

Spontaneous Passage of a Retained Small-Bowel Capsule after Corticosteroid Therapy in Refractory Celiac Disease: A Case Report

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Abstract

Capsule retention is a rare but clinically significant complication of video capsule endoscopy (VCE), particularly in patients with refractory celiac disease. We report the case of a 42-year-old man with type II refractory celiac disease who developed jejunal capsule retention after VCE despite a negative magnetic resonance enterography (MRE). Conservative treatment with prednisone and laxatives resulted in spontaneous capsule passage after 30 days, avoiding endoscopic or surgical retrieval. This case highlights the importance of careful patient selection, pre-procedural risk assessment, and individualized management of capsule retention in inflammatory small bowel disease.

Keywords: Celiac disease, Video capsule endoscopy, Small Bowel, Refractory celiac disease, Capsule retention.

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INTRODUCTION

VCE has transformed the evaluation of the small bowel by providing a noninvasive, well-tolerated method with high-resolution, wide-angle imaging.

In celiac disease (CD), which primarily affects the small intestinal mucosa, VCE enables the detection of macroscopic lesions and villous abnormalities, thereby complementing traditional diagnostic tools. It also allows assessment of disease extent and severity, as well as identification of complications such as ulcerative jejunoileitis and enteropathy-associated T-cell lymphoma, supporting both diagnosis and long-term monitoring. Although generally safe, VCE carries a risk of capsule retention, particularly in patients with refractory CD or associated complications, which may require endoscopic, surgical, or medical management. We report a rare case of prolonged capsule retention successfully managed conservatively after corticosteroid therapy.

CASE REPORT

A 42-year-old man with a history of seropositive celiac disease (Marsh stage IIIb) had been following a poorly monitored gluten-free diet (GFD)

since 2018. In August 2018, he underwent surgery for bowel obstruction due to small-bowel stenosis, including a 70-cm resection of stenotic segments with associated mesenteric lymphadenopathy. Histopathological examination revealed acute ulcerated enteritis with reactive lymphadenopathy. In 2019, he underwent surgery for an uncomplicated umbilical hernia.

He presented with Koenig-type periumbilical pain, intermittent abdominal bloating, and mucous diarrhea, associated with a 4% weight loss over one month.

The patient subsequently developed painless lower-limb edema extending to the lumbar region, resistant to albumin infusion despite six months of a well-conducted GFD. Prophylactic anticoagulation with heparin was initiated.

Baseline laboratory investigations are summarized in Table 1. They demonstrated normocytic normochromic anemia, thrombocytosis, elevated inflammatory markers, increased fecal calprotectin, negative celiac serology, elevated alpha-1 antitrypsin clearance, and increased β 2-microglobulin levels.

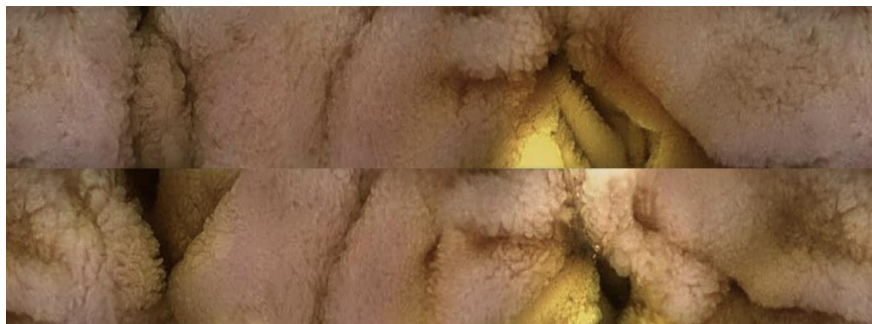
Table 1: Baseline Laboratory Findings at Presentation

Parameter	Result	Reference range
Hemoglobin	11 g/dL	13.5–17.5 g/dL
Platelet count	$713 \times 10^9/L$	$150-400 \times 10^9/L$
C-reactive protein (CRP)	43.63 mg/L	<5 mg/L
Fecal calprotectin	>1000 $\mu\text{g/g}$	<50 $\mu\text{g/g}$
IgA anti-tissue transglutaminase	Negative	Negative
IgA anti-endomysial antibodies	Negative	Negative
Alpha-1 antitrypsin clearance	42.5 mL/day	<27 mL/day
$\beta 2$ -microglobulin	>4 mg/L (on two occasions)	0.8–2.2 mg/L

