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Pseudoinvasion and squamous metaplasia/morules in colorectal adenomatous polyp: a case report and literature review

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Abstract

Background Submucosal pseudoinvasion and squamous metaplasia (SM) are incidental and special morphological findings in colorectal adenomas, and both can mimic invasive carcinoma. The coexistence of these two findings further increases the risk of misdiagnosis, posing a great diagnostic challenge to pathologists. From 1979 to 2022, only 8 cases have been reported, which was extremely rare. In this report, we presented a case of sigmoid colon adenoma accompanied by pseudoinvasion and SM. Additionally, relevant literature was analyzed to summarize the clinical and pathological characteristics.

Case presentation A 51-year-old Chinese male patient presented with fresh blood after defecation. Electronic colonoscopy revealed multiple polyps, which were removed using a snare and subjected to high-frequency electrocoagulation resection. The largest polyp, located in the sigmoid colon, was a thick pedunculated and lobulated polyp with a maximum diameter of 2.8 cm. The surface of the polyp showed slight ruggedness and redness, and it was sent for pathological examination. Grossly, the polyp had a lobulated and slightly rough surface. Microscopically, it showed a tubulovillous adenoma with focal high-grade dysplasia and mucosal muscle hyperplasia. Glandular elements were observed in the submucosal layer, forming a well-defined lobular structure. Some of the glands displayed cystic change, and focal SM could be seen within the adenoma. SM could manifest as discrete solid cell nests of varying sizes or cribriform-morular-like structures. Immunohistochemical staining showed that SM cells were diffusely positive for cytokeratin 5/6 (CK5/6); p40, p63, and cytokeratin 20 (CK20) were negative; while caudal type homeobox 2 (CDX2) was weakly positive. β-catenin showed abnormal nuclear expression, and an extremely low Ki67 proliferation index was observed.

Conclusions Coexistence of SM and pseudoinvasion in colorectal adenomas is highly rare. It is more commonly observed in males and tends to occur in the sigmoid colon. It primarily manifests in tubulovillous adenoma and tubular adenoma, with a majority of cases exhibiting a pedicle. Histologically, it is similar to invasive lesions. The cystic dilation of the submucosal glands, hemosiderin deposition, and the presence of a lamina propria around the submucosal glands without adjacent desmoplastic reaction, suggest pseudoinvasion rather than cancer. The bland cytological morphology and Immunohistochemical markers play a crucial role in distinguishing SM from true invasive lesions.

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Keywords Colorectal adenoma, Colorectal polyp, Pseudoinvasion, Squamous metaplasia, Squamoid morule, Case report

Introduction

Adenoma is the most common benign epithelial tumor in the colon and rectum and is a precursor lesion of colon cancer. According to the diagnostic criteria of world health organization (WHO), invasive cancer is diagnosed when the tumor penetrates the muscularis mucosae into the submucosa. In rare cases, pseudoinvasion of submucosal glands, which mimics the histological morphology of invasive cancer, can occur in colorectal adenomas [1, 2]. Furthermore, with the advancement of rectal cancer screening in the population, an increasing number of adenomas are identified and removed, leading to the recognition of less common histological features of adenomas [3–5]. SM/ squamous morules can be rarely found in colorectal adenomas. Previous large-scale studies have reported an incidence rate of approximately 0.4% [4, 5], highlighting the need to differentiate it from malignant lesions. Particularly when pseudoinvasion coexists with adenomas, it is more likely to be misdiagnosed as invasive cancer [6]. In this case report, we present an extremely rare case of sigmoid colon adenoma accompanied by pseudoinvasion and SM. We also review relevant literature to contextualize our findings, aiming to enhance clinical doctors' and pathologists' awareness of this distinctive morphology of colorectal adenomas and to prevent overdiagnosis.

Case presentation

A 51-year-old male was admitted to the hospital for 3 days with intermittent chest pain and a history of hypertension and coronary heart disease. During his hospitalization, fresh blood was observed after defecation, attributed to hemorrhoids. The stool occult blood test also yielded a positive result, prompting the performance of an electronic colonoscopy. The colonoscopy revealed multiple diverticula throughout the colon, as well as 10 polyps ranging in diameter from 0.2 cm to 2.8 cm. The largest polyp, located in the sigmoid colon, measured approximately 2.8 cm in maximum diameter and showed a thick pedicle, lobulated appearance, and flushed appearance (Fig. 1). All polyps were removed using a snare device and subjected to high-frequency electrocoagulation resection. The largest polyp was sent for pathological examination. Macroscopically, a grayish-red pedicle polyp (Fig. 2) measuring 2.8 cm × 2.6 cm × 1.9 cm was observed. The length and width of the pedicle were 0.8 cm and 1.5 cm, respectively, and the surface appeared lobulated and slightly rough.

