Abdom Imaging 20:307–311 (1995)

Abdominal Imaging

Obstructive jaundice in gastric carcinoma: cause, site, and relationship to the primary lesion

Byung Hee Lee, Soo Yil Chin, Sun A. Kim, Kie Hwan Kim, Young Soo Do

Department of Diagnostic Radiology, Korea Cancer Center Hospital, 215-4 Gongneung-dong, Nowon-gu, Seoul, 139-240, Korea

Received: 5 May 1994/Accepted: 8 June 1994

Abstract

Objective: Obstructive jaundice is frequently present in patients with advanced gastric carcinoma. The purpose of this study was to assess the cause and preferential site of bile duct obstruction in patients with gastric carcinoma and to evaluate correlativity of biliary obstruction with the nature of the primary gastric lesion.

Methods: Cholangiographic findings of 54 patients with metastatic gastric carcinoma presenting with obstructive jaundice were reviewed retrospectively.

The level of the bile duct obstruction was divided into four segments: segment 1, from an individual intrahepatic duct to the biliary hilum; segment 2, common hepatic duct (CHD) involvement from the biliary hilum to the level of the cystic duct; segment 3, the proximal half of the common bile duct (CBD); segment 4, the distal half of the CBD. To evaluate the characteristics of the primary gastric lesion, operative records and pathologic findings were reviewed.

Results: Obstruction sites were segment 1 in eight patients (15%), segment 2 in 25 (46%), segment 3 in 17 (32%), and segment 4 in four (7%). The causes of obstruction were metastatic lymphadenopathy in the hepatoduodenal ligament (50 of 54) and direct invasion of the primary or recurrent tumor (four of 54). The location of the primary gastric lesions was the antrum, antrum and body, and body in 36 (67%), 17 (31%), and 1 (2%), respectively. Borrmann type 3 lesions were present in 72% of cases, and type 2 lesions in the remaining 24%. Histologic type was undifferentiated adenocarcinoma in 91% of patients, and differentiated adenocarcinoma in the remaining. Serosal invasion was shown in 96% of the patients.

Conclusion: Our results show that the cause of bile duct obstruction in advanced gastric carcinoma is predominantly metastatic lymphadenopathy in the hepatoduodenal ligament, and its preferential site is around the level of the cystic duct. Obstructing lesions showed characteristic cholangiographic findings.

Key words: Obstructive jaundice, cholangiography—Gastric carcinoma.

Correspondence to: B. H. Lee

Gastric carcinoma is one of the most common malignancies in Asian countries. Recent advances resulting in earlier detection of gastric carcinoma coupled with improved surgical and chemotherapeutic management have replaced many of the more traditional diagnostic and treatment regimens for gastric carcinoma. Nevertheless, many patients progress and present with evidence of recurrent or metastatic disease. Often, symptomatic obstructive jaundice is one of the most important clinical manifestations. It is generally considered that metastatic lymphadenopathy in the hepatoduodenal ligament (HDL) is a main cause of biliary obstruction [1–7]. However, diagnostic modalities including ultrasonography (US), computed tomography (CT), and magnetic resonance (MR) imaging have some limitations in evaluating the precise cause and site of bile duct obstruction [8–13]. To our knowledge, there has been little in the radiologic literature evaluating a prevalent site of bile duct obstruction and the risk factors associating the site of obstruction with the nature of the primary gastric tumor.

This study was designed to assess the prevalent site and cause of bile duct obstruction by cholangiographic findings, and to evaluate grade and location of the primary lesion in patients with metastatic obstruction.

Table 1. The site of biliary obstruction according to the risk factors of gastric carcinoma

Risk factors	Segment of bile duct obstruction				Total (N = 54)
	1 (N = 8)	2 (N = 25)	3 (N = 17)	4 (N = 4)	
Primary site					
Cardia, fundus	0	0	0	0	0
Body	0	1	0	0	1
Body, antrum	3	6	5	3	17
Antrum	5	18	12	1	36
Borrmann type					
1	0	0	0	0	0
2	1	8	3	1	13
3	7	15	14	3	39
4	0	2	0	0	2
Depth of invasion					
No serosal invasion	0	0	1	1	2
Serosal invasion	8	20	13	3	44
Invasion to adjacent structure	0	5	3	0	8
Histologic type					
Undifferentiated	8	23	14	4	49
Differentiated	0	2	3	0	5
Gastric resection					
No	3	7	0	1	11
Partial gastrectomy	3	16	14	3	36
Total gastrectomy	2	2	3	0	7

Materials and Methods

The study group included 54 patients with histologically proven gastric carcinoma, who subsequently underwent percutaneous transhepatic biliary drainage (PTBD) due to obstructive jaundice related to metastatic disease.

