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A Case Report on Ulcerative Colitis with Special Reference to Heredity

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Abstract: ***This case report discusses a 17 - year - old male who presented with constipation, severe abdominal pain, bloody diarrhea, and significant weight loss, all suggestive of ulcerative colitis or inflammatory bowel disease (IBD). His father had previously been diagnosed with ulcerative colitis (UC) and renal cell carcinoma, indicating a possible hereditary predisposition to the condition. Blood and biochemical tests revealed anemia, hypoalbuminemia, transferrin, iron - binding capacity, and Vitamin D_3, along with elevated C - reactive protein (CRP). Stool studies showed increased inflammatory markers. Sigmoidoscopy with biopsies from the colon and rectum confirmed the diagnosis of ulcerative colitis. This report highlights the clinical presentation, hereditary factors, diagnostic approach, and therapeutic management of ulcerative colitis. Keywords: Constipation, Bloody stool, Diagnosis, Heredity, Treatment, Ulcerative colitis.***

Keywords: Constipation, Blood in stool, diagnosis, Heredity, Treatment, Ulcerative

1. Introduction

Ulcerative colitis (UC) is a chronic, complex inflammatory bowel disease (IBD) that affects the epithelium of the rectum and colon. It is characterized by symptoms such as perianal swelling or bleeding, cramping abdominal pain, bowel urgency, frequent stools, diarrhea (often with blood, pus, or mucus), tenesmus, fever, weight loss, and anemia [(1).] UC is an autoimmune disease, marked by the infiltration of T - cells into the colon [(2).] Although the exact cause of UC is unknown, it is speculated that factors such as an overactive immune system, genetics, changes in normal gut bacteria, and environmental triggers contribute to its development [(3).] Genetic studies have identified variations in dozens of genes linked to ulcerative colitis, and persons with a family history of IBD are at increased risk of developing the disease. About 10% to 25% of people diagnosed with UC have a parent or sibling with IBD, making them four to eight times more likely to develop some form of the disease [(4).]

In the pathogenesis of ulcerative colitis, colonic epithelial cells play a crucial role by impairing the expression of peroxisome proliferator - activated receptor γ (PPAR - γ), a nuclear receptor that downregulates inflammation through nuclear factor kB (NF - kB) [(5).] Due to variations in clinical manifestations and the lack of uniform diagnostic criteria, the incidence of IBD ranges from 1% to 20% [(6).] Diagnosing UC can be challenging, as it requires a combination of clinical manifestations, laboratory tests, sigmoidoscopy/colonoscopy, and histopathology. During acute flare - ups, inflammatory markers such as ESR, CRP, and leukocytosis are typically elevated. Other tests, including perinuclear antineutrophil cytoplasmic antibodies (P - ANCA) and anti - Saccharomyces cerevisiae antibodies (ASCA), are helpful in differentiating UC from Crohn's disease. Testing for fecal calprotectin also has diagnostic utility, although it is nonspecific [(7).]

Treatment options for UC may include oral, systemic, and topical therapies, as well as surgery. Medications such as mesalazine, sulfasalazine, steroids, immunosuppressants (e. g., azathioprine), and biological agents are commonly used to induce and maintain remission. In severe cases where medical therapy fails, or if complications like colon cancer arise, surgery becomes the primary option [(8).] UC leads to chronic and persistent gastrointestinal symptoms, requiring long - term treatment and monitoring [(9).] Although UC is a lifelong disease without a permanent cure, it has a significant impact on both mental and physical health. This case report highlights nonspecific clinical signs, a parental history of UC, along with the diagnostic workup and therapeutic management of the disease

