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Serrated polyposis: rapid and relentless development of colorectal neoplasia

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Abstract

Objective—Serrated (hyperplastic) polyposis (SP) is a rare disorder with multiple colorectal hyperplastic polyps and often sessile serrated adenomas/polyps (SSA/P) or adenomas. Although associated with colorectal cancer, the course of SP is not well described.

Design—44 patients with SP were studied. The results of 146 colonoscopies with median follow-up of 2.0 years (range 0–30) and a median of 1.0 years (range 0.5–6) between surveillance colonoscopies were evaluated. Findings from oesophagastroduodenoscopy examinations were analysed.

Results—The mean age at diagnosis of SP was 52.5±11.9 years (range 22–78). In two pedigrees (5%) another family member had SP. None of 22 patients had gastroduodenal polyps. All patients had additional colorectal polyps at surveillance colonoscopy. SSA/P or adenomas were found in 25 patients (61%) at first colonoscopy and 83% at last colonoscopy. Recurrent SSA/P or adenomas occurred in 68% of patients at surveillance colonoscopy. Three patients had colorectal cancer. Eleven patients (25%) underwent surgery (mean time from diagnosis of SP 2.0±0.9 years). After surgery all seven surveyed patients developed recurrent polyps in the retained colorectum (4/7 had SSA/P or adenomas). No association was found between colorectal neoplasia and sex, age at diagnosis of SP or initial number of colorectal polyps.

Conclusions—In SP, rapid and unrelenting colorectal neoplasia development continues in the intact colorectum and retained segment after surgery. These findings support the possibility of annual colonoscopic surveillance, consideration for colectomy when SSA/P or adenomas are encountered and frequent postoperative endoscopic surveillance of the retained colorectum.

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INTRODUCTION

Colorectal cancer (CRC) is an important global health problem. Worldwide, this tumour is the third most commonly diagnosed malignancy after lung and breast cancer.[1] Traditionally, the colorectal adenoma is considered the precursor lesion to CRC, and two pathways of colorectal carcinogenesis— chromosomal instability and microsatellite instability—are described to explain the adenoma-carcinoma transition.[23] Germline mutations in the former pathway cause familial adenomatous polyposis (FAP) and, in the latter, Lynch syndrome. These two conditions are characterised by young individuals with multiple colorectal neoplasia, high risk for CRC and extracolonic manifestations.[45]

Over the past decade some serrated polyps have been recognised as premalignant lesions and as markers for synchronous and metachronous colorectal neoplasia.[6–12] These observations have given rise to a third pathway in which the 'serrated polyp' is a precursor lesion for CRC.[6–9] Pathologically, the serrated polyp is characterised by sawtooth infolding of the crypt epithelium.[13] This histological definition also includes the hyperplastic polyp thought for generations to be a non-premalignant lesion.

Hyperplastic polyposis syndrome is a relatively rare colorectal condition typically characterised by several dozen hyperplastic polyps scattered throughout the colon and associated with CRC.[13] Recently, the name 'hyperplastic polyposis syndrome' has been changed to 'serrated polyposis' (SP) since a spectrum of serrated lesions, not just hyperplastic polyps,[14] can be noted on colonoscopy. In parallel to FAP and Lynch syndrome which characterise the extreme phenotype of the two classical colorectal carcinogenesis pathways, SP may be such an exaggerated representation of the serrated pathway since recent investigations have found a predominantly serrated pathway in this condition.[15] Unlike, FAP and Lynch syndrome which are caused by specific germline mutations, no genetic abnormality has consistently been noted in patients with serrated polyposis (SP), but inheritance is seen in a small percentage of these pedigrees.[14]

There are sparse data concerning the phenotypic expression of SP over time.[1416–18] The present investigation therefore evaluated in patients with SP, as defined by WHO criteria, the colorectal phenotype including the multiplicity and temporal development of colorectal neoplasia and extracolonic polyps.

