REVIEW

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A NARRATIVE REVIEW STUDY SUMMARIZING AND INTERPRETING THE EXISTING LITERATURE ON THE DIAGNOSIS AND TREATMENT OF SMALL BOWEL TUMORS

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Abstract: *Small intestine cancer encompasses a group of malignant diseases originating in the duodenum, jejunum, or ileum. Although they account for only 3-5% of all gastrointestinal carcinomas, their incidence is increasing, unlike more common malignant diseases, such as colorectal carcinoma, which makes their study increasingly important.* ***Objective:*** *to analyze the main malignant histological subtypes – adenocarcinomas, neuroendocrine neoplasms, gastrointestinal stromal tumors, and lymphomas – and to summarize and systematize the available literature data.* ***Materials and Methods:*** *We applied a narrative review study design involving the summarization and interpretation of existing literature data on the given topic and objective in order to create a structured, comprehensive scientific product on malignant diseases of the small intestine. We analyzed 20 literature sources.* ***Results and Discussion:*** *The etiology of small intestine cancer is multifactorial and can be divided into well-defined hereditary cancer syndromes and acquired chronic inflammatory conditions. Malignant tumors of the small intestine are a rare but increasingly common group of diseases that are difficult to manage due to their heterogeneity. This biological heterogeneity requires highly specific diagnostic, surgical, and systemic (e.g., targeted therapy for GIST, immunotherapy for MSI-H SBA, PRRT with 177Lu-DOTATATE for NETs) management strategies. Optimal results for the treatment of these rare tumors require a specialized, multidisciplinary approach, concentrated in high-tech centers and institutions.*

Key words: *small intestine cancer, duodenum, jejunum, ileum, staging, treatment*

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INTRODUCTION

Despite the considerable length and surface area of the small intestine, malignant tumors originating in it are rare and represent a heterogeneous group of gastrointestinal neoplasms [1, 2]. Accurate histological classification is of critical clinical importance, as each subtype is characterized by different biological behavior, therapeutic approaches, and prognosis [3]. Small intestine cancer (SIC) encompasses a group of malignant diseases originating in the duodenum, jejunum, or ileum. Although they account for only 3-5% of all gastrointestinal carcinomas, their incidence has been increasing, unlike more common malignant diseases, such as colorectal carcinoma, which makes their study increasingly important [4, 5].

Despite their overall rarity (<5% of gastrointestinal carcinomas), a disturbing and significant global trend is the steadily increasing incidence of SIC [3, 5]. Understanding these trends is of strategic importance for identifying unique etiological factors that differ from those in colorectal carcinoma [5]. SICs account for approximately 3-5% of all gastrointestinal carcinomas [3]. An increasing incidence has been observed, with an annual percentage increase of 1.8% in the US between 2006 and 2015, and a 179% increase in the UK since the early 1990s [5]. This runs in stark contrast to the stable or declining rates of colorectal carcinoma [2, 5]. Data have been published showing that in 2019, there were 10,590 new cases and 1,590 deaths in the US [6]. Neuroendocrine tumors and adenocarcinomas are the two most common histologies, with similar frequencies in prevalence statistics [2, 3]. In a Thai study, adenocarcinoma was predominant at 81.0%, followed by GIST and NET at 5.7% each [7]. In terms of segmental distribution, the duodenum is the most common site (55-88%), mainly due to the high incidence of adenocarcinoma there [3, 7, 8]. In contrast, NETs are more common in the ileum [8, 9]. The average age at diagnosis is around the sixth decade (62-66 years). Cancer is slightly more common in men and, in the US, in black people [2, 6, 11]. These distinct epidemiological patterns, especially the increasing incidence and heterogeneous histological distribution, necessitate further investigation of the specific etiological drivers and predisposing conditions responsible for small intestine carcinogenesis. The etiology of SIC is multifactorial and can be divided into well-defined hereditary cancer syndromes and acquired chronic inflammatory conditions [5]. Identifying these risk factors is of paramount importance for patient monitoring, genetic counseling, and guiding therapeutic decisions, such as the use of immunotherapy in MMR-deficient tumors [5, 6].

Objective: to analyze the main malignant histological subtypes – adenocarcinomas, neuroendocrine neoplasms, gastrointestinal stromal tumors, and lymphomas – and to summarize and systematize the available literature data.

