LETTERS TO THE EDITOR
EDİTÖRE MEKTUPLAR
Smoking as an important factor increasing risk of *Helicobacter pylori*

Helicobacter pylori riskini yükselten önemli bir risk faktörü olarak sigara içimi

To the Editor

The prevalence of *Helicobacter pylori* as a major cause of gastroduodenal disease is rather high in developing countries (1, 2). *H. pylori* prevalence is known to be related with multiple factors (1-3). For many years, cigarette smoking has been regarded as an important risk factor for the development of peptic ulcer disease (4). Early studies showed that cigarette smoking was associated with increased prevalence of duodenal and gastric ulcers and delayed ulcer healing and increased the rate of ulcer recurrence following anti-secretory treatment (4, 5). Several mechanisms by which cigarette smoking adversely affects the gastric mucosa have been suggested (4, 5). Some studies have disputed the significance of continued smoking in ulcer relapse and have shown that treatment of infection produces a dramatically reduced relapse rate of peptic ulcer disease regardless of patients' smoking habits (6, 7). Although some studies showed an increased rate of *H. pylori* infection among smokers (8), other studies failed to find such a relation (4, 5, 9).

Table 1. Characteristics of patients with and without *Helicobacter pylori* infection with C–14 urea breath test

	***H. pylori* Positive (n=277)**		***H. pylori* Negative (n=78)**		
	(n)	**%**	**(n)**	**%**	***p***
Age (mean±SD)[a]	46.25±12.11		43.46±14.04		0.091
≤ 24	11	55.0	9	45.0	**0.042**
25-44	114	82.0	25	18.0	
45-64	138	80.7	33	19.3	
≥ 65	19	76.0	6	24.0	
Sex					
Male	134	82.7	28	17.3	0.161
Female	148	76.7	45	23.3	
Education status					
Illiterate	18	85.7	3	14.3	0.466
Primary	98	81.7	22	18.3	
Middle-high school	66	81.5	15	18.5	
University	100	75.2	33	24.8	
Marital status					
Married	240	81.4	55	18.6	**0.047**
Single	42	70.0	18	30.0	
Dyspepsia					
Yes	232	89.9	26	10.1	**0.000**
No	50	51.5	47	48.5	
Daily cigarette consumption (number)					
No	107	70.9	44	29.1	**0.005**
1-10	60	87.0	9	13.0	
11-20	86	83.5	17	16.5	
21 and over	29	90.6	3	9.4	

[a] Student-t test was used; chi-square test was used in the others.

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Our aim in this study was to determine *H. pylori* existence by C-14 urea breath test (UBT) and to evaluate the association with smoking, dyspeptic symptoms and some sociodemographic statuses using a questionnaire. Three hundred and fifty-five outpatients who were examined for routine check-ups were included in the study between May-September 2005. The study was approved by the Selcuk University Medical Faculty Ethics Committee, and written consent was obtained from patients.

The characteristics of patients with and without *H. pylori* infection with C–14 UBT are shown in Table 1. Table 2 demonstrates logistic regression analysis results for factors affecting *H. pylori* frequency in our patient group. Although analysis with chi-square test revealed a lower ratio of *H. pylori* positivity for the patients younger than 24 years old, this effect was not compatible with logistic regression test results. Educational status was negatively associated with *H. pylori* frequency in our patient group. The odds ratio of illiterate cases in the patient group was 4.49 with a 95% confidence interval (CI) of 1.05-19.30. In other words, illiterate patients had 4.5-fold increased frequency rates of *H. pylori* compared to the group of patients with university education. The odds ratios (95% CI) of dyspepsia symptoms in the patients was 8.20 (4.40-15.27) and the patients who had dyspeptic symptomatology had approximately 8-fold increased *H. pylori* frequency compared to the group of patients who did not. Smoking status and the amount of smoked cigarettes per day were positively associated with *H. pylori* frequency. The odds ratios (95% CI) according to consumption of cigarettes per day in the smoker groups (divided as 1-10, 11-20, and 21 and over per day) were 2.46 (1.02-5.10), 1.68 (0.80-3.54), and 5.21 (1.29-21.00), respectively. The smoker group had an approximately 2.5- to 5-fold increased risk of *H. pylori* existence than non-smokers (95% CI; 1.02-21.0) (Table 2).