METHODS

Study population

Patients with SP enrolled in the Johns Hopkins Hereditary Colorectal Cancer Registry between 1 January 2001 and 1 October 2010 were included in the study. These patients self-enrolled in the Registry without physician referral or were asked to enroll by a Johns Hopkins physician who had seen the patient in consultation. None of the patients were referred to the Registry because of a family history of SP and none were obtained from the mining of an independent endoscopic database. Patients met the WHO criteria for this disorder with: (1) at least five serrated polyps proximal to the sigmoid colon with two or more of these being >10 mm; or (2) any number of serrated polyps proximal to the sigmoid colon in an individual who had a first-degree relative with SP; or (3) >20 serrated polyps of any size but distributed throughout the colon.[13] This study was approved by the Johns Hopkins Joint Committee on Clinical Investigation (institutional review board).

Study design

Data were collected on each patient from medical records including colonoscopy, oesophogastroduodenoscopy (EGD) (if performed) and histopathology reports. Abstracted

information included sex; age; date of colonoscopy; age at each colonoscopy; number of colonoscopies; number, type, and location of colorectal neoplasia; date of EGD, findings of EGD; family history of SP; and date and type of surgery. In accordance with WHO guidelines,[13] serrated colorectal polyps were histologically classified as hyperplastic polyps, sessile serrated adenomas/polyps (SSA/P) or traditional serrated adenomas. Hyperplastic polyps are characterised by elongation of the crypts with variable degrees of serration located in the more luminal aspects of the crypt and with narrow crypt bases. SSA/P are characterised by elongation of the crypts with prominent serration with crypt distortion and dilation at the base along with excess serration near the base and without cytological dysplasia. Traditional serrated adenomas are defined as SSA/P with cytological dysplasia, conventional adenomas with an overall serrated architecture or a lesion characterised by an overall complex and villiform pattern with cells showing cytological features characterised as dysplasia. In addition to the above neoplasms, colorectal adenomas or adenocarcinomas were also identified. From these data the mean average increase in polyp number per year was calculated.

Statistical analysis

Mean, SD, median and range were reported where appropriate. Statistical analysis was conducted using the Fisher exact test and Student t tests. Statistical significance was defined as a p value <0.05. The statistical analyses were performed using the statistical software STATA V.11.

RESULTS

In total, 44 patients from 42 pedigrees met the definition of SP and had one or more colonoscopies (table 1). Two of 10 patients initially diagnosed with SP by WHO criterion I then met criterion III during surveillance. Two of 44 families (5%) had one other first-degree family member (a sibling) with SP (two sisters in one family and a sister and brother in the other). The median cumulative number of polyps is shown in table 2.

These patients underwent a total of 146 colonoscopies (table 3). All polyps found on colonoscopy were removed and the numbers and types of polyps were confirmed histologically. The indications for the first colonoscopy were CRC screening in 30 patients (68%), gastrointestinal bleeding in seven (16%), abdominal pain in four (9%), anaemia in two (5%) and change in bowel habits in one (2%). The mean±SD age at first colonoscopy was 50.8±11.2 years and at last colonoscopy was 52.5±12.6 years. The median colonoscopic follow-up was 2.0 years (range 0–30) and the mean interval between colonoscopies in this patient group was 1.6±1.2 years.

In 24 of the 44 patients (55%) the diagnosis of SP was made at the first colonoscopy which was done for CRC screening. Six additional patients who had a first colonoscopy for CRC screening were found to have colorectal polyps and were diagnosed with SPS on a subsequent surveillance colonoscopy.

A subset of 22 patients underwent EGD at an average age of 56.2 years. The indications for EGD were screening for upper tract polyps in nine (41%), unabating symptoms of gastrooesophageal reflux in four (18%), gastrointestinal bleeding in three (14%), abdominal pain in three (14%), anaemia in two (9%) and nausea and vomiting in one (5%). None of the patients had serrated polyps or adenomas noted on examination.

