<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1375</td>
<td>GUIDELINE INTERPRETATION</td>
<td>Li Y, Wang HL</td>
</tr>
<tr>
<td>1388</td>
<td>REVIEW</td>
<td>Wang S, Chen J, Guo XZ</td>
</tr>
<tr>
<td>1406</td>
<td>Poorly cohesive cells gastric carcinoma including signet-ring cell cancer: Updated review of definition, classification and therapeutic management</td>
<td>Drubay V, Nuytens F, Renaud F, Adenis A, Eveno C, Piessen G</td>
</tr>
<tr>
<td>1446</td>
<td>MINIREVIEWS</td>
<td>Uson Junior PLS, Araujo RL</td>
</tr>
<tr>
<td>1456</td>
<td>Crosstalk between gut microbiota and COVID-19 impacts pancreatic cancer progression</td>
<td>Zhang CY, Liu S, Yang M</td>
</tr>
<tr>
<td>1469</td>
<td>Angiogenesis in gastrointestinal stromal tumors: From bench to bedside</td>
<td>Papadakos SP, Tsagkaris C, Papadakis M, Papazoglou AS, Moysidis DV, Zografos CG, Theocharis S</td>
</tr>
<tr>
<td>1478</td>
<td>Stereotactic radiotherapy for intrahepatic cholangiocarcinoma</td>
<td>Borakati A, Froghi F, Bhogal RH, Mavroidis VK</td>
</tr>
<tr>
<td>1499</td>
<td>ORIGINAL ARTICLE</td>
<td>Safety and feasibility of irreversible electroporation for the pancreatic head in a porcine model</td>
</tr>
</tbody>
</table>
Retrospective Cohort Study

1510 Second-line therapy for advanced hepatocellular carcinoma with regorafenib or cabozatinib: Multicenter French clinical experience in real-life after matching

Retrospective Study

1528 Profiling of gene fusion involving targetable genes in Chinese gastric cancer
Liu ZH, Zhu BW, Shi M, Qu YR, He XJ, Yuan HL, Ma J, Li W, Zhao DD, Liu ZC, Wang BM, Wang CY, Tao HQ, Ma TH

1540 Adjuvant chemoradiotherapy vs adjuvant chemotherapy in locally advanced Siewert type II/III adenocarcinoma of gastroesophageal junction after D2/R0 resection

Observational Study

1552 Duodenal-type follicular lymphoma more than 10 years after treatment intervention: A retrospective single-center analysis
Saito M, Mori A, Tsukamoto S, Ishio T, Yokoyama E, Izumiyama K, Morioka M, Kondo T, Sugino H

1562 Evaluation of the diagnostic value of serum-based proteomics for colorectal cancer
Wang HJ, Xie YB, Zhang PJ, Jiang T

1574 RASSF1A methylation as a biomarker for detection of colorectal cancer and hepatocellular carcinoma
Li J, Li H, Run ZC, Wang ZL, Jiang T, An Y, Li Z

CASE REPORT

1585 Ewing sarcoma of the ileum with wide multiorgan metastases: A case report and review of literature
Guo AW, Liu YS, Li H, Yuan Y, Li SX

LETTER TO THE EDITOR

1594 Exosomes: Promising biomarkers and targets for cancer
Fang Z, Ding YX, Li F

1597 Colitis and colorectal tumors should be further explored and differentiated
Xu DH, Zhou B, Li ZP, He LP, Wang XJ

1600 Acute or chronic inflammation role in gastrointestinal oncology
Chen HJ, Liang GY, Chen X, Du Z
ABOUT COVER
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Colitis and colorectal tumors should be further explored and differentiated

Dong-Hui Xu, Bo Zhou, Zhi-Peng Li, Lian-Ping He, Xin-Juan Wang

Abstract

The original article by Yuichi et al explored whether the Japan Narrow-Band Imaging Expert Team classification and the pit pattern classification are suitable for diagnosing neoplastic lesions in patients with ulcerative colitis. In this letter, we offer some other perspectives. Risk factors for colorectal tumors include type 2 diabetes. Among genetic factors, the deletion or mutation of some genes, such as the p53 gene, can lead to colorectal tumors. There are significant gender differences in the occurrence and development of colorectal tumors. Some non-genetic factors, such as smoking, are also associated with the development of colorectal tumors. These all suggest that colorectal tumors are not only caused by ulcerative colitis, and we suggest further exploration and differentiation between colitis and colorectal tumors.