Microscopically, the head of the polyp showed a lobular structure. The adenoma contained branching and proliferating mucosal muscles, along with normal glands and proliferating mucosal muscles extending into the submucosal layer of the pedicle, resulting in a disorganized distribution of neoplastic glands and normal mucosal glands within the lesion. The intricate mucosal muscles divided or surrounded the glandular clusters, resulting in lobular shapes with smooth contours. The stroma surrounding the submucosal glands or lobules appeared loose and similar to the lamina propria in adenomatous polyps (Fig. 3A). No desmoplastic stroma observed. The surface of the pedicle was covered with normal mucosal epithelium, showing conspicuous submucosal bleeding and cystic expansion of certain glands, forming mucinous or bloody cysts (Fig. 3A). Within the mucinous blood pool of the largest cyst, a neoplastic gland could be observed. The glandular epithelia displayed low-grade cytological characteristics, with a small amount of inflammatory stroma surrounding the gland. The cystic dilated gland was enveloped by fibrous stroma with hemosiderosis (Fig. 3B), and the interstitial space was infiltrated by chronic inflammatory cells, mainly comprising plasma cells and some eosinophils.

The tumor manifested as a tubulovillous adenoma with focal severe dysplasia (Fig. 3C). Amidst the alkaline-stained adenomas, there were sporadic regions consisting of nests with amphophilic cytoplasm (Fig. 3D). These cell nests varied in size and had distinct boundaries. They were either dispersed within loose stroma or densely arranged (Fig. 3E). The cells within the nests displayed cytologically bland features, appearing polygonal or short spindle-shaped, with abundant cytoplasm. The nuclei were round or ovoid, vesicular, with inconspicuous nucleoli, and no mitosis was observed. The cells exhibited a syncytial-like arrangement with poorly defined cell borders, forming a solid or fenestrated pattern (Fig. 3F), occasionally revealing small foci of necrosis composed of eosinophilic granular material and fragmented nuclear debris. In some solid nests, the cell boundaries could be recognized, and occasional intercellular bridges were visible, arranged in characteristic whorls similar to squamous epithelium. The cell nests were connected to the adenomatous gland or extended into the glandular cavity, sometimes replacing the glandular epithelium (Fig. 3G). The junction areas could form cribriform-morular-like structures with uniformly sized glandular cavities (Fig. 3H). Numerous pink-stained fibrinoid casts were present in the glands adjacent to the nests (Fig. 3I).

The immunohistochemical staining results were as follows (shown in the Supplementary Table 1): Desmin demonstrated complex hyperplasia of mucosal muscles,

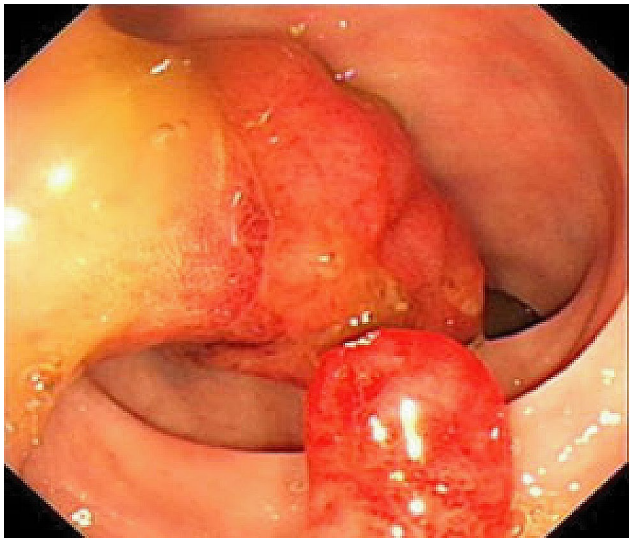

Fig. 1 Electronic colonoscopy. The largest polyp showed a thick pedicle, lobulated appearance, and flushed appearance in the upper left. Another polyp was shown below the image