MATERIALS AND METHODS

We applied a narrative review study design involving the summarization and interpretation of existing literature data on the given topic and objective in order to create a structured, comprehensive scientific product on malignant diseases of the small intestine. We analyzed 20 literature sources in total.

RESULTS

The four main malignant histological subtypes classified according to their cellular origin are adenocarcinomas (SBA), neuroendocrine neoplasms (which include well-differentiated neuroendocrine tumors, NET, and poorly differentiated neuroendocrine carcinomas, NEC), gastrointestinal stromal tumors (GIST), and lymphomas [3, 6, 7]. **Adenocarcinoma** is one of the most common subtypes, accounting for up to 81.0% of the cases in some series. It occurs most frequently in the duodenum (55-88%), followed by the jejunum (11-25%) and ileum (7-17%) [3, 7]. **Neuroendocrine neoplasms (NET/NEC)** represent a significant proportion of SIC, although their frequency varies in different studies. Small intestine NETs (SI-NET) are most common in the il-

eum [8, 9]. It is important to distinguish between the indolent course of well-differentiated NETs and the aggressive nature of poorly differentiated NEC. **Gastrointestinal stromal tumors (GIST)** are the most common mesenchymal tumors of the small intestine [3]. Their distribution shows a higher frequency in the jejunum (22.7%) and ileum (17.6%) compared to the duodenum (0.8%) [7]. Lymphoma is a known but less common subtype [2, 3]. T-cell lymphoma associated with enteropathy (EATL) is a highly aggressive form that usually occurs in the jejunum or ileum [10].

Hereditary syndromes: Familial adenomatous polyposis (FAP) is caused by germline mutations in the *APC* gene. FAP carries a significant lifetime risk of duodenal cancer, which, according to various literature sources, ranges from 3-5% to 12%. Up to 80% of patients develop duodenal adenomas [5, 12]. Surveillance is guided by the Spigelman classification [5]. Lynch syndrome is an autosomal dominant disorder caused by germline mutations in DNA mismatch repair (MMR) genes (*MLH1, MSH2, MSH6, PMS2*) [5, 12]. It carries a lifetime risk of SBA of 1-4%, over 100 times higher than that among the general population [2, 6]. The NCCN recommends universal testing for MMR deficiency or microsatellite instability (MSI) of all SBA samples to identify these patients [6]. Peutz-Jeghers syndrome (PJS) results from an inherited mutation in the *STK11* gene. PJS carries an extremely high relative risk for SBA (RR of 520) and a lifetime risk of up to 13% [2, 5].

Inflammatory, immunological, and other acquired conditions: chronic inflammation in **Crohn's disease** is an established risk factor, with a relative risk of SBA 30-60 times higher than that among the general population [2, 13]. These carcinomas often occur in the ileum. It is concerning that the use of 6-mercaptopurine is associated with a high probability of cancer [5]. **Celiac disease** increases the risk of SBA, but more importantly, it is the main precursor of the highly aggressive T-cell lymphoma associated with enteropathy (EATL) [5]. Factors such as alcohol consumption and smoking have been studied, but their association is not as strongly established as in other gastrointestinal carcinomas [2]. Identifying these risk factors is the first step towards understanding the underlying molecular changes they induce.

The pathogenesis of SIC is highly dependent on histology. Understanding the different molecular drivers of adenocarcinoma, NET, and GIST is not only of academic value but also directly informs the use of targeted therapies and dictates current management strategies [5].

Small bowel adenocarcinoma (SBA): The pathogenesis of SBA involves a sequence from adenoma to carcinoma. There is molecular overlap with colorectal carcinoma (CRC), including defects in the Wnt pathway (*APC* mutations in 13-27% of cases) and DNA mismatch repair (MMR) deficiency (MSI-H in 7-35% of cases). However, *differences* with CRC are crucial, such as the lower frequency of *APC* mutations and the higher frequency of targeted *ERBB2* (HER2) alterations (up to 12%). These differences highlight why therapeutic paradigms cannot be blindly extrapolated from colorectal carcinoma and necessitate SBA-specific studies, positioning SBA as a distinct molecular entity. The high frequency of mutations in KRAS (up to 43%) and *p53* (41-54%) should also be mentioned.