Table 2. A logistic regression analysis of factors affecting presence of *H. pylori*

Variable	Odds ratio	*p*
Sex	0.768 (0.401–1.469)	0.425
Age		
≤ 24	1.000	0.707
25-44	1.579 (0.446-5.597)	0.479
45-64	1.231 (0.341-4.444)	0.751
≥ 65	0.858 (0.180-4.083)	0.847
Education status		
University	1.000	0.240
Illiterate	4.492 (1.046-19.301)	**0.043**
Primary	1.262 (0.605-2.635)	0.535
Middle-High School	1.324 (0.595-2.944)	0.491
Marital status	0.584 (0.251-1.362)	0.213
Dyspepsia	8.201 (4.403-15.275)	**0.000**
Cigarette consumption (per day)		
No	1.000	**0.043**
1-10	2.46 (1.022-5.995)	**0.045**
11-20	1.683 (0.800-3.542)	0.170
21 and over	5.214 (1.295-20.982)	**0.020**

In our current study, while *H. pylori* existence was positively related with smoking and dyspepsia, a negative relation was determined with advanced educational status. The risk of *H. pylori* was dependent on the amount of cigarette consumption. This chaotic relationship between two important carcinogens, *H. pylori* and cigarettes, may be broken, either with smoking cessation or by combating smoking epidemics.

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Coincidental occurrence of granular cell tumor and gastrointestinal stromal tumor in a patient

Granüler hücreli tümör ve gastrointestinal stromal tümörün bir hastada rastlantısal birlikteliği

To the Editor

A yellow-colored firm-sessile polyp was excised from the sigmoid colon of a 61-year-old male patient during colonoscopy. Histopathological evaluation yielded granular cell tumor (GCT). Four months later, non-colicky left lower quadrant abdominal pain complaint began and an abdominal mass was palpated, which was also detected on ultrasound and magnetic resonance (6x8.5x10 cm) as well as multiple irregular cystic liver lesions. With laparotomy, a proximal ileal mass and the biggest cystic lesion of the liver were excised. Histopathologic diagnosis of mass was gastrointestinal stromal tumor (GIST) with high malignant potential. Pericentral sinusoidal dilatations and thinning of parenchymal liver cordons were detected in liver specimen.

GCT has been seen in numerous locations such as tongue, mediastinum, skin, vulva, breast, larynx, bronchus, pituitary gland and other sites (1). Gastrointestinal involvement is uncommon (5–9%), mostly in esophagus, duodenum, anus and stomach, rarely in rectum and sigmoid (2). Presence in sigmoid colon is very rare in the literature and only a few reports were abstracted (3). GCTs are mostly benign (96–98%) and endoscopic removal is sufficient. Regarding the histogenesis of GCT, most authors favor a Schwann cell origin; however, in some lesions, there is no evidence of Schwann cell participation. The GCTs characteristically react with S–100 and neuron-specific enolase, both of which are Schwann cell markers (4). Immunohistochemically, the patient's specimen stained positively with S–100 (Figure 1). There was no staining with desmin, smooth muscle actin (SMA), CD117 and CD34. Ki-67 proliferative index was 1%.

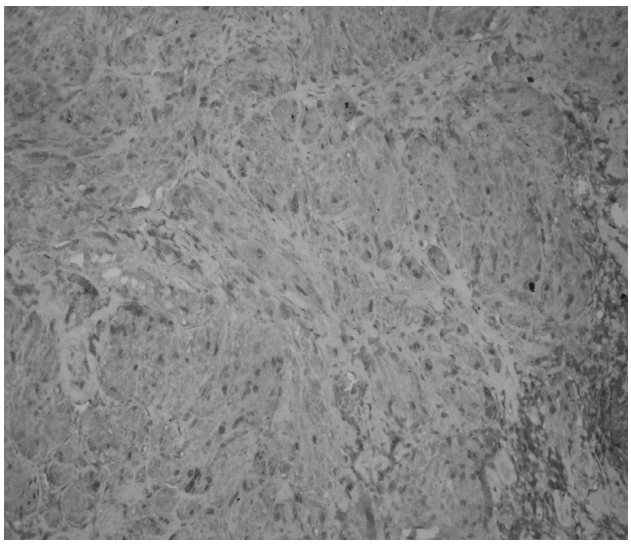

Figure 1. Immunohistochemically positively stained with S–100 of granulary cell tumor.

