Case Report

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20162963

Synchronous colonic malignancy

Prasad Dasharath Hake*, Narayan Pundalik Umale, Atul Uttamrao Yadgire, Kaustubh Madhusudan Sarda

Department of Surgery, Dr. Panjabrao Deshmukh Hospital, Amravati, Maharashtra, India

Received: 02 July 2016 Revised: 09 August 2016 Accepted: 17 August 2016

*Correspondence:

Dr. Prasad Dasharath Hake, E-mail: dr.hakeprasad@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Synchronous colorectal neoplasias, defined as 2 or more primary tumors identified in the same patient and at the same time, are caused by common genetic and environmental factors. Since intraoperative palpation can miss up to 69% of the SN, currently, synchronous neoplastic lesions are usually diagnosed at a preoperative staging by colonoscopy or virtual colonoscopy; according to data from literature, 3% of the patients with SN are affected by different types of malignant lesions while 33-55% shows villous adenomas. Literature also confirms the presence of primitive synchronous cancers; malignant synchronous lesions are very rare, showing the following incidence: between 0,17% and 0.69% in case of 2-3 synchronous lesions, 0.19% in case of 4-5 synchronous lesions. The most voluminous synchronous cancer is called "first primitive" or "index" cancer. When the index cancer is located in the caecum, the incidence of left colon synchronous cancers is higher than when the index cancer is located in the left colon. Colorectal adenomas standard treatment is usually represented by endoscopic polypectomy; indeed only 5% of synchronous colorectal lesions require a surgical treatment.

Keywords: Abdominal discomfort, Caecum and descending colon, Bowel thickening, Adenocarcinoma, Chemotherapy

INTRODUCTION

Colorectal cancer is the third most common malignancy worldwide and the fourth most common in the United States, with estimated 146,970 new cases diagnosed in 2009. In the United States, approximately 49,920 cancerrelated deaths were attributed to a colorectal malignancy in 2009, making it second only to mortality from lung cancer.¹

Despite the high incidence of colorectal carcinoma that is diagnosed yearly, the majority of lesions are resected with curative intent (70%-80%) and colorectal cancerrelated deaths account for 20%-30% of those diagnosed and treated surgically, making it a highly curable malignancy if identified in its early-stages. Approximately, 55% of colorectal cancers diagnosed on

screening are found to present with Stage I or II disease.^{2,3} Patients with Stage I or Stage II colon cancer have a greater than 60% 5-year survival, and for patients with Stage I or Stage II rectal cancer, there is a greater than 50% 5-year survival.^{4,5}

Because of this, colorectal screening exams in asymptomatic patients have become the recommended standard of care for average risk patients at the age of 50 and at the age of 40 or younger for those in the moderate and high risk groups.

Approximately, 70% of colorectal lesions occur distal to the splenic flexure, and looking at colon cancer alone, approximately 25% are found in the sigmoid, 10% at the rectosigmoid junction, and 4%-6% located in the descending colon.⁶ Anatomically, the left colon has a

smaller diameter than the right, and as a result, left-sided carcinomas can cause varying degrees of intraluminal occlusion and patients more frequently present with obstructive symptoms. Large bowel obstructions present a challenging clinical scenario for the physician in the diagnosis, operative management, and the timing of colonic surveillance. Patients presenting with advanced lesions causing partial or high-grade large bowel obstruction commonly have a distant history or no history of previous colonic surveillance.

This fact is of clinical importance because the diagnosis of a primary colorectal malignancy is accompanied by an overall incidence of synchronous colorectal cancers between 2% and 10% and a frequency of synchronous adenomatous polyps ranging from 15% to 50%. ⁷⁻¹³ Interestingly, when compared by stage, no statistically significant difference has been found in the overall and disease-free survival between synchronous and primary cancers. ¹⁴⁻¹⁶

CASE REPORT

47 years old male residing at Chandur Bazar farmer by occupation was presented with chief complaints of vague abdominal discomfort since 12 months; incomplete bowel evacuation since 12 months; decreased appetite since 12 months; and loss of weight since 12 months.

There was no history of vomiting, hemetemesis, malena, bleeding per rectum, fever, breathlessness, hemoptysis, chest pain, giddiness, headache, backache or urinary complaints. With the past history of pulmonary tuberculosis for which CAT 1 AKT was taken 15 years ago. General conditions was satisfactory as afebrile, with pulse rate of 96/min; blood pressure 130/80 mm Hg and paleness was present, and no signs of lymphadenopathy, icterus, oedema, and signs of dehydration.



