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

WJGS mainly publishes articles reporting research results and findings obtained in the field of gastrointestinal surgery and covering a wide range of topics including biliary tract surgical procedures, biliopancreatic diversion, colectomy, esophagectomy, esophagostomy, pancreas transplantation, and pancreatectomy, *etc*.

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CASE REPORT

Primary coexisting adenocarcinoma of the colon and neuroendocrine tumor of the duodenum: A case report and review of the literature

Song Fei, Wei-Dong Wu, Han-Shuo Zhang, Shao-Jie Liu, Dan Li, Bo Jin

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Abstract

BACKGROUND

Neuroendocrine tumors (NETs) arise from the body's diffuse endocrine system. Coexisting primary adenocarcinoma of the colon and NETs of the duodenum (D-NETs) is a rare occurrence in clinical practice. The classification and treatment criteria for D-NETs combined with a second primary cancer have not yet been determined.

CASE SUMMARY

We report the details of a case involving female patient with coexisting primary adenocarcinoma of the colon and a D-NET diagnosed by imaging and surgical specimens. The tumors were treated by surgery and four courses of chemotherapy. The patient achieved a favorable clinical prognosis.

CONCLUSION

Coexisting primary adenocarcinoma of the colon and D-NET were diagnosed by imaging, laboratory indicators, and surgical specimens. Surgical resection combined with chemotherapy was a safe, clinically effective, and cost-effective treatment.

Key Words: Neuroendocrine tumor of the duodenum; Multiple primary tumors; Colorectal cancer; Endoscopic resection; Case report

Core Tip: Coexisting primary adenocarcinoma of the colon and neuroendocrine tumor of the duodenum (D-NET) is a rare occurrence in clinical practice. We report the details of a case involving a female patient with coexisting primary adenocarcinoma of the colon and D-NET diagnosed by imaging and surgical specimens. The tumors were treated by surgery and four courses of chemotherapy. The patient achieved a favorable clinical prognosis. The classification and treatment criteria for D-NETs combined with a second primary cancer have not yet been determined. Our experience may help others to diagnose and manage similar patients.

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INTRODUCTION

Neuroendocrine tumors (NETs) originate from endocrine organs and thus may arise from almost any location in the body. They are most commonly found in the gastrointestinal tract (*i.e.* GEP-NETs) and respiratory system. Neuroendocrine cells produce neuroregulators, neuropeptides, or neurotransmitter hormones. Multiple primary tumors are a unique occurrence in medical practice. The clinical features of D-NETs combined with a second primary malignant tumor lack specificity. Diagnosis of coexisting primary adenocarcinoma of the colon and NET of the duodenum (D-NET) can be diagnosed by imaging, laboratory indicators, and from surgical specimens.

Different D-NETs have different treatments, but surgery remains the best method. Endoscopic resection (ESD) is safe and effective for duodenal carcinoid tumors that are ≤ 10 mm in diameter and limited to the submucosal layer. For tumors between 10 mm and 20 mm in diameter, endoscopic or surgical treatment can be used, and surgical treatment is performed for suspected tumors > 10 mm or tumors with positive margins after resection. Surgery is the only treatment for local early colon cancer (stages I and II). Chemotherapy is the standard treatment for patients with locally advanced stage III and IV after radical surgery. For some stage-2 colon cancer patients, systemic treatment with surgery is based on risk factors and microsatellite instability (*MSI*) gene status.

We describe the treatment of a female patient diagnosed with coexisting primary adenocarcinoma of the colon and D-NET by imaging and examination of surgical specimens. The tumors were treated by surgery and four courses of chemotherapy, and the patient achieved a favorable clinical prognosis (Table 1)[1,2]. The patient's clinical, laboratory, and imaging features, and pathophysiology are discussed, with a short review of the recent literature on coexisting primary adenocarcinoma of the colon and a D-NET.

CASE PRESENTATION

Chief complaints

A 66-year-old female patient was admitted to the hospital on April 25, 2023 because of abdominal pain for 4 mon and had worsened in the previous 7 days.

History of present illness

Four months prior to admission, the patient experienced intermittent periumbilical pain and abdominal distension without obvious reasons, accompanied by a sense of urgency and incomplete defecation, and her stools became thinner. The symptoms worsened 7 days before admission.

History of past illness

The patient had no history of hypertension, diabetes, or other major organ disease such as cardiopulmonary disease.

Personal and family history

The patient's father had a history of nasopharyngeal carcinoma, and there was no other family history of cancer. The patient had no history of smoking or drinking.

Physical examination

Mild tenderness in the upper abdomen, pains around the umbilicus, no rebound pain or muscle tension, and no obvious abnormalities in the rest of the physical examination.

Laboratory examinations

Except for positive fecal occult blood, the laboratory examination found no abnormalities.

