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Sessile serrated adenomas: high-risk lesions?

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Summary

Sessile serrated adenomas (SSAs) were unrecognized in pathology and gastroenterology practice until about 2005; we have diagnosed them since 2001, allowing up to 10 years of follow-up. We evaluated follow-up of patients with sessile serrated adenoma diagnosed between 2002 and 2004 in our teaching institution and compared it to follow-up of randomly selected tubular adenomas. Materials from patients diagnosed with sessile serrated adenoma from January 2002 to December 2004 were reviewed. A control group of patients with sporadic tubular adenomas was selected. Ninety-nine sessile serrated adenomas from 93 patients were diagnosed between January 2002 and December 2004. Forty three patients (46.2%) had follow-up colonoscopy. One or more lesions were found in 42 (97.6%) of 43 patients. Mucinous adenocarcinoma was diagnosed in 1 (2.3%) of 43 patients, and 1 (2.3%) of 43 patients had high-grade dysplasia in an sessile serrated adenoma. Sessile serrated adenomas were found in 22 (51.2%) of 43 patients, 16 (37.2%) of 43 patients had tubular adenomas, and hyperplastic polyps were diagnosed in 18 (41.9%) of 43. Ninety-two patients with tubular adenomas between January 2002 and December 2004 formed the control group. Sixty-six patients (71.7%) received follow-up colonoscopy. Most (53/66, 80.3%) patients had tubular adenomas on follow-up, 12 (18.2%) of 66 patients had hyperplastic polyps, and 2 (3.0%) of 66 patients had a sessile serrated adenoma. The follow-up of sessile serrated adenomas from the study period (2002 to 2004) was more rigorous than proposed for sporadic tubular adenomas (patients with sporadic tubular adenomas were also followed up more aggressively than suggested by guidelines). Those with follow-up were managed as per advanced adenomas; their clinical outcomes supported this. These results suggest that guidelines for following up patients with sessile serrated adenomas as per advanced adenomas are warranted.

Keywords

Sessile serrated adenoma; Serrated polyp; Serrated neoplasia; Colorectal carcinoma; Follow-up colonoscopy

1. Introduction

Sessile serrated adenomas (SSAs) are colonic polyps with a serrated glandular pattern and architectural features that overlap with those of hyperplastic polyps. They are less common than conventional colorectal adenomas and were largely undiagnosed in general pathology

and gastroenterology practice until about 2005 [1]. At The Johns Hopkins Hospital, we have diagnosed SSAs since 2001 based on awareness of work by Torlakavik and Snover [2]. Sessile serrated adenomas can progress to dysplastic lesions ultimately producing microsatellite unstable carcinomas [3], and have been shown to have *BRAF* mutations and DNA hypermethylation [4–11].

The aims of the present study were to (1) evaluate the outcome of patients receiving an initial diagnosis of SSA on a screening colonoscopy, (2) determine how many of the patients were brought back for subsequent follow-up colonoscopy, recognizing that we would be unaware of follow-up data from other institutions, (3) compare follow-up data and patient demographics alongside a control group of patients receiving an initial diagnosis of tubular adenoma (TA) on screening colonoscopy, and (4) determine whether guidelines enforcing a follow-up protocol for patients diagnosed with SSA on screening colonoscopy are defensible.

2. Materials and methods

2.1. Study design

For our study we identified patients diagnosed with SSA from January 2002 to December 2004 by review of an institutional pathology database. The selection of this period allowed for adequate follow-up time. The lesions were classified as SSAs based on histologic features. SSA showed architectural and cytologic alterations including elongated crypts with micropapillary growth of epithelium, broad based crypts, mitoses in upper crypts, goblet cell mitoses, paucity or lack of endocrine cells, gastric type mucin, free floating goblet cells, and mild cytologic atypia. The size and location of the SSAs were not used as exclusion criteria if the morphological parameters were met.