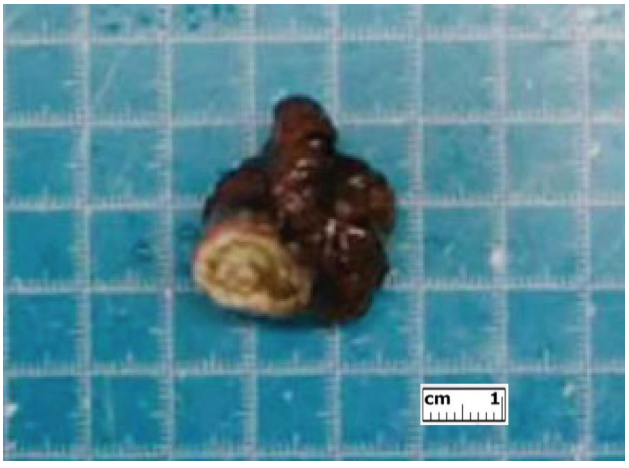

Fig. 2 Gross appearance. The polyp was lobulated with slightly rough surface, and display a thick pedicle

with the glandular component trapped in the submucosa (Fig. 3A). The SM cells were diffusely positive for cytokeratin 5/6 (CK5/6) (Fig. 4A) and P16 (Supplementary Fig. 1A). P40 (Fig. 4B), P63 (Fig. 4C), and cytokeratin 20 (CK20) were not expressed (Supplementary Fig. 1B), while caudal type homeobox 2 (CDX2) was uniformly weak positive expression (Fig. 4D). The nuclei, cytoplasm, and membrane showed diffuse and strong positivity for β-catenin (Fig. 4E). The Ki67 positive index was approximately 2%, and the adenomatous gland varied from 30 to 70% (Fig. 4F). A few cells were positive for synaptophysin (Syn) and CD56 in the solid nests, while being negative for chromogranin A (CGA) and INSM transcriptional repressor 1 (INSM1) (Supplementary Fig. 1C-F). P53 was found to be wild-type (Supplementary Fig. 1G). There was no loss of mismatch repair proteins(Supplementary Fig. 2). Based on the morphological findings above, this case does not have the characteristics of invasive cancer, namely, irregular and spiked epithelial components accompanied by desmoplastic stroma, is consistent with pseudoinfiltration and SM of the adenoma, and immunohistochemical results support this consideration. We finally concluded that this was a case of colonic adenoma with pseudoinvasion and SM.

We sent the tissue embedded in paraffin to an external laboratory (Zhejiang Topgen Medical Laboratory Co., Ltd, Huzhou, China) for molecular testing. This detection was based on the liquid-phase probe hybridization method for targeted capture of nucleic acid sequences and high-throughput sequencing. The sequencing platform was MGISEQ_T7_R1100600200017. We analyzed hotspot gene variations in 52 genes highly associated with solid tumors and microsatellite status. Only one tumor related gene variation, PMS2, c.1738 A>T (p.K580X) (Supplementary Fig. 3), was detected, and the microsatellite status was microsatellite stability.

Discussion

In the bowel cancer screening in the UK, an Expert Board (EB) consisting of three specialist gastrointestinal pathologists was established to distinguish between pseudoinfiltration and adenocarcinoma. The assessment results of the EB showed that complete consistency rate between originating pathologist with the EB was only 33% (68/206), while the proportion of cases with equivocal original diagnosis but confirmed EB diagnosis was 35.9% (74/206) and that with completely opposite original diagnosis to EB accounted for 23.8% (49/206) [7]. From this, it can be seen that this is indeed an extraordinary diagnostic conundrum. In 1973, Muto et al. [1] first described colorectal adenomas with glandular tubules entering the submucosal layer, introducing the concept of "*pseudo infiltration of colorectal adenomatous polyps*". The incidence rate was 2–3% among polypectomy cases, and the most common site was the sigmoid colon. 90% of the adenomas were larger than 1 cm, with a pedicle length greater than 1 cm. The main histological characteristic was the presence of adenoma tubules in the submucosal layer, similar to those found in the head of the polyp. The gland was clearly demarcated from the surrounding stroma. The cystic dilation of glands caused by mucus retention or bleeding was prominent. Hemosiderosis in the submucosal layer and the presence of a lamina propria around the submucosal gland without an immediately adjacent desmoplastic reaction indicated pseudoinvasion rather than carcinoma. Recently, Hou et al. [2] summarized two patterns of pseudoinvasion by observing the clinical pathological morphological characteristics of colorectal adenomas involving the submucosa.