Table 1 Clinical and pathological data of the patient

Feature	Colon adenocarcinoma	Neuroendocrine tumor
Tumor size	6 cm × 4 cm × 1 cm	9 mm
Lymph node invasion	- (0/20)	Not applicable
Fat tissue invasion	+	-
Perineural invasion	+	-
Vascular invasion	+	-
Lymphatic vessel invasion	+	-
Muscularis propria invasion	+	-
Serosal invasion	+	-
Resection margins	-	-
Tumor necrosis	+	-
P53	90% (+)	10% (+)
Ki-67	70% (+)	2% (+)
Mitoses/2 mm^2	> 20	1
Immunocytochemistry	Syn (-), MLH1 (+), MSH2 (+), MSH6 (+), PMS2 (+), BRAFV600E (-), CD34 (+), D2-40 (+)	CK (+), CK7 (+), CgA (+), Syn (+), NSE (+)
Grade of the cancer	High grade (G3)	Low grade (G1)
TNM stage	pT4apN0pM0	pT1pN0pM0
Astler-Coller classification[1]	B	Not applicable
AJCC clinical stage[2]	IIB	I

AJCC: American Joint Committee on Cancer; BRAFV600E: B-Raf proto-oncogene, serine/threonine kinase 600E; CgA: Chromogranin A; CK: Cytokeratin; CK7: Cytokeratin 7; Ki-67: Antigen Ki-67; MLH1: MutL homologue 1; MLH3: MutS homologue 3; MSH2: MutS homologue 2; MSH6: MutS homologue 6; NSE: Neuron-specific enolase; PMS2: PMS1 homolog 2 mismatch repair system component; Syn: Synapsin; TNM: Tumor, node, metastasis classification of malignant tumors.

Imaging examinations

Enhanced computed tomography (CT) of the entire abdomen on admission showed intestinal cancer invading the serosal layer of transverse colon near the hepatic flexure, small mesenteric lymph node metastasis that needed to be ruled out (Figure 1A-C), and abnormal enhancement of the duodenal bulb (Figure 2). Gastroscopy and endoscopic ultrasonography showed a slightly hypoechoic lesion with a broad base measuring approximately 8.9 mm × 6.3 mm in the mucosal layer of the anterior wall of the duodenum. The remaining mucosal layer appeared normal, but a large polyp in the duodenum was of possible concern. Multiple attempts to remove the polyp by endoscopic procedures were unsuccessful. Chronic gastritis with erosion was visible (Figure 3). Colonoscopy found an irregular mass with a diameter > 2 cm located 70 cm from the anus. It had a rough surface, hard texture, bled easily, and obstructed the intestinal lumen, making it difficult for the endoscope to enter. A biopsy was taken, and the pathological diagnosis was mucosal adenocarcinoma of the transverse colon (Figure 1D).

FINAL DIAGNOSIS

Considering the clinical history, CT imaging manifestations, endoscopic visualization, EUS, and the postoperative pathology report (Figure 4), the diagnosis was malignant tumor of the hepatic flexure of the colon (T4 N0 M0, IIB) with a concurrent gastric neuroendocrine tumor (NET, G1).

TREATMENT

The preoperative diagnosis was a malignant tumor located at the hepatic flexure of the colon and a polyp in the duodenal bulb. Intraoperative exploration revealed a mass with a hard texture and a diameter of approximately 1 cm in the duodenal bulb. The spleen showed no evidence of metastasis, and there were no enlarged lymph nodes were palpable near the abdominal aorta. A mass of approximately 4.0 cm × 4.0 cm was palpated at the hepatic flexure of the colon. It had

