Journal of Inflammation Research

Dovepress
open access to scientific and medical research

Open Access Full Text Article

CASE REPORT

Unexpected Foreign Body-Induced Small Bowel adenocarcinoma: A Case Report

Yu Zhang[1], Hong-Hai Li[1], Min-Feng Ye[1], Wei-Wei Li[2], Chu Zhang[3], Hai-Feng Wang[1]

[1]Department of Gastrointestinal Surgery, Shaoxing People's Hospital, Shaoxing, Zhejiang Province, 312000, People's Republic of China; [2]Department of Gastroenterology, Shaoxing People's Hospital, Shaoxing, Zhejiang Province, 312000, People's Republic of China; [3]Department of Thoracic Surgery, Shaoxing People's Hospital, Shaoxing, Zhejiang Province, 312000, People's Republic of China

Correspondence: Hai-Feng Wang, Department of Gastrointestinal Surgery, Shaoxing People's Hospital, No. 568 Zhongxing North Road, Shaoxing, Zhejiang Province, 312000, People's Republic of China, Email drwanghaifeng01@163.com

Background: Foreign body-induced cancer is a traditional way of understanding cancer development. The induction of cancers by exogenous foreign bodies has been identified in many organs. However, small bowel adenocarcinoma induced by foreign bodies has not been reported in the literature, although the incidence of small bowel adenocarcinoma is increasing globally.

Case Presentation: A 70-year-old man was hospitalized for persistent right-sided abdominal pain for 3 months. Abdominal computed tomography revealed localized thickening and clustering of the small bowel wall in the right abdominal cavity. A comminuted fracture of the right 11th rib protruding into the abdominal cavity was observed, with a bone fragment located within the intestinal mass. Exploratory laparotomy was performed, and extensive adhesions were noted among the greater omentum, small bowel, mesentery, and right abdominal wall. Radical resection and lymph node dissection of the affected small bowel and appendix were performed. We also excised the rib end and repaired the abdominal wall to prevent further irritation. The patient was discharged 12 days post-surgery and follow-up assessments revealed no reported discomfort.

Conclusion: We first report a case of small bowel adenocarcinoma induced by self-bone tissue, along with successful radical tumor excision and thorough foreign body removal. This case highlights the significant role of chronic inflammation in carcinogenesis.

Keywords: foreign body, small bowel adenocarcinoma, chronic inflammation, rib fracture, case report

Introduction

Since Virchow's seminal work in 1863 linking inflammation to cancer, the concept that many cancers originate from infection, chronic irritation, and inflammatory sites has gained prominence.[1] Research indicates that over 15% of cancers are caused by inflammation or are inflammation-related. Foreign bodies inadvertently introduced into the body or for medical purposes have been confirmed to be carcinogenic through sustained chronic inflammatory reactions.[2]

Small bowel adenocarcinoma (SBA) is a rare fatal disease that has increased in incidence in recent years due to improvements in diagnostic tools. However, there are still no reports about foreign body-induced SBA. Here, we report a case in which a patient experienced occult rib fracture with penetration into the abdominal cavity. The absence of conspicuous abdominal symptoms has led to inadequate attention from both patients and medical professionals for many years. The patient underwent surgical treatment and postoperative pathology was confirmed to be SBA. We believe this is the first case of foreign body induced SBA after a retrospective review of the patient's medical history.

Case Report

A 70-year-old man was hospitalized for persistent right-sided abdominal pain for 3 months. The patient had no accompanying symptoms such as vomiting, diarrhea, or fever. He had previously self-administered antispasmodic drugs and probiotics for enteritis but was not significantly relieved. Additionally, he had a history of heavy alcohol consumption and smoking for more than 30 years, had quit smoking 2 years prior, and continued drinking alcohol. Laboratory tests revealed a hemoglobin level of 107 (reference: 130–175 g/L) and a C-reactive protein level of 16.71 (reference: 0.00–7.00 mg/L), and no tumor markers or T-cell tests

Received: 11 May 2024
Accepted: 5 September 2024
Published: 24 September 2024

for tuberculosis revealed abnormal elevation. Abdominal computed tomography (CT) revealed localized thickening and clustering of the small bowel (SB) wall in the right abdominal cavity. A comminuted fracture of the right 11th rib protruding into the abdominal cavity was observed, with a bone fragment located within the intestinal mass (Figure 1). Further enteroscopy was unable to reach the lesion in the SB due to adhesions and torsion. We conducted a retrospective review of the patient's medical history and reported that the patient had been hospitalized 10 years prior owing to kidney and ureteral stones. During that hospitalization, an abdominal CT revealed a preexisting rib fracture accompanied by symptoms of intestinal obstruction. However, rib fracture was overlooked because of a lack of history of trauma. With subsequent internal medical intervention, the symptoms of intestinal obstruction quickly resolved, and no recurrence was observed. The patient's treatment subsequently focused primarily on managing urinary tract stones. Thus, prolonged stimulation of intra-abdominal bone tissue against the

