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Case Report

Bile Acid Diarrhea: A Case Report

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Abstract

Background: Chronic diarrhea, defined as the passage of loose stools for more than four weeks, is a common clinical problem with a broad differential diagnosis. Bile acid diarrhea (BAD), also known as bile acid malabsorption, is an under-recognized cause of chronic secretory diarrhea and accounts for a substantial proportion of patients previously labelled as having functional diarrhea or diarrhea-predominant irritable bowel syndrome. The diagnosis remains challenging in countries where specialized investigations such as SeHCAT scanning and serum 7α-hydroxy-4-cholesten-3-one (C4) estimation are not readily available.

Case Presentation: A 43-year-old woman presented with profuse watery diarrhea for two months associated with marked weight loss. Extensive clinical evaluation, laboratory investigations, endoscopic examination, radiological imaging, and duodenal biopsy failed to reveal an infectious, inflammatory, endocrine, pancreatic, or structural cause. Considering the chronic secretory nature of diarrhea, previous cholecystectomy, and exclusion of other etiologies, bile acid diarrhea was suspected. A therapeutic trial with a bile acid sequestrant resulted in significant clinical improvement, supporting the diagnosis.

Conclusion: This case highlights the importance of considering bile acid diarrhea in the differential diagnosis of chronic watery diarrhea. Early recognition and an empirical trial of bile acid sequestrants can avoid unnecessary investigations and provide rapid symptomatic relief.

Keywords: Chronic Diarrhea, Bile Acid Diarrhea, Secretory Diarrhea, Cholestyramine, Bile Acid Malabsorption.

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Introduction

Chronic diarrhoea is defined as the passage of loose or watery stools for more than four weeks and is a common gastrointestinal disorder with multiple underlying causes. The condition may arise from secretory, osmotic, inflammatory, fatty, or motility-related disorders. A systematic diagnostic approach is necessary because clinical manifestations frequently overlap.[1,2]

Bile acid diarrhea is increasingly recognized as an important cause of chronic secretory diarrhea. Normally, approximately 95% of bile acids are actively reabsorbed in the terminal ileum and recycled through the enterohepatic circulation.

Failure of this mechanism or excessive hepatic bile acid synthesis results in increased delivery of bile acids to the colon. Colonic bile acids stimulate chloride secretion, inhibit sodium absorption, increase mucosal permeability, and accelerate colonic motility, producing persistent watery diarrhea.[3,4]Despite its relatively high prevalence, BAD remains underdiagnosed because specialized diagnostic tests are unavailable in many healthcare settings. Consequently, an empirical therapeutic trial with bile acid-binding resins often serves as both a diagnostic and therapeutic strategy.[2,4,5]

Etiology: Chronic diarrhoea has a broad differential diagnosis and is commonly classified as secretory, osmotic, inflammatory, fatty (malabsorptive), or functional. Secretory diarrhoea includes bile acid diarrhoea, endocrine disorders, and microscopic colitis, whereas osmotic diarrhoea results from carbohydrate malabsorption or laxative use. Inflammatory diarrhoea is seen in inflammatory bowel disease and infections, while fatty diarrhoea is associated with pancreatic insufficiency and celiac disease. Functional disorders such as diarrhoea-predominant irritable bowel syndrome (IBS-D) are also common causes.[1,2]

Table 1: Etiology of Chronic Diarrhoea

Category	Common Causes
Secretory	Bile acid diarrhoea, Microscopic colitis, Endocrine tumors (VIPoma, gastrinoma), Hyperthyroidism
Osmotic	Lactose intolerance, Fructose malabsorption, Laxative abuse
Inflammatory	Ulcerative colitis, Crohn's disease, Infectious colitis
Fatty (Malabsorptive)	Celiac disease, Chronic pancreatitis, Pancreatic insufficiency, Small intestinal bacterial overgrowth
Functional/ Factitious	IBS-D, Factitious diarrhoea due to laxative misuse

Causes of Bile Acid Diarrhoea

Mechanism	Causes
Hepatic Overproduction of Bile Acids	• Idiopathic (Primary bile acid diarrhoea) • Post-cholecystectomy • Irritable bowel syndrome–diarrhoea predominant (IBS-D) • Pancreatic insufficiency
True Bile Acid Malabsorption	• Crohn's disease involving the terminal ileum • Right hemicolectomy or ileal resection • Enteropathy (e.g., HIV enteropathy) • Pelvic radiation therapy • Bariatric surgery • Microscopic colitis • Small intestinal bacterial overgrowth (SIBO)

Primary bile acid diarrhoea results from excessive hepatic bile acid synthesis due to impaired feedback regulation, whereas secondary bile acid diarrhoea (true bile acid malabsorption) occurs because of impaired ileal reabsorption or conditions affecting the enterohepatic circulation. In the present case, the patient's history of cholecystectomy made secondary bile acid diarrhoea the most likely diagnosis.

