

When Two Silent Killers Collide: Profound Anemia from Myelodysplastic Syndrome and Occult Small-Bowel Bleeding in a Patient with Carcinoid Tumor

Khiem Phan*, Rabia Naheed, Colin Sauter, Alexandra Young, Abdallah Izmegna, Tasha McAdams, Ismail Ganim, and David E Martin

Unity Health White County Medical Center, Searcy, Arkansas, USA

*Corresponding author: Khiem Phan, MD, Department of Medicine, Unity Health- White County Medical Center, 3214 E. Race Ave, Searcy, Arkansas 72143 USA, Tel: +1- 225-239-0169; E-mail: kvphan10@gmail.com

Received: September 10, 2026; Accepted: October 05, 2026; Published: October 15, 2026

Abstract

Background: Myelodysplastic syndrome (MDS) commonly causes chronic anemia through ineffective hematopoiesis, while gastrointestinal bleeding from small-bowel neuroendocrine tumors (NETs) is uncommon and may evade conventional endoscopic evaluation.

Case Presentation: An 85-year-old man with MDS, small-bowel carcinoid tumor, atrial fibrillation on apixaban, and systolic heart failure presented with dizziness, dyspnea, and weakness. Hemoglobin was 4.1 g/dL, accompanied by hypotension, atrial fibrillation with rapid ventricular response, melena, and positive fecal occult blood testing. Troponin increased from 2,865 to >50,000 ng/L without chest pain, consistent with severe supply-demand myocardial injury from profound anemia, hypotension, and tachyarrhythmia. Apixaban was withheld, and packed red blood cell transfusions increased hemoglobin to 9.1 g/dL. Esophagogastroduodenoscopy revealed no bleeding source. Given persistent suspicion for small-bowel hemorrhage and the known carcinoid tumor, mesenteric angiography was performed with selective transcatheter arterial embolization. Hemoglobin remained stable without recurrent overt bleeding.

Conclusion: Worsening anemia in patients with MDS should not automatically be attributed to ineffective hematopoiesis. Concurrent gastrointestinal blood loss, including bleeding associated with small-bowel NETs, should be investigated when clinical findings suggest an additional source.

Keywords: *Myelodysplastic syndrome; Neuroendocrine tumor; Carcinoid tumor; Gastrointestinal bleeding; Acute anemia; Transcatheter arterial embolization*

1. Introduction

Small-intestinal neuroendocrine tumors (SI-NETs), historically called small-bowel carcinoid tumors, are uncommon gastrointestinal neoplasms with an increasing incidence over recent decades [1]. The ileum is the most frequent small-bowel primary site [2]. Although SI-NETs often follow an indolent course, they may cause abdominal pain, obstruction, mesenteric ischemia, weight loss, malabsorption, and hormone-mediated symptoms. Mesenteric tumor deposits may provoke a marked desmoplastic reaction, leading to vascular encasement, venous congestion, ischemia, and chronic intestinal complications. Overt gastrointestinal hemorrhage is less common but has been previously noted [3,4].

Diagnosis is challenging because routine esophagogastroduodenoscopy (EGD) and colonoscopy do not visualize most of the jejunum and ileum [5,6]. Small-bowel bleeding accounts for approximately 5%-10% of gastrointestinal bleeding and should be considered after an appropriate upper and lower endoscopic evaluation fails to identify a source [7,8]. Diagnostic difficulty is further amplified when another established cause of anemia is present [9]. MDS comprises clonal hematopoietic neoplasms characterized by ineffective hematopoiesis and peripheral cytopenias [10]. Anemia predominates in lower-risk disease and may produce chronic transfusion dependence [11]. Consequently, a sudden decline in hemoglobin may be incorrectly attributed to marrow failure alone [12].

We report a patient with MDS and a known stable SI-NET who developed profound acute-on-chronic anemia, melena, hemodynamic instability, atrial fibrillation with rapid ventricular response, and marked acute myocardial injury. Negative EGD prompted evaluation of the small bowel and selective transcatheter arterial embolization (TAE) [13,14]. This case illustrates how an established chronic cause of anemia does not exclude a second, potentially treatable source of blood loss.

