optimize immunotherapeutic outcomes. In addition to systemic and immunotherapy-based strategies, pressurized intraperitoneal aerosol chemotherapy has emerged as an investigational locoregional approach for patients with peritoneal metastases and may serve as a potential adjunct within multimodal treatment strategies in highly selected patients (48,49). Further studies are warranted to better define predictive factors associated with durable response to immune checkpoint blockade in MSI-H gastric cancer and to optimize multimodal approaches for patients with peritoneal dissemination.

The present case has several strengths, including real-world evidence of sustained long-term disease control >5 years in a patient with stage IV MSI-H/dMMR gastric cancer treated with sintilimab plus chemotherapy, supporting the clinical relevance of biomarker-guided immunotherapy in advanced gastric cancer. Similar durable responses have been reported in MSI-H/dMMR gastric cancer treated with PD-1 inhibitors, including case reports describing responses lasting >24 months (50,51), a retrospective analysis demonstrating favorable long-term outcomes in dMMR/MSI-H gastrointestinal cancer treated with immune checkpoint blockade (52),

and clinical trials identifying a subset of patients achieving prolonged clinical benefit and extended progression-free survival (19,53), collectively underscoring the exceptional nature of sustained responses in biomarker-selected populations. However, MSI-H gastric cancer exhibits marked response heterogeneity, with a substantial proportion of patients demonstrating primary resistance to PD-1 blockade (53). In this context, long-term disease control >5 years, as observed in the current case, remains uncommon and represents an extreme spectrum of durable clinical benefit. This may reflect a highly immunogenic tumor microenvironment and sustained immune surveillance. The inherent limitations of a single case report preclude generalization to broader populations. In addition, the absence of certain biomarkers, including PD-L1 expression and claudin 18.2 status, may limit comprehensive molecular characterization.

The optimal duration of immunotherapy in patients achieving complete or durable responses remains uncertain (54). While some reports describe treatment discontinuation after sustained complete response, others continue therapy in the absence of validated predictive biomarkers for durable remission, reflecting persistent uncertainty and the need for individualized treatment decisions (55,56).

In conclusion, the present case illustrates the potential of biomarker-driven PD-1 blockade-based combination therapy to achieve durable disease control in molecularly defined MSI-H gastric cancer, highlighting both the promise and current limitations of precision immunotherapy in advanced disease.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

JH and HX designed the study. ZZ analyzed and interpreted data. CZ performed the pathological analysis. HM performed the radiological analysis. LiaZ and LihZ participated in clinical decision-making. YT performed the surgery and follow-up. LH analyzed data. JH and ZZ wrote the original draft of the manuscript. ZZ, LH, LiaZ, LihZ, YT and HX revised the manuscript. JH and HX confirm the authenticity of all the raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

The present case report was approved by the Ethics Committee of Tongji Hospital, Huazhong University of Science and Technology (approval no. TJ-IRB202603110).

Patient consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Competing interests

The authors declare that they have no competing interests.

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