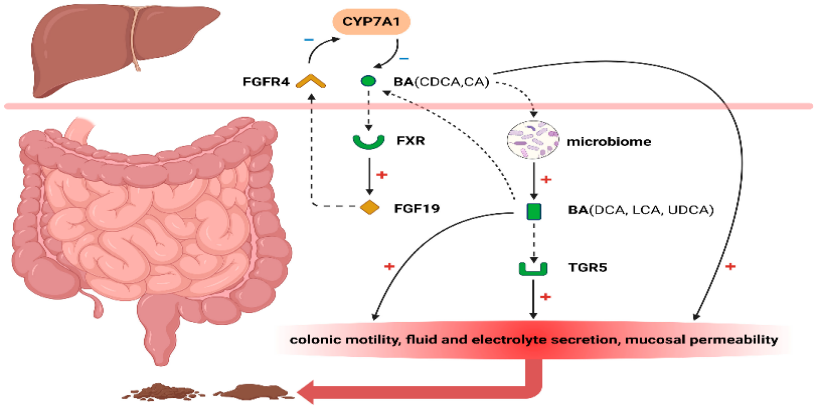

Figure 1: Bile Acid Diarrhea: From Molecular Mechanismsto Clinical Diagnosis and Treatment.

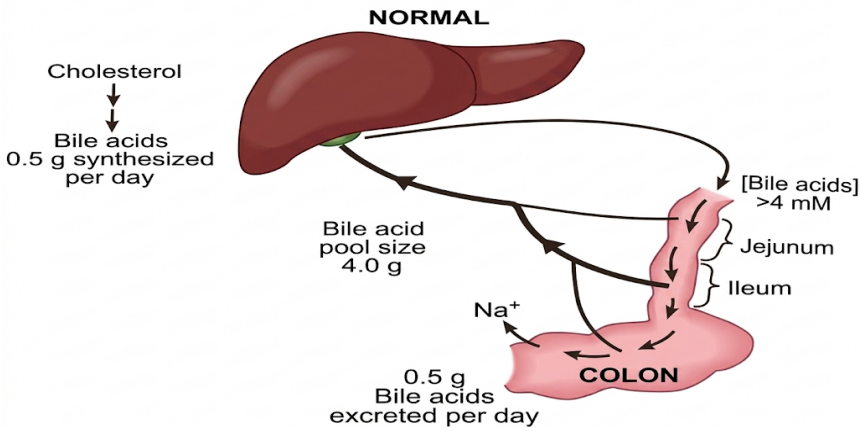

Figure 2: Schematic representation of the enterohepatic circulation of bile acids

Bile acids are synthesized from cholesterol in the liver, stored in the gallbladder, and released into the duodenum after meals. Approximately 95% are reabsorbed in the terminal ileum via sodium-dependent transport and returned to the liver through the enterohepatic circulation. Only about 0.5 g/day is excreted in stool and replaced by new hepatic synthesis, maintaining a bile acid pool of approximately 4 g.

Case History: A 43-year-old female presented to the medicine outpatient department with complaints of frequent watery diarrhea for two months.

She reported more than fifteen episodes of loose stools daily. The stools were watery without blood, mucus, pus, or visible fat droplets. The diarrhea occurred both during the day and night and was unrelated to food intake. Associated symptoms included abdominal discomfort, excessive flatulence, and progressive weight loss of more than 10 kg during the preceding three months. There was no history of fever, chronic cough, vomiting, gastrointestinal bleeding, recent antibiotic intake, or similar illness among family members.

Past medical history was unremarkable for diabetes mellitus, hypertension, tuberculosis, thyroid disease, inflammatory bowel disease, or chronic liver disease. The patient had previously undergone Hysterectomy, Appendicectomy, and Cholecystectomy. There was no history suggestive of vitamin B12 deficiency, peripheral neuropathy, gait abnormality, or malnutrition.