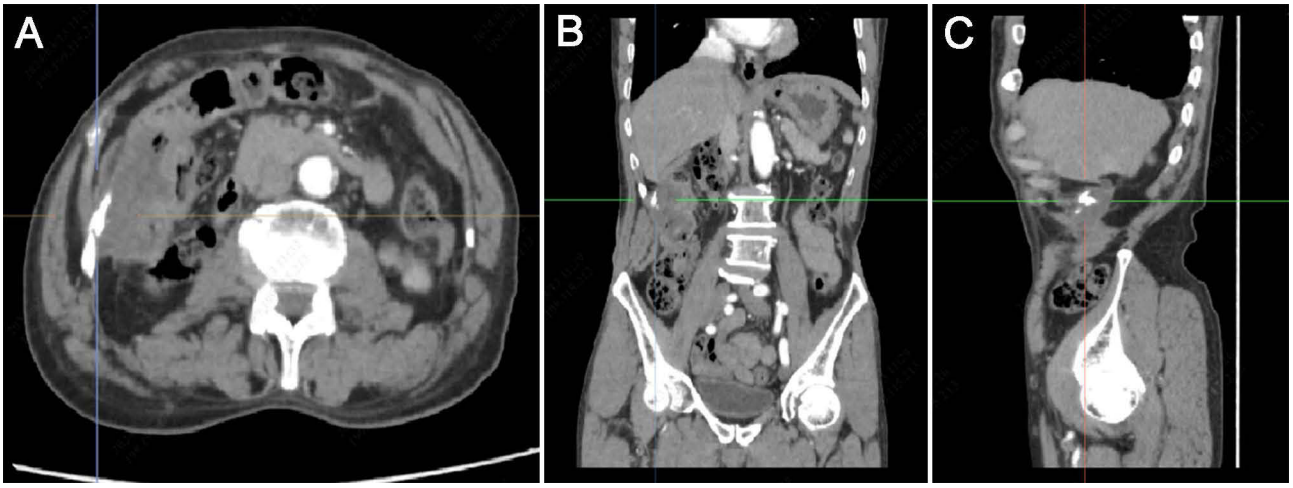

Figure 1 The abdominal CT findings revealed localized thickening and clustering of the small bowel wall in the right abdominal cavity with comminuted fracture of the right 11th rib protruding into the abdominal cavity. (**A**): Axial view of an abdominal CT scan; (**B**): Coronal view of an abdominal CT scan; (**C**): Sagittal of an abdominal CT scan.