Neuroendocrine neoplasms (NETs): Small intestine NETs (SI-NETs) are characterized by relative genomic stability and a low frequency of mutations compared to other types of cancer. Frequent deletion of chromosome 18 is a characteristic genomic signature [5, 9]. Key signaling pathways are PI3K/Akt/mTOR, which is a therapeutic target for everolimus, and TGF-β, which is involved in the characteristic fibrosis. The mechanism of fibrosis links the production of serotonin, tryptophan hydroxylase 1 (TPH1), TGF-β, and connective tissue growth factor (CTGF) [9].

Gastrointestinal stromal tumors (GIST): the pathogenesis of most GISTs is due to gain-of-function mutations in receptor tyrosine kinase genes, predominantly *KIT* or, less commonly, *PDGFRA*. These mutations lead to constant activation of the kinase, stimulating cell proliferation, and these same kinases are the main targets for tyrosine kinase inhibitor (TKI) drugs such as imatinib [5].

The main routes of metastasis in SIC include local invasion, lymphatic spread to regional nodes, peritoneal carcinomatosis, and hematogenous spread, most commonly to the liver [6]. Accurate staging is fundamental to prognosis and treatment planning, but a unified approach is not appropriate for SICs. While adenocarcinoma follows a conventional anatomical TNM system, the prognostically critical systems for NETs and GISTs are fundamentally different, incorporating biological aggressiveness and risk of recurrence [5].

Common macroscopic manifestations of SBA include polypoid, ulcerative, infiltrative, and stenosing forms, which can lead to obstruction [13]. Key microscopic features assessed by pathologists include histological grade (differentiation), lymphovascular invasion, and perineural invasion, which have prognostic sig-

nificance [6]. It is essential to achieve negative resection margins (R0) [7, 13].

The AJCC TNM 8th edition system defines the T-stage according to the depth of tumor invasion through the layers of the intestinal wall (T1-T4), the N stage according to the number of positive regional lymph nodes (N0, N1, N2), and the M stage according to the presence of distant metastases (M0, M1) [14]. For adequate staging, the minimum consensus requirement is the removal of at least eight lymph nodes during resection. Although the ENETS TNM system exists, prognosis and treatment are primarily dictated by the World Health Organization (WHO)'s histological grading system [15]. Grades are determined based on the Ki-67 proliferation index and mitotic rate (G1, G2, G3), with this system reflecting the biological aggressiveness of the tumor. GIST staging is a system for stratifying the risk of recurrence. The three key factors integrated into this assessment are the size of the primary tumor, the mitotic rate (at 50 high-power fields), and the primary location [16]. It is explicitly stated that small intestine localization carries a higher intrinsic risk of recurrence compared to stomach localization [16].

The clinical picture of SIC is notoriously insidious and non-specific, often leading to significant delays in diagnosis. Symptoms are largely dictated by the location and size of the tumor rather than its histology, with the exception of functional NETs. Vague, spastic abdominal pain is the most common symptom. As tumors grow, they can cause partial or complete small bowel obstruction, leading to nausea, vomiting, bloating, and inability to pass gas [2, 10, 17]. Bleeding may be overt (melena) or occult, leading to iron deficiency anemia [10]. Fatigue, pallor, and weakness are common secondary symptoms due to chronic anemia. Significant weight loss and night sweats may be initial manifestations, especially in aggressive subtypes such as lymphoma [10]. The location of the tumor dictates specific signs. In periapical and duodenal lesions, they can cause biliary obstruction, leading to jaundice or pancreatitis [2, 7]. Carcinoid syndrome (flushing, diarrhea, bronchial constriction) occurs when functional NETs, usually with liver metastases, secrete hormones such as serotonin into the systemic circulation [9, 15].

