Case Report

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When Joint Swelling Hides A Secret: The Unexpected Discovery Of Rice Bodies And Literature Review

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1. Abstract
Cronkhite-Canada syndrome (CCS) is an extremely rare acquired polyposis syndrome of unknown etiology. It was first reported in 1955 by Richard L. Cronkhite and Wilma Canada. Its characteristic features include diarrhea and cutaneous changes (such as alopecia, hyperpigmentation, and onychodystrophy). It is also known as the polyposis-pigmentation-alopecia-onychodystrophy syndrome. Polyps are diffusely distributed throughout the entire gastrointestinal tract, except for the esophagus. Familial Adenomatous Polyposis (FAP) is an autosomal dominant hereditary disorder typically manifesting around the age of 20, with adenomas predominantly concentrated in the rectum and colon. This article presents a case of CCS misdiagnosed as FAP, aiming to enhance clinicians' understanding of both conditions and improve the diagnosis and treatment of this rare disease.

2. Keywords
Cronkhite–Canada syndrome, Familial Adenomatous Polyposis, gastrointestinal polyps

3. Case Data
A 61-year-old male patient was admitted due to "hematochezia, acid reflux, alopecia, onychodystrophy, hypogeusia, decreased appetite and weight loss, and bilateral lower limb edema." The symptoms had intermittent episodes over a course of more than one year. On November 10, 2020, the patient underwent gastroscopy, which revealed polypoid hyperplastic lesions in the cardia, gastric body, gastric angle, and gastric antrum, with mucosal erythema and stromal edema in the lamina propria. On November 23, 2020, colonoscopy showed over 100 polyps (approximately 0.2-2.0 cm in size) in the ileocecal region, entire colon, and rectum, which were flat and some with short stalks. In December 2020, the patient underwent polypectomy, during which more than 30 polyps were removed. After discharge, his alopecia showed significant improvement.

On March 10, 2021, follow-up gastroscopy revealed a 0.6 cm submucosal bulge at the 9 o'clock position of the cardia, rough and uneven mucosa in the gastric fundus with patchy bleeding and erosions, and rough, fish-scale-like changes in the gastric angle mucosa, which was firm on biopsy. The folds in the upper and middle gastric body were markedly edematous, and the mucosa from the lower gastric body to the antrum was rough, dry, thin, with reduced secretions and active peristalsis. Colonoscopy showed several 0.5-1.5 cm Yamada type I-III polyps in the terminal ileum and scattered diffuse Yamada type I-IV polyps (0.5-3.0 cm) throughout the entire colon. Familial polyposis was considered, and the symptoms alleviated after albumin infusion and diuretic therapy. After another polypectomy, hematochezia and abdominal pain improved.

In June 2021, the patient experienced recurrent hematochezia and bilateral lower limb edema. A external hospital prescribed prednisone acetate 20 mg once daily for two weeks. During oral medication, the frequency of hematochezia increased. Improvement was observed only after albumin infusion and diuretic therapy. The poor response to steroids may have been due to insufficient dosage. According to a retrospective analysis of 210 CCS patients, oral corticosteroids (30-49 mg/day) are the most effective treatment for active disease, and adjunct nutritional support is also considered beneficial for patients [1].

In October 2021, the patient presented with fresh blood in stool, 4-5 times daily, accompanied by generalized fatigue and bilateral lower limb edema. No abnormalities were found in complete blood count, liver and kidney function, blood glucose and lipids, antinuclear antibody profile, or anti-neutrophil cytoplasmic antibodies. Since the onset of the disease, the patient had poor sleep, lethargy, and fatigue, with a weight loss of 15 kg over the past year. He denied a history of smoking or alcohol consumption.

Physical examination on admission: temperature 36.4°C, heart rate 74 beats/min, respiratory rate 18 breaths/min, SpO2 98%; symmetric blood pressure in both upper limbs (84/48 mmHg), height 167 cm, weight 52 kg, BMI: 18.64. The patient was well-developed but emaciated. Hair, eyebrows, and eyelashes were lost, with visible newborn vellus hair; no beard. The skin on the dorsum of both hands was hyperpigmented, and the fingernails and toenails appeared yellowish-white, with partial loss. Heart

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rhythm was regular, and lung sounds were clear. The abdomen was soft without tenderness or rebound tenderness. Bilateral lower limb edema was present. The Nutritional Risk Screening (NRS-2002) score was 4, indicating malnutrition.