2. Case Presentation

A 17 - year - old male, weighing 48 kg, with a pulse of 102/min, respiratory rate 22/min and blood pressure of 120/80 mmHg, presented to Bawa Hospital, Ludhiana, with a 6 - month history of constipation. The patient was under acute stress due to prolonged sitting and irregular food intake. He was treated for constipation and haemorrhoids. However, there was no relief from constipation and the patient spent up to one hour in the toilet for defecation. At the time of presentation, he complained of mild abdominal pain, anal irritation and weight loss. The patient's father had a history of ulcerative colitis (UC) at the age of 45 and renal cell carcinoma at age 70. Upon physical examination, no cyanosis, icterus, clubbing, skin lesions, or lymphadenopathy were noted. Local perianal examination revealed a thrombosed external hemorrhoid and swelling. The abdomen was soft and non - tender. No abnormalities were found in other systems. The patient reported no previous similar complaints, and there was no significant past medical or surgical history. He was advised to undergo ultrasonography of the whole abdomen, colonoscopy, as well as biochemical

and hematological examinations. Stool examination and fecal calprotectin levels were not performed. Ultrasound revealed borderline hepatomegaly without other abnormalities. Hematological findings (Table 1) and biochemical examinations were normal, except for a significant rise in ALP (Table 2). Colonoscopy up to the ascending colon showed multiple erosions with erythema and small ulcerations in the rectum and sigmoid colon. The descending, transverse, and ascending colon appeared normal, and inflammatory bowel disease was suspected (Fig 1). Biopsy samples, approximately 1x1 cm multiple greyish - white pieces from the colon, were submitted to the pathology laboratory. Histopathological examination revealed ulceration of the lining with a large number of acute inflammatory cells in the lamina propria. Focal areas of cryptitis and crypt abscesses were noted. Areas of crypt distortion and crypt loss were observed, indicating active colitis. Tuberculosis and HIV were ruled out. Based on clinical and histopathological examination, the case was diagnosed as ulcerative colitis in the terminal part of the sigmoid colon and rectum, with external hemorrhoid. The patient was treated with Tab. mesalamine 1.2 gm bid, tab. folvite, and a probiotic. He recovered within two weeks and was advised to continue the treatment for life.

Table 1: Haematological evaluation in a case of ulcerative colitis

Biomarkers	Reference value	19/02/2022 (1st episode)	19/11/2022 (3nd episode)	22/11/22 (Date of discharge)
Haematological				
Hb gm/dl	12.0 - 17.0	14.60	7	11.5
TLCx10^3/ul	4.0 - 10.0	5.40	5.7	11.2
DLC %				
N	40.0 - 80.0	61	79.7	82.8
L	20.0 - 40.0	32	8.3	12.4
M	2.0 - 10.0	03	10.5	4.6
E	1.0 - 6.0	04	1.4	0.1
B	0.0 - 1.0	0	0.1	0.1
PCV%	45.0 - 55.0	41.60	22.2	36.9
MCV fl	83.0 - 101.0	81.10	90.3	85.9
Plateletsx10^3/ul	150.0 - 450.0	234	127	236
TECx10^6/ul	4.5 - 5.5	5.13	2.46	4.25
ESR	00 - 10mm/hr	05	-	-
Rapid HBs Ag		Non - Reactive	-	-
Rapid ANTI HCV		Non - Reactive	-	-
Rapid Anti HIV		Non - Reactive	-	-
Mantoux test	10mm	5x7mm Negative	-	-
Wt.		49kg	-	-
Urine				
Color	-	-	straw	-
Glucose	-	-	-	-
Protein	-	-	+1	-
RBC/HPF	0.0 - 2.0	-	28.9	-
WBC/HPF	0.0 - 5.0	-	6.9	-
Epith. Cell/HPF	0.0 - 0.0	-	1.1	-
Casts/LPF	0.0 - 5	-	40	-
crystal	0.0 - 0.0	-	0	-
Bacteria	-	-	4.1 bacilli	-