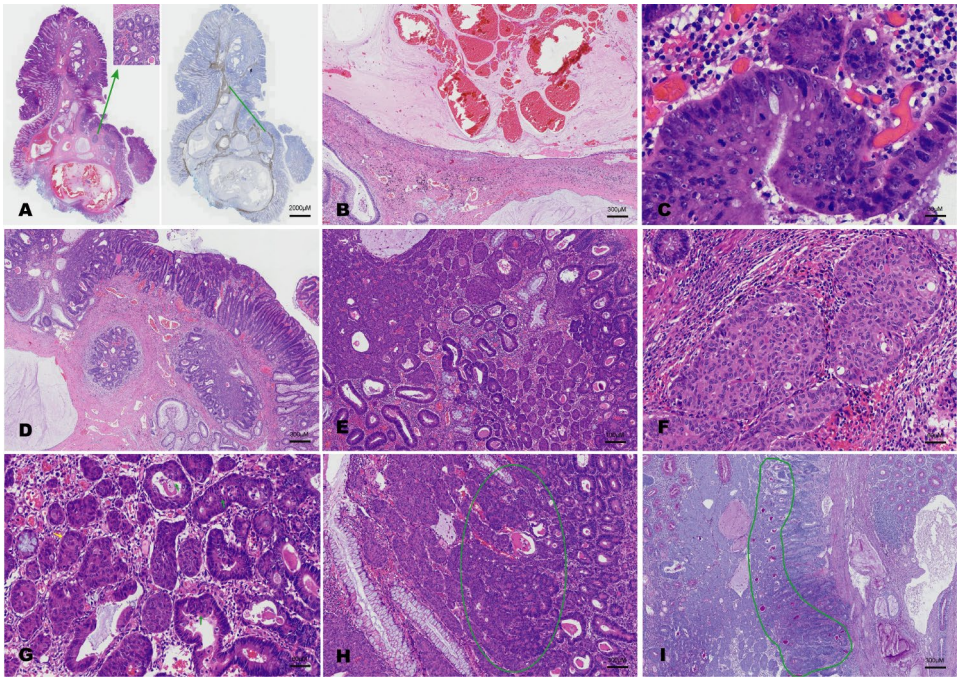

Fig. 3 Morphological features of the adenomatous polyp. (**A**) Polyp with pseudoinvision of the stalk. Glandular component and proliferating mucosal muscles extended into the submucosal layer of the pedicle and cystic expansion of certain glands formed mucinous or bloody cysts (H&E, magnification ×5). Desmin immunostaining in the right image highlighted mucosal muscle rupture (green line) and the intricate mucosal muscles. (**B**) The cysts filled with mucus or blood were surrounded by fibrous stroma with hemosiderosis (H&E, magnification ×40). (**C**) High-grade dysplasia within the adenoma (H&E, magnification ×400). (**D**) The submucosal lobules surrounded by lose connective tissue stroma had smooth contour. Amidst the alkaline-stained adenomas, sporadic regions consisting of nests with amphophilic cytoplasm distributed on both sides of muscularis mucosa (arrowheads) (H&E, magnification ×40). (**E**) The cell nests with clear boundaries varied in size. They were either dispersed within loose stroma (lower right) or densely arranged (upper left). Numerous pink-stained fibrinoid casts were present in the glands adjacent to the nests (H&E, magnification ×100). (**F**) Syncytial-like cells with ill-define cell border formed a solid or fenestrated pattern (H&E, magnification ×200). (**G**) The cell nests were connected to the adenomatous gland, extending into the glandular cavity, or replacing the polarized glandular epithelium (arrowhead), which could easily be misdiagnosed as invasive components or adenomas with high-grade dysplasia (H&E, magnification ×200). (**H**) The junction area between cell nests and neoplastic glands formed cribriform-morular-like structure with uniformly sized glandular cavities (H&E, magnification ×40). (**I**) A large number of red-stained fibroin-like casts (PAS staining, magnification ×40)

Our case involved the sigmoid colon. The polyp was large and had a thick peduncle. The pseudoinvasive glands were lobulated and appeared as single cysts containing mucus or blood. Mucus pools were also observed. The intricate mucosal muscles divided the glandular clusters into lobules. These characteristics were consistent with the description of "Pattern 1".

