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The primary aim of *World Journal of Gastrointestinal Oncology* (*WJGO*, *World J Gastrointest Oncol*) is to provide scholars and readers from various fields of gastrointestinal oncology with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

WJGO mainly publishes articles reporting research results and findings obtained in the field of gastrointestinal oncology and covering a wide range of topics including liver cell adenoma, gastric neoplasms, appendiceal neoplasms, biliary tract neoplasms, hepatocellular carcinoma, pancreatic carcinoma, cecal neoplasms, colonic neoplasms, colorectal neoplasms, duodenal neoplasms, esophageal neoplasms, gallbladder neoplasms, *etc*.

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EDITORIAL

Navigating the complex landscape of crawling-type gastric adenocarcinomas: Insights and implications for clinical practice

Hai-Bo Yu, Ke-Feng Jia, Xing-Fen Wang, Bao-Yu Li, Qi Xin

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Abstract

In this editorial, we comment on an article by Xu *et al*. This article describes a case of crawling-type gastric adenocarcinoma (CRA) distinguished by its rare occurrence and diagnostic complexity. We reviewed the detailed case-report findings showcasing clinical, pathological, and molecular characteristics of CRA that shed light on its elusive nature and challenges for early detection and treatment. This case underscored the significance of advanced diagnostic tools such as endoscopic submucosal dissection. Emphasis was placed on the molecular peculiarities of CRA, including the higher mutation rates of genes such as *TP53* and *RHOA* and the notable absence of *HER2* amplification, differentiating it from more conventional forms of gastric adenocarcinoma. In this editorial, we advocate for a multidisciplinary approach to effectively manage this rare subtype and highlight the necessity for precision in both diagnostic and therapeutic strategies. Moreover, a heightened awareness urging the adoption of advanced diagnostic techniques and collaborative approaches is necessary among clinicians and researchers. We aim to contribute to the ongoing discourse in gastrointestinal oncology, emphasizing the importance of recognizing and addressing the complexities associated with rare cancer subtypes such as CRA.

Key Words: Crawling-type gastric adenocarcinoma; Diagnosis; Pathology; Endoscopy;

Treatment

Core Tip: Crawling-type gastric adenocarcinoma (CRA) is characterized by its elusive presentation and propensity for lateral expansion within the mucosal layer, posing significant challenges to early detection. This study emphasizes the need for a multidisciplinary approach to achieve accurate diagnosis and effective treatment. The findings reveal distinct molecular features of CRA including the notably increased mutation frequencies in the *TP53* and *RHOA* genes, and the absence of *HER2* amplification. These characteristics highlight the critical need for precision in diagnostic and therapeutic modalities. The objective is to augment clinical awareness, thereby facilitating prompt identification and efficient management of this subtype of gastric cancer.

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INTRODUCTION

In the dynamic field of gastrointestinal oncology, enhancing patient outcomes depends on identification and a thorough understanding of rare cancer subtypes. This expertise is crucial for advancing clinical practice[1]. A recent case report on crawling-type gastric adenocarcinoma (CRA) presented in the *World Journal of Gastrointestinal Oncology* exemplifies this crucial endeavor[2]. CRA is a rare subtype of gastric cancer (GC) with an elusive presentation that presents diagnostic challenges and offers profound insights into the complexities of the pathology, endoscopy, and management of GC[3,4].

In 1999, Endoh *et al*[5] described eight cases of well-differentiated gastric adenocarcinoma resembling intestinal metaplasia. These cases were distinguished by mild cytological atypia and structurally complex glands with characteristics including branching, tortuous formations, anastomoses, and a plexiform arrangement, in addition to an intestinal immunophenotype. Subsequently, Okamoto *et al*[6] referred to these gastric adenocarcinomas as the "crawling type," alternatively known as "shaking-hands carcinoma," a term not widely recognized in the medical community. CRA is distinguished by its propensity for lateral spread within the mucosal layer and is characterized by low-grade cellular atypia. Its deceptive presentation, which mimics benign conditions, underscores the challenges faced in early diagnoses using conventional diagnostic methods. This case, emblematic of the elusive presentation and diagnostic challenges, highlights the necessity for precision in diagnostic and therapeutic strategies, thus enhancing clinical knowledge of the diverse pathology of GC in the medical community.