The patients ranged from 25–65 years of age (mean, 53 years), and included 34 men and 20 women. Pathologic proof of gastric adenocarcinoma was obtained by surgical exploration in 47 patients and endoscopic biopsy in seven. Distal subtotal gastrectomy was performed in 36 patients, total gastrectomy in seven, and palliative gastrojejunostomy in four. The time lag between the diagnosis or operation for gastric carcinoma and PTBD was 1 month to 6.5 years (mean, 15 months).

The cholangiograms performed via PTBD tube were reviewed, and the cause and site of bile duct obstruction were assessed.

The causes of bile duct obstruction were confirmed by exploratory laparotomy in 10 patients, endoscopy in four, and characteristic cholangiographic findings in the remainder. Exploratory laparotomy, which was performed in the cases with obstruction in segment 1 to segment 3 revealed metastatic lymphadenopathy in the HDL and lesser omentum.

The level of obstruction was divided into four segments by anatomic landmarks: segment 1, involvement from the individual intrahepatic ducts to the biliary hilum; segment 2, common hepatic duct (CHD) involvement from the biliary hilum to the level of the cystic duct; segment 3, involvement of the proximal half of the common bile duct (CBD) from the cystic duct to the upper margin of the pancreas; and segment 4, involvement of the distal half of the CBD, representing the intrapancreatic portion of the CBD.

To evaluate the characteristics of the primary gastric cancer, operative records and pathologic findings were analyzed with regard to the lesion site, macroscopic type, histologic type, and depth of invasion. The macroscopic features of advanced gastric carcinoma were categorized according to the Borrmann classification: type 1, a broad-base, protruded, polypoid lesion with less ulceration; type 2, a protruded or elevated lesion with ulceration and well-demarcated margins; type 3, a protruded or elevated lesion with ulceration and diffuse margins; and type 4, a diffuse, rather flat lesion with less ulceration [14].

Results

The level of the bile duct obstruction was segment 1 in eight cases (15%), segment 2 in 25 (46%), segment 3 in 17 (32%), and segment 4 in four (7%) (Table 1).

Cholangiographic findings tended to vary according to the level of obstruction. Most segment 1 obstructions showed smooth concentric luminal narrowings (Fig. 1). Segment 2 and 3 obstructions predominantly showed eccentric smooth round or oval-shaped defects on the right lateral wall of the bile duct, presumably due to extrinsic compression from enlarged periductal lymph nodes (Figs. 2 and 3A). This semilunar appearance was conspicuous during balloon dilation in all cases of segment 2 and 3 obstructions (Fig. 3B). All segment 4 obstructions showed encircling irregular defects with diffuse luminal narrowing in the distal CBD (Fig. 4), and all were confirmed to be serosal infiltration to the distal CBD and duodenum by endoscopic biopsy.

The primary lesions of the stomach were all advanced gastric carcinomas, and none were early gastric carcinomas. Most of the lesions involved the gastric antrum (98%), and there were no cases involving the cardia or fundus (Table 1).

Macroscopic types showed Borrmann type 3 in 39 patients (72%), type 2 in 13 patients (24%), and type 4 in two patients. There were no cases of type 1.