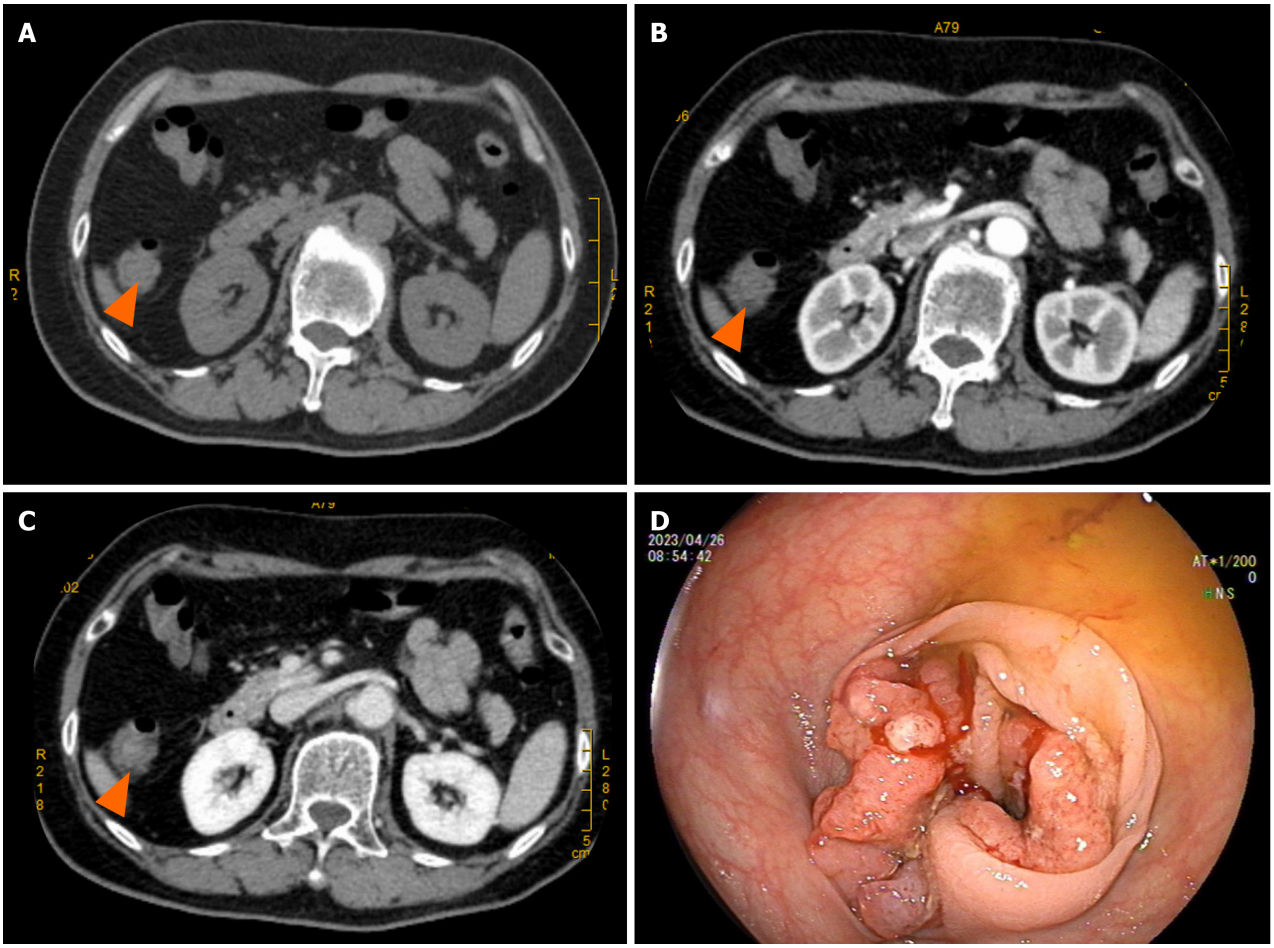

Figure 1 Colonoscopy and enhanced total abdominal computed tomography. A-C: Computed tomography (CT) shows that the colon cancer near the hepatic flexure of the transverse colon invaded the serosal layer, with a maximum invasion depth of approximately 2 cm, with small mesenteric lymph node metastasis (orange triangles) to be ruled out; CT plain scan (A), CT enhanced scan (B), and CT venous phase of the mass (C), respectively; D: Colonoscopy shows an irregular mass with a diameter of > 2 cm located 70 cm from the anus.

invaded the serosal layer, and the proximal cecum was slightly dilated, but no mass was palpated. It was decided to perform hand-assisted laparoscopic surgery (HALS) of right hemicolectomy, peri-intestinal lymph node dissection, ileotransverse colon anastomosis, distal gastrectomy, and gastrojejunostomy. The gastric tube was removed on the fifth postoperative day, and the patient started taking fluids. The patient recovered by the eighth postoperative day and was discharged from the hospital without fever, jaundice, abdominal distension, abdominal pain, hematemesis, diarrhea, black stools or other symptoms.

OUTCOME AND FOLLOW-UP

The patient was followed-up after discharge and given four courses of oxaliplatin plus capecitabine chemotherapy. At the time of writing, the patient has been followed-up for 11 mon, with CT scans and laboratory examination every 3 mon. The patient's overall condition is good, with no signs of tumor progression or additional metastasis.

DISCUSSION

As NETs originate in endocrine organs, they can be found in nearly any location in the body, with most arising in the gastrointestinal tract (*e.g.*, GEP-NETs) and respiratory system. Neuroendocrine cells produce neuroregulators, neuropeptides, or neurotransmitter hormones[3]. The fifth edition 2019 of World Health Organization (WHO)[4] and the 2016 European Neuroendocrine Tumor Society (EUETS)[5] classification of digestive system tumors divides gastric NETs into three types by their differentiation, well-differentiated G-NETs and neuroendocrine carcinomas. However, because few duodenal NETs have been reported, the WHO classifies and grades NETs of the gastrointestinal and hepatopancreatobiliary tracts, including duodenal NETs, by their Ki67 index and mitotic rate. The 2016 EUETS classification lists it as a separate category without classification. The 2020 Chinese Society of Gastroenterology Expert Consensus on the Diagnosis and Treatment of Gastrointestinal Pancreatic Neuroendocrine Tumors was based on the above classification[6], but

