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Contents

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OPINION REVIEW

- 5124** Malignant insulinoma: Can we predict the long-term outcomes?
Cigrovski Berkovic M, Ulamec M, Marinovic S, Balen I, Mrzljak A

MINIREVIEWS

- 5133** Practical points that gastrointestinal fellows should know in management of COVID-19
Sahin T, Simsek C, Balaban HY
- 5146** Nanotechnology in diagnosis and therapy of gastrointestinal cancer
Liang M, Li LD, Li L, Li S
- 5156** Advances in the clinical application of oxycodone in the perioperative period
Chen HY, Wang ZN, Zhang WY, Zhu T

ORIGINAL ARTICLE

Clinical and Translational Research

- 5165** Circulating miR-627-5p and miR-199a-5p are promising diagnostic biomarkers of colorectal neoplasia
Zhao DY, Zhou L, Yin TF, Zhou YC, Zhou GYJ, Wang QQ, Yao SK

Retrospective Cohort Study

- 5185** Management and outcome of bronchial trauma due to blunt *versus* penetrating injuries
Gao JM, Li H, Du DY, Yang J, Kong LW, Wang JB, He P, Wei GB

Retrospective Study

- 5196** Ovarian teratoma related anti-N-methyl-D-aspartate receptor encephalitis: A case series and review of the literature
Li SJ, Yu MH, Cheng J, Bai WX, Di W
- 5208** Endoscopic surgery for intraventricular hemorrhage: A comparative study and single center surgical experience
Wang FB, Yuan XW, Li JX, Zhang M, Xiang ZH
- 5217** Protective effects of female reproductive factors on gastric signet-ring cell carcinoma
Li Y, Zhong YX, Xu Q, Tian YT
- 5230** Risk factors of mortality and severe disability in the patients with cerebrovascular diseases treated with perioperative mechanical ventilation
Zhang JZ, Chen H, Wang X, Xu K

- 5241** Awareness of initiative practice for health in the Chinese population: A questionnaire survey based on a network platform

Zhang YQ, Zhou MY, Jiang MY, Zhang XY, Wang X, Wang BG

- 5253** Effectiveness and safety of chemotherapy for patients with malignant gastrointestinal obstruction: A Japanese population-based cohort study

Fujisawa G, Niikura R, Kawahara T, Honda T, Hasatani K, Yoshida N, Nishida T, Sumiyoshi T, Kiyotoki S, Ikeya T, Arai M, Hayakawa Y, Kawai T, Fujishiro M

Observational Study

- 5266** Long-term outcomes of high-risk percutaneous coronary interventions under extracorporeal membrane oxygenation support: An observational study

Huang YX, Xu ZM, Zhao L, Cao Y, Chen Y, Qiu YG, Liu YM, Zhang PY, He JC, Li TC

- 5275** Health care worker occupational experiences during the COVID-19 outbreak: A cross-sectional study

Li XF, Zhou XL, Zhao SX, Li YM, Pan SQ

Prospective Study

- 5287** Enhanced recovery after surgery strategy to shorten perioperative fasting in children undergoing non-gastrointestinal surgery: A prospective study

Ying Y, Xu HZ, Han ML

- 5297** Orthodontic treatment combined with 3D printing guide plate implant restoration for edentulism and its influence on mastication and phonic function

Yan LB, Zhou YC, Wang Y, Li LX

Randomized Controlled Trial

- 5306** Effectiveness of psychosocial intervention for internalizing behavior problems among children of parents with alcohol dependence: Randomized controlled trial

Omkarappa DB, Rentala S, Nattala P

CASE REPORT

- 5317** Crouzon syndrome in a fraternal twin: A case report and review of the literature

Li XJ, Su JM, Ye XW

- 5324** Laparoscopic duodenojejunostomy for malignant stenosis as a part of multimodal therapy: A case report

Murakami T, Matsui Y

- 5331** Chordoma of petrosal mastoid region: A case report

Hua JJ, Ying ML, Chen ZW, Huang C, Zheng CS, Wang YJ

- 5337** Pneumatosis intestinalis after systemic chemotherapy for colorectal cancer: A case report