-; Sample not collected/examined

Three months later, during the second episode, the patient experienced bloody diarrhea, fever, severe abdominal pain, and 2 - 4 loose bloody stools per day. Investigations were repeated, and flexible sigmoidoscopy revealed grade 2 - 3 hemorrhoids in the anal canal, erosions in the distal rectum, and mucus with hyperemia in the proximal rectum. The sigmoid colon and distal 5 cm of the descending colon also showed mucus and hyperemia (Fig 2). The patient was again diagnosed with grade 2 - 3 hemorrhoids and left - sided ulcerative colitis in partial remission. He was treated with injections of calcirol 6 lac units followed by oral cholecalciferol once every two weeks, tab. meconerve 1500 mg, tab. mesalamine 1.2 gm twice daily, and Anovate ointment for local application. After 15 days, there was no response, and azathioprine 50 mg twice daily was added for an additional 15 days, along with potassium permanganate ($KMnO_4$) crystal warm water baths. Despite two months of treatment, symptoms subsided but full recovery was not achieved. Seven months later, during the third episode, the patient was admitted to the emergency department with severe abdominal pain, vomiting, fever, perianal irritation, and 2 - 3 loose stools per day. The patient was on mesalamine and azathioprine at the time of admission. There was no history of smoking, alcohol use, palpitations, cough, or syncope. Upon examination, the patient was conscious, alert, oriented, and cooperative with pallor but no edema, cyanosis, or clubbing. His blood pressure was 110/70 mm Hg, pulse 92 beats per minute, respiration rate 24 breaths per minute, oxygen saturation 98%, and temperature 98.4°F. Other vitals were within normal limits. Respiratory, cardiovascular, and central nervous system examinations were normal. Abdominal palpation revealed mild tenderness at the epigastrium. Bilateral normal vesicular breathing was observed during chest examination. Chest X - ray showed no consolidation or pleural effusions. Hematological studies revealed hemoglobin (Hb) 7 gm/dl, TLC 5.7 × 103/µl, PCV 2.2%, TEC 2.46 × 106/µl, and a platelet count of 127 × 103/µl (Table 1), indicating anemia. Biochemical investigations

revealed decreased total albumin, transferrin, iron binding capacity (UIBC, TIBC & Iron), and Vitamin D3, along with increased C - reactive protein (CRP). Other parameters were normal (Table 2). Urine examination showed pus cells 6.9/hpf, RBCs 28.9/hpf, renal epithelial cells 1.1, and bacilli 4.1, with other findings being normal (Table 1). Ultrasound of the whole abdomen revealed a normal pancreas, although it showed heterogeneous echotexture. Sigmoidoscopy revealed normal findings in the descending and sigmoid colon. The rectum showed complete loss of vascular pattern, obliteration, edema, hyperemia, and >5 mm ulcers covered with white exudates, with spots of coagulated blood on the mucosal surface, indicating ulcerative colitis and proctitis (Fig.3). Biopsy findings revealed erosion of the epithelium with moderate to marked acute and chronic inflammatory infiltrates, with prominent eosinophils (up to 40/hpf). Focal cryptitis with occasional crypt abscesses was noted, along with mild distortion of crypt architecture. No intraepithelial lymphocytes, basal cell plasma cytosis, metaplasia, or granulomas were observed. There were no signs of CMV inclusions, dysplasia, or parasites. The findings indicated a Geboes score of 5.3, Nancy histological index grade 3, and a Robarts score of 25/33.

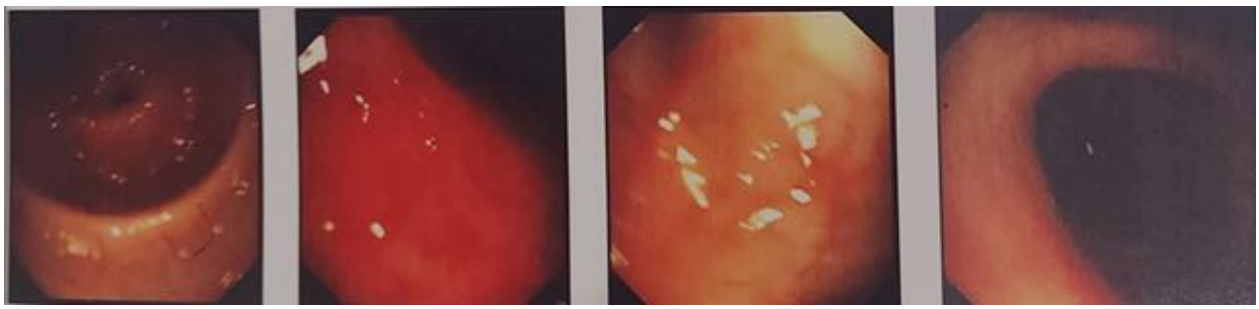

Figure 1: Colonoscopy 1st episode: - Multiple erosions with erythema and small ulceration involving rectum and sigmoid colon in.