SM is rare in colorectal adenomas. Dutra [8] initially described peculiar masses of cells in hyperplastic endometrium as intraglandular morules due to their three-dimensional resemblance to mulberries. Subsequent ultrastructural studies suggested that morules exhibited epithelial differentiation and were related to SM [9]. In 1984, Sarlin et al. [10] first used the term "morules" in the context of SM in colorectal adenomas. Apart from Tetsuya et al. [11] who believed that morules were distinct from SM, all published literature consistently associated SM, squamous differentiation, and squamoid morule with the same morphological changes in colorectal adenomas. In 1979, William et al. [4] investigated the pathogenesis of pure squamous cell carcinoma of the rectum by examining continuous sections of 750 colorectal adenomas from 516 patients. They found three adenomas with squamous differentiation, which was a novel finding at the time. Subsequently, there have been reports on individual cases and small case studies. In 1984, Bansal et al. [5] examined 4644 adenomas, among which 20 had SM. The incidence of SM in adenomas (20/4644) was consistent with the results obtained by William et al. (3/750). To date, by searching PUBMED and CNKI using the keywords "colorectal, adenoma or polyp, SM, and squamous morules," a total of 18 English and Japanese articles can be found through relevant links [4–6, 10–24]. Along with our case, a total of 72 patients with 73 adenomas (including adenomas clearly containing carcinoma in situ/intramucosal carcinoma, while cases with invasive carcinoma in the literature were excluded from this analysis) were included. We reviewed the literature, summarized its clinical and pathological features (Supplementary Table 2), and conducted an analysis.

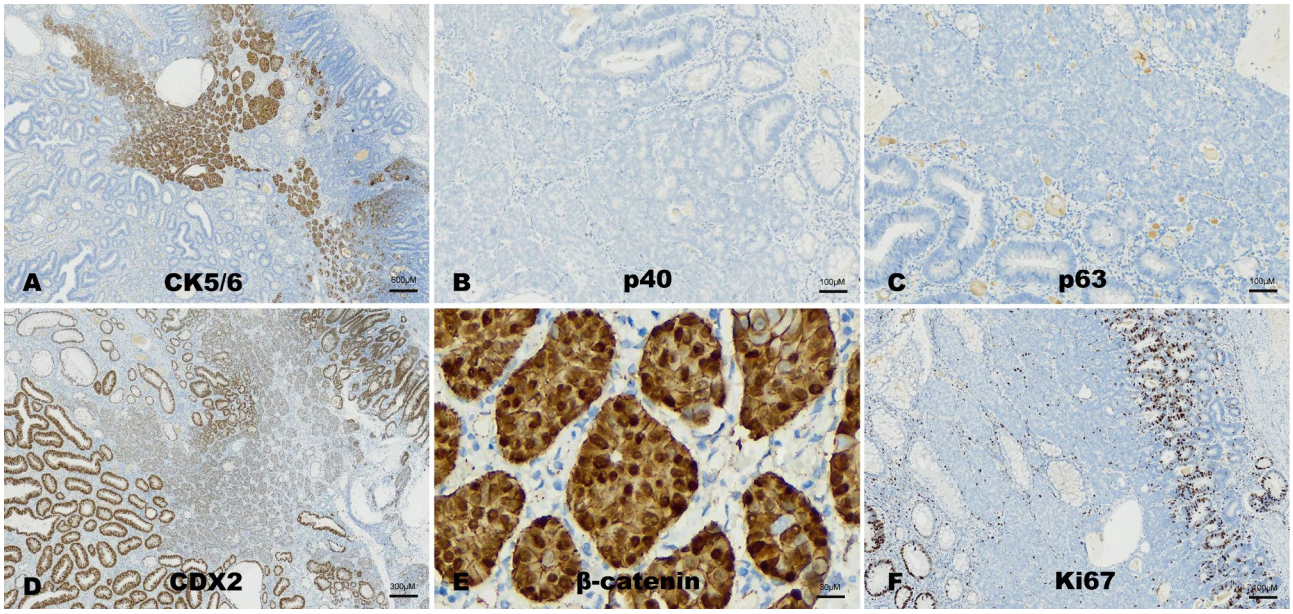

Fig. 4 Immunohistochemical staining results. (**A**) The SM cells were diffusely positive for CK5/6 (magnification ×20). (**B**) Immunostaining of p40 in the areas of squamous metaplasia were negative (magnification ×100). (**C**) The SM cells were negative for p63 (magnification ×100) (D) The SM cells were uniformly weak positive for CDX2, while neoplastic glands and normal mucosal glands were strongly positive (magnification ×40). (E) The nuclei, cytoplasm, and membrane showed diffuse and strong positivity for β-catenin (magnification ×400). (**F**) Ki67 staining showed a very low cell proliferation index whether in the solid nests or cribriform-morular-like structure of the SM (magnification ×100)

