Early Neoplasias of the Gastrointestinal Tract

Endoscopic Diagnosis and Therapeutic Decisions

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Early Neoplasias of the Gastrointestinal Tract

This book is dedicated to Dr. Shin-ei Kudo, Haruhiro Inoue, Yasushi Sano and Shinji Tanaka as exponents of the generation of originary researchers in Japan that has developed image-enhanced endoscopic analysis of early gastrointestinal cancers.

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Preface

We only find what we're prepared to look for

Cancer has traditionally been defined by proof of invasively growing dysplastic epithelia in the Western world, and gastrointestinal oncology has attempted to beat cancer at the invasive stage. Any mucosa-invasive lesion (pM2/3) must yet have a non-invasive precursor lesion. In Japan, therefore, cancer has been defined by cytologic criteria – severely dysplastic epithelial cells – allowing earlier diagnosis in even the pre-invasive stage. Gastrointestinal oncology has emphasized in Japan the concept of early cancer certainly curable by resection, and endoscopic diagnostics have been pushed to detect the earliest, barely visible intraepithelial neoplasias. Hence, in many centers in Japan, more than 70 % of GI cancers are now diagnosed as early cancers, whereas much less (<40 %) in Western countries.

For more than a generation, early diagnosis of cancer has been core research promoted by National Societies for Gastric, Esophageal, and Colorectal Cancer in Japan. Enormous data has accumulated on early cancers resected for cure with extended lymphadenectomy and specimens precisely measured for mucosal or submucosal invasion. Surface microscopic measurements systematically characterized surface structure of early cancer and adjacent mucosa. At the same time, Japan became leading in image-enhanced as well as magnification endoscopy on a superior technical level. Successful research followed on how to accurately predict the histologic type of neoplasias from the endoscopic aspect of mucosal surface and microvascular architecture. Research defined organ-specific criteria for curative snare-resection of small early mucosal cancers, and developed endoscopic electrosurgery – endoscopic submucosal dissection, ESD – in order to resect wider spreading mucosal cancer. Now, Japan's experts mastermind enhanced endoscopic diagnostics and electrosurgery for early cancers.

This is the start of a new era in gastrointestinal oncology and the time to have it transferred from East Asia to the Western world. Western endoscopists are all fascinated by the ease and skill how to resect mucosal cancers with ESD and wish to perform ESD as well. Frankly, diagnosis must always precede treatment – an old

clinical rule – and half of the success of any operator is credited to his competence in diagnosis and decision making before operation. Nevertheless, it takes a sustained training effort to achieve competence and skills to analyze stage and lateral spread of early neoplasias almost as accurately as leading experts from Japan. This book attempts now to convey this endoscopic knowledge and skills also to Western endoscopists, in order to enhance detection and diagnostic accuracy for early gastrointestinal neoplasias.

Based on cooperation with the inventors of hook and dual knife, Drs. Oyama and Yahagi, the co-editors had for the past 5 years the privilege to organize annual training in ESD techniques, and courses in advanced endoscopic detection and decision-making for resection of early GI cancers. Their guidance inspired us to compile the most common endoscopic classifications and diagnostic approaches to early neoplasias.

The aim of this book is to raise the detection rate of minute cancers, size less than 5 mm, and the diagnostic competence for decision making on the resection strategies. Nevertheless, the text is basic and useful for both, to update interventional endoscopists as well as to educate novices striving for endoscopic skills. Those involved in training for clinical ESD should thoroughly sharpen their diagnostic repertoire and proceed to the atlas *Endoscopic Diagnosis of Gastric Adenocarcinoma for ESD* by Tsuneo Oyama. Within the past year, high-end magnifying and image-enhancing endoscopes as good as in Japan have become available all over the Western world. We hope this book comes in time to spur sustainable enthusiasm for accurate image-enhanced magnifying endoscopy of early gastrointestinal cancers. May the effort serve the needs of the patients, and lead the art and science of endoscopy ahead to battle cancer.

