DOI: 10.7860/JCDR/2026/82239.22589

Case Report

Pathology Section

Filiform Polyposis (A Rare Form of Pseudopolyps) with Co-existing Cytomegalovirus Infection in a Case of Ulcerative Colitis

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ABSTRACT

Filiform Polyposis (FP) is a rare form of pseudopolyps, occurring most commonly in the large intestine. It usually occurs as a rare complication of inflammatory bowel disease (Crohn's disease or, more commonly, ulcerative colitis). But it can also occur in other chronic inflammatory or infectious diseases affecting the large bowel. Most common locations are the rectum and sigmoid colon for FP, but they can involve any length of the large intestine. They can occur as finger-like projections, sometimes with interconnections and can affect the entire colonic mucosa in a diffuse manner. Colonoscopy is the usual and most common modality for diagnosis. They are known to occur as a reparative process to chronic inflammatory conditions affecting the bowel. Inflammatory Bowel Disease (IBD) patients harbouring Cytomegalovirus (CMV) infection can also predispose the colonic mucosa to develop FP. Management usually depends on the symptoms. Surgical management is restricted in cases of complications like intestinal obstruction or bleeding and pain. Here, we report a case of a 55-year-old male with pre-existing ulcerative colitis with co-existing CMV infection, now presenting with severe intestinal obstruction. Colonoscopy revealed many polypoidal projections involving the rectum and sigmoid colon, following which he underwent subtotal colectomy. The colon specimen histopathologically revealed to have multiple interconnecting polypoidal projections lined by normal colonic mucosa with areas of chronic inflammatory changes, which was confirmed to be FP in a known case of ulcerative colitis. Though FP has no malignant potential, it can lead to intestinal obstruction and other complications, making routine follow-up necessary for patients with IBD.

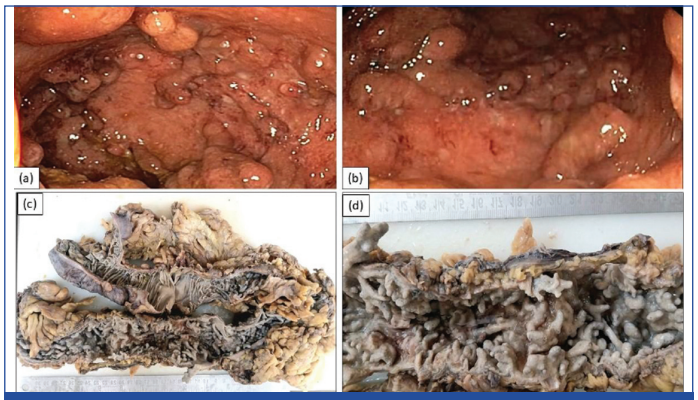
Keywords: Colonic biopsy, Colonoscopy, Complication, Cryptitis, Immunosuppressive drugs, Inflammatory bowel disease

CASE REPORT

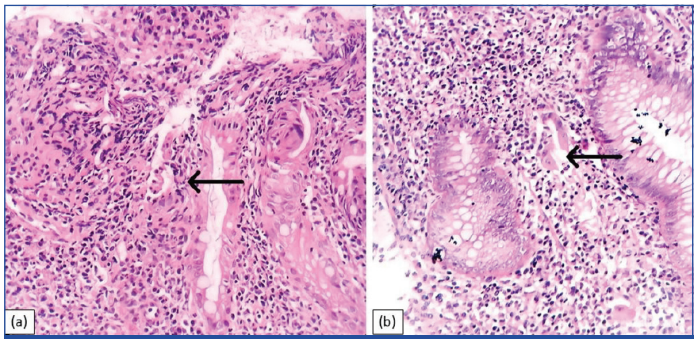
A 55-year-old male who is a diagnosed case of Inflammatory Bowel Disease (IBD) (ulcerative colitis) has been under immunotherapeutic treatment with adalimumab for the last two years. He had a poor response (increased stool frequency and blood in stools) to immunotherapy, along with irregularity in medications. Therefore, he was recently supplemented with oral methyl-prednisolone. He now presented with colicky pain and melena for four days, for which he underwent endoscopic evaluation. Sigmoidoscopy was done and colonic biopsy samples were sent for histopathological study and Cytomegalovirus (CMV) DNA Polymerase Chain Reaction (PCR) assay.

Sigmoidoscopy revealed numerous polypoidal structures beyond the rectal mucosa with superficial ulceration and adjacent colonic mucosa showed few superficial ulcerations [Table/Fig-1a,b]. A provisional diagnosis of acute severe pancolitis with pseudopolyps involving the entire colon was made from the sigmoidoscopic findings. Histopathological examination of the colonoscopic biopsy sample revealed cryptitis, crypt abscess, crypt budding and branching, suggesting Ulcerative Colitis (UC) in active phase. Intranuclear viral inclusion bodies were also seen in the endothelial cells of blood vessels in colonic epithelium [Table/Fig-2]. CMV DNA PCR assay on the colonic tissue came out to be positive for CMV infection. The patient was treated with an injection of Ganciclovir for 14 days. However, a month later, he presented with a sudden onset of acute intestinal pain and obstruction with abdominal distension. Ultrasonogram done for acute abdomen revealed hepatomegaly and moderate pleural effusion. He immediately underwent subtotal colectomy with end ileostomy and the resected colon specimen was sent for histopathological analysis.