CASE PRESENTATION

This report meticulously described the case from patient presentation to diagnosis and treatment, providing valuable lessons at each step. The initial lack of symptoms, a common scenario in early GC, and the subsequent findings from routine examinations illustrated the silent character of this disease and the serendipitous nature of its detection. The narrative further explored the diagnostic enigma posed by this cancer subtype, highlighting the critical role of endoscopic submucosal dissection (ESD) as a definitive diagnostic tool, not just a therapeutic intervention.

This case emphasized the necessity for a multidisciplinary approach to gastrointestinal oncology combining the varied expertise of endoscopists, pathologists, surgeons, and oncologists to navigate the complexities of such rare cancers. Discussions on the use of various diagnostic techniques including narrow-band imaging (NBI), chromoendoscopy, and immunohistochemical markers illustrated the evolving landscape of GC diagnosis and the requirement for a magnitude of clinical suspicion and skill.

Moreover, the successful outcome in this case, with no evidence of recurrence at the one-year follow-up, served as a testament to the potential for a favorable prognosis when CRA is identified early and managed effectively. These findings highlighted the importance of early detection and the role of advanced endoscopic techniques in achieving diagnostic and therapeutic success.

PATHOLOGICAL DIAGNOSIS

CRA, a notable subtype of moderately differentiated adenocarcinoma, is increasingly being recognized for its distinct clinicopathological and molecular features. Unlike typical moderately differentiated adenocarcinomas that progress

through hyperdifferentiation, CRAs originate directly from moderately differentiated adenocarcinomas. This subtype often occurs simultaneously with a pronounced inflammatory backdrop and low-grade cellular heterogeneity closely mimicking intestinal epithelial hyperplasia, leading to frequent misdiagnoses as benign or indeterminate lesions.

CLINICO-PATHOLOGICAL CHARACTERISTICS

Clinically, CRA is characterized by low-grade cellular atypia and pronounced structural anomalies[7]. Cells exhibit minimal dysplasia resembling intestinal metaplasia, or mildly atypical epithelial cells against a background rich in inflammatory cells. Structurally, these adenocarcinomas display irregular proliferation zones, with glands merging in a pattern marked by branching, anastomosis, and dilation. These features result in cystic glandular dilatation with sparse eosinophilic cells, exfoliation, and sharp angles[8]. The unique merging pattern of glands, described as resembling the letters "WHYX,"[5,9] serves as a critical diagnostic tool. Glandular basement membranes exhibit discontinuity and roughness blending into the stroma with protruding "tentacles", occasionally accompanied by undifferentiated elements such as signet ring cells[6] and eventually evolving into a diffuse form. Immunohistochemistry (IHC) assays showing variable expression of protein markers encoded by the *MUC2, MUC5AC, MUC6* genes, and the Ki-67 protein may provide insights into the cellular makeup and proliferative index of tumors. *MUC2* is typically associated with intestinal-type GC s and is rarely expressed in diffuse-type cancers, including CRA. The lack of *MUC2* expression in CRA can help differentiate it from intestinal-type gastric adenocarcinoma, aiding in accurate subtype identification. *MUC5AC* is commonly expressed in the foveolar epithelium of the stomach and is frequently observed in both intestinal and diffuse types of gastric adenocarcinomas, including CRA. Its presence can help confirm the gastric origin of the tumor. *MUC6* is expressed in the deep gastric glands and variably present in gastric adenocarcinomas. In CRA, *MUC6* expression can further characterize tumor differentiation status. The Ki-67 Labeling index provides information on the proliferative activity of the tumor. Higher Ki-67 indices in CRA can indicate aggressive tumor behavior and help gauge the tumor growth rate.

The IHC results contribute to prognostic information. Loss of E-cadherin protein expression is a hallmark of CRA, particularly in diffuse-type and signet ring cell carcinomas. This loss helps identify tumors with a high invasive potential and diffuse growth pattern. While HER2 protein overexpression is less common in CRA compared to intestinal-type gastric adenocarcinoma, its detection can aid in identifying subgroups that may benefit from targeted therapies such as trastuzumab.

MOLECULAR PATHOLOGICAL CHARACTERISTICS

The *TP53* mutation rate in CRAs is significantly higher than that in conventional adenocarcinomas, and predominantly features deletion mutations. Consequently, IHC for p53 shows a negative expression. Moreover, the c.529_546 deletion mutation in the *TP53* gene is notably absent in the conventional types[10]. *TP53* is a tumor suppressor gene, and its mutation leads to the loss of cell cycle regulation, allowing for uncontrolled cell proliferation. In CRA, *TP53* mutations are indicative of genomic instability and are often associated with high-grade tumors. The presence of mutations in *TP53* generally correlate with a more aggressive disease course and poorer overall survival. Identifying *TP53* mutations can help stratify patients into different risk categories and tailor treatment strategies accordingly.