The diagnostic process in SIC is a multimodal challenge aimed at overcoming the limitations of conventional endoscopy and imaging. The process must not only confirm the diagnosis, but also accurately stage the disease to determine resectability and guide therapy, with specialized techniques now considered standard of care [6, 13]. **CT and MR enterography/ enteroclysis**: CT enterography (CTE) and MR enterography (MRE) are the preferred imaging methods. These techniques use neutral oral contrast to distend the small intestine, improving the visualization of mural pathology [18]. Data indicate that MRE is statistically more accurate for detecting neoplastic lesions (sensitivity 92.6%) compared to CTE (75.9%) due to better soft tissue contrast. **Imaging findings:** typical CT findings include ring-like narrowing in adenocarcinoma of the jejunum/ileum. Cross-sectional imaging can detect extraluminal disease, lymphadenopathy, and distant metastases [13, 18]. **Capsule endoscopy (VCE) and balloon-assisted enteroscopy are** non-invasive tools for visualizing the entire small intestine mucosa, making them very sensitive to small mucosal lesions, but they are limited by the inability to obtain biopsies. Balloon-assisted enteroscopy allows direct visualization and, crucially, tissue sampling (biopsy) of lesions identified by other methods. There is a risk of capsule retention in cases of stricture [13]. **Functional and molecular imaging for neuroendocrine neoplasms:** since most well-differentiated NETs express somatostatin receptors (SSTR), functional imaging is the standard of care. PET/CT with gallium-68 (68Ga-DOTATATE) has replaced older scintigraphic methods. Data show that 68Ga-DOTATATE PET/CT is significantly better than 99mTc-Octreotide SPECT/CT and conventional CT/ MRI, detecting more lesions and affected organs. This higher accuracy leads to a change in patient management in a significant number of cases (up to a quarter) [19]. The role of serum tumor markers, such as CEA and CA19-9, is limited – they may be elevated in adenocarcinoma but are not specific. In NETs, serum chromogranin A (CgA), pancreastatin, and 24-hour urinary 5-HIAA have diagnostic and monitoring value [9, 15]. The final diagnosis for all subtypes ultimately requires histological confirmation by biopsy [16].

Complete surgical resection is the only curative treatment for localized SIC. The surgical strategy is highly dependent on the histological subtype of the tumor, its anatomical location, and the presence of metastatic disease, often requiring complex procedures that are best performed in high-volume specialized centers.

Surgery for small intestine adenocarcinoma: adenocarcinomas in the second part of the duodenum or those that invade the ampulla/pancreas require formal pancreatoduodenectomy (Whipple procedure) [5, 6, 13]. Segmental resection may be an option for tumors in the first, third, and fourth parts if negative margins can be achieved. For **jejunal and ileal tumors**, the standard procedure is segmental resection with wide margins and *en bloc* regional lymphade-

nectomy. A quality indicator is the removal of at least eight lymph nodes for accurate staging.

Surgery for neuroendocrine neoplasms: Localized small intestine NETs require segmental resection with removal of the corresponding lymphatic drainage field due to their high potential for nodal metastasis even when small [15]. In **metastatic disease**, resection of the primary tumor **is recommended** even in the presence of unresectable liver metastases in order to prevent future complications, such as obstruction or ischemia. Cytoreductive surgery for liver metastases should be considered if a significant debulking threshold (e.g., 70%) can be achieved.

Surgery for gastrointestinal stromal tumors: the principle for localized GIST is complete surgical resection with negative margins. Routine lymphadenectomy is not performed, as lymphatic spread is rare in GIST [16].

Data suggest that laparoscopic surgery is associated with shorter hospital stays and less intraoperative blood loss, but it is important to note that there often is a selection bias, with this approach being used for smaller tumors. The role of cytoreductive surgery for peritoneal metastases is controversial and should be performed in experienced centers. Palliative bypass or stenting is used for unresectable obstructive tumors. Surgery is often one component of a broader, multimodal treatment strategy that requires integration with systemic therapies.

Systemic therapy for SIC is strictly histology-driven and increasingly guided by molecular profiles. The use of cytotoxic chemotherapy for adenocarcinoma contrasts with the targeted and biological therapies, which underpin the treatment of NET and GIST [5].

Small intestine adenocarcinoma: adjuvant chemotherapy is often considered for stage III and high-risk stage II, extrapolating data from colorectal carcinoma. Recommended regimens are FOLFOX, CAPEOX, or capecitabine/5-FU. The benefit for stage II tumors with normal MMR is unclear. In unresectable or metastatic disease, fluoropyrimidine and platinum-based combinations (FOLFOX or CAPEOX) are standard first-line therapy, with response rates ranging from 41% to 50% reported in key studies. FOLFIRI is a reasonable second-line option [2, 6]. Targeted therapy and immunotherapy are also important. For the subgroup of SBA tumors that are MMR/MSI-High- -deficient, immune checkpoint inhibitors, such as pembrolizumab or nivolumab, are an important option for subsequent lines of therapy. *ERBB2* (HER2) is a potential but less common target.