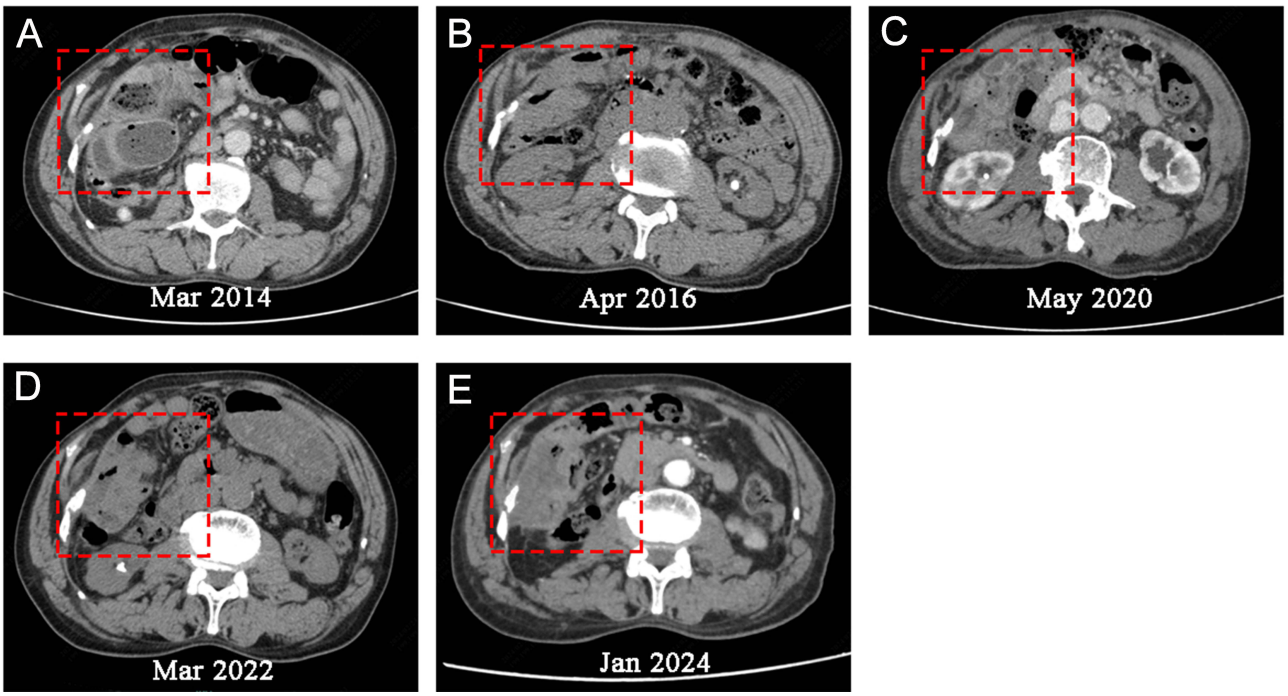

Figure 2 Changes in the small intestine under continuous stimulation of rib tissues (red box). (**A**): First record of a comminuted fracture of the right 11th rib protruding into the abdominal cavity accompanied by intestinal obstruction. (**B**): Small bowel aggregation around bone tissue. (**C**): The inflammatory mass has formed. (**D**): Thickening of the small bowel wall. (**E**): Uneven thickening of the small bowel wall and formation of a tumor.

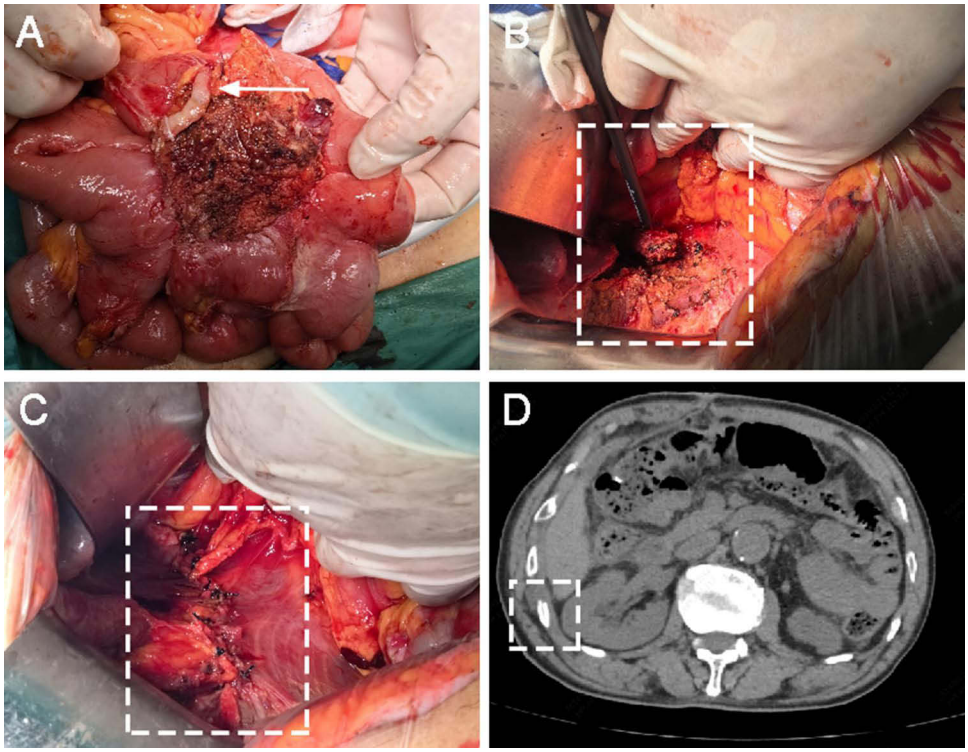

Figure 3 Surgical treatment procedure. (**A**): The rib end penetrates the abdominal cavity and is encircled by intertwined segments of the ileum, forming a mass. The head of the appendix was located within the mass (white arrow). (**B** and **C**): The rib end was removed and the abdominal wall was repaired. (**D**): Confirmed by postoperative CT scan (white box).

adjacent SB induced adhesion, an inflammatory mass, and thickening of the intestinal wall, culminating in the eventual development of tumors (Figure 2).