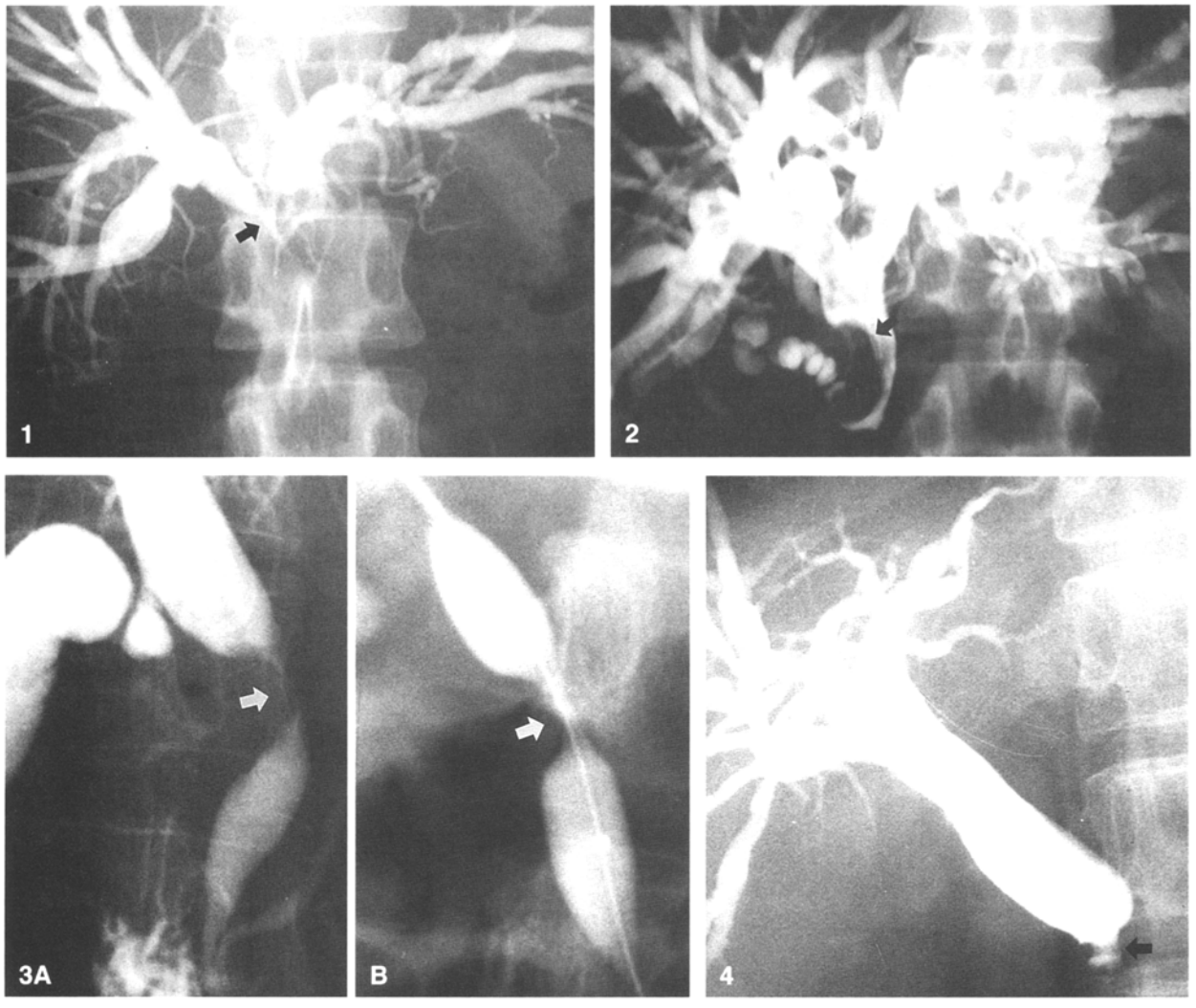

Fig. 1. Obstruction in segment 1. Cholangiogram shows smooth tapered luminal narrowing (*arrow*) in hilar portion of both intrahepatic bile ducts.

Fig. 2. Obstruction in segment 2. Cholangiogram shows smooth ovoid filling defect (*arrow*) on the lateral wall of the distal CBD. Multiple filling defects in both intrahepatic ducts are hematoma.

Fig. 3. Obstruction in segment 3. **A** Cholangiogram shows luminal narrowing in the proximal CBD (*white arrow*). **B** Ballooning at obstruction site demonstrates the extrinsic mass (*white arrow*) on the lateral wall of the proximal CBD.

Fig. 4. Obstruction in segment 4. Cholangiogram shows concentric irregular luminal narrowing in distal CBD (*arrow*).

Histologic differentiation of the primary cancer was undifferentiated adenocarcinoma in 49 patients (91%) and differentiated carcinoma in five (9%).

According to the depth of tumor invasion, 52 patients (96%) showed invasion to the serosa at operation.

Discussion

Gastric carcinoma is the most common malignant neoplasm in Korea accounting for 24% of all malignant neoplasms in the population [15].