Liu H, Hsieh CT, Sun JM

- 5343** Mammary-type myofibroblastoma with infarction and atypical mitosis-a potential diagnostic pitfall: A case report

Zeng YF, Dai YZ, Chen M

- 5352** Comprehensive treatment for primary right renal diffuse large B-cell lymphoma with a renal vein tumor thrombus: A case report
He J, Mu Y, Che BW, Liu M, Zhang WJ, Xu SH, Tang KF
- 5359** Ectopic peritoneal paragonimiasis mimicking tuberculous peritonitis: A case report
Choi JW, Lee CM, Kim SJ, Hah SI, Kwak JY, Cho HC, Ha CY, Jung WT, Lee OJ
- 5365** Neonatal hemorrhage stroke and severe coagulopathy in a late preterm infant after receiving umbilical cord milking: A case report
Lu Y, Zhang ZQ
- 5373** Heel pain caused by os subcalcis: A case report
Saijilafu, Li SY, Yu X, Li ZQ, Yang G, Lv JH, Chen GX, Xu RJ
- 5380** Pulmonary lymphomatoid granulomatosis in a 4-year-old girl: A case report
Yao JW, Qiu L, Liang P, Liu HM, Chen LN
- 5387** Idiopathic membranous nephropathy in children: A case report
Cui KH, Zhang H, Tao YH
- 5394** Successful treatment of aortic dissection with pulmonary embolism: A case report
Chen XG, Shi SY, Ye YY, Wang H, Yao WF, Hu L
- 5400** Renal papillary necrosis with urinary tract obstruction: A case report
Pan HH, Luo YJ, Zhu QG, Ye LF
- 5406** Glomangiomas - immunohistochemical study: A case report
Wu RC, Gao YH, Sun WW, Zhang XY, Zhang SP
- 5414** Successful living donor liver transplantation with a graft-to-recipient weight ratio of 0.41 without portal flow modulation: A case report
Kim SH
- 5420** Treatment of gastric hepatoid adenocarcinoma with pembrolizumab and bevacizumab combination chemotherapy: A case report
Liu M, Luo C, Xie ZZ, Li X
- 5428** Ipsilateral synchronous papillary and clear renal cell carcinoma: A case report and review of literature
Yin J, Zheng M
- 5435** Laparoscopic radical resection for situs inversus totalis with colonic splenic flexure carcinoma: A case report
Zheng ZL, Zhang SR, Sun H, Tang MC, Shang JK
- 5441** PIGN mutation multiple congenital anomalies-hypotonia-seizures syndrome 1: A case report
Hou F, Shan S, Jin H

- 5446** Pediatric acute myeloid leukemia patients with i(17)(q10) mimicking acute promyelocytic leukemia: Two case reports
Yan HX, Zhang WH, Wen JQ, Liu YH, Zhang BJ, Ji AD
- 5456** Fatal left atrial air embolism as a complication of percutaneous transthoracic lung biopsy: A case report
Li YW, Chen C, Xu Y, Weng QP, Qian SX
- 5463** Diagnostic value of bone marrow cell morphology in visceral leishmaniasis-associated hemophagocytic syndrome: Two case reports
Shi SL, Zhao H, Zhou BJ, Ma MB, Li XJ, Xu J, Jiang HC
- 5470** Rare case of hepatocellular carcinoma metastasis to urinary bladder: A case report
Kim Y, Kim YS, Yoo JJ, Kim SG, Chin S, Moon A
- 5479** Osteotomy combined with the trephine technique for invisible implant fracture: A case report
Chen LW, Wang M, Xia HB, Chen D
- 5487** Clinical diagnosis, treatment, and medical identification of specific pulmonary infection in naval pilots: Four case reports
Zeng J, Zhao GL, Yi JC, Liu DD, Jiang YQ, Lu X, Liu YB, Xue F, Dong J
- 5495** Congenital tuberculosis with tuberculous meningitis and situs inversus totalis: A case report
Lin H, Teng S, Wang Z, Liu QY
- 5502** Mixed large and small cell neuroendocrine carcinoma of the stomach: A case report and review of literature
Li ZF, Lu HZ, Chen YT, Bai XF, Wang TB, Fei H, Zhao DB