Two of the 44 families had multiple members with SP. The first family included a 23-year-old woman with 20 hyperplastic polyps increasing cumulatively to 60 hyperplastic polyps by age 26. This patient had a 22-year-old sister with 38 hyperplastic polyps, one

adenomatous polyp and one SSA/P at age 24. Other family members including an additional male sibling and parents were unaffected, and there was no family history of CRC. A second family had a 38-year-old woman with 40 hyperplastic and five adenomatous polyps at colonoscopy. This patient has a 36-year-old brother with 40 hyperplastic polyps, five adenomatous polyps and one SSA when screened. The parents and two sisters were unaffected. There was a family history of CRC in the paternal great uncle.

At first colonoscopy, 61% of patients had SSA/P or adenomas and, by the last colonoscopy, 83% had SSA/P or adenomas (table 4). The mean age at diagnosis of SSA/P or adenomas was 51.7±11.6 years (range 22–71) (table 4). The mean time from colonoscopy without SSA/P or adenomas to colonoscopy with these lesions was 1.8±1.5 years. Nineteen individuals (68%) with SSA/P or adenomas on first colonoscopy had recurrent SSA/P or adenomas on subsequent colonoscopies. The mean time to development of recurrent SSA/P or adenomas was 2.5±2.2 years. All patients had additional polyps at surveillance colonoscopy with a mean increase of 40% in polyp number per year.

Three of the 44 patients (7.6%) were diagnosed with CRC at colonoscopy, one at first colonoscopy and two at surveillance colonoscopy. The first patient was a 45-year-old woman who underwent CRC screening. On colonoscopy the patient had a large sigmoid mass diagnosed on colonoscopic biopsy as adenocarcinoma. The patient subsequently had an additional 24 serrated polyps confirmed by pathological review which were located in all segments of the colon and measured 3–20 mm, of which nine were SSA/P. The second patient was a 50-year-old woman undergoing colorectal surveillance because of a history of 20 hyperplastic polyps and a SSA/P, which had all been previously removed. At the next colonoscopy 34 months after the previous endoscopic procedure the patient had a 2.5 cm sessile polyp in the caecum. Biopsies revealed tubular villous adenoma with high-grade dysplasia. The patient underwent colectomy and the resection specimen revealed tubulovillous adenoma with infiltrating adenocarcinoma (T1, N0, M0). The third patient was a 75-year-old woman undergoing colorectal surveillance because previous endoscopy had revealed 13 hyper-plastic polyps, four SSA/P and two adenomas which had all been removed. On colonoscopy 22 months later the patient had a 3 cm mass in the ascending colon. Colonoscopic biopsies revealed SSA/P with adenocarcinoma and the surgical specimen revealed a T1, N0, M0 cancer.

Twelve of the 44 patients (27%) underwent surgery for SP during an average follow-up of 5.3 years (tables 5 and 6). The indications for surgery were: individual polyps unable to be endoscopically removed in four patients, polyps too numerous to remove endoscopically in three patients, cancer noted on colonoscopic biopsy in two patients, tubular villous adenoma with high-grade dysplasia in one patient, a traditional serrated adenoma (dysplasia noted in a SSA/P) in one patient and endoscopic surveillance could not be performed secondary to tortuosity of the colon in one patient. After surgery all patients had retained colorectum. Of the 12 patients undergoing surgery five were lost to postoperative follow-up. All seven patients who underwent postoperative surveillance of the retained colorectum had polyps identified on endoscopy. Of these, four (57%) had SSA/P or adenomas noted a mean of 2.5 years after surgery.

DISCUSSION

Few data exist on the natural history of patients with SP. The present study evaluated the colorectal phenotype in patients who met WHO criteria for the diagnosis of SP. These individuals were monitored—but not as part of a protocol—by serial colonoscopy with an average interval between procedures of 1.6 years for a median average of 2.0 years (range

0–30) of follow-up. Although not part of a formal procotocol, endoscopists removed all polyps during each colonoscopy except when patients were immediately sent for colectomy.