Generally accepted important prognostic factors in gastric carcinoma are gross appearance, anatomic site, degree of local invasion, and lymph node metastases [4]. Additionally, there are five main patterns of recurrence after surgical removal of gastric cancer: lymph node (10%), local (10%), remnant stomach (8%), peritoneal (50%), and hematogenous recurrence (22%) [5].

Among them, obstructive jaundice is one of the most debilitating complications. Obstruction of the extrahepatic biliary tree may occur by metastatic replacement of periportal lymph nodes, or by direct neoplastic invasion from the primary or recurrent malignancy in the stomach [16]. The lymphatics in the gastrohepatic lig-

ament are frequently the site of metastasis from a gastric cancer. The HDL is the free edge of the gastrohepatic ligament, extending from the flexure between the first and second portion of the duodenum to the porta hepatis, and transporting the portal vein, hepatic artery, CBD, and lymphatic vessels.

In general, however, diagnostic modalities including US and CT have limitations in detecting the obstructive lesion, and in evaluating the precise obstruction level. Proximity to the duodenal bulb, together with obscuring gas, sometimes makes ultrasonic visualization difficult. The skill and experience of the ultrasonographer are also important factors in the detection of lymph nodes in the HDL [11]. Also, the HDL, containing the extrahepatic bile duct, is difficult to evaluate using axial CT because of its oblique orientation, anatomic variability, paucity of fat, and compact volume [17]. Therefore, we used cholangiograms to evaluate the cause and site of obstruction.

The Japanese Research Society for Gastric Cancer (JRSGC) reported 16 lymph node stations as possible locations of metastasis and classified them into three groups (N1–N3), which form the anatomic basis for systematic lymph node dissection in the surgical treatment of gastric cancer [7].

According to the JRSGC, lymph nodes related to obstruction of the extrahepatic biliary tree are stations 12 (lymph nodes in the HDL) and 13 (retropancreatic lymph nodes), which belong to the N3 node group (Fig. 5). The incidence of metastasis from gastric carcinoma to station 12 is from 5–8% with a 5-year survival rate of 9%, and to station 13 is 2% with a 5-year survival rate of 2%. There is, however, little correlation with positivity between stations 12 and 13, because they belong in a different lymphatic pathway [1, 4].

The Japanese literature has divided the lymph nodes in station 12 into five groups [7]: 12h, nodes in the porta hepatis; 12a, nodes along the left side of the hepatic artery; 12b, nodes along the right side of the bile duct; 12p, nodes along the posterior to the portal vein; and 12c, nodes along the cystic duct. Groups a, b, and p are further subdivided into 1 and 2, each according to the mid-point of the bile duct from the biliary hilum to the upper margin of the pancreas.

Among them, the groups resulting in obstructive jaundice are considered to be 12h, 12b, 12c, and 13. Stations 12h, 12b1, 12b2, and 13 match segments 1, 2, 3, 4 in our study, respectively.

In the cases with lymphadenopathy in stations 12b and 12c, the obstructing node demonstrates a characteristic cholangiographic feature that shows a sharply demarcated, eccentric round, or ovoid defect on the right lateral wall of the bile duct.

It was interesting that the cause of distal CBD obstruction was not metastatic lymphadenopathy in station 13, but direct invasion from recurrent cancer, which was proved by endoscopy.

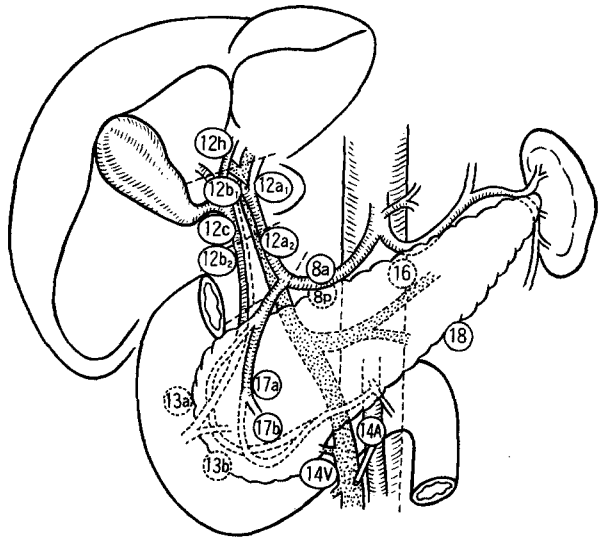

Fig. 5. Lymph node stations in a gastric cancer (cited from ref. [7]).