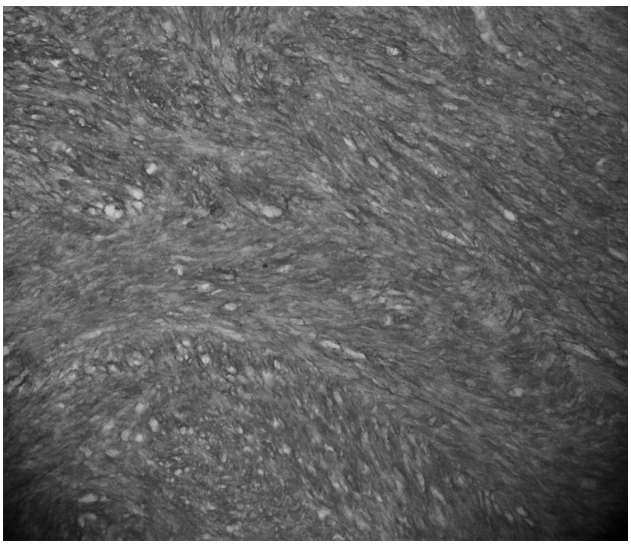

Figure 2. CD34 Stain of ileal gastrointestinal stromal tumor.

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GISTs are rare mesenchymal tumors (10–20/106), mostly found in stomach, small intestine, and colon. One-third of GISTs are malignant. GISTs are believed to arise from intestinal pacemaker cells of Cajal. Most GISTs express c-KIT, a type III tyrosine kinase receptor, which has a role in the development of GISTs. KIT is important for gametogenesis, hematopoiesis, melanogenesis, mast cell growth and differentiation and development of interstitial cells of Cajal (5). Spindle cells of the small intestinal mass stained positively with c-KIT (67%) and CD34 (100%) (Figure 2) and stained focal positively with SMA; there was no staining with S–100, desmin, or Ki–67.

GCTs and GISTs are rare tumors that can bear malignant potential. Tumor size, mitotic count, and presence of necrosis and hemorrhage in lesion are the important factors for progression to malignancy (6). Simultaneous presence of GCT and GIST in the same patient is extremely rare. Only one case report was detected in MEDLINE (7). Sigmoid colonic location of the GCT and simultaneous presence of this tumor with GIST in a patient is an extremely rare finding in the literature.

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Gastrointestinal sarcoidosis mimicking colonic cancer

Kolon kanserini taklit eden gastrointestinal sarkoidozis

To the Editor

Sarcoidosis is a chronic multisystemic granulomatous disease with unknown etiology. The characteristic histological lesions are noncaseating granulomas containing multinucleated giant cells in the absence of tuberculosis, fungal infections, and foreign bodies. Although disease is frequently diagnosed in the lungs, mediastinal lymph nodes, skin and eyes, it rarely involves the esophagus, small bowel and colon. However, the stomach is one of the gastrointestinal organs in which sarcoidosis is

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frequently diagnosed (1, 2).

A 49-year-old woman presented with watery diarrhea for two months. The diarrhea was mild to six watery stools per day without mucus and blood, and occasionally awoke her from sleep. Her past medical history showed total abdominal hysterectomy and bilateral oophorectomy for cervical cancer 14 years earlier for which she was treated with radiotherapy and chemotherapy. During the postoperative follow-up, her chest X-ray revealed pulmonary mass and she was diagnosed with metastases to the lung. A scalene lymph node biopsy revealed noncaseating granuloma with multinucleated giant cells and no tuberculosis bacilli; she was diagnosed with pulmonary sarcoidosis in 1999. After diagnosis of pulmonary sarcoidosis, she was treated with a tapering dose of oral methylprednisolone and inhaler budesonide with a complete symptomatic remission. She was asymptomatic until two months before presentation of new-onset diarrhea.

On examination, there was no palpable lymphadenopathy. Chest auscultation revealed rare dry rales bilaterally, and cardiac examination was normal. There was no abdominal mass, organomegaly or ascites. Tumor markers including CEA, CA19-9, and CA 125 were normal.

Results of routine hematology and biochemistry investigations were all within normal limits except for mildly elevated alkaline phosphatase (279 U/L), total cholesterol (241 mg/dl), triglyceride (265 mg/dl) and glucose (146 mg/dl). Barium enema examination showed traction toward left side to the hepatic flexure of colon. Abdominal ultrasonography showed mild hepatomegaly, multiple periportal lymph nodes and pseudo kidney appearance (diameter 6x4 cm) in the right upper quadrant. Hence, diagnosis of right colon cancer was considered.

