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Case Report

A Case Report on Arthritis Induced Inflammatory Bowel Disease

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Abstract: IBD is a recurrent inflammation of the gastrointestinal tract caused by an abnormal immune response to gut microbiota. It is a genetically predisposed condition, with incidence rates ranging from 4 to 10 per 100,000 people annually. It is more prevalent in highly industrialized countries and affects the entire bowel wall. Both forms are categorized by their location and degree of involvement. IBD is characterized by diffuse inflammation of the intestinal mucosa, proctitis, and transmural ulceration. Treatment focuses on immunosuppressive drugs and anti-inflammatory compounds, but achieving remission remains a clinical challenge. A 23-year-old female patient with pat history of Arthritis was admitted to the hospital. She complained of weight loss (lost about 5 – 10 kg in the last month), diminished appetite, weakness, blood in her faeces, and abdominal pain. The patient had significant anaemia and a tentative diagnosis of UGI-bleed. She is married, has a son who is nine years old, has regular bowel and bladder habits. The patient's MCHC was 29.4 g/dl, mean MCV was 72.8 fL, and haemoglobin was 11.1%. The patient's colonoscopy revealed Crohn's disease and colitis with rectal sparing. ANA screening tested positive and Negative for RF-IgM. The Patient's medication regimen includes Intravenous dextrose, Vitamin supplementation, Magnesium sulphate, tranexamic acid, Metronidazole, Cefoperazone & Sulbactam, and Pantoprazole. The oral supplementations include Syrup Sucralfate, probiotic capsule, Tab. Hydroxychloroquine, Tab. Tramadol & Acetaminophen, Mesalamine in tablet and sachet usually taken by mixing in water.

Keywords: Anti-nuclear antibodies, Crohn's disease, Modified Schober's test, Mesalamine, Schober's test, Inflammatory Bowel Disease.

1. INTRODUCTION

The hallmark of inflammatory bowel disease (IBD) is recurrent episodes of gastrointestinal tract inflammation brought on by an aberrant immune response to gut microbiota. Two forms of idiopathic intestinal disease that are distinguished from one another by their location and degree of involvement in the intestine wall are combined to form inflammatory bowel disease. Diffuse inflammation of the intestinal mucosa is a symptom of ulcerative colitis (UC). Proctitis, the most common form of ulcerative colitis (UC), can also affect the sigmoid (proctosigmoiditis), the entire colon up to the cecum (pancolitis), or somewhere in between. Transmural ulceration of any part of the gastrointestinal tract (GI) is a result of Crohn's disease (CD), but it most frequently affects the colon and terminal ileum. Both

conditions are categorized according to their location and degree (mild, moderate, or severe) [1].

IBD is a condition that affects people all over the world, while it is more prevalent in some areas than others (the United States, the United Kingdom, and Scandinavia). Incidence rates range from 4 to 10 per 100,000 people annually, whereas prevalence rates range from 40 to 100 per 100,000 people. There is no discernible gender difference in the prevalence of IBD diagnoses, which occur most frequently in the third and fourth decades of life. About 5% of patients with IBD receive a diagnosis before the age of ten, while 20% of patients experience symptoms throughout childhood [2].