RHOA mutations are significantly more prevalent in CRA than in conventional tubular adenocarcinoma, similar to the pattern observed in diffuse-type GC[8]. A higher incidence of *RHOA* mutations and *CLDN18-ARHGAP* gene fusions has been found in CRA, mirroring the genetic phenotype of diffuse-type GC (poorly cohesive carcinoma)[11]. *RHOA* is involved in regulating the cytoskeleton, cell motility, and epithelial-mesenchymal transition. Mutations in *RHOA* disrupt these processes, leading to enhanced invasiveness and metastasis in CRA. *RHOA* mutations are specific to diffuse-type GCs and can serve as a molecular marker for this subtype. They are associated with distinctive histopathological features and worse prognosis, emphasizing the need for targeted therapeutic approaches and rigorous follow-up. (4) CRA shows non-amplification of HER2[8].

ENDOSCOPIC DIAGNOSIS

CRA is predominantly found in the middle third of the stomach, particularly in the transitional zones. Furthermore, CRA can be categorized into two types: Superficial depression (0-IIc) or superficial flatness (0-IIb). The boundaries of CRA can sometimes appear indistinct. This characteristic feature is likely due to the "creeping" infiltration of the CRA tumor glands into the proliferative zone of the epithelium, where they are partially covered by the non-tumorous pit epithelium. Traditional white-light endoscopy may not adequately reveal the distinct features of the tumor, often presenting as flat or slightly depressed lesions with an indistinct border[6] and closely mimicking benign gastric conditions such as gastritis or intestinal metaplasia[3]. NBI, magnifying endoscopy, and chromoendoscopy enhance the visualization of mucosal patterns and vascular architecture, which are indicative of neoplastic changes[12]. NBI enhances the contrast between neoplastic and non-neoplastic tissues, revealing irregular microvascular patterns and mucosal structures possibly indicating the presence of this cancer subtype[13]. Chromoendoscopy, which involves the application of special dyes, further delineates subtle mucosal abnormalities, aiding in the identification and targeted biopsy of suspicious areas.

The specificity of endoscopic findings for CRA is relatively low due to its resemblance to benign conditions and other types of GC. The irregular and subtle appearance can lead to misdiagnosis or underdiagnosis, especially in the early stages. Sensitivity for detecting CRA is also a challenge. Standard white-light endoscopy may miss CRA due to its flat and infiltrative nature. Sensitivity improves with the use of enhanced imaging techniques. Studies have shown that NBI and chromoendoscopy can significantly increase the detection rates of early GCs, including CRA, by highlighting abnormal mucosal structures and vascular patterns that are not visible with conventional endoscopy. While benign conditions like gastritis and intestinal metaplasia appear similar to CRA, they lack the irregular, invasive margins characteristic of CRA. In contrast, intestinal-type gastric adenocarcinomas present as raised, ulcerative, or polypoid lesions with well-defined edges. Endoscopic ultrasonography aids differentiation by providing detailed images of submucosal invasion depth.

The propensity of CRA for lateral spread, rather than deeper penetration into the gastric layers, necessitates a high degree of vigilance and expertise from endoscopists. Therefore, special attention must be paid to accurately determining tumor margins. Given the potential difficulty in endoscopically identifying CRA margins, the possibility of CRA should be considered when superficial depressed or flat lesions are detected in the middle third of the stomach, necessitating a careful boundary assessment.

TREATMENT

The borders of intramucosal CRA are often poorly defined because of the lack of contrast from the surrounding non-neoplastic mucosa. This feature of CRA often causes failure of complete resection after ESD[14]. CRA treatment is complicated by the high rate of margin positivity, which is reported to be approximately 30%, even after meticulous examination. This requires a careful and tailored treatment approach that emphasizes the need for complete tumor resection with clear margins to minimize the risk of recurrence. ESD has been employed in selected cases of CRA in which the tumor is confined to the mucosa or superficial submucosa and has well-defined margins. However, the high risk of incomplete resection necessitates close postoperative surveillance and, in some cases, additional surgical intervention. For more advanced cases or when endoscopic resection is not feasible, surgical resection remains the cornerstone of treatment, often accompanied by lymph node dissection to address potential metastatic spread.