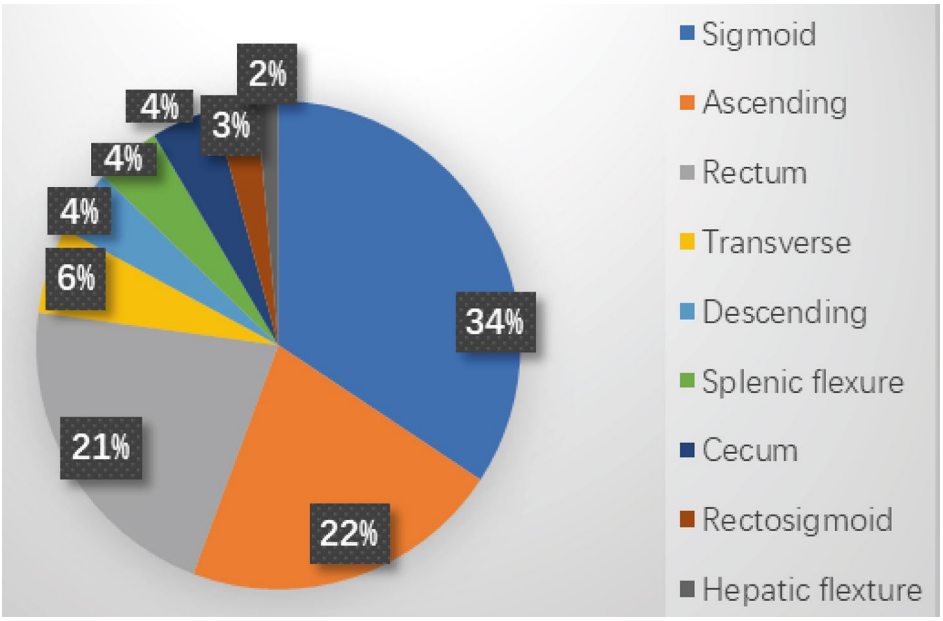

Fig. 5 Location distribution of colorectal adenomas with SM. **Note:** A total of 70 cases were counted and 3 cases were not obtained

Our results showed that the average age of colorectal adenoma patients with squamous differentiation was 63 years (range 31–89), which was similar to the average age of 61 years (range 39–80 years) analyzed by Pantanowitz et al. [12] in 2009. The gender ratio was about 2:1 for males and females, with a clear advantage in males. These adenomas could occur in the entire large intestine (Fig. 5: Chart 1), with the sigmoid colon being the most common (34%), followed by the ascending colon and rectum (both accounting for 22%), while other sites were rare. Grossly, the ratio of pedunculated adenoma to sessile adenoma was 6.5:1, and the average size of the adenoma was 2.3 cm (0.9–7 cm). Histologically, the histological types of adenomas were tubular villous adenomas (61%), tubular adenomas (29%), and villous adenomas (11%) (Table 1). The ratio of low-grade dysplasia to high-grade dysplasia was

Table 1 Summary of macro- and microscopic findings of colorectal adenomas with SM

Macroscopic			
Type	**Pedicle***	**Size****	
Pedunculated (including semi-pedunculated)	26	2.3 cm (0.9–7 cm)	
Sessile	4		
Microscopic features (Histological type) ***			
Type	**LGD**	**HGD** (Including cancer in situ/ intramucosal cancer)	**Total**
TVA	27	13	40(61%)
TA	13	6	19(29%)
VA	3	4	7(11%)
Total	43(65%)	23(35%)	66

Note *A tatal of 30 were counted and 43 cases were not obtained; ** A tatal of 51 were counted and 22 cases were not obtained; *** A tatal of 66 were counted, and 7 cases were not obtained