The mean age at diagnosis of SP was 52.5 years. This finding is similar to two other large studies of SP[16,17] in which the mean age at diagnosis was 56 and 49 years. In our cohort, CRC screening guidelines appeared to influence the age at diagnosis, since the majority of patients were diagnosed at screening colonoscopy. Of note, one patient in the present study was diagnosed with SP at the age of 22 years, similar to the young age reported by several others.[18,19] However, the age at diagnosis of SP varies greatly with some patients first presenting at age 10 and others in the eighth decade of life.[14,15]

In the current study, two of the 44 families (5%) had one other first-degree family member with SP (one family with two affected sisters and one family with an affected brother and sister). Chow *et al* also reported two of 38 (5.3%) pedigrees with multiple members with the condition.[14] This included one with possible autosomal dominant and the other autosomal recessive inheritance. Pedigree aggregation with possible dominant inheritance is also reported in five other cases.[19–21] Lage *et al* performed CRC screening in 17 at-risk members of six families with SP. Of these, seven had adenomas and/or serrated adenomas, four of whom were aged <50 years, and one 42-year-old patient was newly diagnosed with SP. Moreover, a >5 times increased risk of CRC has been noted in first-degree relatives of patients with SP.[22] These data, in addition to the natural history of this disorder described below, suggest an inherited genetic basis for this condition. Although patients with MUTYH-associated polyposis can have a similar colonic phenotype with concomitant adenomas and serrated neoplasms,[23] biallelic deleterious germline MUTYH mutations have been noted in only five patients with SP.[14,17,23] Nevertheless, prudence would dictate continuous colonoscopic screening of at-risk family members of the proband with SP starting at age 35 or 5 years younger than the lowest age at diagnosis of SP in the family, as suggested by Boparai *et al*.[22]

Patients with the classic inherited colorectal polyposis syndromes can have extracolonic polyposis in the small intestine and/or stomach. In SP, polyps outside the colorectum have not been reported but a systematic evaluation has not been performed. Also, cancers of the stomach or small bowel have not been reported in these patients. In the current study a subset of 22 patients had EGD at a mean age of 55.6 years. None of these individuals had adenomas or serrated polyps on examination. These data support the previous impression of an absence of gastroduodenal polyps in this condition obtained from large phenotypic studies of SP by investigators including Buchanan *et al*.[17]

In the three largest series of patients with SP a high percentage of patients (26% and 28.5%) were diagnosed with CRC at the time of initial colonoscopy[14,15] or before the diagnosis of SP.[18] The circumstances of cancer diagnosis in these studies probably represent an important selection bias inflating the perception of CRC in SP. Of note, Ferrandez *et al* reported a smaller series of 15 patients with SP followed for 33 months in which no CRC occurred.[24] Nevertheless, the preponderance of evidence suggests that patients with SP have at least a moderately increased risk of CRC. In our cohort, progression to CRC appeared high with a rapid and unrelenting development of colorectal neoplasm. At first colonoscopy 61% of patients had SSA/P or adenomas and by last colonoscopy (mean of 5.3 years later) 83% had these lesions. The mean time from colonoscopy without SSA/P or adenomas to the identification of SSA/P or adenomas was 1.8 years and mean time to recurrence of these lesions was 2.5 years. In addition, surveillance colonoscopy revealed that every patient had additional polyps at the next surveillance colonoscopy (mean interval of 1.8 years) with an average 40% increase per year in polyp number over the baseline polyp number. Moreover, one patient (2%) at initial colonoscopy and 11 patients (23%) during surveillance

colonoscopy underwent surgery; all had retained colorectum. All seven patients who had postoperative colorectal endoscopic surveillance had recurrent polyps and four of them had SSA/P or adenomas (occurring at a mean of 2.5 years postoperatively). Also, two patients in this study developed CRC at 22 and 34 months after the index colonoscopy. A literature review by Orlowska[25] revealed that five of 27 patients (18.5%) with SP without CRC on index colonoscopy developed CRC on follow-up. Also, Bopari *et al* calculated a worrisome risk of CRC during surveillance of 7% at 5 years in those with intact colons.[16]

The findings in this retrospective investigation are limited by several considerations. A small number of patients were evaluated in this study. As in other retrospective studies, selection bias can influence the data. Yet this cohort had only one patient presenting with CRC in comparison to other studies in which a significant minority of patients had CRC at initial colonoscopy. Although complete information was obtained and verified on all participants, the accuracy of the data was dependent on the medical record. In our investigation the patients came to a specialised centre for management and, consequently, the element of referral bias cannot be discounted, although none of the patients was enrolled in the study because of colorectal adenomas or cancer.