LETTER TO THE EDITOR

- 5510** Pleural involvement in cryptococcal infection
Georgakopoulou VE, Damaskos C, Sklapani P, Trakas N, Gkoufa A
- 5515** Electroconvulsive therapy plays an irreplaceable role in treatment of major depressive disorder
Ma ML, He LP

ABOUT COVER

Editorial Board Member of *World Journal of Clinical Cases*, Shivanshu Misra, MBBS, MCh, MS, Assistant Professor, Surgeon, Department of Minimal Access and Bariatric Surgery, Shivani Hospital and IVF, Kanpur 208005, Uttar Pradesh, India. shivanshu_medico@rediffmail.com

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Pneumatosis intestinalis after systemic chemotherapy for colorectal cancer: A case report

Hsien Liu, Cheng-Ta Hsieh, Jui-Ming Sun

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Hsien Liu, Division of General Surgery, Department of Surgery, Ditmanson Medical Foundation, Chia-Yi Christian Hospital, Chia-Yi City 600, Taiwan

Cheng-Ta Hsieh, Division of Neurosurgery, Department of Surgery, Sijhih Cathay General Hospital, New Taipei 22174, Taiwan

Cheng-Ta Hsieh, Department of Medicine, School of Medicine, Fu Jen Catholic University, New Taipei City 24205, Taiwan

Cheng-Ta Hsieh, School of Medicine, National Tsing Hua University, Hsinchu City 300044, Taiwan

Jui-Ming Sun, Section of Neurosurgery, Department of Surgery, Ditmanson Medical Foundation, Chia-Yi Christian Hospital, Chiayi City 600, Taiwan

Jui-Ming Sun, Department of Biotechnology, Asia University, Taichung City 41354, Taiwan

Corresponding author: Jui-Ming Sun, MD, Director, Section of Neurosurgery, Department of Surgery, Ditmanson Medical Foundation, Chia-Yi Christian Hospital, No. 539 Zhongxiao Road, East District, Chiayi City 600, Taiwan. 07178@cych.org.tw

Abstract

BACKGROUND

Pneumatosis intestinalis (PI), also known as intramural gas in the small intestine, is a rare condition encountered by patients with cancer after receiving chemotherapy.

CASE SUMMARY

A 78-year-old man with a history of colorectal cancer developed epigastric pain and diarrhea after receiving combination chemotherapy of fluorouracil, leucovorin, irinotecan, and cetuximab. Abdomen radiography revealed in-tramural air in the small intestinal wall. A computed tomography scan of the abdomen revealed the features of PI with air expanding into the mesentery. After surgery, the patient remained symptom-free throughout a 9 mo follow-up period during which he received chemotherapy of fluorouracil, leucovorin, and irinotecan.

CONCLUSION

Although chemotherapy-induced PI is rare among patients with cancer, the differential diagnosis of PI and fulminant complications (such as ischemia, infarction, and perforation of the gastrointestinal tract) should be conducted, in which case

an urgent surgical intervention is required.

Key Words: Pneumatosis intestinalis; Colorectal cancer; Chemotherapy; Surgery; Case report

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Core Tip: Pneumatosis intestinalis (PI) is a rare condition encountered by patients with cancer after receiving chemotherapy. The differential diagnosis of PI and chemotherapy-induced fulminant complications (such as ischemia, infarction, and perforation of the gastrointestinal tract) should be conducted, in which case an urgent surgical intervention is required.