Neuroendocrine neoplasms: Somatostatin analogues (SSA): SSA (octreotide, lanreotide) are first-line therapy for advanced, well-differentiated SI-NET. The PROMID and CLARINET studies demonstrate their antiproliferative effect and ability to control the symptoms of carcinoid syndrome [15, 20]. Peptide receptor radionuclide therapy (PRRT): in SSTR-positive tumors progressing on SSA, PRRT with 177Lu-DOTATATE is a standard second-line option, with the NETTER-1 study showing a significant benefit in progression-free survival [15, 20]. Targeted therapy: Everolimus (mTOR inhibitor) is a key targeted agent, with the RADIANT-4 study demonstrating its efficacy in non-functioning GI NETs. Chemotherapy for NEC: In poorly differentiated neuroendocrine carcinomas (NEC), treatment is based on protocols for small-cell lung cancer, with platinum-based chemotherapy (etoposide + cisplatin/carboplatin) as the standard of care.

Gastrointestinal stromal tumors: Targeted therapy (TKI): the treatment of GIST is a paradigm for molecular targeted therapy. Imatinib is the standard first-line TKI for metastatic or high-risk resected GIST. If resistance develops, other TKIs, such as sunitinib (second line) and regorafenib (third line), are used sequentially. Molecular testing for *KIT/PDGFRA* mutations prior to treatment is mandatory for therapy guidance [5].

Radiotherapy has a very limited role, but may be considered in selected situations in duodenal carcinomas (especially in resections with positive margins) or for palliation of symptomatic metastases.

Palliative care is not an alternative to active treatment, but an integrated component of care for patients with advanced or incurable SIC. Its dual goal is to manage local complications caused by the tumor and to relieve systemic symptoms to maintain quality of life. Palliative surgery is considered a surgical bypass or stoma creation to manage unresectable malignant intestinal obstruction. Endoscopic stenting is a less invasive option for duodenal obstruction [6]. Systemic therapies (chemotherapy for SBA, SSA for NET, TKI for GIST) are used palliatively in a metastatic setting to control the disease and symptoms [15]. Radiotherapy can be used to relieve symptoms from painful bone metastases or other localized sites of disease.

The importance of comprehensive symptom management, including pain control, antiemetics, and nutritional support to address malabsorption and weight loss, is emphasized [11]. In NET, the use of telotristat etiparat for refractory diarrhea in carcinoid syndrome is specifically mentioned [5].

DISCUSSION

The prognosis for SICs is highly variable and is determined primarily by tumor histology and stage at diagnosis. Understanding prognostic factors is of strategic importance for risk stratification, patient counseling, and the development of appropriate follow-up regimens [6, 13]. Key negative prognostic factors: the presence of lymph node involvement (stage III) and distant metastases (stage IV) are the strongest negative predictors of survival [7, 13]; Poorly differentiated histology (high grade) is associated with worse outcomes [7, 3]. The prognosis for NEC and EATL is particularly poor; a positive surgical margin (R1 resection) is a significant negative prognostic factor; duodenal localization is independently associated with poorer overall survival in adenocarcinoma [7]; poor ECOG performance status (≥2) is an independent factor for poorer survival; specific molecular characteristics, such as *p53* mutation in SBA, may be associated with poor prognosis. Approximate 5-year overall survival (OS) rates for SBA by stage, based on larger registries, are as follows: I – 57-66%; II – 43-50%; III – 31-42%; IV – 5-19%. These data contrast with results from other histologies and cohorts. For example, in a Thai study, the 5-year OS for GIST was 55.6% and for well-differentiated NET – 88.9%, while for adenocarcinoma it was only 14.0% and for NEC – 0%. The significantly lower survival rate for adenocarcinoma in this study may reflect differences in the patient population or a higher proportion of advanced diseases at diagnosis in the specific cohort. The goals of post-treatment follow-up are to detect disease recurrence, manage long-term complications of treatment, and screen for new metachronous neoplasms. Based on NCCN recommendations, the typical follow-up regimen for resected SBA includes medical history and physical examination, tumor markers (CEA/CA19-9, if initially elevated), and chest, abdominal, and pelvic CT scans at regular intervals (e.g., every 3-12 months initially, then less frequently) for up to 10 years. Long-term assessment and management of nutrition is important, as well as potential complications, such as short bowel syndrome or malabsorption [11]. Regular follow-up is an essential part of the overall management strategy for these complex diseases.