Key Words: Colorectal cancer; Nicotine; p53; Tobacco; Ulcerative colitis

Core Tip: Among genetic factors, the deletion or mutation of some tumor suppressor genes can lead to colorectal tumors. Non-genetic factors are also associated with the development of colorectal tumors. The underlying disease can be a risk factor for colorectal tumors. There are significant gender differences in the occurrence and development of colorectal tumors. These all suggest that colorectal tumors are not only caused by ulcerative colitis, and we suggest further exploration and differentiation between colitis and colorectal tumors.
TO THE EDITOR

We read with great interest the study by Kida Y et al.[1] which was published in the world journal of gastroenterology. The study focused on whether the Japan Narrow-Band Imaging Expert Team (JNET) classification and pit pattern classification are applicable for diagnosing neoplastic lesions in patients with ulcerative colitis (UC). This study found that The JNET and pit pattern classifications did not show high accuracy in diagnosing the pathology and invasion depth of neoplastic lesions in UC patients. Endoscopic diagnosis of neoplastic lesions in UC patients is still difficult and treatment strategies need to be carefully determined. Although the authors’ findings provide new methods and ideas for existing diagnosis and treatment problems, our team agrees that there are still some issues that need further discussion in this paper.

In the case of genetic factors, environmental factors, living habits, and other adverse factors, everyone is theoretically at risk of developing colorectal tumors. The study by Simon[2] showed that genetic disorders such as Lynch syndrome, a personal history of inflammatory bowel disease, and type 2 diabetes are all predisposing factors for developing colorectal tumors. In genetic factors, deletion or mutation of some genes, such as the p53 gene, can also lead to colorectal tumors[3,4]. There are significant gender differences in the development of colorectal tumors, and the colorectum is a common tumor-producing organ in both men and women[5]. The study by Kim et al[6] showed that women over 65 had higher colorectal cancer mortality compared with men of the same age group. Colorectal cancer detection time and mortality are related to the site of colorectal cancer. Compared with right-sided colon cancer, left-sided colon cancer was detected later and more differentiated. In clinical work, it was found that the proportion of right-sided colorectal cancer in women is much higher than in men. All of the above evidence suggests that the mortality rate of female patients with colorectal cancer may be higher than that of male patients.

Some non-genetic factors, such as smoking, are also associated with the development of colorectal tumors. Among the etiologies of non-hereditary colorectal tumors, smoking has local and systemic effects on the colorectal mucosa through the production of carcinogens[7]. The nicotine in tobacco is potentially addictive and increases the patient’s dependence on tobacco, thereby increasing the risk of colorectal cancer. In addition, the mutation rate of tumor suppressor genes in smokers was significantly higher than in non-smokers. Among the many mutant genes, the p53 gene mutation is the most important. These phenomena are related to the occurrence and development of colorectal tumors. The study by Siegel et al[8] shows that women under 49 are about 3% more likely to die than men.

In summary, colorectal tumors are not only caused by ulcerative colitis. Research by Curtin K shows that smoking (> 20 pack-years vs non-smokers) was associated with TP53 mutations (OR = 1.4, 95%CI 1.02-2.0), BRAF mutations (OR = 4.2, 95%CI 1.3-14.2), and MSI mutations (OR = 1.4, 95%CI 1.02-2.0) in rectal tumors and was associated with an increased risk of rectal cancer. Long-term exposure to > 10 h/wk of environmental tobacco smoke was associated with an increased risk of KRAS2 mutations (OR = 1.5, 95%CI 1.04-2.2)[9]. Colorectal cancer is also related to genetic factors, living habits, eating habits, etc. It may not be clear that patients with chronic ulcerative colitis developed colorectal tumors due to chronic inflammation in this study. To further explore whether chronic ulcerative colitis is a risk factor for colorectal tumors, genetic factors, dietary habits, lifestyle habits and other factors need to be further discussed.

Type 2 diabetes has been shown to be a risk factor for colorectal tumors. Among genetic factors, deletion or mutation of some genes, such as the p53 gene, can lead to colorectal tumors. There are significant gender differences in the occurrence and development of colorectal tumors. Some non-genetic factors, such as smoking, are also associated with the development of colorectal tumors. These all suggest that colorectal tumors are not only caused by ulcerative colitis. Therefore, we suggest further exploration and differentiation between colitis and colorectal tumors.

FOOTNOTES

Author contributions: Wang XJ and He LP conceived of the presented idea and provided critical feedback to the final manuscript, revised the manuscript and approved the main conceptual ideas and outline; Xu DH, Zhou B, and Li ZP wrote the manuscript; all authors provided final edits and approved the manuscript.

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