FOLLOW-UP STRATEGY

The follow-up strategy for CRA patients is crucial for early detection of recurrence and management of any long-term treatment-related complications. A structured follow-up protocol typically includes regular clinical assessments, imaging studies, and endoscopic evaluations. (1) Clinical assessments: Patients are seen every 3-6 months for the first two years following surgery, and every 6-12 months thereafter. During these visits, a thorough physical examination and review of symptoms are conducted to identify any signs of recurrence or metastasis; (2) Imaging studies: Routine imaging, such as computed tomography scans or positron emission tomography scans, are performed at scheduled intervals, usually every 6-12 months in the first two years and annually thereafter. These studies help monitor for any anatomical changes indicative of recurrence; (3) Endoscopic evaluations: Regular endoscopic surveillance is essential for detecting local recurrence, particularly given the submucosal and diffuse-growth patterns characteristic of CRA. Endoscopies are typically performed every 6-12 months in the initial follow-up period and then annually; and (4) Laboratory tests: Periodic blood tests including tumor markers such as carcinoembryonic antigen and carbohydrate antigen 19-9, are also part of the follow-up protocol. These markers can provide early indications of tumor recurrence.

ADJUVANT TREATMENTS AND RATIONALE FOR FOLLOW-UP DURATION

Adjuvant treatments are tailored based on individual patient factors and tumor response to initial therapy. The rationale for follow-up duration and frequency is informed by the aggressive nature of CRA, its potential for late recurrences, and the need for ongoing monitoring to manage any long-term effects of treatment. (1) Adjuvant chemotherapy: Given the high risk of recurrence in CRA, adjuvant chemotherapy is critical. The selected regimens aim to eliminate microscopic residual diseases that might not be addressed by surgery alone; and (2) Radiotherapy: While not routinely used for all patients, radiotherapy may be considered in cases where there is a high risk of local recurrence or where surgical margins are close.

The primary goal of the follow-up strategy is to balance early detection of recurrence with patient quality of life, minimizing unnecessary interventions while ensuring timely identification and treatment of any recurrence or metastasis.

CRA CONTRASTS WITH COMMON-TYPE GC

Endoscopic appearance

The endoscopic features of CRA are distinct when compared with those of more common types of gastric adenocarcinoma. CRA is characterized by its unique "crawling" growth pattern, which is typically observed as a flat or slightly

elevated lesion with an irregular shape and poorly defined margins[15]. The surface may appear granular or nodular, often mimicking benign conditions such as gastritis or intestinal metaplasia. Therefore, distinguishing CRA from non-malignant lesions solely based on endoscopic appearance is challenging. In contrast, more common forms of gastric adenocarcinoma, such as tubular or papillary adenocarcinomas, usually present as raised, ulcerative, or polypoid lesions. These types often exhibit well-defined edges and a more obvious mass effect that is more readily identifiable during endoscopy. The differences in endoscopic appearance are significant for early detection and accurate diagnosis, emphasizing the need for heightened awareness and careful examination when CRA is suspected.

Biological behavior

The biological behavior of CRA also differs notably from other gastric adenocarcinomas. CRA is known for its aggressive infiltration into the submucosa and muscularis propria, often extending laterally over a broad area of the stomach wall [16]. This lateral spread can lead to significant submucosal invasion without forming a prominent mass, complicating detection and staging. This behavior contrasts with the vertical invasion pattern commonly observed in other types of gastric adenocarcinoma, where the tumor tends to penetrate deeper layers more directly.

Moreover, CRA often exhibits a diffuse growth pattern with signet ring cells, a hallmark of diffuse-type GC (Lauren classification). This histological feature is associated with a poorer prognosis and a higher likelihood of peritoneal dissemination and lymph node metastasis when compared with those of intestinal-type GCs, which typically display glandular structures and a more cohesive growth pattern.

CONCLUSION

The CRA case presented in this issue serves as a compelling reminder to clinicians of the diversity of GC and the challenges that are posed by rare subtypes. It advocates increased awareness, improved diagnostic accuracy, and the adoption of multidisciplinary care models to enhance outcomes for patients with this and other rare forms of GC. As we continue to advance our understanding and refine our approaches to gastrointestinal oncology, this case serves as an inspiration for continued research, collaboration, and innovation in the effort to control GC.

FOOTNOTES

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