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Case Report





Case Report: Cap Polyposis in Advanced Pelvic Floor Dysfunction - Stronger Evidence of a Mechanical Prolapse-Related Pathology

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Abstract

We describe a case of diffuse rectal involvement with cap polyposis, manifesting as a protein-losing colopathy and occurring in the setting of advanced mechanical pelvic floor dysfunction. A 59-year-old male with a 5-year history of persistent excessive flatulence, defecatory difficulties and diarrhoea was diagnosed with extensive cap polyposis of the entire rectum. His symptoms progressed to severe faecal incontinence with mucus leakage, pelvic pain, weight loss, and hypoalbuminemia. Clinical examination exhibited severe perineal descent, a large rectocoele, poor anal squeeze, and a poor defecatory technique. After a trial of non-operative therapies addressing his defecatory dysfunction and *Helicobacter Pylori* eradication, surgical resection was offered due to severe symptoms with ongoing incontinence and protein loss with no other reasonable option. A robotic perineal resection with a permanent colostomy was performed, followed by an uncomplicated recovery. Our observation of coexisting mechanical pelvic floor dysfunction floor changes in this patient lends weight to the concept of prolapse-related floor changes in the pathophysiology of this rare condition.

Keywords: Cap polyposis; Case report; Faecal incontinence; Pelvic dysfunction

Introduction

Cap polyposis is a rare, benign, intestinal condition characterised by erythematous, inflammatory polyps covered by a 'cap' of fibrinopurulent mucous. It was initially described by Williams et al, and to date, has been followed by under 100 case reports/series in English literature [1-4]. It affects patients of both genders in a wide age range, usually in the 5th decade, but can also affect the paediatric population [2,4]. Cap polyps may be found throughout the colon, most commonly localised in the rectum and rectosigmoid area. The most common symptoms include abdominal pain, rectal bleeding, and mucoid diarrhoea [1,5] and severe cases of cap polyposis can lead to protein-losing

enteropathy [6]. The pathogenesis of cap polyposis remains unclear and is debated between infectious, inflammatory, and mucosal prolapse aetiologies. We describe a case of diffuse rectal involvement with cap polyposis leading to profuse mucus leakage, faecal incontinence, and protein-losing colopathy in a patient with advanced mechanical pelvic floor dysfunction.

Case Presentation

A 59-year-old male presented with initial complaints of excessive flatulence. He was well apart from having a history of Gastro-Oesophageal Reflux Disease (GORD) and renal calculi. He was an ex-smoker and had moderate alcohol consumption. His regular medication was pantoprazole. The patient described a 5-year history of mixed obstructive defecation symptoms, faecal urgency, and flatus incontinence that worsened gradually

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over time. He tried a low FODMAP and dairy-free diet but had persisting symptoms.

A diagnosis of cap polyposis was made on colonoscopy, with 5-10mm polypoid lesions extending from the anorectal junction proximally to the sigmoid colon, mainly affecting the rectum with diffuse involvement (see Figure 1). Histology confirmed innumerable erythematous polyps with mucus caps, hyperplastic crypts and expanded, inflamed lamina propria. Clinically he exhibited signs of advanced pelvic floor dysfunction with significant perineal descent, a palpable rectocele, poor anal squeeze, and a poor defecatory technique. He was treated with dietary alteration, regular laxatives, biofeedback therapy, and a course of *H.Pylori* eradication.

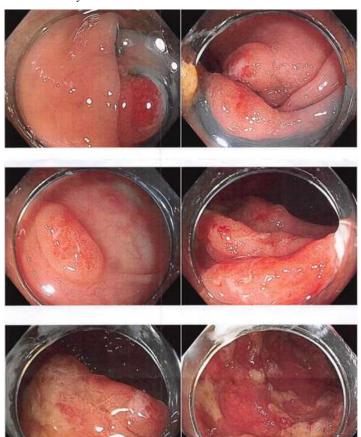


Figure 1: Endoscopic views of rectosigmoid with polyps.

Several months later his condition deteriorated despite the aforementioned treatment. He experienced encore defecation, increased frequency, and faecal incontinence. His bowels worked 35-40 times per day, mostly mucoid in quality, only able to pass small quantities at a time. Rectal pain became a predominant symptom along with tenesmus. He lost 5kg in weight and developed new

hypoalbuminemia (19-26 g/L). A Computed Tomography (CT) scan revealed gross thickening and oedema of the rectosigmoid colon (see Figure 2). A repeat colonoscopy showed progression to almost circumferential polyposis, with polyps up to 2cm in size and causing a degree of luminal narrowing (see Figure 3).