The colon specimen, when opened, revealed numerous polypoidal lesions (more than 100 in number) occupying more than 70% of the colonic mucosa [Table/Fig-1c,d]. Microscopic examination of the

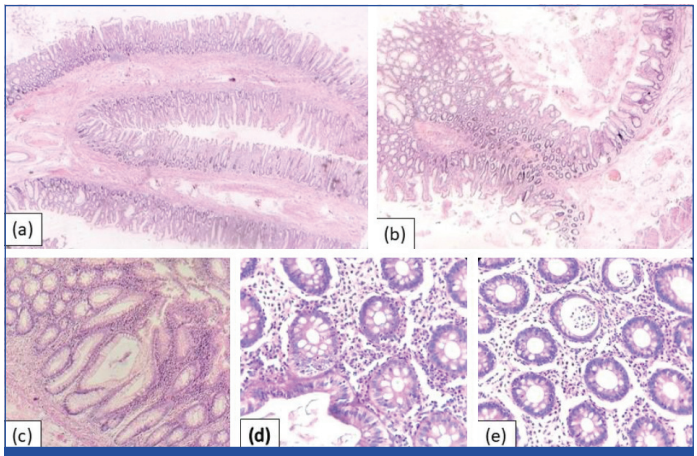


[Table/Fig-1]: (a,b) Sigmoidoscopy showing multiple numerous finger-like polyps arising from the colonic mucosa and some showing mild superficial ulceration; (c) Gross specimen of subtotal colectomy specimen from a patient of refractory ulcerative colitis showing features of Filiform Polyposis (FP); (d) Colectomy specimen of FP in a patient of ulcerative colitis showing presence of numerous finger-like projections (pseudopolyps) covering the entire colonic mucosa. These polyps are long, slender, branching and have a long stalk and can range from a few to numerous in number.



[Table/Fig-2]: (a,b): Micrographs of a small biopsy of colon showing the presence of CMV cytopathic effect, which is usually encountered in the endothelial cells of blood vessels in colonic mucosa (H&E, 400x magnification).

sampled polyps revealed projections of the colonic mucosa with elongated, branched glands and dilated crypts. The central stalk of these polyps contained dilated and congested blood vessels, nerve bundles and inflammatory cells comprising of lymphocytes, plasma cells and few neutrophils [Table/Fig-3a,b]. Focal surface ulceration was observed. Crypt architectural distortion in the form of crypt budding and branching was noted [Table/Fig-3c]. The adjacent colonic mucosa showed cryptitis, crypt abscess and basal plasmacytosis [Table/Fig-3d,e]. No evidence of dysplasia or malignancy was noted in the sections studied.



[Table/Fig-3]: (a,b): Micrographs of the sampled polyps showing elongated finger-like projections lined by normal colonic mucosa and having a layer of submucosa with blood vessels in the core of the polyps. (H&E stain, 200X low power magnification) (c): Micrographs of thin, slender polyps lined by intestinal mucosa showing features of chronic active colitis, like crypt architecture distortion, crypt budding and branching and moderate lymphoplasmacytic inflammatory cells infiltration in the lamina propria. (H&E stain, 400x high power magnification) (d,e) Colonic crypts showing cryptitis and crypt abscess with moderate lymphoplasmacytic inflammatory cells infiltration in lamina propria (H&E, 400X)

A final diagnosis of FP in a known case of UC was made. IV steroids and antibiotics were continued for 15 days postoperatively. The patient was asymptomatic in routine follow-ups and was continued on immunotherapy and steroids.

DISCUSSION

Filiform Polyposis (FP) is an unusual entity occurring as a complication in 10 to 15% of patients with UC and can be associated with other forms of IBD [1]. The term was first coined by Appleman et al., to describe the radiological appearance of numerous worm-like filiform projections in the colon [1]. Most common locations are the rectum and sigmoid colon for FP, but they can involve any length of large intestine [2]. They can occur as finger-like projections, sometimes with interconnections and can affect the entire colonic mucosa in a diffuse manner depending on the chronicity of inflammation [3,4]. FP usually occurs due to an inflammatory process, which can cause mucosal ulceration. The ulceration undergoes re-epithelialisation with time, separated by edematous mucosa that in due course of time takes the shape of a polyp [2].

Ali Z and Iltaf S described a case of a 45-year-old man who presented with bleeding per rectum. His endoscopic colonic biopsies revealed chronic active colitis and the subtotal colectomy specimen histopathologically revealed Giant FP. The polyps were numerous in number and lined by normal colonic mucosa [1]. In our present case, more than 100 finger-like projections were seen in the colon grossly, which were lined microscopically by normal colonic mucosa without any dysplasia.