2. Case Presentation

An 85-year-old man with myelodysplastic syndrome (MDS) and chronic anemia, a known small-intestinal neuroendocrine tumor (SI-NET), atrial fibrillation on apixaban, and heart failure with reduced ejection fraction (LVEF 30%-35%) presented with several days of progressive weakness, dizziness, and dyspnea. Although his MDS and SI-NET had recently been considered clinically stable, he had experienced approximately 50 lbs of unintentional weight loss and required three red blood cell transfusions during the preceding year.

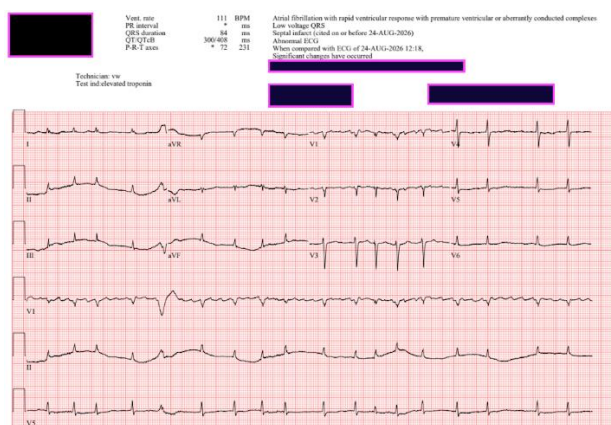


FIG. 1. ECG on admission shows atrial fibrillation (Black boxes to cover personal information).

On presentation, he was hypotensive (93/45 mmHg) and tachycardic (125 beats/min) with atrial fibrillation and rapid ventricular response. Hemoglobin was 4.1 g/dL, hematocrit 13.1%, platelet count $251 \times 10^3/\mu\text{L}$, lactate 2.5 mmol/L, and troponin 2,865 ng/L. Fecal occult blood testing was positive, and melena was observed, suggesting acute gastrointestinal hemorrhage superimposed on chronic MDS-associated anemia.

TABLE 1. Admission laboratory findings.

Laboratory test	Admission value
Hemoglobin	4.1 g/dL
Hematocrit	13.1%
Platelet count	$251 \times 10^3/\mu\text{L}$
INR	1.3
PT	14.1 s
aPTT	33.1 s
Total bilirubin	1.5 mg/dL
Troponin	2,865 ng/L
BNP	916 pg/mL
Lactate	2.5 mmol/L

He received intravenous crystalloid and four units of packed red blood cells, repeated lab 9 hours after showed increasing hemoglobin to 9.1 g/dL. Apixaban was withheld because of suspected major gastrointestinal bleeding. Troponin subsequently increased to >50,000 ng/L without chest pain or other ischemic symptoms. Cardiology attributed the marked myocardial injury primarily to severe oxygen supply-demand mismatch from profound anemia, hypotension, and persistent tachyarrhythmia. Echocardiography demonstrated an unchanged LVEF of 30%-35%, and an amiodarone infusion was initiated for atrial fibrillation.

TABLE 2. Troponin trend overnight.

Ferritin (22.0-275.0 ng/mL)	623.9 H					993.0 H			
Total Bilirubin (0.1-1.3 mg/dL)	1.1		1.5 H			2.0 H			
AST (5-34 U/L)	17		38 H			131 H			
ALT (11-55 U/L)	<4 L		5 L			11			
Alkaline Phosphatase (38-126 U/L)	87		63			65			
Lactate Dehydrogenase (125-243 U/L)						421 H			
Troponin I Hi Sens Ba... (0.0-35.0 ng/L)			2865.8 H*			>50000.0 H *			
Troponin I Hi Sens 2 Hr (0.0-35.0 ng/L)					7404.5 H*			>50000.0 H *	
B-Natriuretic Peptide (0-135 pg/mL)			916 H						
Total Protein (6.3-8.2 g/dL)	7.3		5.8 L			5.8 L			
Albumin (3.2-5.0 g/dL)	3.8		3.1 L			3.1 L			
Lipase (8-78 U/L)			23						
Vitamin B12 (213.0-816.0 pg/mL)	1388.0 H		1431.0 H						
Folate (7.0-31.4 ng/mL)	15.3		12.1						