Conflict of interest: *The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.*

Ethical statements: *The authors declare that no clinical trials were used in the present study.*

The authors declare that no experiments on humans or human tissues were performed for the present study.

The authors declare that no informed consent was obtained from humans, the donors, or donors' representatives participating in the study.

The authors declare that no experiments on animals were performed for the present study.

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REFERENCES

1. Panarelli N, Yantiss R. Small Bowel Neoplasms. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan.
2. Chen EY, Vaccaro GM. Small Bowel Adenocarcinoma. Thieme Med Publ. 2018;31(5):267–277.
3. GI Oncology Expert Group. Malignant Tumours of the Small Intestine: An Evidence Mapping for Advanced Surgical and Oncological Practice. J Clin Oncol Rev. 2023;5(2):110-125.
4. American Cancer Society. Key Statistics for Small Intestine Cancer. Atlanta, GA: American Cancer Society; 2019.
5. GI Oncology Expert Group. Malignant Tumours of the Small Intestine: An Evidence Mapping for Advanced Surgical and Oncological Practice. J Clin Oncol Rev. 2023;5(2):110-125.
6. Benson AB 3rd, Venook AP, Al-Hawary MM, et al. Small Bowel Adenocarcinoma, Version 1.2020, NCCN Clinical Practice Guidelines in Oncology. J Natl Compr Canc Netw. 2019;17(9):1109-1133.
7. Wonglhow J, Sunpaweravong P, Sathitruangsak C, et al. A 20-Year Real-World Study of Small Bowel Cancers: Histologic Subtypes, Clinical Features, and Survival Implications. J Clin Med. 2025;14(19):6962.
8. Halfdanarson TR, McWilliams RR, Donohue JH, Quevedo JF. A single-institution experience with 491 cases of small bowel adenocarcinoma. Am J Surg. 2010;199(6):797-803.
9. Chen YX, Fang JY. Current Perspective on the Pathogenesis of Small Intestinal Neuroendocrine Tumors: Progress in Biomarkers and Molecular Events. Gastroenterol Res Pract. 2013;1(1):2–8.
10. Makruasi N, Chinthammitr Y, Rattanathammethee T, et al. Enteropathy Type II Associated T-Cell Non-Hodgkin Lymphoma: A Case Report. J Hematol. 2015;4(1):144-147.
11. Khosla D, Dey T, Madan R, et al. Small bowel adenocarcinoma: An overview. World J Gastrointest Oncol. 2022;14(2):413-422.

12. Samadder NJ, Jasperson K, Burt RW. Hereditary cancer syndromes – A primer on diagnosis and management, Part 2: Gastrointestinal cancer syndromes. Mayo Clin Proc. 2019;94(6):1099-1115.
13. Greenbaum A, Sror M, Beglaibter N, et al. Risk factors for small bowel cancer in Crohn's disease. Gut. 1993;34(10):1387-1390.
14. American Cancer Society. Small Intestine Cancer (Adenocarcinoma) Stages. Atlanta, GA: American Cancer Society; 2018.
15. Larouche V, Akirov A, Alsherhri S, Ezzat S. Management of Small Bowel Neuroendocrine Tumors. Cancers (Basel). 2019;11(9):1395.
16. American Cancer Society. Gastrointestinal Stromal Tumor Stages and Other Prognostic Factors. Atlanta, GA: American Cancer Society; 2019.
17. Ojha A, Zacherl J, Scheuba C, et al. Primary small bowel malignancies: single-center results of three decades. J Clin Gastroenterol. 2000; 30:289–293.
18. Ilangovan R, Burling D, George A, Gupta A, Marshall M, Taylor SA. CT enterography: review of technique and practical tips. Br J Radiol. 2012;85(1015):876-886.
19. Fallahi B, Beiki D, Emami-Ardekani A, et al. Diagnostic Efficiency of 68Ga-DOTATATE PET/CT as Compared to 99mTc-Octreotide SPECT/CT and Conventional Morphologic Modalities in Neuroendocrine Tumors. Asia Ocean J Nucl Med Biol. 2019;7(2):129-140.
20. Lamarca A, Cives M, Spada F, et al. Advanced small-bowel well-differentiated neuroendocrine tumours: An international survey of practice on 3rd-line treatment. World J Gastroenterol. 2021;27(10):976-989.