about 1.9:1, indicating that SM was most commonly seen in tubular villous adenomas and was not significantly related to severe dysplasia of adenomas. Morphologically, the SM cells were distributed in a dispersed small nest pattern in the loose stroma. Cell differentiation can be relatively mature, such as arranging in whorls, intercellular bridges, and keratinization [20, 24], or immature. The cell nests can surround the glandular cavity, protrude into the glandular cavity, or replace the glandular gland. The presence of small glandular cavities within some nests was first observed in 1982 [15]. The cells consisting of the glandular cavity had nuclei similar to glandular epithelium but rounder, and cytoplasm similar to squamous cells. In this case, cribriform-morular-like structures with glandular cavities could be seen in the junction area between the cell nests and the adenomatous glands. These cells were similar to adjacent glandular epithelial cells but had more rounded nuclei, unclear cell boundaries, and weak expression of CK5/6, which conformed to the transitional form between squamous differentiation and adenoma. The CDX2 expression patterns of solid nests and cribriform-morular-like structures were diffusely and weakly positive, which was significantly different from the strong positive expression patterns of adenomatous glands. It also supported the idea that squamous differentiation could appear as nests and adenoid form. The fenestrated structure observed by Sarlin et al. [10] was consistent with the "pseudocribriform" shown in the image in Lee et al.'s study [6] and also existed in our case. Sarlin et al. believed that these small, rounded spaces in the morules were produced by dropout of these zones of focal necrosis affecting single or groups of cells. We believed this form represents relatively mature differentiation. The koilocytic-like cells mentioned by Simona et al. [24] and the transparent inclusion bodies in the nuclei observed by Tetsuya et al. [11] were not obvious in this case. In our case, we also observed a large number of red-stained fibroin-like casts in the glands adjacent to the nests of SMcells, with positive PAS staining (Fig. 3H). This has not been described in previous literature, and no similar secretion has been observed in glands of normal adenomas. It is speculated that it was produced by abnormally differentiated glandular epithelium.

Colorectal adenomas with both pseudoinvasion and SM are extremely rare. In 1983, Japanese scholars first described the simultaneous presence of these changes in colorectal adenomas [16]. We reviewed a total of 9 cases out of 73 adenomas with SM and pseudoinvasion, and summarized their clinical and pathological features in Table 2 (we were unable to obtain the original literature for Case 1, thus detailed information is unknown). The average age was approximately 52 years old, which was younger than patients with adenomas accompanied only by SM. Men had a higher representation (7 out of 8 cases), and the sigmoid colon was the most commonly affected site (5 out of 9 cases). 7 cases had a pedicle, with 2 cases specifically described as having a thick pedicle. Most colorectal adenomas with pseudoinvasion occurred in the pedunculated polyps of the sigmoid colon. Cystic dilation in the gland of adenomatous pedicle could even result in the formation of larger cysts, as observed in this case, leading to a thick and bulging stalk [22, 25, 26]. Histologically, neoplastic glands entering the submucosa of

Table 2 Clinicopathological characteristics of colorectal adenomas with pseudoinfiltration and SM

Case	Author	Age/Sex	Site	With/without a pedicle	Size	Histological type
1	Sirouzu K, et al. [16]	?	Sigmoid	?	?	?
2	Hayashi, et al. [17]	44/F	Sigmoid	pedunculated	1.2 cm X 1.7 cm X 1.7 cm	TA(focal HG)
3	Hirasaki S, et al. [22]	65/M	Sigmoid	pedunculated(1.3 cm)	1.6 cm	TA(LG)
4	Lee HE, et al. [6]	45/M	Ascending	sessile	2 cm	TA(LG)
5		53/M	descending	pedunculated	1.1 cm	TVA(LG)
6		67/M	Sigmoid	pedunculated	1 cm	TA(LG)
7		69/M	Ascending	pedunculated	3.5 cm	TVA(focal HG)
8		73/M	Ascending	pedunculated	2.5 cm	TVA(focal HG)
9	Our case	51/M	Sigmoid	pedunculated (1.5 cm)	2.8 cm*2.6 cm*1.9 cm	TVA(focal HG)

Abbreviation TA, Tubular adenoma; TVA, tubular villous adenomas; LGD, low-grade dysplasia; HGD, high-grade dysplasia

the pedicle, accompanied by nested or cribriform-morular like structures, were highly prone to misdiagnosis as malignant lesions. Pathologists need to remain vigilant. Under endoscopic ultrasound, dilated cysts appeared as hypoechoic masses in the pedicle, with hyperechoic areas visible when there was fluid or bleeding inside the cyst. This finding suggests pseudoinvasion of colorectal adenomas rather than malignant submucosal invasion [25].