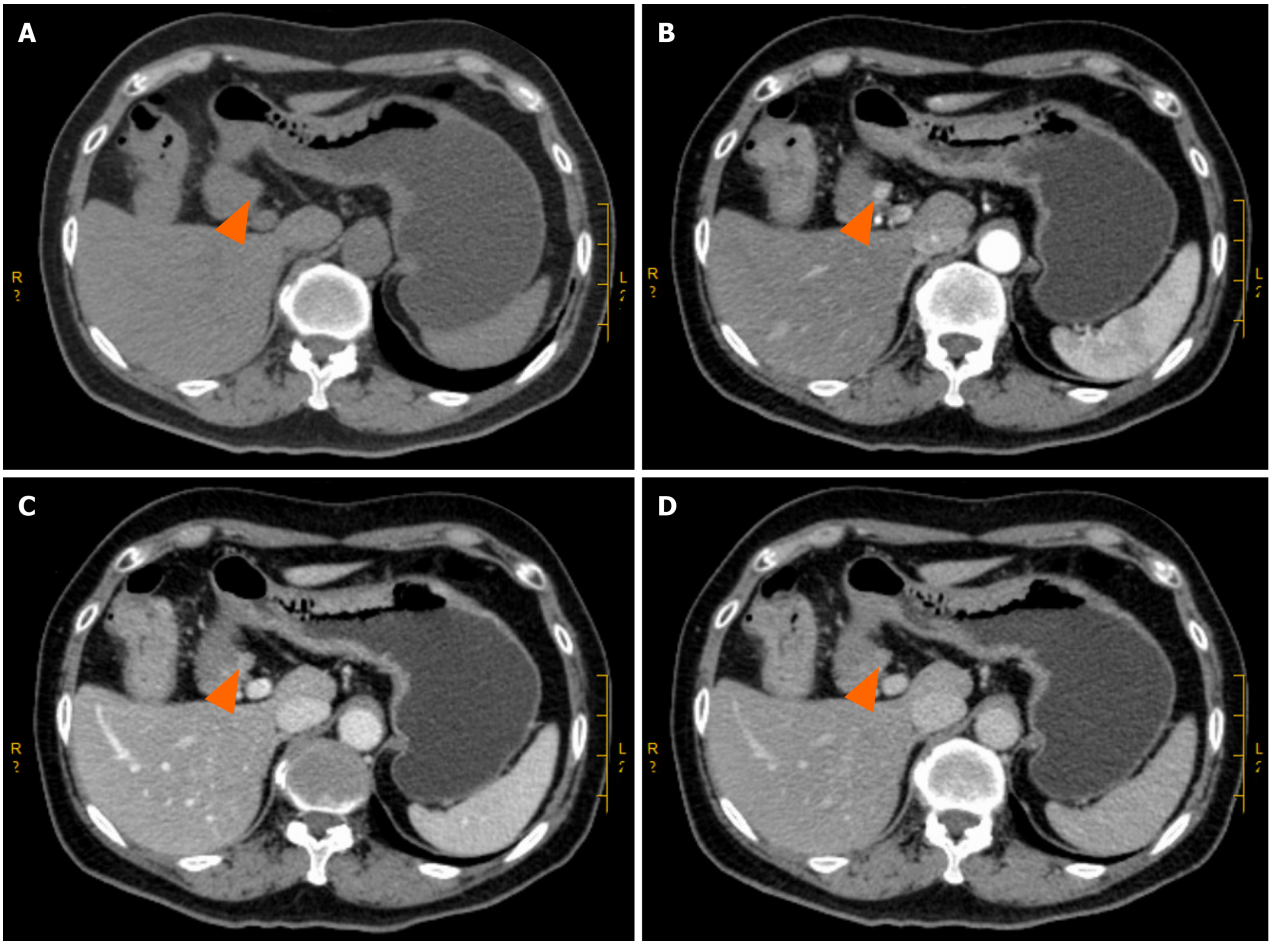

Figure 2 Enhanced total abdominal computed tomography findings. A: Computed tomography (CT) plain scan of a mass in the duodenal bulb; B: Abnormal enhancement of the duodenal bulb (orange triangles); C and D: CT venous phase and CT venous delayed phase of the mass, respectively.

duodenal NETs were not mentioned. Gastrointestinal NETs can be divided into functional and nonfunctional subtypes according to whether secreted hormones can be detected and by the presence of related endocrine symptoms. Functional tumors are slow growing and their onset is usually caused by the secreted hormones rather than tumor proliferation. In the case of nonfunctional tumors, it is generally believed that hormones are secreted but cannot be detected by existing assays. Nonfunctional tumors tend to be more aggressive and invasive than functional tumors[3]. According to the Surveillance, Epidemiology, and End Results (SEER) database of National Cancer Institute, the incidence of G-NETs has risen rapidly in the United States in the past 40 years, and was 0.62/100000 in 2016[7,8]. A study in Argentina[9] reported that G-NETs and D-NETs accounted for 6.9% and 2.0% of all digestive tract NETs, respectively. The most recent study in Japan[10] reported that D-NETs accounted for approximately 5% of GEP-NETs. Epidemiological studies of G-NETs show that the incidence of D-NETs is increasing in many countries worldwide[9-12].