Esophagogastroduodenoscopy (EGD) showed a cracked appearance of the duodenal mucosa. Histopathological analysis revealed subtotal villous atrophy with 42% intraepithelial lymphocytes, consistent with refractory celiac disease type 2. Ileocolonoscopy with intubation of the terminal ileum showed macroscopically normal mucosa; however, biopsies demonstrated partial villous atrophy with 34% intraepithelial lymphocytes and edematous, congestive colitis. Positron Emission Tomography–Computed Tomography (PET-CT) revealed no abnormal metabolic uptake. Small-bowel VCE was indicated to assess mucosal involvement, evaluate the extent and severity of lesions, and screen for complications such as ulceration or lymphomatous transformation. MRE showed a small peritoneal effusion without bowel dilatation or stenosis. As no contraindication to capsule endoscopy was identified, VCE was performed using the Capsocam® system. Two weeks after capsule ingestion, the patient reported delayed capsule excretion but remained asymptomatic. Computed tomography (CT) enterography confirmed capsule retention in the proximal jejunum at the site of non-stenosing

inflammatory lesions, associated with abundant ascites. A conservative approach was adopted because the patient showed no signs of bowel obstruction. Oral prednisone was initiated at a dose of 30 mg/day in combination with a laxative, with close clinical monitoring and serial abdominal radiographs every 3 days to assess capsule progression. One week after initiating corticosteroid therapy, the patient developed mild abdominal pain without clinical or radiological evidence of intestinal obstruction. Given the patient's previous surgical history, the visceral surgeon recommended postponing endoscopic retrieval. After 30 days of capsule retention, corresponding to two weeks after initiation of corticosteroid therapy, the capsule was spontaneously expelled, thereby avoiding endoscopic or surgical intervention.

VCE demonstrated severe small-bowel mucosal involvement, characterized by localized congestive and nodular gastritis, ulcerative duodenitis, extensive ulcerative and hemorrhagic jejunitis, and an ulcerated jejunal stricture (Figures 1–3).

**Figure 1: VCE showing diffuse ulcerative duodenitis with mucosal edema and erosions****Figure 2: VCE showing extensive ulcerative and hemorrhagic jejunitis with diffuse mucosal inflammation**

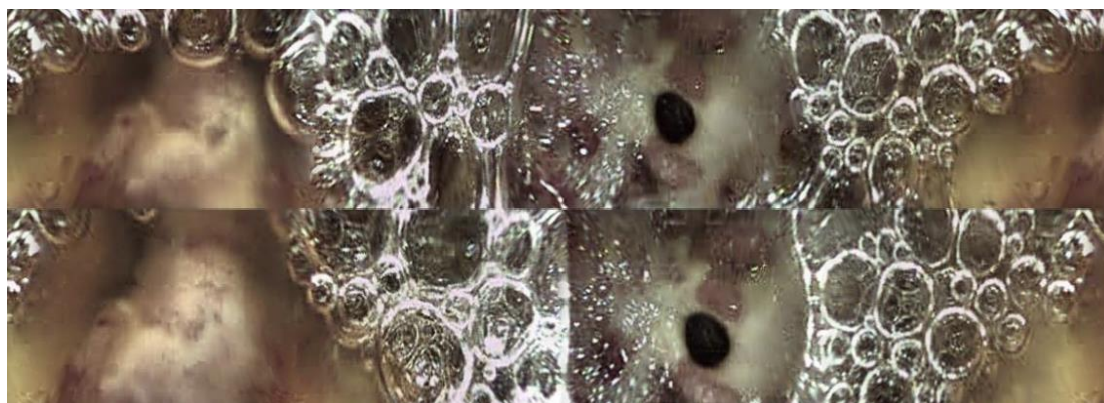


Figure 3: VCE showing an ulcerated jejunal stricture responsible for capsule retention

DISCUSSION

VCE has become a key diagnostic tool for evaluating the small intestine. However, capsule retention remains the main adverse event associated with VCE. Despite this limitation, VCE is considered the most sensitive modality for detecting inflammatory lesions of the small bowel, with a reported sensitivity and specificity approaching 90% [1].

Previously, direct endoscopic examination of the small bowel was limited, with only the duodenum, proximal jejunum and terminal ileum being accessible for visualization using a conventional endoscope. Since its introduction in 2000, small-bowel VCE has become the gold standard for evaluating small-bowel diseases [2]. It is typically not recommended for patients with known or suspected gastrointestinal (GI) obstruction, strictures, fistulas, swallowing difficulties, implanted electronic medical devices, or during pregnancy [3].

Capsule retention is defined as persistence of the capsule within the gastrointestinal tract for at least two weeks or when directed medical, endoscopic, or surgical intervention is required for its retrieval [4].

Most studies have reported a capsule retention (CR) rate ranging from 0.3% to 2.5% across various indications [5, 6]. The small bowel, particularly the ileum, is the most common site of retention, followed by the esophagus [7, 8, 9]. Most patients with CR are asymptomatic, with studies indicating that 38% of patients experience symptoms of obstruction due to the retained capsule [10].