On the basis of clinical evaluation, laboratory assessments, and imaging observations, the patient received a diagnosis of an SB tumor, a comminuted fracture of the right 11th rib, and anemia. Exploratory laparotomy was performed and extensive adhesions were noted among the greater omentum, SB, mesentery, and right abdominal wall. After adhesions were separated, the rib end penetrated the abdomen and was encircled by intertwined segments of the ileum, forming a mass. The head of the appendix was located within the mass. Radical resection and lymph node dissection of the affected SB and appendix were performed. We also excised the rib end and repaired the abdominal wall to prevent further irritation (Figure 3).

Gross examination revealed irregular thickening of the SB wall, internal fistula formation, and rib fragments within the tumor cavity (Figure 4). The final pathological diagnosis was adenocarcinoma of the small bowel, which was staged as $pT_4N_0M_0$ (II) according to Version 1. 2020 of the NCCN Clinical Practice Guidelines in Oncology.[3]

The patient was discharged 12 days post-surgery and follow-up assessments revealed no reported discomfort one month later. Although adjuvant chemotherapy was recommended, the patient and his family declined, opting for regular outpatient monitoring instead.

Discussion and Conclusions

The concept of foreign body induced carcinogenesis represents a traditional, simplistic understanding of cancer development, which has historical significance. A multitude of methodologies are available today to validate and elucidate cancer development.[2] Many foreign bodies, including particulate carcinogens, asbestos fibers, metals, and medical devices, have been shown to induce cancer.[4] Furthermore, some chronic inflammatory diseases, such as Crohn's disease, ulcerative colitis, and chronic anal fistula, without specific bacterial infections, may also lead to rectal, colon, and anal cancers.[5–7] The recognized theory suggests that chronic inflammation, marked by repeated episodes of intestinal mucosal damage, results in prolonged mucosal repair and atypical hyperplasia, ultimately leading to carcinogenesis. Foreign