Multiple primary tumors are a unique phenomenon in medicine and are divided into two categories by their time of onset. Synchronous tumors occur simultaneously and heterochronous tumors occur in chronological order. Warren and Gates[13] conducted autopsies of 1078 cancer patients and found that 40 (3.7%) had either occult or clinically apparent second primary tumors. Some studies have reported a correlation of NETs with an increased risk of developing secondary gastrointestinal malignancies. A study of 58596 patients with NETs found that 4612 (7.9%) developed a second primary malignancy during follow-up. Patients with different types of NETs had different incidences of second primary malignancies. G-NET patients had an increased risk of developing second primary malignancies in the esophagus, small intestine, pancreas, and liver but no such risk was reported for D-NETs[14]. In a study of 459 patients Kamp *et al*[15] found a correlation between GEP-NETs and a second primary malignancy in which the incidence of a GEP-NET combined with a second primary malignancy (mainly colorectal cancer) was increased. The pathogenesis of secondary cancers associated with D-NETs was not clear but it involved the interaction of genetic, environmental, hormonal, medical, and sex-related factors[16-18].

The clinical features of GEP-NETs combined with a second primary malignant tumor lack specificity. A previous study reported that gastrin and cholecystokinin were associated with NETs and induction of tissue growth and cellular malignant transformation in the gastrointestinal tract, leading to colorectal and gastric cancer[19]. In patients with NETs, the possibility of a hormone hypersecretion syndrome, such as paraneoplastic or carcinoid syndrome, must be strongly considered. Vilallonga *et al*[20] found that 5 of 2155 patients diagnosed with colorectal cancer presented with paraneoplastic or carcinoid syndrome. This study of NETs did not include carcinoid tumors. However, the clinical manifestations of D-NETs are not specific, and not all tumors have the above manifestations. A retrospective study of 927 patients with

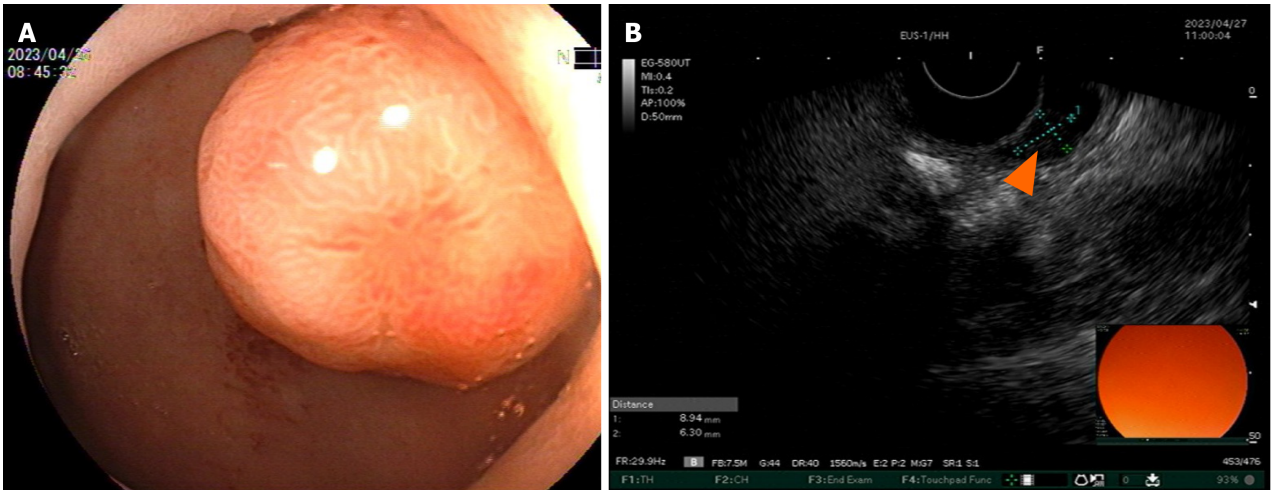

Figure 3 Gastroscopy and endoscopic ultrasonography. A: Gastroscopy findings of the mass; B: A slightly hypoechoic lesion with a broad base and approximately 8.9 mm × 6.3 mm is seen in the mucosal layer of the anterior wall of the duodenum (orange triangles).

duodenal endocrine tumors found that the incidence of carcinoid syndrome was only 3.1%[21], and that the incidence of carcinoid syndrome in duodenal NETs may have been even lower. According to the most recent classification of gastric NETs, types I and III often do not present with paraneoplastic or carcinoid syndrome, which makes their diagnosis more difficult. Our patient was admitted to the hospital with abdominal pain. Except for positive fecal occult blood, there were no obvious abnormalities o tumor markers or blood-test indicators. Because a duodenal NET was not considered before surgery, serum chromogranin A (CgA), neuron-specific enolase, and related hormone levels were not tested. However, preoperative enhanced CT of the abdomen showed an abnormal enhancement sign in the duodenal bulb, and gastroscopy and endoscopic ultrasound revealed a slightly hypoechoic lesion with a wide base in the mucosal layer of the anterior wall of the duodenum. Most NETs are hypervascular, and their obvious enhancement characteristics in the arterial phase have a highly suggestive role in qualitative diagnosis. Many studies and current guidelines indicate that enhanced CT and gastroscopy have reference value for the diagnosis of gastrointestinal NETs[22-25]. The WHO and EUETS criteria and the condition of our patient, it can be classified as a G1 NET. However, the classification criteria for D-NETs combined with a second primary cancer have not yet been explicit conclusions.