A control group of patients with a diagnosis of TA was randomly selected from the same time period. We chose examples that had been removed by the same team of gastroenterologists in the same time frame rather than select for specific age matching—our hope was to learn if the demographics for the 2 types of polyps were similar. For the purposes of this study, TAs had low-grade dysplasia. A subset of patients diagnosed with tubular adenomas (6/92 [6.5%]) had three or more tubular adenomas on colonoscopy (range, 3–6 tubular adenomas).

The authors reviewed slides and records for both the SSA and control group for increased accuracy of polyp classification.

2.2. Initial colonoscopy data

The demographics of patients with an initial diagnosis of SSA and control patients with an initial diagnosis of TA from specimens obtained during colonoscopy were analyzed. Demographics included age, race, location of lesion, and size of lesion. The size of the lesion was calculated based on macroscopic specimen measurement by review of colonoscopy procedural report and pathology gross specimen description.

2.3. Follow-up data

Follow-up data included mean and median time from initial diagnosis to first follow-up colonoscopy, diagnosis on follow-up colonoscopy, number and percentage of patients receiving follow-up colonoscopy, race, and gender. These data from both case and control groups were compared.

2.4. Statistical analysis

Demographic features, pathological features, and clinical outcome differences between SSA and TA patients were tested using Mann-Whitney U test and χ^2 test, as appropriate. $P < .05$ was considered statistically significant.

3. Results

3.1. Initial colonoscopy

A total of 93 patients were diagnosed with SSA (Figure A and B) between January 1, 2002, and December 31, 2004. Two patients presented with an adenocarcinoma arising in association with an SSA (see Figure C–E). These carcinomas were found in the ileocecal junction and sigmoid colon. Another patient presented with a colonic adenocarcinoma arising from a TA (see Figure F). Fifty (53.8%) were men and 43 (46.2%) were women. The median age of the patients was 60.0 years; the mean age was 63 years (range, 31–90 years). Seventy nine were white (85%); 42 (53%) of 79, men; and 37 (47%) and 79, female. Eight were black (8.6%); 3 (37.5%) of 8, male; 5 (62.5%) of 8, female. Two were Asian males (2.1%). The race of four patients (4.3%) was unknown (Table 1).

A total of 99 SSAs were diagnosed in the SSA group. Most of the SSAs were found in the cecum (20/99) and right (40/99) and transverse colon (25/99). The remaining SSAs were diagnosed in the ileocecal junction (1/99), left colon (12/99), and rectum (1/99) (Table 1). The average size of the SSAs was 0.46 cm (range, 0.20–2.00 cm) (Table 1). A subset of patients diagnosed with SSA had synchronous TAs (12/93, 13%), and 18/93 (19%) of patients diagnosed with SSA had received a diagnosis of TA on prior colonoscopy. Review of endoscopy procedural notes revealed that 65 (70%) of 93 patients had complete removal of their SSAs, 13 (14%) of 93 patients did not have completely excised SSAs, and procedural notes were not available for 15 (16%) of 93 patients.

For a control group, 92 patients with a diagnosis of tubular adenoma, made from January 1, 2002, to December 31, 2004, were randomly selected. Fifty-two (56.5%) were male, and 40 (43.5%) were female. The median age of the patients was 66.5 years; the mean age was 66.8 year (range, 36–86 years). Seventy-one patients (77.2%) were white, 20 patients (21.7%) were black, and one of the patients (1.1%) was Asian. One hundred seventeen TAs were diagnosed in the control group. Most TAs arose in the transverse (33/117) and left colon (39/117) but a sizable minority were in the right colon (26/117) or cecum (9/117). The average size of the TAs was 0.47 cm (range, 0.10–2.30 cm) (Table 1). A subset (21/92, 23%) of the control patients with tubular adenomas had received a diagnosis of tubular adenoma on a prior colonoscopy. Review of endoscopy procedural notes revealed that 69 (75%) of 92 patients had their tubular adenoma completely excised, 6 (6.5%) of 92 patients did not have complete removal of their tubular adenomas and procedural notes were not available on 17 (18.5%) of 92 patients.