Genetically predisposed people develop inflammatory bowel disease (IBD) as a result of an improper immune response to the intestinal flora. The exact cause of IBD is still unknown. Numerous factors have been suggested; however none of them apply to every patient. The one constant in Crohn's disease research is the disease's close association with tobacco use. However, smoking seems to offer protection against ulcerative colitis. Dietary factors are still up for debate. Although the CARD15 gene has been linked to IBD, it is impossible to predict which region of the GI tract will be impacted due to its polymorphism characteristics. Compared to Crohn's disease, ulcerative colitis is less strongly influenced by genes [1]. Defective intercellular connections in the intestinal epithelium allow germs or antigens to enter the body, which is the cause of IBD. A-defensin secretion and mucus formation are examples of protective mechanisms. Overwhelming inflammation exacerbates the condition and increases the risk of infection. In ulcerative colitis, inflammation results in bleeding, ulceration, oedema, and electrolyte imbalances. Chronic cases cause the colon to grow short and stiff, giving the appearance of a lead pipe. Crohn disease affects the gastrointestinal tract, affecting all layers of the bowel and causing strictures, inflammation, and fistulas. It primarily affects the colon and ileum, with only 5% affecting the gastro duodenal segments. It predisposes patients to extraintestinal complications like inflammatory arthropathies and primary sclerosing cholangitis. Crohn disease also increases the incidence of kidney disease and gallstones due to malabsorption of bile salts and fatty acids [1]. Crohn's disease and ulcerative colitis are similar gastrointestinal diseases (IBDs) with different inflammatory changes. Crohn's disease affects any part of the gastrointestinal tract, while ulcerative colitis is localized to the large intestine. The disease is prevalent in highly industrialized countries and affects the entire bowel wall, while ulcerative colitis is restricted to the gut's epithelial lining. Diagnosing one form of IBD is challenging due to similar symptoms. Treatment relies on immunosuppressive drugs and anti-inflammatory compounds, but achieving remission remains a clinical challenge. Symptoms vary depending on the type of IBD. Crohn's disease often leads to blockage of the intestine due to swelling, leading to thickening of the bowel wall. People with Crohn's disease also face comorbidities like colorectal cancer, cardiovascular disease, and respiratory disease [3].

2. CASE REPORT

Subjective Findings

A 23 year old female presented to the hospital on 16th September 2023 complaining about blood in stools [Haematochezia] for 2 days 15 days ago and now for 2 days associated with abdominal pain since 15 days, generalized weakness. Decreased appetite since 20 days and loss of weight since 1 month (lost about 5-10 kg). On examination the patient was afebrile and chest was clear. The patient's BP was 90/60 mmHg and pulse rate of 86 bpm.

Past Medication History

Patient is a known case of Rheumatoid Arthritis (pain in proximal interphalangeal joint and bilateral knee joint) since 7 years and on irregular treatment since 1 month. She consumes alcohol occasionally and had a history of similar complaints 15 days back

The patient's condition was provisionally diagnosed as UGI– bleed with severe anaemia for evaluation.

Her personal history states: She is married at her 13 and also has a male child of 9 years.

Social History: Mixed diet, Adequate sleep, Regular bowel and bladder habits, and addictions: Alcoholic (consumes occasionally) and non-smoker.

Table 1: Follow Up Details of Patient

Vitals	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
On Examination/Complaints	c/c	c/c	c/c	c/c	c/c	c/c	c/c
Temperature	Afebrile	Afebrile	Afebrile	Afebrile	Afebrile	Afebrile	Afebrile
Blood Pressure (mmHg)	90/60	100/60	110/70	100/60	90/40	80/50	90/60
Pulse Rate (bpm)	86	116	80	94	93	94	88
GRBS (mg/dl)	61	-	-	-	60	71	-

Vitals	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Respiratory System	BAE +	BAE +	BAE +	BAE +	BAE +	BAE +	BAE +
CVS	S1 & S2 +	S1 & S2 +	S1 & S2 +	S1 & S2 +	S1 & S2 +	S1 & S2 +	S1 & S2 +
PER Abdomen	Tender	Tender	Tender	Tender	Tender	Tender	Tender

Laboratory Investigations

Table 2: Haemogram & Electrolytes

Haemoglobin			11.1 gms% (12 - 18)		
MCV			72.8 fL (76 - 96)		
MCH			21.3 pg (27 - 32)		
MCHC			29.4 g/dl (31 - 37)		
Electrolyte/ Date	17/09	18/09	19/09	20/09	21/09
Potassium	3.4 mmol/L	2.5 mmol/L	-	2.8 mmol/L	3.1 mmol/L
Sodium	-	-	-	-	-
Chloride	113 mmol/L	112 mmol/L	-	-	-
Magnesium	-	1.3 mEq/L	-	-	-

Vitamin D
25- Hydroxy (OG) Vit D: 10.55 ng/ml (<20ng/ml)

Peripheral Blood Smear
- **RBC:** Microcytic, Hypochromic.
- **WBC:** Normal in morphology and distribution.
- **Platelets:** Thrombocytopenia.
- **Parasite:** Not seen on present smear.