Different D-NETs have different treatments, but surgery is still the best method for treat D-NETs. Endoscopic submucosal dissection is a safe and efficacious treatment for duodenal carcinoid tumors that are ≤ 10 mm in diameter and limited to the submucosal layer[5,6,26]. For tumors between 10 mm and 20 mm in diameter, endoscopic or surgical treatment can be chosen, but surgery is performed for suspected tumors > 10 mm or tumors with positive margins after resection. The surgery that is selected (*e.g.*, local resection, gastrectomy, or total gastrectomy) is guided by the pathological characteristics and invasiveness of the tumor[2,5]. A literature review of 44 duodenal small-papilla NETs with an average tumor size of 14.0 mm (range: 2-27 mm) found that most were pathological type G1 tumors (20/22). More than half of the lesions (58.3%; 14/24) that were ≥ 10 mm had lymph node metastasis, and duodenal small-papilla NETs had a higher probability of lymph node metastasis[27]. The results have a reference value for the selection of the treatment method for this type of tumor in the future, such as sentinel lymph node biopsy to determine whether to perform radical tumor resection. At present, tumor resection, chemotherapy, targeted therapy, and radionuclide therapy are the conventional treatment methods for GEP-NETs. For localized tumors, only surgery provides the possibility of complete cure[28, 29]. However, lymph node metastasis or distant organ micrometastasis may have occurred during or before surgery. Such tumors are often neuroendocrine carcinomas and the treatment of that type of tumor currently relatively consistent. For localized lesions, treatment by local or radical surgery and adjuvant chemotherapy after surgery are relatively common. However, most patients with neuroendocrine carcinoma have distant metastasis at the time of diagnosis, and the tumor is highly malignant and unresectable. In those cases, chemotherapy is the first choice for treatment. Etoposide + cisplatin (EP) is a chemotherapy regimen for small cell lung cancer and was shown to be effective in 67% of cases[30]. Epirubicin + cyclophosphamide (EC) and irinotecan + cisplatin (IP) can be selected as first-line regimens. Ooxaliplatin + leucovorin + 5-fluorouracil (FOLFOX), irinotecan+calcium folinate + 5-fluorouracil (FOLFIRI), and Capecitabine + temozolomide. (CAPTEM) can be used as second-line chemotherapy regimens. Anti-PD-1 therapy can be considered as a third-line treatment in patients with high MSI (MSI-H), mismatch repair deficiency (d-MMR), and high tumor mutation burden (TMB-H)[6]. In this case, the patient did not have secretory NETs, paraneoplastic symptoms or signs, or any ectopic hormone secretion. She was considered to have a well-differentiated G1-like D-NET, and surgical resection was the first choice of treatment. However, during the operation, laparoscopic exploration showed that the texture of the mass was hard, and no enlarged lymph nodes were found near the abdominal aorta. Combined with the preoperative CT and gastroscopy, it was suggested that this type of mass was possibly malignant. In the absence of a pathological diagnosis, distal gastrectomy + gastrojejunostomy was selected for the mass, and the distal stomach, with a volume of 11.5 cm × 8.5 cm × 1.5 cm was resected. Complete resection of the mass may improve the overall prognosis of the patient.