The average age was higher in the TA group than in the SSA group: 66.8 versus 62.8 years ($P = .033$). Interestingly, only 8.6% of the SSA patients were black compared to 21.7% of the TA patients ($P = .013$) (Table 1).

Furthermore, SSAs were significantly more often located in the cecum ($P = .028$) and right colon ($P = .036$). In contrast, TAs were more frequently seen in the left colon ($P < .001$) and rectum ($P = .029$) (Table 1).

3.2. Follow-up colonoscopy

Forty-three patients (46.2%) diagnosed with SSA received a follow-up colonoscopy at our hospital. Twenty-four patients (55.8%) were men and 19 (44.2%) were women. The median age of the patients was 60.0 years; the mean age was 62 years (range, 31.0–87.0 years). Thirty-seven patients were white (86.0%), and 4 were black (9.3%). The race of 2 patients (4.7%) was unknown (Table 2).

The average time from initial SSA diagnosis to follow-up colonoscopy was 2.72 years (range, 1–6 years). Most of the follow-up lesions were right sided: in the right colon (18/46) and cecum (11/46). The remaining lesions were in the transverse colon (12/46) and left colon (5/46). The average size of the follow-up lesions was 0.42 cm (range, 0.2–1.2 cm).

Follow-up colonoscopy in these 43 patients showed lesions in virtually all of them: 42 (98%) of 43. These lesions included 1 mucinous adenocarcinoma (2.3%) located in the sigmoid colon (Figure A, C, and E), high-grade dysplasia arising from a tubular adenoma in 1 (2.3%) of 43 patients, additional or persistent SSAs in 22 (51.2%) of 43, tubular adenomas in 16 (37.2%) of 43, hyperplastic polyps in 18 (41.9%) of 43, and an inflammatory polyp in 3 (7.0%) of 43 (Table 2). The mucinous adenocarcinoma on follow-up developed in a patient who had an initial diagnosis of SSA at the splenic flexure 1 year prior. Most patient (12/20, 60%) had SSAs at the same site as the initial SSA, and 6 (30%) of 20 had SSAs on follow-up colonoscopy at a site different from the initial lesion.

From the control group of patients diagnosed with a TA, 66 (71.0%) received a follow-up colonoscopy at our hospital. Thirty-three (50.0%) of these patients were male, and 33 (50.0%) were female. The median age of the patients was 67 years, the mean age was 67 years (range 36–86 years). Fifty-three (80.3%) patients were white, and 13 patients (19.7%) were black (Table 2).

The average time from a diagnosis of initial tubular adenoma to follow-up colonoscopy was 3.1 years, a median of 3.0 years (range, 1.0–6.0 years), which approached statistical significance for longer follow-up than for the SSA group ($P = .071$). The majority of the follow-up lesions were left sided: in the left colon (30/85) and rectum (5/85). The remaining follow up lesions were present in the transverse colon (24/85), right colon (19/85), and cecum (5/85). The location of 2 follow-up lesions was unknown. The average size of the follow-up lesions was 0.49 cm (range, 0.1 cm–2.3 cm). Fifty-three patients (80.3%) were diagnosed with tubular adenoma on follow-up colonoscopy, 12 (18.2%) were diagnosed with hyperplastic polyps, 2 (3.0%) patients were diagnosed with a SSA, and 3 (4.5%) patients had lymphoid aggregates (Table 2).

As expected, SSAs were statistically significantly more often seen on follow-up in the SSA group compared to the TA group ($P < .001$), whereas TAs were more frequently diagnosed in TA patients during the follow-up ($P < .001$). In addition, hyperplastic polyps were more common in SSA patients than TA patients with P values of .007 (Table 2).