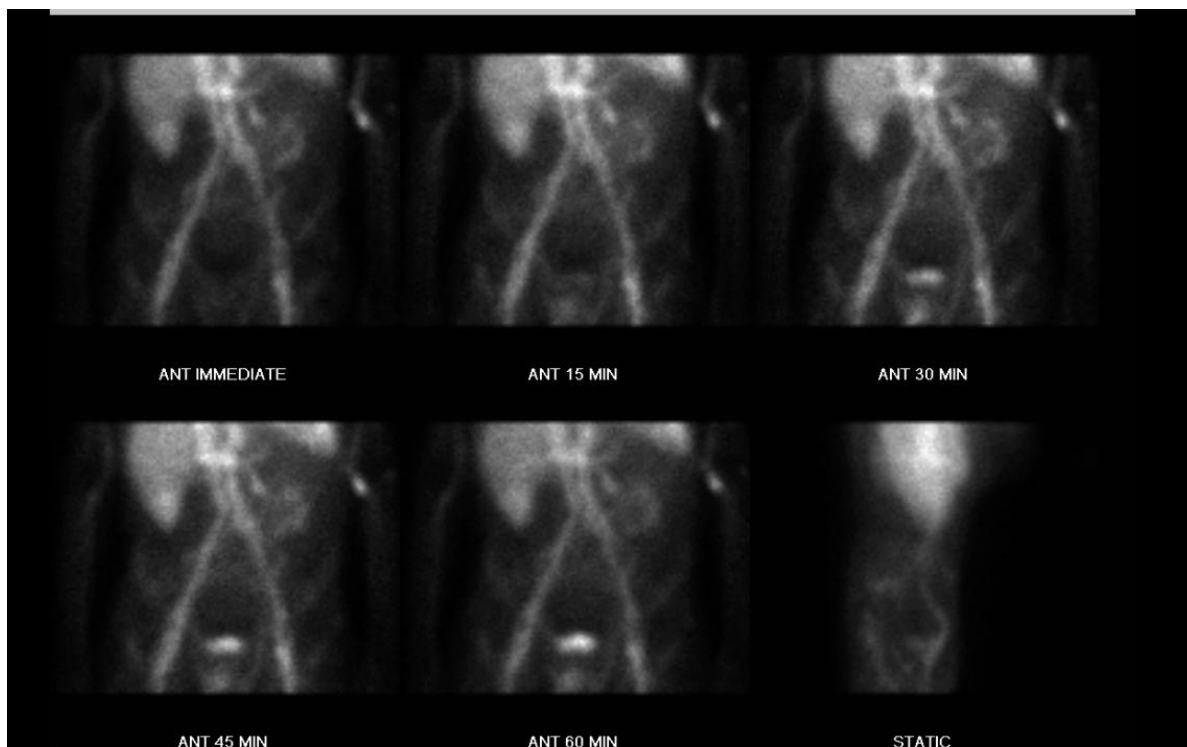


FIG. 2. Radiotracer accumulation within the left hemiabdomen suggestive of gastrointestinal hemorrhage.

Esophagogastroduodenoscopy revealed no upper gastrointestinal bleeding source, raising suspicion for small-bowel hemorrhage given the known SI-NET. The patient was transferred for interventional radiology evaluation and underwent mesenteric angiography with selective transarterial embolization (TAE) of the suspected bleeding territory. Hemoglobin remained approximately 9 g/dL over the following two days without clinically significant recurrent bleeding, and he was subsequently discharged with close outpatient follow-up.

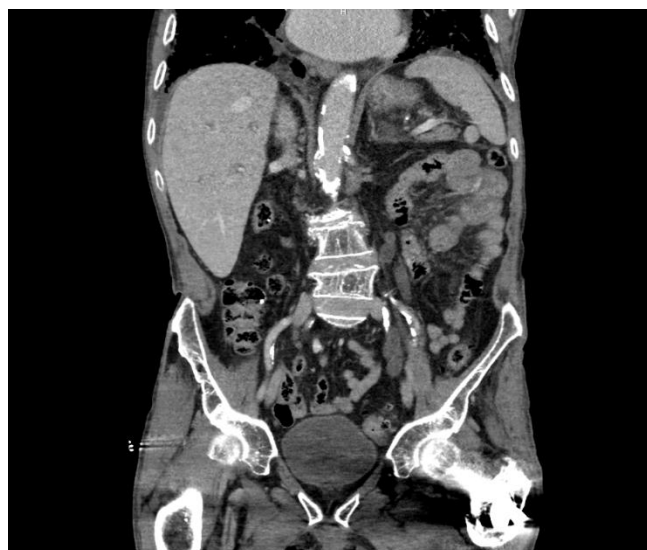


FIG. 3. Abdominal/Pelvic CT shows diverticulosis of the sigmoid colon without diverticulitis, finding compatible with small bowel carcinoid.