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INTRODUCTION

Pneumatosis intestinalis (PI), which is characterized by the presence of submucosal or subserosal gas in the intestinal wall, is an uncommon disease[1]. It can occur as an idiopathic disease, or more commonly, as a secondary condition associated with the coexistence of gastrointestinal tract disorders or respiratory system diseases such as chronic obstructive pulmonary disease, ischemic bowel disease, intestinal obstruction, necrotizing enterocolitis, immunodeficiency, bacterial infection, and viral infection[2,3]. However, chemotherapy-induced PI is relatively rare in patients with cancer. Chemotherapy agent-induced cytotoxic damage and mucosal ischemia in the gastrointestinal tract may also lead to gastrointestinal perforation, necrotizing enterocolitis, or ischemic bowel[4]. Prompt surgical intervention may be required to manage the perforation or ischemic necrosis of the gastrointestinal tract [3]. Therefore, the differential diagnosis of PI and bowel perforation in patients with cancer after chemotherapy is essential for clinical physicians to treat acute abdomen[4]. Herein, we report a rare case of a patient with colon cancer who presented with acute abdomen after systemic chemotherapy. PI was diagnosed following radiological studies and surgical intervention. The possible mechanisms of PI in patients with cancer receiving chemotherapy are discussed and reviewed.

CASE PRESENTATION

Chief complaints

A 78-year-old man complained of acute lower abdominal pain with cramping pain and diarrhea lasting for 3 d.

History of present illness

A 78-year-old man with a history of mucinous adenocarcinoma (pT3N2bcM1b, stage IVB) in the descending colon and upper rectum underwent laparoscopic radical left hemicolectomy, sigmoidectomy, and loop T-colostomy. He then underwent chemotherapy comprising fluorouracil, leucovorin, and irinotecan (FOLFIRI); intravenous injections of 400 mg/m² fluorouracil, 200 mg/m² leucovorin, and 120 mg/m² irinotecan were administered every 2 wk. Starting from the second course of FOLFIRI treatment, the adjuvant chemo-agent (cetuximab) was administered at a dose of 500 mg/m². One week after the third course of FOLFIRI combined with the second course of cetuximab chemotherapy, he complained of acute lower abdominal pain with cramping pain and diarrhea lasting for 3 days.

History of past illness

The patient had a history of mucinous adenocarcinoma (pT3N2bcM1b, stage IVB) in the descending colon and upper rectum and underwent laparoscopic radical left hemicolectomy, sigmoidectomy, and loop T-colostomy.

Physical examination

The patient presented at our emergency department with a body temperature of 36.7 °C, heart rate of 127 bpm, blood pressure of 180/108 mmHg, and respiratory rate of 18 breaths per minute. A physical

examination revealed abdominal distention, tenderness in the epigastric region, and hypoactive bowel sounds. No rebounding pain was observed. The colostomy exhibited good perfusion with brown soft stool passage.

Laboratory examinations

Laboratory examinations revealed a white blood cell count of 1200/ μ L, platelet count of 245000/ μ L, hemoglobin level of 10.3 mg/dL, total bilirubin of 0.6 mg/dL, lipase level of 34 mg/dL, lactate level of 1.0 mg/dL, and C-reactive protein level of 146 mg/dL.

Imaging examinations

Abdominal radiograph revealed gas in the small intestinal wall (Figure 1). A computed tomography (CT) scan of the abdomen with contrast enhancement indicated intramural gas in the small intestine that expanded into the mesentery (Figure 2).

FINAL DIAGNOSIS

A pathological examination confirmed the diagnosis of PI with focal ischemic and ulcerative changes.

TREATMENT

The patient underwent upper midline mini-laparotomy surgery because bowel perforation or necrotizing enterocolitis was suspected. During surgery, expanded intraluminal air spaces in the small intestine were observed (Figure 3). An approximately 50 cm length of the proximal jejunum exhibited severe edematous changes and compromised mild blood circulation without necrosis. Segmental resection of the small bowel with end-to-end anastomosis was performed.

OUTCOME AND FOLLOW-UP

Postoperatively, the patient's previously observed symptoms improved after 2 wk of parenteral nutrition, antibiotic treatment, and oxygen therapy. The FOLFIRI treatment for his colorectal cancer was started 3 mo after discharge. No recurrence of PI was observed during the 9 mo follow-up.