4. Discussion

Although SSAs have been known since at least 1996 [2], general pathologists and gastroenterologists became aware of them much later. SSAs are more likely to result in symptoms than hyperplastic polyps and more likely to be encountered in the proximal colon. Over half are larger than 5 mm, and about 20% are larger than 10 mm [12]. They have subtle endoscopic characteristics, appearing flat to sessile and either the same as the background mucosa or slightly red. They accounted for 9% of sampled polyps in one prospective screening study [12]. They are now believed to be the precursors to sporadic

microsatellite unstable colorectal carcinomas (MSI-hi neoplasms) and the precursors to CpG island-methylated microsatellite stable cancers [13]. Colonoscopists have become more proficient at recognizing and biopsying them. For example, during the study period of the current study between 2002 and 2004 (inclusive), 93 patients were diagnosed with such lesions in our hospital whereas >350 were detected between 2008 and 2010 (inclusive).

Microscopically, SSAs feature bland cytology, elongated crypts, and prominent serrations. They differ from hyperplastic polyps by having an overall architectural distortion resulting from altered proliferation. It has been noted that the crypts often assume unusual contours resembling an inverted T or an L [2,14]. As per hyperplastic polyps, there is no cytologic dysplasia in uncomplicated SSAs.

Cytologic dysplasia develops in SSAs as they progress toward adenocarcinoma. The WHO has suggested the term “SSAs with cytological dysplasia” for this possibility [15], whereas in the past, such polyps have been termed *mixed hyperplastic-adenomatous polyp* [1]. The appearances of this change are similar or identical to those of conventional adenomas, but there are substantially different molecular underpinnings. SSAs are prone to promoter methylation, which can result in inactivation of *MLH1* [3], leading to microsatellite instability, or methylation of a variety of other genes (but not *MLH1*) can lead to CIMP-H microsatellite stable neoplasia. Both of these phenomena are mirrored by the development of dysplasia microscopically. In contrast, sporadic adenomas have a wholly different molecular signature (*APC*, *KRAS*, and *P53* mutations).

We believe our study provides a thorough review of initial and follow-up data from patients histologically diagnosed with SSA on initial colonoscopy. Our study patients were selected specifically based on an initial diagnosis of SSA on screening colonoscopy before SSA was widely recognized in the community. At our hospital, pathologists have been diagnosing these lesions since late 2001. Thus, we used January 2002–December 2004 as our time frame for initial diagnosis of SSA on colonoscopy as it allowed for 6 to 8 years of follow-up data. Most studies have relied on generalized database searches using “serrated polyp,” “mixed polyp,” or “hyperplastic polyp” in their selection of patients or retrospective review [16]. Our study was aimed at following up patients by means of review both of their initial and follow-up pathology specimens in addition to chart and record review. Moreover, often the size and location rather than histologic evaluation have been pedantic factors used in classifying serrated lesions of the colon as SSAs [17,18]. Endoscopy procedure notes and gross pictures have been used as an assessment of size in these studies; in our opinion this may lead to an overestimation of lesion size. Recognizing limitations, we have combined review of procedure notes along with pathology gross specimen measurements for the determination of lesion size. In fact, our study classified SSAs by review of pathology slides strictly on the basis of morphology; the tumor size and location were not used to exclude or include lesions if they met SSA histologic classification criteria although we recognize that there are limitations in histologic classification of these serrated lesions in that some can be difficult to separate from hyperplastic polyps. Our study is among the first to compare patients with initial diagnoses of SSA to those with an initial diagnosis of TA. Demographic data in addition to follow-up time and diagnoses were analyzed in tandem. We found that patients with an initial SSA diagnosis are on average 4 years younger than those with an initial diagnosis of TA on screening colonoscopy. This reached statistical significance but may be an artifact of relatively small numbers of patients. In our population, SSA were more common in whites in comparison to black or Asian races at our hospital.