Although the clinical course supported a small-bowel bleeding source, available procedural documentation did not demonstrate direct tumor-associated extravasation, tumor blush, or pathologic confirmation. Therefore, the SI-NET remained a suspected rather than definitively proven source of hemorrhage.

3. Discussion

The central diagnostic challenge was the coexistence of two plausible mechanisms of anemia. MDS produces chronic anemia through ineffective erythropoiesis, abnormal hematopoietic differentiation, intramedullary cell death, and progressive marrow failure. In older adults, anemia commonly becomes the dominant manifestation of lower-risk MDS and may lead to repeated transfusion [15]. This history made marrow failure an intuitive explanation for the patient's recurrent anemia [15].

The acute phenotype, however, was not adequately explained by chronic ineffective erythropoiesis alone [16]. Hemoglobin of 4.1 g/dL accompanied by melena, positive fecal occult blood testing, hypotension, and tachycardia indicated a superimposed process until proven otherwise [17]. The more useful model was additive: reduced red blood cell production from MDS, increased red blood cell loss from suspected small-bowel hemorrhage, and amplification of bleeding due to apixaban use [18]. His recurrent transfusions during the preceding year may likewise have reflected variable contributions from both marrow dysfunction and occult blood loss.

SI-NETs most often present with abdominal pain, obstruction, mesenteric fibrosis, ischemia, weight loss, metastatic disease, or carcinoid syndrome [19]. Bleeding is less characteristic but is well documented [18]. Proposed mechanisms include mucosal ulceration over the tumor, vascular erosion, tumor-associated neovascularity, venous congestion, local ischemia, and vascular compromise caused by mesenteric desmoplasia [20,21]. A lesion appearing radiographically stable may therefore remain clinically consequential [22]. Size stability does not rule out mucosal injury, vascular involvement, or intermittent hemorrhage [22].

After nondiagnostic EGD and appropriate lower gastrointestinal assessment, available small-bowel modalities include video capsule endoscopy, device-assisted enteroscopy, CT enterography, computed tomography angiography, nuclear medicine studies, and catheter angiography [7,23-25]. Selection depends on hemodynamic stability, bleeding activity, suspected pathology, local expertise, and whether immediate therapy is required [26]. In a stable patient, capsule endoscopy is often an early test [27,28]. In severe or ongoing bleeding, catheter angiography offers the advantage of proceeding directly to embolization [13,14, 27,28].

An oxygen saturation of 99% was falsely reassuring regarding systemic oxygen delivery. Arterial oxygen content is approximated by $\text{CaO}_2 = (1.34 \times \text{hemoglobin} \times \text{arterial oxygen saturation}) + (0.003 \times \text{arterial oxygen tension})$ [29]. Because hemoglobin is the principal determinant, a normal saturation cannot compensate for a hemoglobin concentration of 4.1 g/dL [29]. Tachycardia increased myocardial oxygen demand, while hypotension reduced coronary perfusion [30,31]. Pre-existing systolic heart failure limited the patient's ability to augment cardiac output [30,31].

Type 2 myocardial infarction requires acute ischemic myocardial injury caused by an oxygen supply-demand imbalance unrelated to acute coronary atherothrombosis; recognized triggers include severe anemia, hypotension, hypoxemia, and

sustained tachyarrhythmia [30,31]. Troponin magnitude alone cannot identify the mechanism [30]. Symptoms, electrocardiographic findings, imaging, hemodynamics, and the overall context remain essential [31]. Because the supplied record did not establish objective ischemia or coronary thrombosis, marked acute myocardial injury attributed clinically to supply-demand mismatch is the most defensible description [30,31].

Apixaban does not create a structural gastrointestinal lesion, but it can increase the severity or persistence of hemorrhage from an existing lesion [32]. PT, INR, and aPTT are not reliable measures of clinically relevant apixaban activity; an INR of 1.3 therefore did not exclude an anticoagulant contribution [33]. Temporary interruption was appropriate during evaluation of suspected major bleeding [34]. The timing of resumption should be individualized according to hemostasis, the likelihood of recurrent bleeding, and thromboembolic risk from atrial fibrillation [34].