In fact, for suspected CD, the first line evaluation involves serologic testing. In clinical practice, IgA anti-endomysial (EMA) and IgA anti-tissue transglutaminase antibodies, both with reported sensitivity and specificity exceeding 95%, are the standard screening tools for symptomatic patients [11,12]. Patients with positive serology, or those with strong clinical suspicion despite negative tests, should proceed to EGD with small bowel biopsy for definitive diagnosis.

About 30% of CD patients experience persistent or recurrent symptoms despite a GFD [11,13]. Non-responsive CD (NRCD) is defined as ongoing symptoms, signs, or laboratory abnormalities typical of CD after 6–12 months of strict gluten avoidance [14], most often due to unintentional gluten ingestion.

When dietary adherence is confirmed and symptoms persist, the original diagnosis should be reconsidered and alternative causes such as pancreatic insufficiency, irritable bowel syndrome, bacterial overgrowth, inflammatory bowel disease, microscopic colitis, tropical sprue, or small bowel neoplasms should be excluded. Refractory CD (RCD) is diagnosed when symptoms and histologic abnormalities persist beyond 12 months despite strict GFD [11, 13, 14], representing only 1–2% of CD cases but carrying a higher risk of ulcerative jejunitis and enteropathy-associated T-cell lymphoma (EATL) [11].

Small bowel VCE has proven useful in identifying high-risk subgroups, particularly RCD or patients with suspected small bowel neoplasms. Villous atrophy, distal ulcers, and extensive mucosal damage occur more often in RCD type II, and VCE has revealed critical complications like ulcerative jejunitis and lymphoma [15].

Van Weyenberg *et al.*, [16] further demonstrated that absent capsule progression and focal erythema predict poor prognosis. Although ulcerative jejunitis and EATL increase stricture risk, no capsule retentions were reported in published series likely due to careful patient selection. Therefore, in clinical practice, small bowel strictures should be excluded before VCE is performed in NRCD or RCD patients. In the present case, MRE did not demonstrate a significant stricture despite subsequent capsule retention, suggesting that transient inflammatory narrowing may not always be detected by cross-sectional imaging. This finding underscores the limitations of MRE in excluding functionally significant inflammatory strictures and highlights the importance of careful patient selection and close post-procedural monitoring.

In patients with celiac disease, capsule retention may result from transient inflammatory edema or fixed stenosis caused by ulcerative jejunoileitis or malignancy. Differentiating between these mechanisms is crucial for management: while fibrotic strictures generally require endoscopic or surgical removal, transient inflammatory narrowing may resolve with medical therapy, allowing spontaneous passage of the capsule [17,18].

Several studies and case series have documented successful conservative management of capsule retention when the underlying obstruction is inflammatory. Corticosteroid therapy or optimization of disease specific treatment can lead to resolution of mucosal edema and spontaneous evacuation of the device [18,19]. However, this approach requires careful monitoring, as persistence of the capsule beyond several weeks or clinical deterioration mandates active retrieval.

Preventive strategies remain the cornerstone of management in at-risk patients. The use of cross-sectional imaging (CT Enterography or MRE) prior to VCE is strongly recommended in patients with suspected small bowel strictures, as it significantly reduces the risk of capsule retention [20]. In CD, performing such screening is especially important when symptoms such as abdominal pain, bloating, or sub-obstructive episodes are present [20, 21].

Once retention is confirmed, management depends on clinical presentation and the suspected etiology. In asymptomatic patients, a conservative approach with medical therapy (corticosteroids or gluten-free diet reinforcement) and radiologic monitoring may be justified, as illustrated in this case. Conversely, development of obstructive symptoms, capsule migration failure, or imaging evidence of fixed stenosis should prompt intervention either via device assisted enteroscopy or surgery [17,19].

Long term retentions have been reported, with capsules remaining in the bowel for months or even years without symptoms, but these cases highlight the importance of individualized follow-up and a predefined extraction plan [22].

Patients should be informed about the risk of capsule retention before VCE and advised to seek prompt medical attention if capsule excretion is delayed or symptoms of bowel obstruction develop.

CONCLUSION

Capsule retention in celiac disease represents a rare but significant diagnostic and therapeutic challenge. Medical management may be successful when the underlying process is predominantly inflammatory, as illustrated in the present case. However, careful individual assessment, pre-procedural risk stratification, and close clinical follow-up remain essential. Preventive imaging and/or patency capsule testing should be

systematically considered in patients with celiac disease who present with obstructive features or suspected stricturing disease. This case suggests that carefully selected asymptomatic patients with inflammatory capsule retention may benefit from an initial conservative approach before invasive retrieval is considered.

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