Figure 2: Axial and sagittal views revealing thickened rectum.

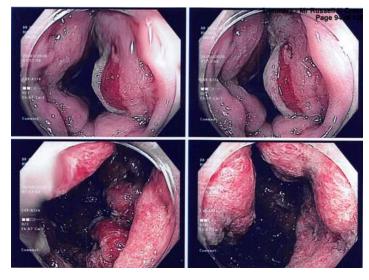


Figure 3: Endoscopic views of progression.

A decision was made to proceed with surgical resection due to his severe symptoms and protein loss. Another course of antibiotics or a trial of an immunomodulator was deemed unlikely to be of benefit. A robotic abdominoperineal resection with a permanent colostomy was performed. Operative findings included a megacolon, supporting his history of straining and constipation, and a rectum containing extensive polyposis (see Figure 4). The procedure was straightforward and the patient had an uncomplicated recovery with regular follow-up and stoma care.

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Figure 4: Abdominoperineal resection specimen. A. Anorectum B. specimen opened revealing extensive polyposis

Discussion

Cap polyposis is a rare condition first described in 1985 by Williams et al and has been followed by several case reports and series. It is characterised by multiple inflammatory polyps covered by a cap of fibrinopurulent exudate and surface granulation. These polyps can be found anywhere along the colon but are more commonly seen between the rectum and distal colon. The most common are abdominal pain, rectal bleeding, and mucoid diarrhoea [4,6,7]. It usually affects adults in a wide age range between 16-76 years old but is also observed in the paediatric population. The pathogenesis of cap polyposis remains poorly understood. The formation of polyps may represent a reaction to mucosal injury mainly attributed to infection, inflammatory bowel disease, and localised mucosal prolapse.

Multiple case reports have reported on the relationship with *Helicobacter pylori*, some showing impressive cure rates with subsequent eradication [3,8]. A case of complete resolution from treatment with Metronidazole even though no causative organism was identified suggested that the anti0inflammatory actions of the drug may play a central role in its healing [9]. Successful treatment with infliximab, a monoclonal antibody against tumour necrosis factor (TNF)- α , suggests a T-cell mediated enteropathy has also been described. A case of surgical resection followed by confined recurrence along the anastomotic line suggested that inflammatory processes in wound healing instead were implied in its pathogenesis [10].

The initial association of cap polyposis to the spectrum of mucosal prolapse syndromes was postulated based on histologic similarities. Cap polyps exhibit elongated, hyperplastic glands with fibromuscular obliteration and mixed inflammatory infiltrates in the lamina propria as can be seen in Solitary Rectal Ulcer Syndrome (SRUS) and severe mucosal prolapse [5,11,12]. It has been hypothesised that chronic mechanical straining and recurrent mucosal trauma lead to prolapse at redundant folds, followed by ischemia, subsequent inflammatory reaction and polyp formation

[1,13,14]. Protein-losing enteropathy occurs in advanced cases as persistent mechanical stimulation leads to mucosal hyperplasia and hypersecretion of serum proteins into the lumen [6]. The most commonly reported symptoms of abdominal pain, rectal bleeding, and mucoid diarrhoea potentially lead to a misdiagnosis of Inflammatory bowel disease as the clinical and endoscopic features can be similar. Notwithstanding, symptoms of pelvic floor dysfunction including constipation, tenesmus, and habitual straining at defaecation have also been consistently reported [7,15] and raise the possibility that cap polyposis belongs to the spectrum of mucosal prolapse, solitary rectal ulcer syndrome, and other related pathologies.

Consequently, optimal treatment for cap polyposis has not been established given its incidence and unclear pathogenesis. Different approaches have been met with mixed success [3]. These range from close observation in asymptomatic patients to relieving straining in patients with colonic dysmotility, eradication of *H. pylori*, treatment with steroids and/or immunomodulators as per inflammatory bowel disease, to endoscopic polypectomy or surgical resections in those not responding to medical therapy [4,7,10,13,14,16-21].

Conclusion

Our case of cap polyposis presented with signs of severe pelvic floor dysfunction and impaired rectal biomechanics. These examination findings have not been previously reported in cap polyposis and support the concept of prolapse syndrome playing a major role in its pathogenesis. Exhaustive work-up to identify the initial injury -whether it be infection, inflammatory, or mechanical - and establishing early treatment may prevent disease progression and preclude surgical intervention.

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