Forty-six percent of patients diagnosed with SSA on an initial colonoscopy were brought back for follow-up at our hospital as compared to the patients with a diagnosis of TA, more than half of whom (72%) were brought back for a subsequent colonoscopy.

The follow-up colonoscopy for SSA in our study was more rigorous than that traditionally recommended for tubular adenomas. Additionally, our clinicians were aware of the need for follow-up of SSAs, and the SSAs in this study were managed as per high-risk adenomas with an average follow-up of time of 2.7 years. The outcomes of one of the SSA patients developing colonic carcinoma and one progressing to high grade dysplasia support this follow-up protocol. The fact that one additional SSA patient presented with an adenocarcinoma in the same anatomic area 1 year after receiving the initial diagnosis of SSA further supports that SSAs are high-risk lesions, and indicates that endoscopic surveillance of SSAs is justified. This patient was an 87-year-old black woman at the time of her initial SSA in 2004 (Figure A). It seems unlikely that the lesion progressed to the carcinoma depicted in Figure C and E in only 1 year, and presumably, sampling error was a factor. This underscores the sometimes subtle endoscopic features of serrated lesions. Given that virtually all patients with follow-up with SSAs had lesions, we would be concerned that only 46% (43/93) of patients had follow-up colonoscopy. However, our hospital is a tertiary care center and many of the remaining patients may have had additional studies in their communities.

However, our clinical colleagues also had a low threshold for follow-up of conventional adenomas and followed them up as per advanced adenomas. The average size of the lesions followed at an average of about 3 years was 0.5 cm (range, 0.1–1.8 cm) and, despite our selecting 92 control patients randomly, 66 had undergone follow-up colonoscopy at 3 years. This may be a result of our role as a tertiary care center and the availability of an “on-demand” colonoscopy center.

Clinical guidelines divide patients with a post colonoscopy diagnosis of adenoma into low and high risk categories based on the number, size, villous features, and absence or presence of high grade dysplasia [19]. High-risk patients are followed up on a 3-year protocol; those who are low risk or with hyperplastic polyps are followed up in 5 to 10 years.

In 2008, consensus guidelines published for follow-up of colon adenomas suggested that screening colonoscopy begin at age 50 for individuals with average risk for colorectal carcinoma [20]. Individuals with the occasional hyperplastic polyp are considered average risk, and the recommended colonoscopic screening interval is 10 years. Patients with increased risk of colorectal carcinoma are stratified into abbreviated screening intervals based on their risk category. For patients with known colorectal carcinoma, status post resection, a 1-year screening interval is recommended. In contrast, patients with an SSA removed piecemeal are recommended to have a colonoscopic screening interval of *2 to 6 months*. Of note, there are no screening recommendations for completely removed SSA.

In 2005, Lazarus et al provided evidence that patients with SSA may need to be surveyed at least as closely as those patients with conventional adenomas [21]. These authors found that patients with excised SSA had subsequent lesions develop at twice the rate of individuals with conventional adenomas, and SSA grew at a faster rate than both conventional adenomas and hyperplastic polyps. Moreover, patients with SSA had a higher rate of malignant progression. Although long-term clinical follow-up data are not yet available for patients with SSA and traditional serrated adenoma (TSA), the most prudent approach may be to ensure complete removal of the SSA and TSA, and to follow the patient at least as closely as those patients with conventional adenomas.

Further evidence that SSAs are clinically important comes from both a US Veteran's Administration multicenter study and a Japanese multicenter study in which both groups found that the presence of large serrated polyps were risk factors for colorectal carcinoma [17,18]. As noted above, currently there are no established clinical guidelines for follow-up

of patients diagnosed with SSAs [22]. Moreover a majority of SSAs are misdiagnosed as hyperplastic polyps, which was shown to have consequences in a retrospective study [16]. From both a clinical and pathological perspective there is a need for improved recognition of these lesions as they are precursors to potential malignant transformation; a set of guidelines for both screening and clinical follow-up are warranted [23]. Our data, like those of others, suggest that follow-up as per advanced adenomas after assuring complete removal of the polyp may be warranted.