DISCUSSION

PI was first reported radiologically in 1946 by Lerner and Gazin[5]. It is an uncommon disease characterized by the presence of gas and free air in the extraluminal spaces of the intestines from the mucosa to mesenteric vessels[3]. Several terms have been used to describe PI, including pneumatosis cystoides intestinalis, intramural gas, pneumatosis coli, pseudolipomatosis, intestinal emphysema, bullous emphysema of the intestine, and lymphopneumatosis[1]. The incidence of PI is 0.03% according to the literature; however, this is believed to be an underestimation because most patients are asymptomatic and, consequently, do not receive clinical attention[6].

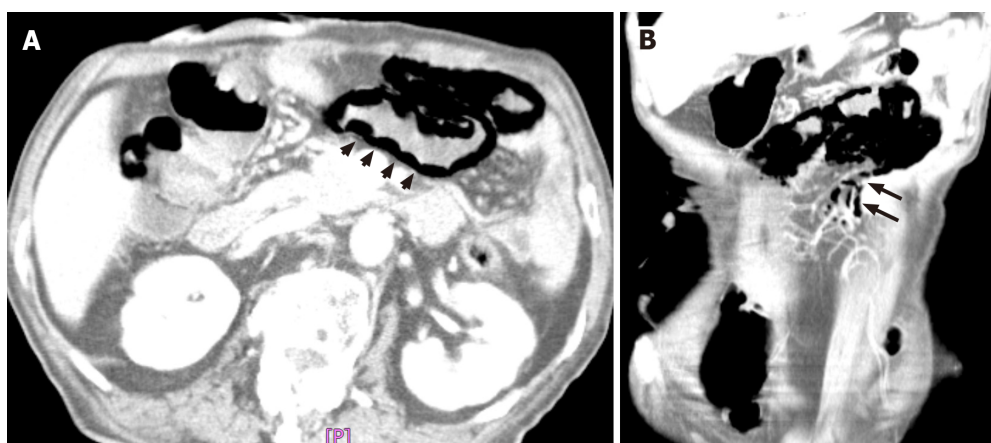
PI is a radiological finding and not a diagnosis[7], and it is commonly classified as primary and secondary forms[8]. Secondary PI is found in approximately 80% to 85% of patients and is often associated with bowel ischemia, trauma, inflammatory bowel diseases, chronic obstructive pulmonary disorder, use of specific medications, immunocompromised disease, and immunosuppressive therapy [8,9]. Although the exact pathogenesis of PI remains unclear, four categories are used to describe the pathogenesis of PI, namely, bowel necrosis, mucosal disruption, increased mucosal permeability, and pulmonary disease[10]. Mechanical and bacterial theories have been proposed to explain the development of PI[6,9]. A mechanical theory suggests that a defect in mucosal integrity allows for gas to be transmitted through the dissection of submucosal or subserosal layers and that peristalsis then propagates the gas to distant sites[6]. A bacterial theory proposes that some intramural gas is normally present in the gastrointestinal tract and that the overgrowth of normal bacteria (such as *Escherichia coli* and *Clostridium difficile*) leads to bacterial invasion of the bowel wall and lymphatic system through mucosal defects and the subsequent production of excess gas.

Chemotherapy-induced PI in patients with cancer is rare[4,11-15]. Many cytotoxic chemotherapeutic agents have been reported to be associated with PI; they include cyclophosphamide, cytarabine, vincristine, doxorubicin, daunorubicin, etoposide, docetaxel, irinotecan, cisplatin, methotrexate, fluorouracil, paclitaxel, tyrosine kinase inhibitors (imatinib, sunitinib, sorafenib, and erlotinib), bevacizumab (a monoclonal body to vascular endothelial growth factor), and cetuximab (a monoclonal



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Figure 1 Plain radiograph of the abdomen revealing intramural gas in the small intestine.