In general, unfortunately, group 12 nodes are not included in lymph node dissection at gastric resection. Recently, in some institutions including our hospital, radical lymph node dissection has been performed extending to the medial group of HDL, a1 and a2. It is, however, very difficult to dissect group b and c nodes due to technical inaccessibility.

In our study, patients with metastatic obstruction had a primary gastric cancer predominantly located in the antrum, and of Borrmann types 2 or 3 consisting of the undifferentiated cell type with serosal invasion. These risk factors are the same as previously reported factors related to peritoneal dissemination or hematogenous metastasis to major organs [5, 18].

In conclusion, lymphadenopathy in the HDL was the major cause of bile duct obstruction and was metastatic from predominantly advanced gastric carcinomas, with the preferential obstructing site at the level of the cystic duct which showed typical cholangiographic findings.

References

1. Maruyama K, Gunven P, Okabayashi K, Sasako M, Kinoshita T. Lymph node metastases of gastric cancer. *Ann Surg* 1989;210:596–602
2. Koga S, Kishimoto H, Tanaka K, Kawaguchi H. Clinical and pathologic evaluation of patients with recurrence of gastric cancer more than five years postoperatively. *Am J Surg* 1978;136:317–321
3. Wagner PK, Ramaswamy A, Ruschoff J, Schmitz-Moormann P, Rothmund M. Lymph node counts in the upper abdomen: ana-

tomical basis for lymphadenectomy in gastric cancer. *Br J Surg* 1991;78:825–827
4. Noguchi Y, Imada T, Matsumoto A, Coit DG, Brennan MF. Radical surgery for gastric cancer. *Cancer* 1989;64:2053–2062
5. Koga S, Takebayashi M, Kaibara N, et al. Pathological characteristics of gastric cancer that develop hematogenous recurrence, with special reference to the site of recurrence. *J Surg Oncol* 1987;36:239–242
6. Soga J, Ohyama S, Miyashita K, et al. A statistical evaluation of advancement gastric cancer surgery with special reference to the significance of lymphadenectomy for cure. *World J Surg* 1989;12:398–405
7. Japanese research society for gastric cancer. *General rules for the gastric cancer study,* 11th ed. Tokyo: Kanehara & Co. Ltd., 1985:4–29
8. Baron RL, Stanley RJ, Lee JKT, Koehler RE, Levitt RG. Computed tomographic features of biliary obstruction. *AJR* 1983;140:1173–1178
9. Honickman SP, Mueller PR, Wittenberg J, et al. Ultrasound in obstructive jaundice: prospective evaluation of site and cause. *Radiology* 1983;147:511–515
10. Lyttkens K, Forsberg L, Hederstrom E. Ultrasound examination of lymph nodes in the hepatoduodenal ligament. *Br J Radiol* 1990;63:26–30
11. Pedrosa CS, Casanova R, Rodriguez R. Computed tomography in obstructive jaundice. *Radiology* 1981;139:627–634
12. Pedrosa CS, Casanova R, Lezana AH, Fernandez MC. Computed tomography in obstructive jaundice. *Radiology* 1981;139:635–645
13. Gibson RN, Yeung E, Thompson JN, et al. Bile duct obstruction: radiologic evaluation of level, cause and tumor resectability. *Radiology* 1986;160:43–47
14. Borrmann R. Geschwulste des Magens. In: Henke FU, Lubarsch O, eds. *Handbuch der speziellen patologischen anatomie und histologie.* Berlin: Springer-Verlag, 1926:864–871
15. Ahn YO. Gastric cancer etiology and prevention. *J Korean Med Assoc* 1992;35:820–827
16. Ferrucci JT, Mueller PR, vanSonnenberg E. Transhepatic cholangiography. In: Berk RN, Ferrucci JT, Leopold GR, eds. *Radiology of the gallbladder and bile ducts.* Philadelphia: WB Saunders. 1983:347–350
17. Baker ME, Silverman PM, Halvorsen RA, Cohan RH. Computed tomography of masses in periportal/hepatoduodenal ligament. *J Comput Assist Tomogr* 1987;11:258–263
18. Yoshihiko M, Sunao M, Yoshihiro K, Shunji K, et al. Pertinent risk factors and gastric carcinoma with synchronous peritoneal dissemination or liver metastasis. *Surgery* 1991;110:820–823