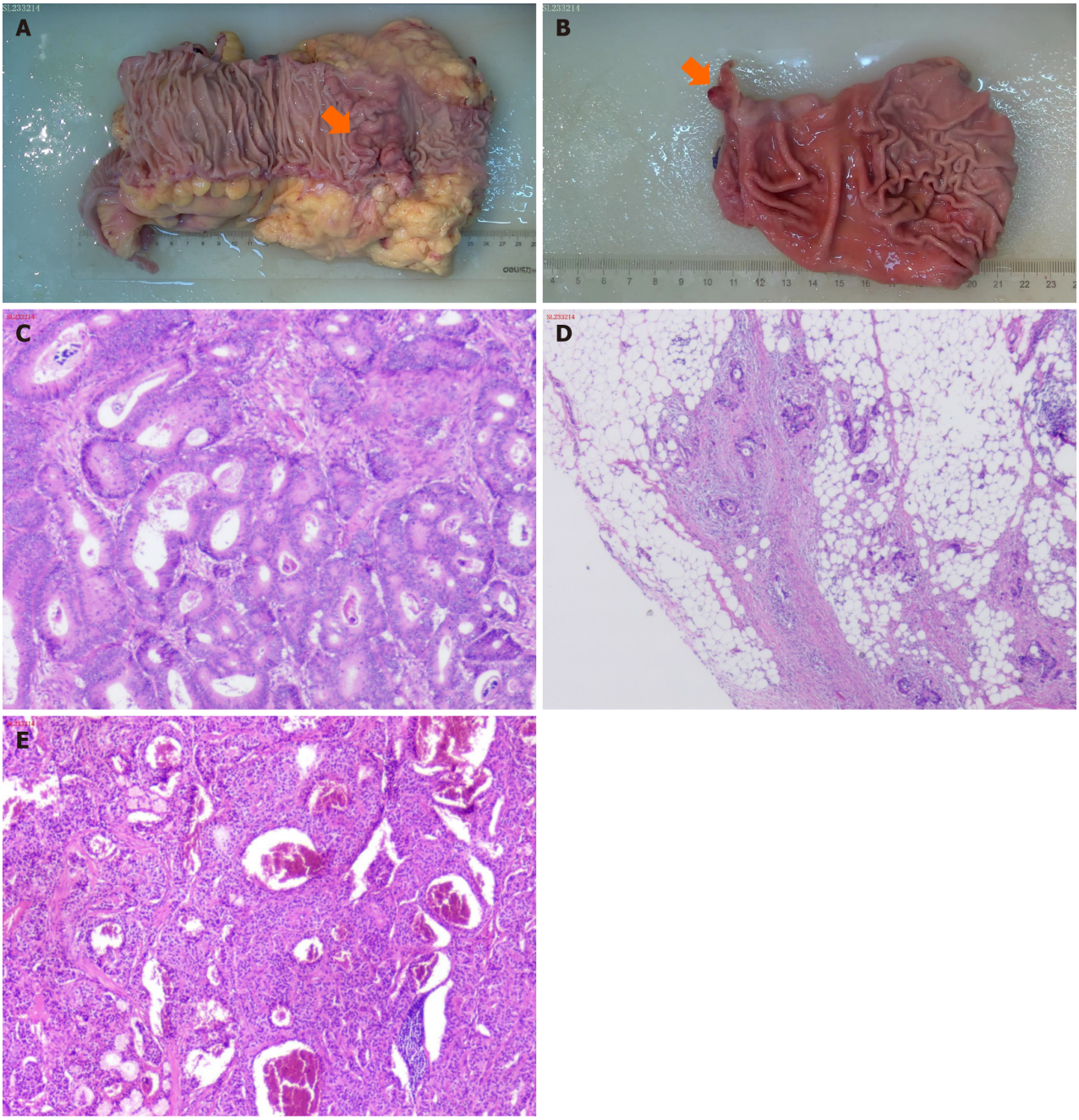

Figure 4 Postoperative pathology. Tumors are indicated by orange arrows. A: Resected colon and mass during surgery; B: Resected distal stomach and mass during surgery; C and D: Hematoxylin and eosin-stained tissue from the colon mass; E: Hematoxylin and eosin-stained tissue from the distal stomach mass.

In patients with NETs positive for somatostatin receptors, subcutaneous or intramuscular administration of somatostatin analogs (octreotide LAR 10, 20, or 30 mg intramuscularly every 4 weeks, or lanreotide 60, 90, or 120 mg subcutaneously every 4 weeks) can relieve the symptoms by blocking hormone release[6,31,32]. In addition to being suitable for patients with neuroendocrine carcinoma, chemotherapy and targeted therapy are not currently used as first-line treatment regimens for NETs. In radiological and serological studies, more than half of the patients achieved partial remission (PR) with combined use of cisplatin and etoposide, streptozocin and doxorubicin or 5-fluorouracil[33-35]. Targeted chemotherapeutic drugs (*e.g.*, everolimus or sunitinib) have been used in some studies to improve progression-free survival (PFS), Continuous administration of 37.5 mg daily improved the PFS, overall survival, and objective response rates in patients with advanced NETs. Everolimus was significantly correlated with a median PFS of more than 6.4 mon longer than placebo[36-41]. Isolated liver metastases can be improved with radiofrequency ablation or hepatic artery embolization, or by combining hepatic artery embolization with hepatic artery chemotherapy perfusion[42]. The internal radiation therapy chosen for neuroendocrine liver metastases can be given by injection of radioactive microspheres directly into the hepatic artery. The available isotopes for radiotherapy include yttrium-90, lutetium-177-labeled analogs, and iodine-131-meta-iodobenzylguanidine[43-45].