In summary, the natural history of SP is consistent with an inherited genetic syndrome marked by unrelenting and rapid development of colorectal neoplasia and a significant risk of CRC. Unlike other polyposis conditions, gastroduodenal lesions were not found in this condition. Also, recurrence of colorectal neoplasia postoperatively in retained colorectal segments occurs rapidly. The characteristics of SP support the possibility of annual colonoscopy surveillance in patients with intact colons and biannual endoscopic evaluation in those with colectomy and retained colorectal segments with removal of polyps at the time of these procedures. These recommendations are consistent with those of experts who promote close surveillance in those treated endoscopically for SP[26] or suggest surveillance similar to patients with an adenomatous polyposis syndrome.[27] Surveillance of the retained rectal segment every 6–12 months is specifically advocated by East *et al*.[26] Surveillance colonoscopy should be performed with high quality standards. Although none of the patients in this study had colonoscopy performed with narrow band imaging or chromoendoscopy, the addition of these techniques should be considered since serrated polyps are particularly difficult to detect endoscopically.

At present the management strategy of patients with SP as delineated by Young and Parry[28] is to recommend surgery when polyps cannot be controlled endoscopically, particularly if adenomatous features are present. We agree that the decision for colectomy needs to be individualised for each patient based on the results of surveillance colonoscopies. Based on expert opinion and because of the rapid and relentless natural history of this condition in most patients, clinicians should begin to consider colectomy when SSA/P, traditional serrated adenomas or adenomas are encountered in those meeting WHO criterion III.

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Significance of this study

What is already known on this subject?

- Serrated polyposis is a rare colorectal condition with association to colorectal cancer.
- Serrated polyposis is associated with colorectal adenomas and sessile serrated adenomas/polyps.
- The presence of extracolonic polyps is unknown in this condition.

What are the new findings?

- High rate of sessile serrated adenomas/polyps or adenomas at diagnosis.
- Rapid and relentless recurrence of colorectal neoplasia on colonoscopic surveillance.
- No gastroduodenal polyps noted on upper endoscopy.
- Rapid development of colorectal neoplasia in retained colorectum postoperatively.

How might it impact on clinical practice in the foreseeable future?

- Consideration for annual colonoscopic surveillance.
- Defer upper endoscopy surveillance.
- Consider colectomy when sessile serrated adenomas/polyps, adenomas or traditional serrated adenomas are encountered in patients meeting WHO criterion III.
- Consideration of postoperative biannual endoscopic surveillance of retained colorectum.

Table 1

Characteristics of study patients (N=44)

Characteristic	
Sex, n (%)	
Female	23 (52)
Male	21 (48)
Caucasian race, n (%)	41 (93)
African-American	3 (7)
No of pedigrees	42
Mean age at diagnosis (years)	
Mean±SD (range)	52.5±11.9 (22–78)
N (%) fulfilling WHO criteria for SPS	
I	10 (23)
II	0 (0)
III	34 (67)

Table 2

Median cumulative no (range) of polyps

	Hyperplastic	SSA/P	Adenoma	Total polyps
First colonoscopy*	13.0 (0–140)	0 (0–10)	1.0 (0–10)	16.0 (1–160)
Diagnostic colonoscopy†	23.0 (0–140)	0 (0–10)	1.5 (0–21)	25.5 (6–160)
Last colonoscopy	37.5 (0–235)	0 (0–10)	2.0 (0–26)	40.0 (6–240)

*First colonoscopy the patient ever had.

†Colonoscopy at which the diagnosis of serrated polyposis was made.