TAE is an established minimally invasive treatment for gastrointestinal hemorrhage that cannot be localized or controlled endoscopically [27,28,35]. Superselective catheterization can achieve hemostasis while limiting non-target embolization and bowel ischemia [36]. Contemporary studies report high technical success, although clinical success is lower because recurrent bleeding and ischemic complications remain possible. For this 85-year-old patient with HFrEF, COPD, atrial fibrillation, MDS, malignancy, and severe myocardial injury, TAE offered an important alternative to exploratory surgery.

4. Conclusion

This case highlights the importance of avoiding diagnostic anchoring in patients with chronic anemia. Despite known MDS, profound anemia (hemoglobin 4.1 g/dL) with melena, positive fecal occult blood testing, hypotension, and tachyarrhythmia strongly suggested superimposed gastrointestinal hemorrhage. A negative EGD and known SI-NET raised suspicion for a small-bowel source, although direct tumor bleeding was not definitively established; mesenteric arterial embolization was followed by early hemoglobin stability. The resulting severe reduction in oxygen-carrying capacity, compounded by hypotension, atrial fibrillation with rapid ventricular response, and limited cardiac reserve, likely contributed to marked acute myocardial injury despite preserved oxygen saturation and absence of chest pain. This case emphasizes the need to search for a treatable “second hit” when anemia is disproportionate to an established chronic diagnosis.

5. Contributors

All authors contributed to the conception, literature review, manuscript preparation, critical revision, and final approval of the manuscript. All authors accept responsibility for the integrity and accuracy of the work.

6. Competing Interests

The authors declare no competing interests.

7. Funding

No external funding was received for this study.

REFERENCES

1. Xavier S, Rosa B, Cotter J. Small bowel neuroendocrine tumors: From pathophysiology to clinical approach. *World J Gastrointest Pathophysiol.* 2016;7(1):117-24.
2. Scherübl H, Jensen RT, Cadiot G, et al. *World J Gastrointest Endosc.* 2010;2(10):325-34.
3. Park A, Martin A, Carlos R, et al. Primary versus secondary nature of mesenteric neuroendocrine tumours. *BMJ Case Rep.* 2021;14(2):e239217.
4. Swafford EP, Magge DR. Acute mesenteric ischemia secondary to metastatic neuroendocrine tumor: a case analysis and review of the literature. *J Surg Case Rep.* 2024;2024(11):rjae725.
5. Dsouza R, Collier SA. Small Bowel Bleeding. [Updated 2026 Jun 13]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026.
6. Kim SE, Kim HJ, Koh M, et al. A practical approach for small bowel bleeding. *Clin Endosc.* 2023;56(3):283-9.
7. Gerson LB, Fidler JL, Cave DR, et al. ACG Clinical Guideline: Diagnosis and Management of Small Bowel Bleeding. *Am J Gastroenterol.* 2015;110(9):1265-87.
8. Murphy B, Winter DC, Kavanagh DO. Small Bowel Gastrointestinal Bleeding Diagnosis and Management-A Narrative Review. *Front Surg.* 2019;6:25.
9. Garcia-Casal MN, Dary O, Jefferds ME, et al. Diagnosing anemia: Challenges selecting methods, addressing underlying causes, and implementing actions at the public health level. *Ann N Y Acad Sci.* 2023;1524(1):37-50.
10. Dotson JL, Lebowicz Y. Myelodysplastic Syndrome. [Updated 2022 Jul 18]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026.
11. Germing U, Oliva EN, Hiwase D, et al. Treatment of Anemia in Transfusion-Dependent and Non-Transfusion-Dependent Lower-Risk MDS: Current and Emerging Strategies. *Hemasphere.* 2019;3(6):e314.
12. Killeen RB, Kaur A, Afzal M. Acute Anemia. [Updated 2025 Feb 26]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026.
13. Kim YS, Kwon JH, Han K, et al. Superselective transcatheter arterial embolization for acute small bowel bleeding: clinical outcomes and prognostic factors for ischemic complications. *Acta Radiol.* 2021;62(5):574-83.
14. Verhalleman Q, Miserez M, Laenen A, et al. Transcatheter arterial embolization for small-bowel bleeding: technical and clinical outcomes and risk factors for early recurrent bleeding. *Ann Gastroenterol.* 2024;37(5):559-66.
15. Patnaik MM, Santini V. Targeting ineffective hematopoiesis in myelodysplastic syndromes. *Am J Hematol.* 2022;97(2):171-3
16. Gupta R, Musallam KM, Taher AT, et al. Ineffective Erythropoiesis: Anemia and Iron Overload. *Hematol Oncol Clin North Am.* 2018;32(2):213-21.
17. Rockey DC. Occult gastrointestinal bleeding. *Gastroenterol Clin North Am.* 2005;34(4):699-718.
18. Sorigue M, Nieto J, Santos-Gomez M, et al. Indications and use of, and incidence of major bleeding with, antithrombotic agents in myelodysplastic syndrome. *Leuk Res.* 2018;73:24-8.
19. Scott AT, Howe JR. Management of Small Bowel Neuroendocrine Tumors. *Surg Oncol Clin N Am.* 2020;29(2):223-41.
20. Huang K, Stuart H, Lyapichev K, et al. Mesenteric desmoid tumour presenting with recurrent abdominal abscess and duodenal fistula: A case report and review of literature. *Int J Surg Case Rep.* 2017;37:119-23.