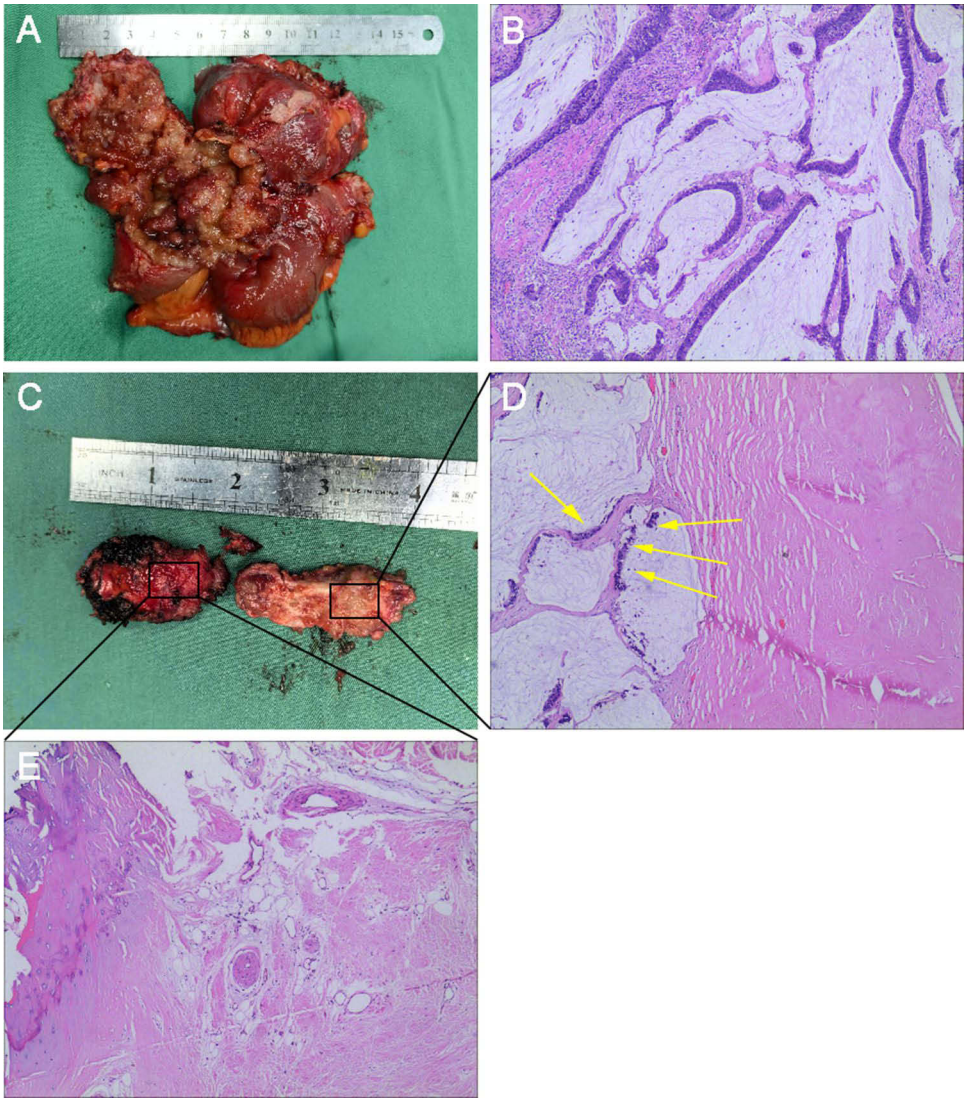

Figure 4 Postoperative specimens and pathology findings. (**A**): Anatomical examination revealed irregular thickening of the small bowel wall with the formation of internal fistulas between the intestinal walls and partial rib fragments located within the tumor cavity. (**B**): Pathological examination revealed raised-type, moderately differentiated adenocarcinoma of the small intestine, with mucinous adenocarcinoma components infiltrating all layers. No lymph node involvement was observed (0/10). IHC: CDX-2(+), CK20(+), CK7(-), Ki-67(+70%), MLH1(+), MSH2(+), MSH6(+), PMS2(+). (**C**): All rib tissues removed from the abdominal cavity. (**D**): Mucinous adenocarcinoma components were found on the surface of the rib within the tumor cavity (yellow arrow), and (**E**): No cancer cells were observed on the rib end.

bodies are triggers of only inflammatory reactions and do not cause cancer themselves.[2,8,9] Therefore, we recommend that patients suspected of having intra-abdominal foreign bodies associated with intestinal lesions be vigilant with respect to the potential for foreign body-induced tumors. Furthermore, when making treatment decisions, two key points should be considered. First, radical surgery should be performed to achieve R0 resection to avoid residual tumor or salvage surgery. Second, complete removal of the foreign bodies is essential to eliminate ongoing inflammatory reactions.

Fractures of the ribs, coupled with intra-abdominal organ injuries, primarily arise from the lower ribs and commonly affect organs such as the liver, spleen, small intestine, and colon.[10] Instances of fractures directly penetrating the abdominal cavity are infrequent, and individuals presenting with such injuries typically require urgent surgical intervention owing to the manifestation of severe acute abdominal symptoms.[11] In our case, subsequent to the fracture of the right 11th rib, the bone tissues did not directly inflict damage upon any organs and persisted within the abdominal cavity. Over time, the rib fragments in the abdominal cavity evolve into "foreign bodies", instigating chronic abdominal inflammation and gradually leading to SBA. The patient's ongoing management of urinary system stones involves regular abdominal

CT scans, enabling us to observe the carcinogenic process dynamically. To the best of our knowledge, this is the first case of foreign body-induced SBA reported worldwide in the English literature.

The patient's progression from the discovery of the rib fracture to the diagnosis of SBA took approximately 10 years, which is shorter than that reported in the literature, and some similar diseases, such as anal fistula-associated mucinous adenocarcinoma and chronic suppurative hidradenitis-associated perineal mucinous adenocarcinoma, were reported.[2] Brand et al[12] reported that of the 98 foreign-body or scar-related cancers reported in the literature, over 25% developed within 15 years, and over 50% developed within 25 years. Slesser et al[13] reviewed the literature and reported that the development of carcinoma in the context of perianal fistulae is associated with perianal disease progression for a period longer than 10 years. On the one hand, we speculate that the uncertainty regarding the precise timing of the rib fracture, which may have potentially occurred earlier, could be a contributing factor. On the other hand, this could also be attributed to patients' prolonged heavy alcohol consumption and smoking habits, both of which are well-established carcinogenic factors.