The pathogenesis of pseudoinvasion in colorectal adenomas is believed to involve repeated torsion of the stalk, mucosal muscle injury, bleeding, and herniation of the epithelium into the submucosa. Histologically, the coexistence of mucosal muscle complex hyperplasia, mixed submucosal adenomatous glands with normal mucosal epithelium, lamina propria, hemosiderosis, and inflammatory proliferative reactions support this viewpoint. However, the histogenesis, mechanism, and significance of SM remain unclear. Its occurrence may be due to abnormal differentiation of adenomatous epithelium or multipotential reserve cells beneath adenomatous epithelium [5, 14, 18, 21]. We have summarized all cases and found that SM can be located on the surface, base, or middle of the adenomas, and even within a pseudoinvasive focus. However, all cases of SM were associated with adenomatous glands, and there have been no reports of its association with residual normal glandular epithelium within the polyp or normal glands misplaced into the submucosa. This supports the above idea. In addition, we observed some CD56 and Syn positive cells within the squamous metaplastic zone of the adenoma, while few cells in both the typical adenoma and normal glandular epithelium. This supports the hypothesis that multipotent undifferentiated reserve cells can differentiate toward different pathways and not only into glandular cells or squamous cells, but also into neuroendocrine cells. Under certain stimuli, the differentiation program is initiated. However, considering that raised polyps are prone to mechanical stimulation and often accompanied by chronic inflammation, and given the rarity of SM in polyps, it does not provide strong support for mechanical stimulation and chronic inflammation being significant inducing factors. Our results showed that the proportion of SM in low-grade and high-grade adenomas was 1.8:1, which does not suggest an increased risk of further malignant transformation in adenomas with SM.

In recent years, there have been reports on immunohistochemical and molecular studies [6, 11, 22–24], which have played a certain role in the diagnosis, differential diagnosis, and understanding of histogenesis and pathogenesis. The SM cells in colorectal adenomas were found to be positive for CK5/6, and in rare cases [11], a few P63-positive cells were scattered. Neuroendocrine markers were not expressed or only expressed in a few cells, which could be distinguished from complex adenoma microcarcinomas of the colon with similar histological morphology [27, 28]. Complex adenoma microcarcinomas are rare colorectal lesions that are more common among middle-aged and elderly patients, with an average age of 62 years. They occur equally in both genders and are more commonly found in the proximal colon. Histologically, complex adenoma microcarcinomas consist of typical adenomatous components and a small number of well-differentiated neuroendocrine cells. Neuroendocrine cells are mainly arranged in small clusters and glandular-like structures, sometimes resembling squamous mulberries. The Ki67 proliferation index is also very low. However, in complex adenomatous microcarcinomas, Syn is diffusely expressed in the carcinoid components, with most cells expressing CGA. On the other hand, the SM cells do not express CGA, but in a few cases, Syn might be scattered. Bland cytological morphology and extremely low Ki67 proliferation index of the SM cells serve as useful immunohistochemical markers for distinguishing them from high-grade components within adenomas and invasive carcinoma. Aberrant nuclear expression of β-catenin has recently been reported in squamous morula-associated neoplasms such as low-grade fetal adenocarcinoma of the lung, endometrial adenocarcinoma, cribriform morular thyroid carcinoma, pyloric adenoma of the gallbladder, and pancreatoblastoma. Kunio et al. [23] conducted mutation analysis through microsurgical dissection of the lesion and did not detect β-catenin, KRAS, or BRAF mutations in the nuclei of SM and adenoma. Our next-generation sequencing results were consistent with theirs. Therefore, there is no evidence to suggest that β-catenin, KRAS, and BRAF mutations are associated with the formation of SM in colorectal adenomas. Nevertheless, further research is still needed to elucidate the pathogenesis and significance of SM in colorectal adenomas.

Pseudoinvasion and SM of colorectal adenomas are both benign processes and that local excision should be sufficient treatment. However, pathologists are prone to misdiagnosis as malignant during diagnosis, especially in small or distorted biopsies. In addition, most authors believe that the SM of colorectal adenomas may be the origin of primary squamous cell carcinoma of the colon and rectum. The histogenesis, significance, and natural history of colonic adenoma with squamous metaplasia are not yet understood very well, further research is still needed.

Abbreviations

SM	Squamous metaplasia
WHO	World Health Organization
HE	Hematoxylin and eosin
PAS	Periodic Acid-Schiff
EB	Expert Board

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13000-024-01535-9.

Supplementary Material 1

Supplementary Material 2

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Author contributions
YW diagnosed the cases. LS contributed to writing the manuscript and constructed the figures. HML reviewed and edited the text. SLL and SYL provided additional clinical data. YW conceived the study design and provided final edits to the manuscript. All authors reviewed the manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This research was conducted under the Declaration of Helsinki. Because this manuscript is only a retrospective description of a rare disease, an ethics committee was unnecessary. In addition, the patients gave written consent to publish the data.

Competing interests
The authors declare no competing interests.

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