21. Furuya M, Nishiyama M, Kasuya Y, et al. Pathophysiology of tumor neovascularization. *Vasc Health Risk Manag.* 2005;1(4):277-90.
22. Maroun J, Kocha W, Kvols L, et al. Guidelines for the diagnosis and management of carcinoid tumours. Part 1: the gastrointestinal tract. A statement from a Canadian National Carcinoid Expert Group. *Curr Oncol.* 2006;13(2):67-76.
23. Scarpa M, Prando D, Pozza A, et al. A systematic review of diagnostic procedures to detect midgut neuroendocrine tumors. *J Surg Oncol.* 2010;102(7):877-88.
24. Mallat J, Vallet B. Ratio of venous-to-arterial PCO₂ to arteriovenous oxygen content difference during regional ischemic or hypoxic hypoxia. *Sci Rep.* 2021;11(1):10172.
25. Martin AC, Benamouzig R, Gouin-Thibault I, et al. Management of Gastrointestinal Bleeding and Resumption of Oral Anticoagulant Therapy in Patients with Atrial Fibrillation: A Multidisciplinary Discussion. *Am J Cardiovasc Drugs.* 2023;23(4):407-18.
26. Ersoy O, Sivri B, Bayraktar Y. How helpful is capsule endoscopy to surgeons? *World J Gastroenterol.* 2007;13(27):3671-6.
27. Fantasia S, Cortegoso Valdivia P, Kayali S, Koulaouzidis G, Pennazio M, Koulaouzidis A. The Role of Capsule Endoscopy in the Diagnosis and Management of Small Bowel Tumors: A Narrative Review. *Cancers (Basel).* 2024;16(2):262.
28. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth Universal Definition of Myocardial Infarction (2018). *J Am Coll Cardiol.* 2018;72(18):2231-64.
29. Boyette LC, Manna B. Physiology, Myocardial Oxygen Demand. [Updated 2023 Jul 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026.
30. Kosaraju A, Pendela VS, Hai O. Cardiogenic Shock. [Updated 2023 Apr 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026.
31. Zappulla P, Calvi V. Gastrointestinal Bleeding and Direct Oral Anticoagulants among Patients with Atrial Fibrillation: Risk, Prevention, Management, and Quality of Life. *TH Open.* 2021;5(2):e200-e210.
32. Cuker A, Siegal DM, Crowther MA, et al. Laboratory measurement of the anticoagulant activity of the non-vitamin K oral anticoagulants. *J Am Coll Cardiol.* 2014;64(11):1128-39.
33. Chu G, Seelig J, Cannegieter SC, et al. Thromboembolic and bleeding complications during interruptions and after discontinuation of anticoagulant treatment in patients with atrial fibrillation and active cancer: A daily practice evaluation. *Thromb Res.* 2023;230:98-104.
34. Park S, Jeong B, Shin JH, et al. Transarterial embolisation for gastroduodenal bleeding following endoscopic resection. *Br J Radiol.* 2021;94(1122):20210062.
35. Funaki B. Superselective embolization of colonic bleeding. *Semin Intervent Radiol.* 2005;22(2):139-40.
36. Lee SM, Jeong SY, Shin JH, et al. Transcatheter arterial embolization for gastrointestinal bleeding related to pancreatic adenocarcinoma: clinical efficacy and predictors of clinical outcome. *Eur J Radiol.* 2020;123:108787.