Figure 1: Specimen removed (caecum, ascending colon, transverse colon, and descending colon).

Per abdomen was soft, non-distended, tenderness in left iliac region, no guarding or rigidity, no palpable lump, tympanic note on percussion all over and bowel sounds was present. Per Rectal was with no ballooning, no palpable mass and no active bleeding. X ray chest PA view was bilateral old kochs; X ray abdomen standing

was within normal limits; USG abdomen/pelvis was diffused, thickening of caecum and descending colon with narrowing of lumen. CECT abdomen/pelvis was significant other circumferential short bowel thickening seen in distal part of descending colon, portion of large bowel proximal to this thickening is dilated, there is mild thickening and oedematous changes in caecum and ascending colon.

Laboratory investigations

Hb- 11.2 gm%; TLC- 13800 cmm; Platelets- 193000 cmm; ESR-14 mm; LFT, KFT- within normal limits; Mauntoux test – negative.

Colonoscopy

Colonoscope could not be passed beyond distal part of descending colon s/o (?) extrinsic mass on descending colon.

Surgical procedure

Under spinal plus epidural anesthesia midline incision taken and peritoneum opened; evidence of grossly distended plastered large bowel with strictures near caecum and near splenic flexure of colon; No evidence of growth on liver surface; No evidence of LN involvement; Caecum, ascending colon, tranverse colon, decsending colon resected and ileo-sigmoid anastomosis done; Resected segment sent for HPE; Two ROMO ADK drains (no.32) were kept; Right drain in pelvic region and left drain in left paracolic gutter.

Post-operative orders (for first 24 hours)

Patient was kept nil by mouth and i.v. fluids-2 RL, 2 DNS, 1 NS; Injection meropenam 1 gm iv 8 hourly; Injection amikacin 500 mg iv 12 hourly; Injection metro 100 ml iv 8 hourly; Injection pantop 40 mg iv OD; Injection tramadol 1cc iv 8 hourly; Injection ondem 4mg iv sos, if vomiting.

On post op day 1

Rt drain 120 ml (serous); Lt drain 100 ml (serous); 1 PCV given. Epidural catheter was removed on day 3, bowel sounds were returned on day 4; liquids started on day 5; soft diet on day 7; right drain removed on day 7 and left drain removed on day 13.

Histopathology report

s/o well differentiated adenocarcinoma of caecum and descending colon with infiltration of 3/4 th thickness of muscle wall; surgical margins, mesocolic tissue and mesocolic LN are free from tumor cells. Suture removal done and patient discharged on post op day 18; As per HPE report patient was referred to oncologist for chemotherapy. 6 cycles of chemotherapy was given for

every 21 days. Patient was advised to follow up after completion of chemotherapy schedule. CECT abdo/pelvis done s/o no locoregional residual tumour, no evidence of distal metastasis or lymphadenopathy.

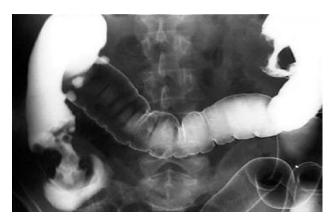


Figure 2: Barium enema showing apple core appearance near caecum and near splenic flexure of colon.

Management

- The choice of type of the operative procedure for synchronous colorectal carcinomas remains controversial.
- Some authors proposed radical operations such as subtotal colectomy to remove enough length of intestines and numbers of local lymph nodes to prevent future development of metachronous tumors.
- Some experts recommended a more conservative approach for older patients and radical procedures for younger patients with regionally confined nonmetastatic disease.
- We agree with the second opinion because major surgery such as subtotal colectomy could increase morbidity in older patients.(17% in patients with age >70 years compared to 3.4% in patients with age <50 years)

Prognosis

- In many studies, there was no difference in survival between the patients with primary multiple and single colorectal cancers and there were only minor differences in stage distribution.
- Postoperative survival in synchronous tumor cases was not worse than that in single tumor cases, and was mainly dependent on the pathological stage and curability of the index lesions.

Follow up

As total or subtotal colectomy performed for these patients, therefore the follow up will be by tumour markers, CT scan, PET scan and MRI.