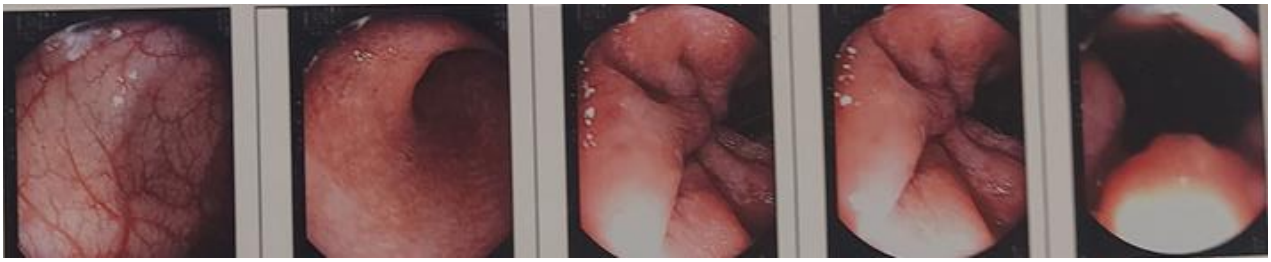

Figure 2: Sigmoidoscopy: 2nd episode Shows erosions in distal part of rectum, lot of mucus and hyperemia sigmoid colon, lot of mucus and hypermia in distal 5cm of descending colon.

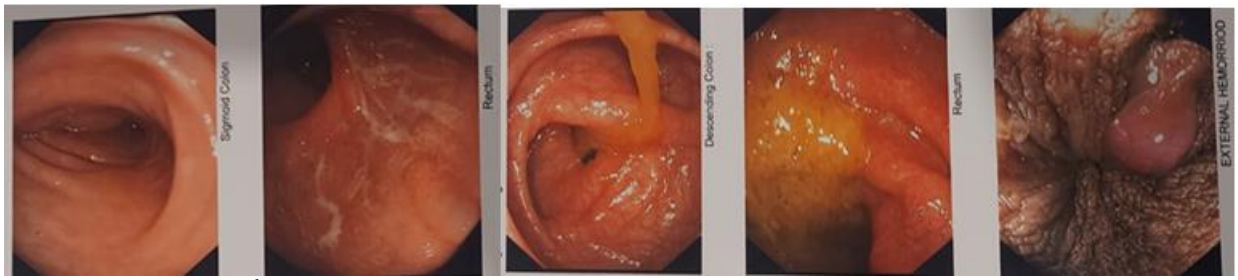

Figure 3: Sigmoidoscopy 3rd episode - Rectum shows complete loss of vascular pattern obliteration, hyperemia, >5mm ulcers on covered with white exudate and coagulated blood on mucosal surface.

Based on the hematological, biochemical, ultrasonographic, and sigmoidoscopy findings, the case was diagnosed as ulcerative colitis with acute intestinal colic and urinary tract infection. The patient was treated with injections of Monocef, PAN, Drotin, Albumin, Metrogyl, Hydrocortisone, Magnesium sulfate, and oral tablets Vegaz, Mesacol suppository, Calgrow, Syrup ranidom, and IV fluids. The patient improved after four days, becoming stable, pain - free, and having normal stools, with normal hematological and biochemical findings (Tables 1 and 2). The patient was discharged after four days with a prescription of Tab. pantocid 40 mg OD, Tab. cortirowa 9 mg OD, Tab. Vegaz - OD 1.2 gm bid, Tab. mesacol suppository 1 gm daily at night, syrup ranidom 2tsf SOS, and Entefoam enema every alternate day. The same treatment was continued for four months. After four months, the patient's stool frequency was 4 - 5 per day without blood, although perianal burning and relapses were noted. A stool calprotectin test was performed every three months, with results of 869 µg/g, 42 µg/g, 1146 µg/g, and 444 µg/g, indicating active inflammatory bowel disease (Table 2). The patient was advised to continue taking Tab. Vegaz - OD 1.2 gm bid, mesacol suppository, Tab. topcid 40 mg, and Syrup Fullyte trio 2 tsf twice daily. The patient eventually recovered fully, with no symptoms, and gained 4 kg in weight. Regular follow - up was advised to monitor the disease. The patient also responded to treatment after relief from stress and change of life style.