Table 2: Baseline Laboratory and Radiological Investigations

Investigation	Result	Interpretation
Complete Blood Count (CBC)		
Haemoglobin (Initial)	**9.6 g/dL**	Mild anemia at presentation
Haemoglobin (Repeat)	**11.5 g/dL**	Improvement on repeat evaluation
Platelet Count	**1,72,000/mm³**	Within normal range
Mean Corpuscular Volume (MCV)	**84 fL**	Normocytic red blood cells
Mean Corpuscular Haemoglobin (MCH)	**26.9 pg**	Within normal limits
Mean Corpuscular Haemoglobin Concentration (MCHC)	**32 g/dL**	Within normal limits
Liver Function Tests (LFT)		
Total Bilirubin	**0.4 mg/dL**	Normal
Aspartate Aminotransferase (AST)	**16 U/L**	Normal
Alanine Aminotransferase (ALT)	**14 U/L**	Normal
Alkaline Phosphatase (ALP)	**134 U/L**	Within normal range
Serum Albumin	**4.6 g/dL**	Normal nutritional status
Lipid Profile		
Total Cholesterol	**159 mg/dL**	Normal
Triglycerides	**118 mg/dL**	Normal
Low-Density Lipoprotein (LDL)	**91 mg/dL**	Normal
Very Low-Density Lipoprotein (VLDL)	**23 mg/dL**	Normal
Renal Function Tests (RFT)		
Serum Creatinine	**0.7 mg/dL**	Normal renal function
Uric Acid	**4.6 mg/dL**	Normal
Serum Calcium	**9.7 mg/dL**	Normal
Serum Sodium	**140 mEq/L**	Normal electrolyte status
Stool Examination		
Stool Routine and Microscopy	**No ova, cysts, or parasitic eggs detected**	No evidence of parasitic infestation
Blood Glucose Profile		
Fasting Blood Sugar (FBS)	**101 mg/dL**	Normal
Glycated Haemoglobin (HbA1c)	**5.5%**	Normal glycaemic control
Thyroid Function Tests		
Thyroid Stimulating Hormone (TSH)	**2.75 µIU/mL**	Normal thyroid function
Triiodothyronine (T3)	**0.95 ng/mL**	Normal
Thyroxine (T4)	**8.40 µg/dL**	Normal
Radiological Investigation		
Ultrasonography (USG) Abdomen	**Grade I fatty liver**	Mild hepatic steatosis; no significant abnormality explaining chronic diarrhoea

Given the unremarkable basic metabolic, endocrine and stool microscopy panel, cross-sectional imaging and endoscopic evaluation were pursued to exclude structural and mucosal disease.

Physical Examination: On examination, blood pressure was 110/80 mmHg. General physical examination revealed no pedal oedema, clubbing or pallor. Chest examination revealed bilateral vesicular breath sounds. Abdominal examination was unremarkable, with no organomegaly, tenderness or palpable lump. Neurological examination showed no abnormal movements or sensory loss, with intact joint position and vibration sense, arguing against a nutritional (B12-deficiency-related) neuropathy.

Imaging and Endoscopy: Contrast-enhanced computed tomography (CECT) of the abdomen showed post-hysterectomy and post-cholecystectomy status, a small calcification in the right adnexa (likely a phlebolith), a distended stomach with normal gastric wall thickness, a well-defined ileocaecal junction, and a predominantly faecal-loaded but otherwise unremarkable colon. The pancreas was normal, with no free fluid or significant mesenteric/retroperitoneal lymphadenopathy.

Upper gastrointestinal endoscopy (UGIE) revealed mild oesophagitis, mild antral gastritis and duodenitis. Duodenal (D2) biopsy was suggestive of lymphoid hyperplasia, without features of villous atrophy to suggest coeliac disease.With infective, structural, pancreatic, hepatobiliary, thyroid and coeliac causes effectively excluded, and given the predominantly secretory, non-fatty, meal-unrelated pattern of watery diarrhoea in a patient with prior cholecystectomy and altered enterohepatic circulation, a diagnosis of primary bile acid diarrhoea was considered and supported by clinical response to empirical bile acid sequestrant therapy.

Differential Diagnoses

Table 3: Differential diagnoses considered and reasons for exclusion

Differential Diagnosis	Basis for Exclusion
Coeliac disease	D2 biopsy showed lymphoid hyperplasia only, without villous atrophy or intraepithelial lymphocytosis
Chronic pancreatitis / exocrine pancreatic insufficiency	Normal pancreas on CECT; no steatorrhoea or oil droplets in stool
Thyrotoxicosis-related diarrhoea	TSH, T3 and T4 within normal limits
Diabetic autonomic diarrhoea	Normal fasting glucose and HbA1c
Infective/parasitic diarrhoea	Stool microscopy negative for ova, cysts and parasites
Inflammatory bowel disease	No blood/mucus in stool; colon unremarkable on CECT; no significant lymphadenopathy
Small intestinal bacterial overgrowth	No predisposing anatomic or motility abnormality identified; considered but not primary explanation
Bile acid diarrhoea (secondary to cholecystectomy)	Consistent with meal-unrelated watery diarrhoea, absent steatorrhoea, and clinical response to empirical therapy

Discussion

Classification of Chronic Diarrhoea: Chronic diarrhoea is broadly classified into secretory, osmotic, inflammatory, fatty (malabsorptive), and functional diarrhoea. Among these, bile acid diarrhoea (BAD) is an important cause of chronic secretory diarrhoea and should be considered in patients with persistent watery stools after common infectious and inflammatory causes have been excluded.[6-9]