CONCLUSION

With the development of early diagnostic technologies, more SCRCs have been diagnosed. Compared with the solitary CRC, SCRCs possess distinctive features. These should be fully investigated in preoperative clinical workups to avoid the possibility of accessory lesions being overlooked and not resected. Older male patients with concurrent adenomas in the colon are at a particularly high risk of SCRCs and close attention should be paid to them.

The extent of surgical resection relies on the location and the number of lesions. The resected region should include enough intestinal length and regional lymph nodes to capture any local spread, thereby guarding against recurrence or subsequent malignant change. Of course, the mechanisms regarding the formation of multicentricity need to be fully elucidated in order to gain insight into SCRC genesis and development, which will ultimately benefit molecular therapies and improve patient survival.

Funding: No funding sources Conflict of interest: None declared Ethical approval: Not required

REFERENCES

- 1. Jemal A, Siegel R, Ward E, Hao Y, Xu J, Thun MJ. Cancer statistics. CA: A Cancer Journal for Clinicians. 2009;59(4):225-49.
- Jessup JM, Menck HR, Fremgen A, Winchester DP. Diagnosing colorectal carcinoma: clinical and molecular approaches. CA: A Cancer Journal for Clinicians. 1997;47(2):70-92.
- 3. Peeters M, Haller DG, ALberts SR, Goldberg RM, Smith R. Therapy for early-stage colorectal cancer. Oncology. 1999;13(3):307-21.
- AJCC Cancer Staging Manual. 2002. Available at: https://cancerstaging.org/ references-tools/ deskreferences/ Documents/ AJCC 6th EdCancer Staging Manual Part1.pdf
- 5. Greene FL, Stewart AK, Norton HJ, Cohen AM. A new TNM staging strategy for node-positive (stage III) colon cancer: an analysis of 50,042 patients. Annals of Surgery. 2002;236(4):416-21.
- Hawk ET, Limburg PJ, Viner JL. Epidemiology and prevention of colorectal cancer," Surgical Clinics of North America. 2002;82(5):905-41.
- 7. Fante R, Roncucci L, Gregorio CD, Tamassia MG, Losi L, Benatti P, et al. Frequency and clinical features of multiple tumors of the large bowel in the general population and in patients with hereditary colorectal carcinoma. Cancer. 1996;77(10):2013-21,
- 8. Arenas RB, Fichera A, Mhoon D, Michelassi F. Incidence and therapeutic implications of synchronous colonic pathology in colorectal adenocarcinoma. Surgery. 1997;122(4):706-10.

- 9. Cunliffe WJ, Hasleton PS, Tweedle DEF, Schofield PF. "Incidence of synchronous and metachronous colorectal carcinoma," British Journal of Surgery. 1984;71(12):941-3.
- Brullet E, Montane JM, Bombardo J, Bonfill X, Nogue M, Bordas JM. Intraoperative colonoscopy in patients with colorectal cancer. British Journal of Surgery. 1992;79(12):1376-8.
- 11. Burns FJ. Synchronous and metachronous malignancies of the colon and rectum," Diseases of the Colon and Rectum. 1998;23(8):578-9.
- 12. Langevin JM, Nivatvongs S. The true incidence of synchronous cancer of the large bowel. A prospective study," American Journal of Surgery. 1984;147(3):330-3.
- 13. Neugut AI, Lautenbach E, Abi-Rached B, Forde KA. Incidence of adenomas after curative resection for

- colorectal cancer. American Journal of Gastroenterology. 1996;91(10):2096-8.
- 14. Passman MA, Pommier RF, Vetto JT. Synchronous colon primaries have the same prognosis as solitary colon cancers. Diseases of the Colon and Rectum. 1996;39(3):329-34.
- 15. Chen HS, Sheen-Chen SM. Synchronous and 'Early' metachronous colorectal adenocarcinoma: analysis of prognosis and current trends. Diseases of the Colon and Rectum. 2000;43(8):1093-9.
- Yang J, Peng JY, Chen W. Synchronous Colorectal Cancers: A Review of Clinical Features, Diagnosis, Treatment, and Prognosis, Dig Surg. 2011;28:379-85.

Cite this article as: Hake PD, Umale NP, Yadgire AU, Sarda KM. Synchronous colonic malignancy. Int J Res Med Sci 2016;4:4212-5.