Table 2: Chronological values of serum biochemistry and associated biomarkers in a case of Ulcerative colitis

Biochemical	Reference value	19/02/22	19/11/22	20/11/22	22/11/22	08/09/23	09/09/23	08/01/24	09/07/24
Glucose mg/dl	70 - 140	125	68	-	-	-	-	-	-
Urea mg/dl	10.0 - 50.0	20.6	12	21	15	-	-	-	-
Creatinine mg/dl	0.67 - 1.17	0.9	0.33	1.01	0.79	-	-	-	-
Nal^+ mmol/	136.0 - 148.0	137	135	135	144	-	-	-	-
K^+ mmol/	3.5 - 5.0	**4.1**	4.75	3.88	3.62	-	-	-	-
Cl mmol/	96.0 - 106.0	99	100	100	104	-	-	-	-
Ferritin	23.9 - 336.2 (ng/ml)	-	65.95	-	-	-	-	-	-
Amylase	28.0 - 100 (U/L)	-	21.0	-	-	-	-	-	-
Lipase	13.0 - 60.0 (U/L	-	10.0	-	-	-	-	-	-
Total Bilirubin mg/dl	0.0 - 1.2	0.6	0.18	-	-	-	-	-	-
SGOT U/L	0.0 - 40.0	30	12	-	-	-	-	-	-
SGPT U/L	0.0 - 41.0	35	7.0	-	-	-	-	-	-
ALP U/L	40.0 - 129	**234**	32	-	-	-	-	-	-
Total Protein g/dl	6.6 - 8.7	7.2	**2.5**	-	5.6	-	-	-	-
Albumin g/dl	3.5 - 5.2	4.2	**1.55**		3.69	-	-	-	-
Calcium mg/dl	8.6 - 10.2	9.92	-	9.2	-	-	-	-	-
Magnesium mg/dl	1.7 - 2.8	-	-	1.68	-	-	-	-	-
Serum folate level	3.1 - 16.0 (ng/ml)	-	7.94	-		-	-	-	-
Vitamin B_{12}	160 to 950 (pg/mL)	-	**88**	-	-	-	-	-	-
Vitamin D_3 ng/ml	>30 sufficient 21 - 30 insufficient <20 deficient	-	15.45	-	-	-	-	-	-
CRP - quantitative	0.0 - 6.0 (mg/l)	-	-	-	**22.63**	-	-	-	-
TIBC panel						-	-	-	-
UIBC	112.0 - 346.0 (ug/dl)	-	-	-	**81.3**	-	-	-	-
TIBC	250.0 - 400.0 (ug/dl)	-	-	-	**104**	-	-	-	-
Iron	59.0 - 158 (ug/dl)	-	-	-	**22.5**	-	-	-	-
% Transferrin saturation estimation	0.0 - 15.0 (%)	-	-	-	21.6	-	-	-	-
EBV - VCA IgG	0 - 18 (U/l)	-	-	-	1.2	-	-	-	-
Fecal Calprotectin a) Consider IBS or non IBD b) Indicate IBD, polyps, infection, malignancy, excess NSAID c) Indicate Active inflammatory bowel disease	 a) 5 - 50 ug/g b) >50 ug/g c) >200		-	-	-	**869**	**42**	**1146**	**444**

3. Discussion

Ulcerative colitis (UC) is a complex, chronic inflammatory bowel disease (IBD) localized to the colon, sparing the upper gastrointestinal tract [(10).] While the direct causes of UC are unknown, it is speculated that an overactive immune system, genetics, changes in normal gut bacteria, and environmental factors contribute to its development [(3).] UC can occur at any age, but it most commonly presents between 15 - 30 years and again in individuals aged 50 - 70 years (4). A study observed that 20% of patients with UC are younger than 20 years of age, 4% are children under 5 years, and 1% are infants [(11).] In the present study, the patient began experiencing symptoms at 17 years of age, with a family history of UC at 45 years and renal cell carcinoma at 70 years, suggesting a hereditary component. Inflammatory bowel disease is reported in 8 - 14% of cases with family history [(12).] Over the past many years, significant progress has been made in understanding the genetics of IBD, with over 200 different gene loci identified [(13).]