With the advent of aggressive surgical intervention and second-line treatment with long-acting somatostatin agonists and targeted drugs, the prognosis and long-term survival of patients with NETs have improved. Studies have shown that

in the case of malignant tumors, the 5-year survival rate can be as high as 77% to 95% following radical resection of the primary tumor and adjuvant therapy[46,47]. For localized and well-differentiated tumors treated by complete surgical resection, the 5-year survival rate of G-NETs is as high as 90%. Radical resection of the primary tumor, absence of liver metastasis, metachronous liver metastasis, and active treatment of liver metastasis are all favorable factors and improve prognosis[48,49]. However, nearly all patients diagnosed with metastatic gastric neuroendocrine cancer have a recurrence within 7 years of follow-up. Recurrence is difficult to avoid even after a complete cure[49], indicating its refractory characteristics.

In the follow-up of NETs, early studies found that octreotide CT or octreotide SPE-CT scans have an important role in detecting the recurrence of NETs[50,51]. Frilling *et al*[50] found that 19 of 35 patients with NETs (54.2%) had extrahepatic tumors that were not detected by other imaging techniques, such as CT, MRI, or ultrasound. Octreo-SPECT/CT imaging can be used to detect and locate suspected NETs before their diagnosis[52,53], and can be used to follow-up and detect tumor recurrence after diagnosis or treatment. In this case, the patient was considered to have an early stage duodenal NET, and only CT and laboratory tests were used for follow-up.

Surgery is the most effective treatment for local early-stage colon cancer (stages I and II). Chemotherapy is the standard treatment for patients with locally advanced stage III and IV cancers after radical surgery. For some stage II colon cancer patients, systemic treatment with surgery is based on risk factors and *MSI* gene status. Commonly used drugs include capecitabine, 5-fluorouracil, irinotecan, and oxaliplatin. Biologics, including bevacizumab, cetuximab, panitumumab, regorafenib, and afatinib are important for the treatment of metastatic colon cancer. Genetic analysis of tumor patients is increasingly used role to guide the selection of treatment plans. The use of radiotherapy is currently limited to palliation of selected metastatic sites (*e.g.*, bone or brain metastases)[44,54-57].

All patients with synchronous colorectal cancer and D-NETs undergo extensive evaluation and clinical monitoring during hospitalization and follow-up to detect disease progression or recurrence. In current practice, patients are followed-up every 3-6 months in the first 3 years and every 6-12 months thereafter. Each follow-up visit includes routine laboratory tests, tumor markers (CEA, CA 19-9, CA 12-5, CgA, and 5-hydroxyindoleacetic acid), gastrointestinal endoscopy, abdominal ultrasound, lymph node ultrasound, chest radiography, and CT or PET/PET-CT scan. Octreotide CT or octreotide SPE-CT scan is also useful to detect recurrence of NETs. In clinical practice, serum CgA is the most commonly used tumor marker for NETs. It can assist in diagnosis, assessment of tumor burden, in treatment, and is helpful for follow-up[55,56,58,59]. We believe that early diagnosis, comprehensive preoperative examination, careful intraoperative exploration, radical resection and regular postoperative monitoring can increase survival time. The treatment strategy depends on many factors, such as the surgical approach, the patient's general condition, tumor grade, extent of disease, and the response to treatment. It should be analyzed in combination with the individual patient's status[60,61].

The appearance of synchronous primary tumors is of interest to surgeons and oncologists and the entire medical field. When such a phenomenon is encountered, questions invariably arise regarding common genetic pathways in the pathogenesis of these tumors. With the increasing incidence of multiple primary tumors, clinicians should be vigilant to the possibility of their occurrence. This case involved several interesting aspects of clinical care. First, few cases of synchronous D-NETs occurring with colon adenocarcinoma have been reported. Second, there are no proven diagnostic criteria, treatment approaches, or follow-up guidelines for patients with synchronous D-NETs and a second primary malignancy. Therefore, our experience may help to inform the diagnosis and management of such patients. We also highlight the advantages of HALS, which allows direct palpation of the mass and assessment of its size, texture, and mobility, as well as the surrounding lymph nodes. This assists surgeons in choosing surgical procedures. In addition, the surgical incision is small and postoperative recovery is fast. It also avoids the need for large surgical incisions, incision infections, and fat liquefaction after traditional open surgery. In the setting of synchronous primary tumors as in this patient, the impact of synchronous cancers on overall prognosis must always be considered when planning patient care. Finally, patients who present with multiple primary tumors are a unique opportunity to study the complex etiology of cancer.

CONCLUSION

There are no clear diagnostic criteria, treatment, or follow-up guidelines for patients with synchronous D-NETs and a second primary malignancy. Our experience may help to inform the diagnosis and management of similar patients. The case also highlights the advantages of HALS, which allows direct palpation of the mass and assessment of its size, texture, and mobility, and evaluation of the surrounding lymph nodes. This is of benefit to surgeons in choosing the surgical procedures. in addition, the surgical incision is small and postoperative recovery is fast. It also avoids the occurrence of large surgical incisions, incision infections, and fat liquefaction after traditional open surgery. In the setting of synchronous primary tumors as in this patient, the effect of the synchronous cancers on prognosis must always be taken into consideration when planning patient care.

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FOOTNOTES

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