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Figure 2 Computed tomographic images of the abdomen. A: Gas entrapment in the wall of the small intestine (arrowheads); B: Air expanding into the mesentery (arrows).

antibody to epidermal growth factor receptor)[11,13-16]. This may contribute to cytotoxic damage, loss of mucosal integrity, mucosal ischemia, and the development of PI. Although four chemoagents (fluorouracil, leucovorin, irinotecan, and cetuximab) might have contributed to the development of PI in our patient, no recurrence of PI was observed after subsequent FOLFIRI treatment without cetuximab. This suggests that cetuximab played an essential role in the development of PI. The perforation of the gastrointestinal tract is also a rare life-threatening complication (due to bowel toxicity) that may occur after molecular-targeted chemotherapy[12]. Therefore, the differential diagnosis of PI and bowel perforation is crucial to the management of acute abdomen in oncologic patients receiving systemic chemotherapy, such as in the case of our patient.

The clinical presentation of patients with PI includes being asymptomatic, vomiting, weight loss, constipation, hematochezia, tenesmus, diarrhea, abdominal pain, and life-threatening peritoneal symptoms such as pneumoperitoneum, intestinal ischemia, peritonitis, and bowel obstruction[1]. The main radiological feature of PI is the presence of circular, linear, or curvilinear gas within the wall of the gastrointestinal tract on plain radiographs or CT scans[17]. CT scan is the most sensitive imaging tool for differentiating intraluminal air from the submucosal layer and identifying additional causes of PI, which include portal air, colonic tissue stranding, and dilated bowel. In a review of 37426 abdominal



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Figure 3 Intraoperative photo revealing intramural gas in the small intestine (arrows).

and pelvic CT scans, Hawn *et al*[18] detected PI in 108 (0.3%) patients. In another review involving 28326 abdominal CT scans, Morris *et al*[17] discovered that out of 104 (0.37%) patients who were diagnosed as having pneumatosis, only 23% had observable PI on their plain radiographs. Cyst-like gas collection is usually benign, whereas linear collection tends to be associated with bowel infarction[10,18]. However, the recognition of these patterns should not be the sole basis for differentiating benign and fulminant conditions.

A consensus regarding the appropriate management of PI has not been reached due to the difficulties of delineating the underlying etiologies[17]. An initial clarification to differentiate life-threatening conditions from nonurgent ones is crucial[8]. Approximately 50% of patients with PI can be successfully managed nonoperatively. Because PI indicates that necrotic tissue is allowing gas to enter the extraluminal space, ischemia or infarction of the gastrointestinal tract system should be suspected, in which case an urgent surgical intervention is required. Based on the underlying diseases leading to PI, surgical procedures such as bowel resection, lysis of adhesion, bowel biopsies, or nontherapeutic exploratory laparotomy should be performed. However, patients with concomitant PI and portal venous gas tend to have bowel ischemia, which is associated with a high mortality rate. Patients with a serum lactic acid level of more than 2 mmol/L at the time of diagnosis have an overall mortality rate of more than 80%[18]. Patients with no signs of peritonism or sepsis can usually be managed conservatively[9]. Therefore, the occurrence of PI should trigger a clinical physician to investigate the possible causes, ensure a correct diagnosis, and develop an appropriate management protocol.

CONCLUSION

PI is a radiologic sign and not a diagnosis. Although chemotherapy-induced PI is rare among patients with cancer, the differential diagnosis of PI and life-threatening conditions (such as ischemia, infarction, and perforation of the gastrointestinal tract) should be conducted for patients who present with acute abdomen.

FOOTNOTES

Author contributions: Liu H and Hsieh CT drafted the manuscript; Liu H performed the surgery; Liu H collected the clinical data and performed the follow-up; Hsieh CT and Sun JM conducted the literature review; and Sun JM revised the manuscript critically for intellectual content; all authors have read and approved the final manuscript.

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Country/Territory of origin: Taiwan

ORCID number: Hsien Liu 0000-0002-3005-4056; Cheng-Ta Hsieh 0000-0002-2018-7015; Jui-Ming Sun 0000-0002-4878-5084.

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