Abdominal computed tomography (CT) showed a few lymph nodes in the hepatic hilus, calcifications of the abdominal aorta and diffuse thickening of the rectum wall.

Colonoscopy revealed mucosal edema and hyperemia, loss of vascularity, rare ulcers, and submucosal hemorrhage from the rectum to the cecum; no obstructive lesions were observed on sides of the colon. Biopsy specimens obtained from various sites of the colon showed noncaseating granuloma with multinucleated giant cells with typical sarcoidosis (Figure 1). Search for foreign bodies and special stains for tuberculosis were all negative. Her upper gastrointestinal endoscopy showed mucosal edema and hyperemia of the antrum and corpus and also upper part of the duodenum. Histology of gastric biopsy showed noncaseating granuloma with multinucleated giant cells (Figure 2). Biopsy of duodenum was normal. Small bowel follow-through X-ray was normal.

A diagnosis of gastrointestinal sarcoidosis was made, and the patient was treated medically with oral prednisolone (30 mg/day). One month after the initiation of medical therapy, diarrhea disappeared and the prednisolone dose was tapered to a maintenance dose of 5 mg/day with a sustained symptomatic remission.

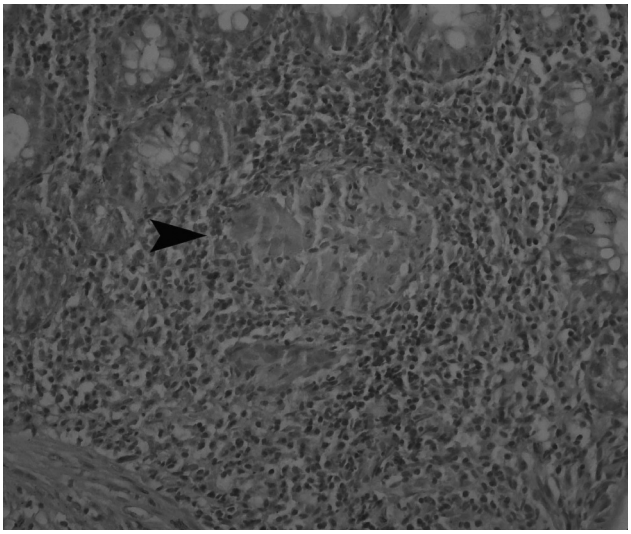

Figure 1. Noncaseating epithelioid granuloma and multinucleated giant cells in the colonic mucosa (hematoxylin and eosin [HE], X100).

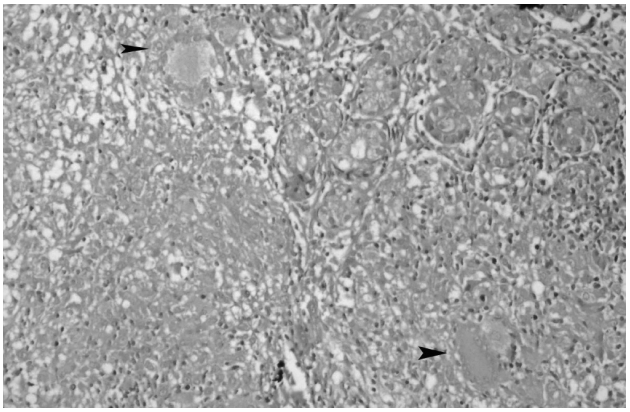

Figure 2. Noncaseating epithelioid granuloma with multinucleated giant cells in the gastric mucosa (HE, X100).

Interestingly, the diagnosis of sarcoidosis in our patient was made when investigating pulmonary mass on the chest X-ray. In the literature, there are reports of multi-organ involvement, especially in non-respiratory sites, which can confuse the clinical presentation or delay diagnosis. Prior diagnosis of malignancy creates more diagnostic uncertainty (1). Clinicians must be alert to atypical cases and unexpected variations in the presentation of sarcoidosis.

While involvement of the liver and spleen is common, other gastrointestinal manifestations of sarcoidosis are very rare (2, 3). Gastric sarcoidosis may manifest as ulcerative lesions, cone-shaped antral deformities, polyposis, diffuse nodular lesions, or a linitis plastica type lesion (4-6). Our patient's gastroscopy showed no specific findings. Colonic lesions may present as a colonic mass, polyposis or colitis (7-9). Our case presented as granulomatous colitis. Tuberculosis was ruled out in our case by special stains. The histological differentiation of Crohn's disease and systemic sarcoidosis is not simple. Nonnecrotizing granulomas of the entire digestive tract can be found in both conditions (10). In the present case, the previous granulomatous involvement of her scalene lymph node and pulmonary involvement and extreme responsiveness to corticosteroid therapy could not be explained by Crohn's disease. Steroids have been used with success in gastrointestinal sarcoidosis. The ultimate duration of therapy has not been reported.