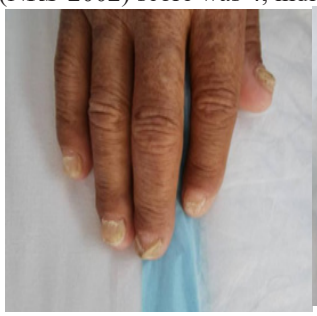


Figure 1: Fingernail Loss

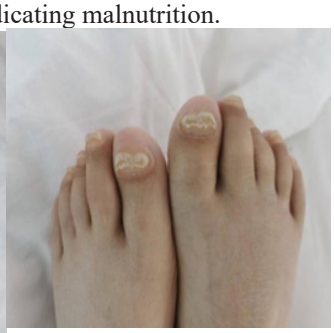
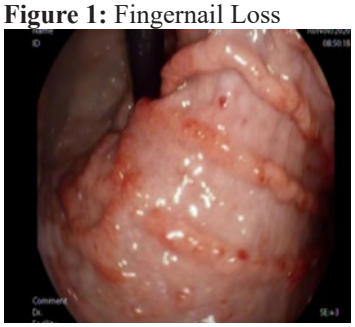
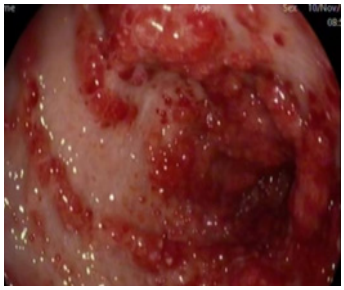
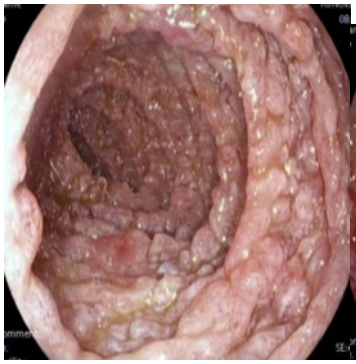


Figure 2: Toenail Loss







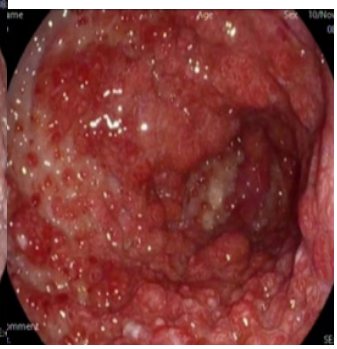


Figure 3-4: Multiple gastric polyps with hyperemia and edema
Figure 5-6: Multiple colonic polyps with hyperemia, edema, and ulceration

4. Discussion

The patient is an elderly male with an acute disease course, presenting with hematochezia, fatigue, onychodystrophy, weight loss, and hyperpigmentation on the dorsum of both hands. Diffuse polyps were observed in the stomach, entire colon, and rectum. Due to limited awareness of CCS, the initial diagnosis was mistaken for FAP. The most prominent feature of FAP is the presence of thousands of adenomatous polyps in the colon and rectum, varying in size and distribution. These polyps typically begin on the right side of the colon and extend to the left colon or rectum, primarily concentrating in the colon and rectum, with most being adenomatous [2]. Early polyps are usually small and flat with a smooth surface. As the disease progresses, some polyps may enlarge, become raised, and exhibit varying degrees of erosion or ulceration, particularly larger or dysplastic polyps. Most are tubular adenomas, though villous adenomas may also occur, and the majority exhibit intraepithelial neoplasia [3]. However, FAP is usually diagnosed in adolescence or early adulthood [3]. It is an autosomal dominant disorder caused by APC gene mutations, often with a clear family history. Onychodystrophy is rare, and significant cutaneous or alopecia changes are uncommon unless complicated by Gardner syndrome (a subtype of FAP).