Salzburg, Austria
January 26, 2014

Frieder Berr, on behalf of the editors

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The Editors

Contents

Part I
General Principles of Endoscopy for Early Gastrointestinal Neoplasias

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Chapter 1
Endoscopic Screening and Surveillance: Indications and Standards

Frieder Berr, Thierry Ponchon, and Tsuneo Oyama

1.1 Introduction

The gastrointestinal (GI) tract is the organ system bearing the highest cancer incidence ($1.0–1.4 \times 10^3$) and mortality ($0.7–0.9 \times 10^3$ per 10^5 and year). Annual mortality-to-incidence ratio ranges from 42 % for colorectal cancer to 82 % for esophageal cancer and exceeds 66 % for gastric cancer in the West but has fallen below 40 % in Japan [1, 2]. Curative radical surgery with complete removal of first- and second-tier lymph nodes for early gastric cancer (≤pT1) achieved 5-year overall survival rates (OS) exceeding 90 % [3, 4]. Endoscopic resection en bloc yielded comparable 5-year OS (92–93 % without mortality from cancer) for early gastric cancers selected according to the criteria of the Japanese Gastric Cancer Association or expanded criteria of National Cancer Center (NCC), Tokyo [5, 6].

Early GI cancers mostly (>95 %) show differentiated grading, except gastric cancer (in only ~60 %). Early cancer when differentiated (HGIN, G1, G2) progresses slower to systemic disease, e.g., within 3 years, than undifferentiated cancer [3]. This allows some time for detection of early cancer – as necessary for screening and surveillance programs.

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1.2 Rationale for Endoscopic Screening and Surveillance

Detection of precursors or cancer in *early stage* (pT1a, differentiated grade) is critical to reduce mortality from gastrointestinal cancers. Endoscopy is best for detection of early GI cancer and precursor lesions, much better than fecal screening for occult blood loss or attempts of serum screening tests [7–10]. Endoscopic screening of the population aims to reduce mortality from frequent GI cancers. Beyond the average risk of GI cancers in the general population, there are many individuals with high-risk profile depending on environmental factors (e.g., carcinogen exposure, smoking, alcohol abuse) or/and individual disposition (familial inheritance, chronic GI inflammatory diseases). Such individuals require opportunistic screening endoscopy earlier in life and surveillance at more frequent intervals than the general population [8, 10–14]. However, even in specialty practice up to 39 % of patients had CRC screening without taking the risk profile and family history, and 55 % of patients with strong family history had received inappropriate screening and surveillance [11]. And after complete resection of early cancer or precursor lesion, the interval for follow-up endoscopy depends on the risk for recurrence [8–10].

Note
Taking the history of carcinogenic risk factors including family history is a prerequisite for any screening endoscopy as well as for scheduling follow-up examinations (endoscopic surveillance).

Colorectal cancer (CRC) is the third most common cause of cancer-related death worldwide ranking second in Western countries and third in Japan [15], with similar yearly incidence rates (cases/100,000/year) in the USA (range 28–38), Western Europe (33–50), and Japan (22–58) [1, 7, 15, 16]. In the US National Polyp Study, the incidence rate of CRC was much lower after clearing colonoscopy (with resection of all neoplasias) than predicted from the US population [14]. This delivered the rationale for nationwide colonoscopy screening programs in many countries, to reduce mortality from CRC.

Gastric cancer is frequent in Japan (incidence ~25/100,000/year) justifying screening of the general population [15, 16]. Screening endoscopy is recommended to start at the age of 40 years and has decreased cancer-related mortality [17–19]. The incidence of gastric cancer (GC) also is high in China, Chile, and Eastern Europe [1, 16, 17, 20]. However, in most Western countries, GC is too rare (e.g., ≤5/100,000/year in the USA) to start an endoscopic screening program [1, 12, 16]. Nowadays, the epidemiology of gastrointestinal cancers is becoming more similar in Japan and Western countries because of a global trend for similar lifestyle and nutrition, rising prevalence of chronic gastroesophageal reflux disease, and rapidly declining prevalence of *Helicobacter pylori* infection. In Western countries, evidence can be claimed

for endoscopic surveillance of Barrett's esophagus to detect early malignancies [12]. As endoscopic screening and surveillance for GI cancers is an evolving topic, we emphasize to refer to your national guidelines.