If patients with diagnosis of pulmonary sarcoidosis have gastrointestinal symptoms such as diarrhea or abdominal pain, they should be evaluated for gastrointestinal sarcoidosis.

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Prevalence of cholelithiasis in a Turkish population of end stage renal failure patients and related risk factors: Experience of a center from Turkey

Son dönem böbrek yetmezlikli Türk hasta popülasyonunda safra kesesi taşı sıklığı ve ilişkili risk faktörleri: Türkiye'de tek merkez deneyimi

To the Editor

Low protein diet is traditionally used to delay progression of chronic renal failure (CRF) to end stage renal disease (ESRD) (1). Patients on hemodialysis (HD) therapy were found to have increased bile cholesterol and an increased saturation index in bile, i.e. changes implying increased risk of cholelithiasis. These changes were further enhanced by the effect of low protein diet (2). In this study, we aimed to evaluate the incidence of cholelithiasis in patients with ESRD (CrCl <10 ml/min) receiving or not receiving HD therapy. Furthermore, we analyzed the risk factors causing gallstones.

Fifty-eight ESRD patients not receiving HD therapy and 146 patients receiving HD were included into the study. Of the 204 patients, 91 (44.6%) were female and 113 (55.4%) were male. Twenty-nine patients (19 female, 10 male) were found to have biliary lithiasis with an overall prevalence of 14.2% (20.9%, 8.85%), which was 3 times higher in women and 2.5 times higher in men and twice as high as in the general population (3, 4).

Similar to the study of Li Vecchi et al. (5), the duration of HD did not influence the occurrence of gallstones. Furthermore, HD itself did not affect gallstone formation, similar to results of Bektaş et al. (6).

It is well known that the prevalence of cholelithiasis increases with age (7). Patients were divided into 10-year groups and prevalence of biliary stones in the various age groups was determined. Prevalence reached maximum frequency in the 5th decade of life ($p<0.05$).

Compared to the nonbiliary lithiasis group, age, body mass index (BMI) and parity were significantly higher in the biliary lithiasis group ($p<0.05$) (Table 1).

Table 1. Demographic characteristics and some laboratory parameters in CRF patients with and without gallstone disease

Cholelithiasis	Gallstone Disease (+)	Gallstone Disease (-)	P
Sex (F/M)	29 (19/10)	175 (72/103)	P=0.014
Age (years)	57.1±11.59	53.8±14.54	P=0.031
Duration of hemodialysis (months)	27.9±26.28	31.3±42.17	NS
PTH (pg/ml)	182.9±228.9	16.9±177.7	NS
BMI (kg/m^2)	25.4±4.2	23.6±3.5	P=0.031
Cholesterol (mg/dl)	176.9±34.27	176.4±48.47	NS
Triglyceride (mg/dl)	160.9±48	167.9±71.77	NS
Uric acid (mg/dl)	6±1.2	5.7±0.9	NS
Ca (mg/dl)	9.2±1.17	9±1.2	NS
P (mg/dl)	5.3±1.2	5.7±1.8	NS
CaxP	48.27±10.8	51.68±15.8	NS
Parity	5±2.26	3.5±2.23	P=0.007

PTH: Parathormone. BMI: Body mass index. Ca: Calcium. P: Phosphorus.

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Among the other potential risk factors, there was no association between serum concentrations of Ca (calcium), P (phosphorus), CaxP product, serum lipid levels, uric acid, parathormone (PTH) and oral contraceptive use and prevalence of gallstone disease.

In the present study, BMI and parity were important risk factors for gallstone disease. Pregnancy is one of the risk factors held responsible for increased gallbladder stones in females (7).

In conclusion, HD therapy had no effect on prevalence of gallstone disease. Female sex, parity and BMI seemed to be important risk factors for development of gallstones in patients with CRF. Further studies in large series of patients are necessary to further clarify this issue.