ECG
- Atrial fibrillation.
- Ventricular premature complexes.
- Non-specific intraventricular conductive depolarisation.
- Repolarisation abnormal suggest ischemia.
- Artefact in lead(s) II, III, avL, V1, V2, V3, V4, V5, V6 and I, III, avF, V5, V6.
- Abnormal ECG.
- Lateral infarct, acute.

Urine for Culture and Sensitivity
Bacterial growth and Isolated: E. coli
Colony count: 1, 00,000 CFU/ml significant bacteriuria.
Shows sensitivity to Fosfomycin, Gentamicin, Ertapenem, Imipenem and Amikacin and is resistance to Colistin.
To rule out Ankylosing Spondylitis, HLA-B27 was done and was negative.

CECT Abdomen
Impression:
- Circumferential mural thickening of terminal ileum, caecum and ascending colon and mild wall thickening with non-enhancing areas in transverge and descending colon with peri colonic fat stranding and engorgement of vasarecta as described – likely Inflammatory Bowel Disease.
- Multiple non enhancing hypodense areas within the wall of terminal ileum, caecum, and ascending colon? Abscesses
- Few non enhancing wedge shaped hypodense area in segment IV-? Etiology.
- Non obstructive left renal calculi.
- Likely bilateral sacroiliitis.
- GB sludge/Cholelithiasis
- ? Splenic cyst
- Bilateral minimal pleural effusion.
  - ✓ In view of multiple joint pain and RA, patient was advised for ANA Screening, RF - IgM which was positive for ANA screening and negative for RF - IgM.

Ultrasound Abdomen: 17/09
IMP: Minimal GB sludge
Non obstructive left renal calculi
Cystitis

Colonoscopy Report
Impression: Colitis with rectal sparing
Crohn's disease

Upper GI Endoscopy Report
IMP: Antral ulcers and Gastritis
Erosive Duodenitis

Table 3: Prothrombin Time & LFT

APTT	34 sec (24 - 36)
Prothrombin Time	16.5 sec (11-15)
Total bilirubin	0.5 mg/dl (0.1- 1.2)
ALT (SGPT)	18 Units/L (up to 42)
AST(SGOT)	20 Units/L (0-35)

Spine Examination
Inspection: No Scordiosis/Kyphosis
1. Occipital to wall distance/Flesche test – Negative

2. Schober's test (+ve) –18 cm
3. Modified Schober's test (+ve) – 12.5 cm
4. SLRT - +ve Sciatic pain between 30- 40…….
5. Particles (figure of H test) – positive
6. Chest Examination: Normal 25 cm
   S/o restricted chest expansion