1.2.1 Screening Colonoscopy for Prevention of CRC

1.2.1.1 Colonoscopy

Colonoscopy performed in due quality is the *best diagnostic standard* for detection of neoplasias in the entire colon [8, 10, 17] – and combined with polypectomy of all detected adenomas (clearing colonoscopy) reduces the risk of colon cancer by 66–71 % for 10 years after colonoscopy [14]. Annual fecal occult blood test (FOBT) screening reduced this risk by 23 %, because one-third of participants received colonoscopy (and polypectomy of neoplasias) [21]. The risk of complications of screening colonoscopy is low (overall 0.39 % for diagnostic, 1.02 % for therapeutic screening colonoscopy; mortality 1:150,000) [10, 22, 23].

Note
Recommendations for asymptomatic, average-risk individuals:

- Age ≥50 years [≥40 years in Japan] → screening colonoscopy (every 10 years)
- [Aim: prevention and early detection of colon cancer]
- If not → annual FOBT → colonoscopy, if FOBT is positive
- [Aim: (early) detection of asymptomatic colon cancer] [10, 23]

1.2.2 Individuals with Increased Risk for Colorectal Cancer

Approximately 75 % of CRC occur sporadically in average-risk individuals and up to 25 % in persons with positive family history (FH) for colon adenomas or cancer, i.e., increased risk profile [8, 10, 17]. Monogenic autosomal dominant inherited familial cancer syndromes account for less than 10 % of all CRC – familial adenomatous polyposis coli (FAP) for 1 % and hereditary nonpolyposis colon cancer (HNPCC) for 5 % – and another 15–20 % of all CRC cases report colon cancer or adenomas in the family history (FH) [24]. The lifetime risk for CRC ranges from 60 to 80 % with HNPCC and is up to nearly 100 % with classical FAP [>100 colon adenomas] by age 40–50 years, and the onset is at young age [13, 25] (Fig. 1.1). Attenuated FAP (with less adenomas [10–99] and later onset) is suggested by the following criteria: (a) at least 2 FDRs with 10–99 adenomas at the age >30 years (none under age 30 years) or (b) one FDR with 10–99 adenomas and one FDR with

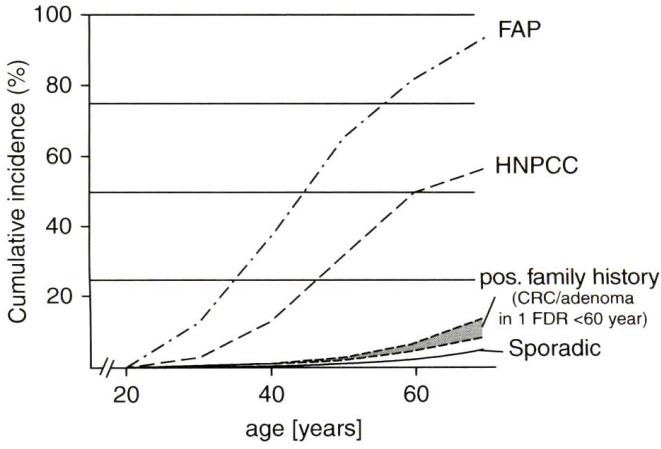

Fig. 1.1 Cumulative incidence of CRC by age in different risk groups: FDR, first degree relative; HNPCC, hereditary non-polyposis coli cancer; FAP, familial adenomatous polyposis coli (Modified acc. to Winawer et al. [23]) (Permission granted by AGA Institute, W.B. Saunders Comp)

Table 1.1 Individuals with increased risk for CRC

Condition	Reference
Family history (FH) of colon adenoma or carcinoma	[25, 30]
Hereditary colorectal carcinomas (rapid progression adenoma → carcinoma)	
HNPCC, autosomal dominant	[24, 26]
FAP, autosomal dominant	[10, 24]
MAP (MUTYH-associated adenomatous polyposis), autosomal recessive	[10, 24]
Peutz-Jeghers syndrome (PJS)	[26, 27]
Familial juvenile (hamartomatous) polyposis (FJP)	[26]
Chronic inflammatory bowel disease (UC, Crohn's colitis)	[28, 29]
Surveillance after polypectomy or surgery for CRC	See [10, 24]