Importantly, the SBA was diagnosed post-surgery, as regular pathological biopsies were not conducted prior to surgery, which limited our ability to understand the tissue changes preceding cancer development. This raises another extreme possibility: the patient already had small bowel lesions or even cancer during or after the time of injury, and the presence of foreign bodies accelerated only tumor progression. Nevertheless, this case underscores the close association between chronic inflammation and cancer, as existing research indicates that inflammation can promote tumor initiation and metastasis.[2,8,9]

In summary, we present a unique case of SBA induced by self-rib tissues, highlighting the role of chronic inflammation in SBA progression. We emphasize the importance of addressing chronic inflammation triggers alongside tumor excision in treatment. Additionally, this case underscores the potential for establishing animal models with a similar process to SBA, further facilitating research into its occurrence, progression, prevention, and treatment.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This study was reviewed and approved by the Institutional Review Board of the Shaoxing People's Hospital and informed written consent was obtained from the patient. Ethical review and approval were not required to publish the case details in accordance with the institutional requirements and informed.

Consent for Publication

Informed written consent was obtained from the patient for publication of this report and any accompanying images.

Acknowledgments

We thank the patient and all colleagues involved in the treatment for their kind cooperation.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This work was supported by Shaoxing Health Science and Technology Program (2022SY016 and 2022KY010).

Disclosure

All authors have no conflicts of interest to declare.

References

1. Coussens LM, Werb Z. Inflammation and cancer. *Nature*. 2002;420(6917):860–867. PMID: 12490959; PMCID: PMC2803035. doi:10.1038/nature01322
2. Okada F. Beyond foreign-body-induced carcinogenesis: impact of reactive oxygen species derived from inflammatory cells in tumorigenic conversion and tumor progression. *Int, J, Cancer*. 2007;121(11):2364–2372. PMID: 17893867. doi:10.1002/ijc.23125
3. Benson AB, 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. PMID: 31487687; PMCID: PMC10191182. doi:10.6004/jnccn.2019.0043
4. Moizhess TG. Carcinogenesis induced by foreign bodies. *Biochemistry*. 2008;73(7):763–775. PMID: 18707584. doi:10.1134/s0006297908070043
5. Iesalnieks I, Gaertner WB, Glass H, et al. Fistula-associated anal adenocarcinoma in Crohn's disease. *Inflamm Bowel Dis*. 2010;16(10):1643–1648. PMID: 20186945. doi:10.1002/ibd.21228
6. Yashiro M. Ulcerative colitis-associated colorectal cancer. *World J Gastroenterol*. 2014;20(44):16389–16397. PMID: 25469007; PMCID: PMC4248182. doi:10.3748/wjg
7. Mukai N, Pinheiro LV, Ayrizono Mde L, et al. Mucinous adenocarcinoma associated with chronic suppurative hidradenitis: report of a case and review of the literature. *Int J Surg Case Rep*. 2016;26:12–16. PMID: 27424105; PMCID: PMC4949808. doi:10.1016/j.ijscr.2016.06.039
8. Song XD, Wang YN, Zhang AL, Liu B. Advances in research on the interaction between inflammation and cancer. *J Int Med Res*. 2020;48(4):300060519895347. PMID: 31885347; PMCID: PMC7686609. doi:10.1177/0300060519895347
9. Greten FR, Grivennikov SI. Inflammation and cancer: triggers, mechanisms, and consequences. *Immunity*. 2019;51(1):27–41. PMID: 31315034; PMCID: PMC6831096. doi:10.1016/j.immuni.2019.06.025
10. Park S. Clinical analysis for the correlation of intra-abdominal organ injury in the patients with rib fracture. *Korean J Thorac Cardiovasc Surg*. 2012;45(4):246–250. PMID: 22880170; PMCID: PMC3413830. doi:10.5090/kjtcs.2012.45.4.246
11. Davoodabadi A, Mosavibioki N, Mashayekhil M, Gilasi H, Kashi EA, Haghpanah B. Correlation of rib fracture patterns with abdominal solid organ injury: a retrospective observational cohort study. *Chin J Traumatol*. 2022;25(1):45–48. PMID: 34303569; PMCID: PMC8787232. doi:10.1016/j.cjtee.2021.07.007
12. Brand KG, Brand I. Risk assessment of carcinogenesis at implantation sites. *Plast Reconstr Surg*. 1980;66(4):591–595. PMID: 7010394. doi:10.1097/00006534-198010000-00015
13. Slesser AA, Bhangu A, Bower M, Goldin R, Tekkis PP. A systematic review of anal squamous cell carcinoma in inflammatory bowel disease. *Surg Oncol*. 2013;22(4):230–237. doi:10.1016/j.suronc.2013.08.002