Table 4: Classification of Chronic Diarrhoea

Type	Common Causes
Secretory	Bile acid diarrhoea, microscopic colitis, endocrine disorders
Osmotic	Lactose intolerance, carbohydrate malabsorption, laxative abuse
Inflammatory	Crohn's disease, ulcerative colitis, infectious colitis
Fatty (Malabsorptive)	Celiac disease, chronic pancreatitis, pancreatic insufficiency
Functional	Irritable bowel syndrome with diarrhoea (IBS-D)

Diagnostic Evaluation: Evaluation of chronic diarrhoea requires careful history, physical examination, and targeted investigations to exclude infectious, inflammatory, endocrine, pancreatic, and malabsorptive disorders. Routine laboratory tests, stool examination, imaging, endoscopy, and small intestinal biopsy help narrow the differential diagnosis. In our patient, all routine investigations were unremarkable, making bile acid diarrhoea a likely diagnosis after excluding other causes.

Table 5. Common Diagnostic Tests for Malabsorptive Diarrhoea

Condition	Diagnostic Test
Lactose malabsorption	Lactose hydrogen breath test
Small intestinal bacterial overgrowth	Glucose hydrogen breath test
Bile acid diarrhoea	SeHCAT scan, Serum C4
Pancreatic insufficiency	Faecal elastase
Vitamin B12 malabsorption	Schilling test

Bile Acid Diarrhoea: Bile acid diarrhoea results from excessive bile acids reaching the colon, where they stimulate electrolyte and water secretion and increase intestinal motility, producing chronic watery diarrhoea.

It is classified as primary, caused by increased hepatic bile acid synthesis due to impaired FGF-19 feedback, and secondary, associated with terminal ileal disease, Crohn's disease, ileal resection, or cholecystectomy. Because our patient had a history of cholecystectomy with normal imaging and endoscopic findings, secondary bile acid diarrhoea was strongly suspected.

Bile Acid Diarrhoea versus Fatty Diarrhoea: Differentiating bile acid diarrhoea from fatty diarrhoea is important because their management differs.

Table 6: Comparison of Bile Acid Diarrhoea and Fatty Diarrhoea

Feature	Bile Acid Diarrhoea	Fatty Diarrhoea
Ileal disease	Limited	Extensive
Steatorrhoea	Absent or mild	Marked (>20 g/day)
Bile acid pool	Normal	Reduced
Response to cholestyramine	Good	Poor
Response to low-fat diet	No	Yes

Final Diagnosis and Management: Based on the patient's clinical presentation of chronic watery secretory diarrhoea, history of previous cholecystectomy, absence of steatorrhoea, and systematic exclusion of infectious, inflammatory, endocrine, pancreatic, and structural gastrointestinal disorders, a diagnosis of secondary bile acid diarrhoea (bile acid malabsorption syndrome) was established.

The diagnosis was further supported by the patient's marked clinical improvement following an empirical therapeutic trial with a bile acid sequestrant, which served as both a diagnostic and therapeutic intervention in the absence of specialized investigations such as the SeHCAT retention scan.

Management of bile acid diarrhoea primarily focuses on reducing the colonic effects of excess bile acids by using bile acid sequestrants, which bind bile acids within the intestinal lumen and prevent their secretory action on the colonic mucosa.

The patient was initiated on cholestyramine 4 g once daily, with the dose adjusted according to clinical response and tolerability. Significant improvement in stool frequency, urgency, and abdominal discomfort was observed following treatment, further confirming the diagnosis. Along with pharmacological therapy, the patient received oral rehydration, dietary counselling, and nutritional support. Patients should be advised to take cholestyramine 2–4 hours apart from other oral medications because it may interfere with their absorption. Common adverse effects such as bloating, constipation, and abdominal discomfort should be monitored during follow-up. In patients who are unable to tolerate cholestyramine, alternative bile acid sequestrants such as colestipol or colesevelam may be considered. Long-term management also includes periodic assessment for nutritional deficiencies, particularly fat-soluble vitamins (A, D, E, and K), vitamin B12, and calcium, with appropriate supplementation when indicated.[10-15]

Conclusion

Chronic diarrhoea demands a systematic, case-based diagnostic approach that sequentially excludes infective, structural, pancreatic, hepatobiliary, endocrine and mucosal causes. Primary bile acid diarrhoea, mediated by reduced FGF19-driven feedback inhibition of hepatic bile acid synthesis, is an important and often underdiagnosed cause of chronic watery, secretory diarrhoea, particularly in patients with a history of cholecystectomy or altered enterohepatic circulation. In settings where confirmatory testing such as SeHCAT scanning is unavailable, a carefully monitored therapeutic trial of bile acid sequestrants offers a practical and effective diagnostic and therapeutic pathway, as illustrated by this case.

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