CRC and few adenomas. There is a 25 % chance of identifying an APC mutation in this attenuated FAP syndrome [13]. A very rare form of adenomatosis coli (10–>100 adenomas) manifested *before* the age of 30 years is MAP (MUTYH-associated adenomatous polyposis), an autosomal recessive disorder due to biallelic MUTYH mutations. MAP persons show predilection of CRC in the right colon as well as adenomas and cancer in the duodenum [13]. Peutz-Jeghers syndrome (PJS) and familial juvenile polyposis (FJP) have a lifetime risk of CRC up to 39 % and 20 %, respectively [26, 27]. Chronic inflammation also increases the probability of cancer, the risk for ulcerative colitis is 7–15 % after 20 years – even higher when combined with primary sclerosing cholangitis – and it is similar for Crohn's colitis [28, 29]. Table 1.1 lists increased risk conditions for CRC.

1.2.2.1 Screening with Positive Family History

The lifetime risk for colon cancer is about 1 % in individuals without increased risk factors and 2 % in individuals with first-degree relatives (FDRs) with colonic

Table 1.2 Clinical criteria for microsatellite instability (MSI) genetic testing (for HNPCC) [13]

Amsterdam criteria II	Revised Bethesda guidelines
At least *3 relatives* with CRC or a Lynch syndrome-associated cancer[a] occurring in the following combinations:	One CRC diagnosed at age <50 years
One is first-degree relative to others	MSI-H-positive CRC at age <60 years
In at least two successive generations	Syn-/metachronous Lynch syndrome-associated tumors[a]
At least one diagnosed at age <50 years	1 CRC and 1 FDR with Lynch syndrome-associated tumor[a], 1 at age < 50 years
FAP excluded in the CRC cases	1 CRC with two or more FDR or SDR with a Lynch syndrome-associated tumor[a]
Tumors verified by histopathology	

[a]These include colorectal, endometrial, stomach, ovarian, pancreas, ureter, renal pelvis, biliary tract, and brain tumors, sebaceous gland adenomas, keratoacanthomas, and carcinoma of the small bowel

Table 1.3 Recommended *screening colonoscopy* for *high risk* of CRC [10, 24, 26, 31]

	Screening colonoscopies	
Risk factors	Age at begin	Intervals (years)
Positive FH only		
1. One SDR or TDR (cousin) with CRC	50 years	10
2. One FDR with CRC/adenoma >60 years or >two SDR with CRC	40 years	10
One FDR with CRC/adenoma <60 years	40 years or 10 years before manifestation in FDR	5
Monogenic hereditary syndromes		
3. FAP (classical form)	12 years	1 or 2
Attenuated FAP (10–100 adenomas)	25 years, or 10 years before CRC in FDR	1 or 2
4. HNPCC	20 or 25 years, or 10 years before earliest CRC in FDR	1 or 2
5. Peutz-Jeghers syndrome (PJS)	18 years	2
6. Familial juvenile polyposis (>10 polyps)	12 years	3–5
Chronic inflammation		
7. Ulcerative colitis, Crohn's colitis	Pan-/colitis for 8–10 years	2 (–1)

See Chap. 11 for surveillance of ulcerative colitis and Crohn's colitis

adenoma or carcinoma at age <60 years (i.e., positive family history FH), and it is 3.5–4 % when one FDR had colon cancer at age <50 years or more than 1 FDR had colon cancer or when two or more second-degree relatives (SDR) had colon cancer [25] (Fig. 1.1). The risk for colon cancer is only marginally increased (~1.5–1.8-fold) when one FDR at age >60 years or one SDR had colon adenoma or cancer [25]. In cases of positive FH and more so in cases with strong hereditary risk for CRC (e.g., positive Amsterdam criteria, Table 1.2) the risk rises earlier in life and becomes very high in the cancer syndromes, e.g., ~60 % in HNPCC and 80–90 % in FAP at age of 60 years (Fig. 1.1) [8, 13, 23]. Recommendations for surveillance are listed in Table 1.3.

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