In contrast, CCS typically occurs in middle-aged and elderly individuals, manifesting as widespread gastrointestinal polyps, chronic diarrhea, hypogeusia, onychodystrophy, alopecia, dry mouth, skin hyperpigmentation, and weight loss [4]. It is a sporadic disease without a clear genetic predisposition, and its pathogenesis remains incompletely understood, though immune dysfunction or environmental factors are suspected. In addition to nonspecific polyps distributed throughout the stomach, small intestine, colon, and rectum, CCS is accompanied by cutaneous and hair changes such as thinning nails, alopecia, and abnormal skin pigmentation (e.g., hyperpigmented patches), which may signal disease progression [5]. Brittle, atrophic, or shedding nails are another common dermatological manifestation in CCS patients, possibly related to malnutrition and chronic illness [6]. In some cases, nail changes are considered an early indicator of CCS, highlighting the need for clinicians to pay attention to these subtle signs during evaluation [7].

A retrospective study of 103 Chinese CCS patients revealed that polyps were most widely distributed in the gastrointestinal tract, primarily in the gastric antrum (77.33%) and duodenum (56%), often appearing as small, flat, or sessile elevated polyps [8]. Histologically, hamartomatous polyps were most common (64.91%), followed by hyperplastic polyps (31.58%), with endoscopic findings showing significant hyperemia and edema on the surface [9]. Another retrospective study of 76 cases found that histological analysis of CCS polyps exhibited notable stromal edema, eosinophilic infiltration, and cystic dilation of glands. Almost all pathological reports indicated stromal edema, while cystic gland dilation was observed in 70.69% and eosinophilic infiltration in 43.10% of cases. These distinctive features differentiate CCS from other gastrointestinal polyposis syndromes and suggest a close association with activated inflammatory responses [10].

Endocrine abnormalities are also relatively common in CCS patients, particularly in cases of long-term malnutrition. A retrospective study by Lanping ZHU et al. indicated that CCS patients may present with hypothyroidism, thyroid cancer, or cervical cancer, often accompanied by hormonal imbalances such as abnormalities in sex hormones and thyroid hormones, which may further impact overall health and quality of life [11]. In this case, the patient experienced malnutrition, protein deficiency, severe diarrhea, and gastrointestinal bleeding caused by polyps. Treatments such as nutritional support (especially protein and vitamin supplementation), antidiarrheal medications, and polypectomy provided limited relief.

To date, approximately 500 cases of CCS have been reported worldwide, with an estimated incidence of one per million [12]. Due to the rarity of the disease, the exact prevalence and incidence remain unclear. However, studies suggest significant regional and

demographic variations. For instance, relatively more cases have been reported in certain Asian countries compared to Western nations [13]. In Japan, CCS cases are primarily concentrated in middle-aged and elderly populations, with a mean age of 63.5 years [6], and a male-to-female ratio of approximately 2:1 [7]. Moreover, due to underreporting and limited awareness among clinicians, some cases may be misdiagnosed or overlooked, suggesting that the actual incidence may be higher than currently reported.

Although no specific genetic mutations have been definitively linked to CCS [7], some case reports indicate a family history of autoimmune diseases in affected individuals. Patients often exhibit manifestations of autoimmunity, such as skin pigmentation changes and alopecia, implying a potential role of the immune system in the disease pathogenesis [3].

In summary, this report presents the clinical case of a 61-year-old male patient, detailing the clinical manifestations, diagnostic pitfalls, and treatment experience of CCS. The case highlights typical symptoms such as hematochezia, alopecia, hypogeusia, onychodystrophy, and bilateral lower limb edema, while emphasizing the critical role of endoscopic examination in diagnosis. Through a thorough review of the patient's history and multiple endoscopic findings, CCS was ultimately diagnosed, and corresponding treatments were administered. As a rare non-hereditary disorder, CCS shares some clinical features with FAP but differs significantly in pathological characteristics and genetic background. Therefore, clinicians must enhance their understanding of CCS, integrate detailed medical histories, clinical presentations, and endoscopic results for comprehensive evaluation to avoid misdiagnosis or missed diagnosis. Future research should further explore the pathogenesis of CCS and identify effective treatment strategies to improve patients' quality of life and survival rates. Additionally, raising awareness and providing education about the disease are crucial for reducing diagnostic errors.

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