Gurung N et al., described a case of segmental FP in a treated case of UC affecting the colon with abrupt normal colonic mucosa in between [2]. However, in our present case, the polypoidal lesions were found in the entire length of the colon with no intervening normal mucosa. Yan W et al., reported a case of a 29-year-old female presenting with worsening abdominal pain, vomiting and decreased appetite. Her colonoscopy revealed a large near-circumferential, heterogeneous, fungating and pseudopolypoidal mass partially obstructing the lumen and extending from the splenic flexure to the hepatic flexure and ascending colon. Microscopically, it showed inflamed polypoidal colonic mucosa with granulation tissue and a final diagnosis of Giant FP was made [3]. However, in our present case, there were multiple interconnecting discrete pseudopolyps instead of a large fungating mass in the colon.

Chronic inflammation of the colonic mucosa in patients with IBD superimposed with ulceration and healing is believed to play a major role in the development of pseudopolyps. Some authors mentioned rare cases of FP occurring in the setting of certain other inflammatory conditions like intestinal Tuberculosis and Histiocytosis X. Moreover, it seems that inflammatory cytokines and the traction of redundant mucosa by intestinal peristalsis with intestinal wall hyperplasia and fibrosis may play a role in the development of FP [5].

Grossly, FP usually presents as few to numerous (more than 100) polyps covering the entire colonic mucosa. These polyps are uniform, thin, sometimes branching projections that resemble the stalk of the polyps without the head (hence referred to as pseudopolyps) [6]. Microscopically, these polyps are lined by normal appearing intestinal mucosa without any dysplasia. Adjacent mucosa may show evidence of IBD [4].

True polyps are abnormal growth or proliferation of cells, which can be a precancerous or cancerous lesion or occur as a part of polyposis syndromes. True polyps can be sessile or pedunculated and can be removed during endoscopy. They can be found anywhere in the gastrointestinal tract, depending on the type of polyp. However, they are more common in the large intestine and can undergo malignant transformation unless removed surgically. Pseudopolyps, however, are lined by normal or inflamed mucosa along with dilated blood vessels in the submucosa, forming the stalk of the pseudopolyp and are seen in patients with IBD. Pseudopolyps do not undergo malignant transformation and do not require polypectomy unless complications arise [6,7].

In our case, UC was treated with immunosuppressive drugs, but the patient showed a poor response to treatment, leading to the formation of FP. In most of the studies done on UC, authors have found that pseudopolyps and FP occur in long-standing cases of IBD and are limited to the left colon. Sometimes pseudopolyps are incidentally diagnosed in patients with bloody diarrhoea and colonoscopy revealing multiple polyps with a suspicion of Familial Adenomatous Polyposis (FAP) or colon carcinoma. However, the absence of dysplasia and presence of histopathological changes of colonic mucosa in IBD prove these to be indolent pseudopolyps in cases of UC [4,7].

Infection with CMV in UC is very rare, accounting for only 1% to 4% of cases with IBD [8]. Treatment of UC with steroids and immunosuppressive drugs may predispose to CMV infection, leading to frequent flare-ups and refractory UC [8]. Diagnosis of CMV in a case of UC is difficult due to the overlapping of clinical symptoms and endoscopic findings of CMV colitis and UC. Patients with UC and coexisting CMV experience poor clinical outcomes. Antiviral therapy significantly reduces the need for colectomy in patients with severe UC and high-grade CMV infection. This indicates that CMV plays a role in the progression and severity of UC [7]. Therefore, detection of CMV is important and can be done by histopathology and immunohistochemistry or viral markers assay on intestinal mucosa.

The most common site for FP is the colon, though filiform polyps have also been described in the oesophagus, gastric and small intestinal mucosa. Sometimes, FP can very rarely occur in the absence of chronic IBD, proceeding bacillary dysentery, necrotising enterocolitis, enema-induced colitis, uretero-sigmoidostomy, Langerhans cell histiocytosis or colonic tuberculosis [7]. Therefore, meticulous examination by clinicians, radiologists and pathologists must be done to find out the etiology of FP and adequate

management and follow-up of such cases must be done to prevent complications.

CONCLUSION(S)

The FP is worth mentioning as it is a highly rare entity in a pre-existing case of UC and it may be a consequence of secondary CMV infection leading to poor response to medical treatment. It is also important to distinguish FP from other intestinal polyps and neoplasms, as FP almost never shows evidence of dysplasia. In all complicated and symptomatic cases of UC with FP, partial or subtotal colectomy is the treatment of choice along with regular follow-up.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Jul 29, 2025
- Manual Googling: Nov 29, 2025
- iThenticate Software: Dec 02, 2025 (7%)

ETYMOLOGY: Author Origin

EMENDATIONS: 5

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Jul 22, 2025**
Date of Peer Review: **Aug 18, 2025**
Date of Acceptance: **Dec 04, 2025**
Date of Publishing: **Mar 01, 2026**