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Figure.

A, Sessile serrated adenoma showing a broad-based crypt (lower right of field). Depending on sampling, such crypts can be numerous or infrequent as in this field (original magnification $\times 20$). B, Higher magnification of an SSA. The epithelium shows mucin reminiscent of gastric type mucin. There are no endocrine cells in this dilated crypt (original magnification $\times 40$). C, Low magnification of an infiltrating mucinous adenocarcinoma (the lesion arose at the same location 1 year after the diagnosis of the SSA depicted in image A) (original magnification $\times 10$). D, Adenocarcinoma arising from a SSA. The SSA is at the right of the field, and polypoid dysplasia is seen at the upper left portion of the field (original magnification $\times 4$). E, Higher magnification of a solid area of the mucinous tumor

seen in image C reveals the presence of tumor infiltrating lymphocytes (TILS) (original magnification $\times 40$). F, Tubular adenoma overlying an infiltrating adenocarcinoma (original magnification $\times 4$).

Table 1

Characteristics SSA and TA patients

	SSA (n = 93)	TA (n = 92)	P
Age			
Mean	63	66.8	.033
Median	60	66.5	
Minimum	31	36	
Maximum	90	86	
Sex			
Male	50 (53.8%)	52 (56.5%)	.706
Female	43 (46.2%)	40 (43.5%)	
Race			
White	79 (84.9%)	71 (77.2%)	.177
Black	8 (8.6%)	20 (21.7%)	.013
Asian	2 (2.2%)	1 (1.1%)	.567
Unknown	4 (4.3%)	0 (0%)	
Polyp size (cm)			
Mean	0.461	0.474	.751
Median	0.400	0.400	
Minimum	0.200	0.100	
Maximum	2.000	2.300	
Localization polyps			
Ileocecal	1	0	.319
Cecum	20	9	.028
Right colon	40	26	.036
Transverse colon	25	33	.188
Left colon	12	39	<.001
Rectum	1	7	.029
Unknown	0	3	
Total	99	117	

Table 2

Characteristics of all follow-up patients

	SSA (n = 43)	TA (n = 66)	P-value
Age			
Mean	62	67	.034
Median	60	67	
Minimum	31	36	
Maximum	87	86	
Sex			
Male	24 (55.8%)	33 (50%)	.553
Female	19 (44.2%)	33 (50%)	
Race			
Caucasian	37 (86.0%)	53 (80.3%)	.44
Black	4 (9.3%)	13 (19.7%)	.012
Asian	0 (0%)	0 (0%)	
Unknown	2 (4.7%)	0 (0%)	
Polyp size (cm)			
Mean	0.42	0.49	.429
Median	0.3	0.4	
Minimum	0.2	0.1	
Maximum	1.2	2.3	
Localization polyps			
Ileocecal	0	0	
Cecum	11	5	.009
Right colon	18	19	.159
Transverse colon	12	24	.359
Left colon	5	30	<.001
Rectum	0	5	.065
Unknown	0	2	
Total	46	85	
Follow-up (years)			
Mean	2.72	3.14	.071
Median	3	3	
Minimum	1	1	
Maximum	6	6	
Lesions during follow-up			
Any lesion	42 (97.6%)	63 (95.5%)	.547
Adenocarcinoma	1 (2.3%)	0 (0%)	.213
High-grade dysplasia	1 (2.3%)	0 (0%)	.213
SSA	22 (51.2%)	2 (3.0%)	<.001
TA	16 (37.2%)	53 (80.3%)	<.001
Hyperplastic polyp	18 (41.9%)	12 (18.2%)	.007

	SSA (n = 43)	TA (n = 66)	P-value
Inflammatory polyp	3 (7.0%)	0 (0%)	.030
Lymphoid aggregate	0 (0%)	3 (4.5%)	.156