In the present study, the patient experienced three episodes of UC, starting at 17 years of age. The first episode presented with nonspecific symptoms of constipation, tenesmus, and perianal swelling, and was managed outpatient. It is reported that tenesmus may be mistaken for constipation due to the tendency to pass small volumes of stool. Blood in the stool and severe abdominal pain are important features of more severe disease [(12).] The third episode was particularly severe, characterized by bloody stools, significant weight loss, perianal swelling, hemorrhoids, and severe abdominal pain. It was managed in the emergency department, marked by diarrhea mixed with blood, which persisted over an extended period (weeks). Diarrhea, which is observed in 90% of patients, and bowel urgency, seen in 75 - 90% of patients, are common [(14).] During the first episode, no significant changes in hematological and biochemical markers were observed. However, in subsequent episodes, there was anemia, decreased total albumin, transferrin, and iron - binding capacity (decreased UIBC, TIBC, and iron), low Vitamin D3, and increased C - reactive protein (CRP), indicating UC. Moderate to severe disease can lead to abdominal pain and anemia, with elevated ESR and CRP often observed [(15).] Chronic blood loss can cause iron deficiency anemia, which is evaluated through serum ferritin, iron, total iron binding capacity, and transferrin saturation. Anemia may also result from treatment complications, such as pancytopenia caused by azathioprine [(16).] The present study's findings align with these observations. Severe UC often results in high ESR, decreased albumin, and electrolyte changes. Elevated alkaline phosphatase and higher fecal calprotectin or lactoferrin levels indicate intestinal inflammation [(17).] The study noted

significant elevation of fecal calprotectin, which is significantly elevated in UC/IBD patients with a sensitivity of 100% and specificity of 67% [18]. Diagnosis in the present case was confirmed using flexible sigmoidoscopy, which revealed a complete loss of vascular pattern, obliteration, edema, hyperemia, ulcers greater than 5mm covered with white exudates in the rectum, and some coagulated blood spots on the mucosal surface, indicative of ulcerative colitis and proctitis. Histopathological examination showed erosion of the epithelium with moderate to marked acute and chronic inflammatory infiltrate, prominent eosinophils (up to 40/hpf), focal cryptitis, occasional crypt abscesses, and mild distortion of crypt architecture. These findings are consistent with those reported in the literature for UC [19, 20].

Based on symptoms, signs, colonoscopy, and histopathology, the patient was diagnosed with severe UC and treated accordingly. Treatment included Tab. Mesalazine 1.2 gm bid, suppository Mesacol, Tab. Meconerve 1500 mg, and Fulllyte Trio powder, with follow - up every month showing satisfactory recovery but with elevated fecal calprotectin, indicating active inflammatory bowel disease. There is no definitive cure for UC. The primary goal of treatment is to control symptoms, restore quality of life, ensure normal growth, and prevent complications [21]. First - line maintenance medication for UC in remission typically includes mesalamine or 5 ASA, often in combination with suppositories [21, 22]. For severe disease or when remission is not achieved with mesalamine and corticosteroids, immunosuppressive medications such as azathioprine and biological agents are considered [23]. To improve clinical outcomes, rectal mesalamine can be used in conjunction with oral medications [20]. Oral glucocorticoids, such as prednisone, are often used as a second - line treatment for mild to moderate UC when unresponsive to mesalamine [24]. To maintain remission in patients dependent on steroids, thiopurine therapy such as Azathioprine or 6 - mercaptopurine may be added [24]. Some studies have observed that broad - spectrum antibiotics can be successful in treating UC in children when other therapies fail [25].

4. Conclusions

As the incidence of ulcerative colitis (UC) increases among the younger population, further research is essential to explore the roles of genetics and environmental factors in its development. UC should be considered in patients presenting with nonspecific clinical signs, especially those with a family history of the disease. Early diagnosis through appropriate testing is crucial for effective treatment and prevention of complications. To ensure a normal, healthy life, patients require comprehensive evaluation, suitable treatment, including immunosuppressive therapy, and regular follow - up. It is important to note that primary sclerosing cholangitis and colon cancer are significant long - term complications associated with UC.

Declarations

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Competing interests: The authors declare that they have no competing interests.

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