Table 4: Treatment Chart

S. No	Brand Name	Generic Name	Dose	ROA	FREQ
1.	Inj. 25% Dextrose	Dextrose	100 ml	IV	TID
2.	Syrup Sucral	Sucralfate	15 ml	PO	QID
3.	Cap-VSL- 3	Bifidobacterium + lactobacillus + streptococcus	1 tab	PO	OD
4.	Tab. HCQ	Hydroxychloroquine	200 mg	PO	OD
5.	Tab. Ultracet	Tramadol + Acetaminophen	1 tab	PO	BD
6.	Inj. Eldervit	Folic acid + Vitamin C +Vitamin B_{12} + Niacinamide	1 amp	IV	OD
7.	Inj.$MgSO_4$	Magnesium sulphate	1 g	IV	BD
8.	Inj. Pan	Pantoprazole	40 mg	IV	BD
9.	Inj. Metrogyl	Metronidazole	100 ml	IV	TID
10.	Inj. Zostum	Cefoperazone + Sulbactam	1.5 g	IV	BD
11.	Inj. Optineuron	Thiamine (Vitamin B1) + Vitamin B6 (Pyridoxine) + D-Panthenol	1 amp	IV	OD
12.	Inj. Arachitol	Calcitriol	6 L	IV	STAT
13.	Tab. Mesachol	Mesalamine	1.2 g (2 tabs)	PO	BD
14.	Pentoza Sachets (in water)	Mesalamine	2 g	PO	BD
15.	Inj. Tranexa	Tranexamic acid	500 mg	IV	TID

3. DISCUSSION

A two-sample Mendelian randomization study was carried out to investigate whether rheumatoid arthritis is causally related to IBD and vice versa. And the results were found to show a positive effect of genetically predicted rheumatoid arthritis on IBD as a whole (OR 1.214; 95% CI 1.134; 1.299; $P = 3 \times 10^{-8}$). In subtype analyses rheumatoid arthritis was suggestively associated with Crohn's disease (CD) (OR 1.108; 95% CI 1.024; 1.199; $P = 0.011$) and ulcerative colitis (UC) (OR 1.082; 95% CI 1.002; 1.168; $P = 0.044$). Regarding the other direction, IBD as a whole as well as both subtypes were not related to rheumatoid arthritis.

The patient in this case, is a known case of Rheumatoid Arthritis and on irregular treatment which gradually developed into Inflammatory Bowel Disease. Treatment plays a vital role in the management of the disease, and hence avoiding regular treatment may also be an aggravating factor for the development of IBD. Our patient has both the risk factors for developing IBD which includes Known case of RA and irregular treatment measures.

Certain investigations have shown that gastrointestinal issues of different kinds are linked to treatment agents for RA, including D-penicillamine, gold salts, and non-steroidal anti-inflammatory medications (NSAIDs). In view of burning Micturition, urine was sent for c/s and showed significant growth of E. coli, Peripheral smear showed microcytic hypochromic RBC. UGIE, CECT & Colonoscopy were planned in View of IBD and Showed antral ulcers, Gastritis, erosive duodenitis and Colitis with rectal sparing which is consider as Inflammatory Bowel Disease. This patient, who has had rheumatoid arthritis for seven years, was sent to a rheumatologist for advice on Anti-nuclear antibodies (ANA) screens and Rheumatoid Factor (RF-IgM) testing. The results of the ANA screening were positive, while the RF-IgM test was negative.

The low prevalence of connection with RA may possibly be related to the usage of immunosuppressive medications in the treatment of UC. This patient's medication chart including corticosteroids and amino salicylates inhibits the inflammatory response in the body, which suggests that the frequency of other autoimmune diseases that coexist with IBD is reduced. To rule out Ankylosing Spondylitis, HLA-B27 was sent and which is negative in this patient. Vitamin D levels were 10.55 and were given Vit.D Supplementations. The Patient was closely monitored & treated with Antibiotics (Inj. Cefoperazone + Sulbactam, Inj. metronidazole), Vitamin Supplementation, Mg. Supplementation, Anti-Inflammatory Drugs and Other Supportive Medication.

4. CONCLUSION

Even though arthritis induced IBD has been seen in a rare scenario. Our patient seems to have developed IBD while on improper treatment for arthritis which might have played a role in the development of IBD. Evidence from previous studies suggests it has a possibility of causal relationship in development of IBD from RA. Likewise there are

several articular and extra articular manifestations which can develop from arthritis; management has always been a key role in control of disease and its associated features. Upon which failed to do so can lead to several comorbidities like inflammatory bowel disease.

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