SSA/P, sessile serrated adenomas/polyps.

Table 3

Characteristics of endoscopic surveillance

Characteristic	
Total no of colonoscopies	146
Median (range) no of colonoscopies per patient	2.0 (1–11)
Mean±SD (range) age at first colonoscopy (years)	50.8±12.2 (22–71)
Mean±SD (range) age at last colonoscopy (years)	56.1±12.6 (24–78)
Median (range) follow-up (years)	2.0 (0–30)
Median (range) colonoscopy surveillance interval (years)	1.0 (0.5–6.0)
No of patients with EGD screening	22
No of patients with serrated polyps/adenomas on EGD	0
Mean±SD (range) age at EGD screening (years)	55.6±12 (29–77)

EGD, oesophogastroduodenoscopy.

Table 4

Characteristics of colorectal neoplasia

Characteristic	
No (%) of patients with SSA/P or adenomas at first colonoscopy	25 (61)
No (%) of patients with SSA/P or adenoma at last colonoscopy	34 (83)
Mean±SD (range) age of SSA/P or adenoma diagnosis (years)	51.7±11.6 (22–71)
Mean±SD (range) time from colonoscopy without SSA/P or adenomas to colonoscopy with SSA/P or adenomas (years)	1.8±1.5 (0.5–5)
No (%) of patients with recurrent SSA/P or adenomas	19 (68)
Mean±SD (range) time to development of any recurrent polyp (years)	2.5±2.2 (0.5–10)
No (%) of patients with colorectal cancer	3 (7.6)

SSA/P, sessile serrated adenomas/polyps.

Table 5

Characteristics of patients undergoing surgery for serrated polyposis

Characteristic	
No (%) of patients undergoing surgery	12 (27)
Mean±SD (range) age at diagnosis of hyperplastic polyposis (years)	51.1±10.3 (36–71)
Mean±SD (range) age at surgery (years)	52.1±11.3 (36–75)
Type of surgery, n (%)	
Colectomy with ileorectal anastomosis	8 (66)
Right hemicolectomy	4 (33)
Patients with endoscopic surveillance after surgery, n (%)	7 (58)
Patients with recurrent polyps, n (%)	7 (100)
Patients with recurrent SSA/P or adenomas, n (%)	4 (57)
Mean±SD (range) time to recurrent SSA/P or adenomas in retained colorectum (years)	2.5±2.4 (1–6)

SSA/P, sessile serrated adenomas/polyps.

Table 6

Indications and surgical pathology of patients undergoing surgery

Age (years)	Indication for surgery	Surgical pathology
55	Polyps too numerous to remove by colonoscopy	>100 HP throughout colon
36	Polyps too numerous to remove by colonoscopy	>140 HP, 10 SSA/P, 10 TA
53	Polyps too numerous to remove by colonoscopy	>100 HPs throughout colon
66	Many polyps/tortuous colon unable to survey	36 HP, 2 SSA/P, 1 TVA, 1 TA
42	Individual polyp(s) unable to be removed by colonoscopy	15 HP with two >1 cm
61	Individual polyp(s) unable to be removed by colonoscopy	7 SSA/P with two >2 cm, 1 TVA
53	Individual polyp(s) unable to be removed by endoscopy and tortuous colon precluded complete colonoscopy	80 HP, 1 adenoma
50	Individual polyp(s) unable to be removed by colonoscopy	13 HP with two >1 cm
50	CRC on colonoscopy biopsy	20 HP, 1 SSA/P, 1 TA, 1 TVA with adenocarcinoma
45	CRC on colonoscopy biopsy	Adenocarcinoma
39	TVA with high grade dysplasia	60 HP, 1 TVA, 5 TA,
51	Dysplasia in SSA/P	13 HP, 4 SSA/P >3 cm, 1 SSA/P with adenocarcioma

CRC, colorectal cancer; HP, hyperplastic polyps; SSA/P, sessile serrated adenomas/polyps; TA, tubular adenomas; TVA, tubulovillous adenomas.