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Polypoid lesions of the gallbladder smaller than 10 mm

10 mm.den küçük safra kesesi polipleri

To the Editor

With the wide use of ultrasonography (USG) in recent years, the detection rate for polypoid lesions of the gallbladder (PLG) has increased up to 4-7% (1-2). Because of the risk of malignancy, cholecystectomy is indicated in polyps exceeding 10 mm in diameter (3,4). On the other hand, asymptomatic patients with polyps smaller than 1 cm usually require repeated USG and follow-up (5). In symptomatic patients with PLG smaller than 1 cm, cholecystectomy is recommended. Our aim was to investigate the relation among the symptoms, gallstones, and histopathological changes of the gallbladder in patients with PLGs smaller than 1 cm.

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Between 2000-2006, 19 patients (5 male, 14 female) underwent cholecystectomy because of PLGs smaller than 1 cm in Suleyman Demirel University General Surgery Department. All patients had symptoms such as abdominal pain, episodic vomiting, bloating, fatty food intolerance, and dyspepsia. No pathologic finding could be detected in upper gastrointestinal endoscopic examination that could be related with these symptoms.

Laparoscopic cholecystectomy was performed in all patients and all materials were examined histopathologically.

Mean age of the patients was 41.89 (range 23-66) years. The size of the polyps ranged between 0.4-9.2 mm and seven patients had multiple polyps. Histopathological analysis of all resected gallbladders revealed chronic cholecystitis. Cholelithiasis was detected in only three patients (Table 1).

In the case of symptomatic patients with PLG, there is no discussion on the necessity of surgery, regardless of the size of the polypoid lesion. Our results suggest that symptomatic gallbladder polyps smaller than 10 mm do not correspond to adenocarcinoma. Chronic cholecystitis was detected in histopathological examination of all resected gallbladders, but only three of our patients had gallstones. Symptoms in patients with PLG cannot be simply explained by concurrent gallstones. It was also reported that the presence of gallstones increased the rate of symptoms in patients with benign PLGs (6), and another study showed that 42 of 45 patients with PLG had resolution of symptoms after cholecystectomy (7). In our series, the symptoms vanished in all of our patients postoperatively.

In conclusion, although the population of the study was too small, these results indicate that there was no relation between small polyps and cholelithiasis, and the symptoms of small polyps could not be explained by gallstones. On the other hand, chronic cholecystitis accompanied all PLGs smaller than 1 cm in symptomatic patients.

Table 1. Ultrasonography findings and pathological features of patients

	Age	Gender	Ultrasonography	Postoperative Pathology	Cholelithiasis
1	39	M	9.2 mm polyp	Chronic cholecystitis, cholesterol polyp	-
2	46	F	Multiple polyps, largest 5 mm	Chronic cholecystitis, cholesterol polyp	-
3	40	F	Multiple polyps, largest 6.5 mm	Chronic cholecystitis	-
4	45	F	9 mm polyp	Chronic cholecystitis	-
5	66	F	8 mm polyp	Chronic cholecystitis	-
6	46	M	Multiple polyps, largest 2 mm	Chronic cholecystitis, adenoma, focal dysplasia	-
7	30	F	3.6 mm polyp	Chronic cholecystitis	-
8	36	M	Multiple polyps, largest 3 mm	Chronic cholecystitis, cholesterol polyp	-
9	38	F	3 mm polyp	Chronic cholecystitis, cholesterol polyp	-
10	41	F	Three polyps, largest 2.1 mm	Chronic cholecystitis, cholesterol polyp	-
11	45	M	3.5 mm polyp	Chronic cholecystitis, cholesterol polyp	-
12	31	F	9 mm polyp	Chronic cholecystitis, cholesterol polyp	-
13	47	F	Multiple polyps, largest 4.5 mm	Chronic cholecystitis, cholesterol polyp	-
14	48	F	5 mm polyp	Chronic cholecystitis, cholesterol polyp	-
15	44	F	Two polyps (5.1 and 3.2 mm)	Adenoma, chronic cholecystitis, cholelithiasis	+
16	23	F	7 mm polyp	Chronic cholecystitis, Cholesterol polyp	-
17	45	F	5 mm polyp	Chronic cholecystitis, cholesterol polyp, cholelithiasis	+
18	55	M	3 mm polyp	Chronic cholecystitis, cholesterol polyp, minimal dysplasia in surface epithelium, adenomyoma, cholelithiasis	+
19	31	F	0.4 mm polyp	Chronic cholecystitis, cholesterol polyp	-

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