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Anemia in Pediatric Inflammatory Bowel Disease

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List of abbreviation:

IBD	Inflammatory Bowel Disease
CD	Crohn's Disease
UC	Ulcerative Colitis
IC	Indeterminate Colitis
IDA	Iron Deficiency Anemia
ACD	Anemia of Chronic Disease
GI	Gastrointestinal
Hb	Hemoglobin
Hct	Hematocrit
MCV	Mean corpuscular volume
TIBC	Total iron binding capacity
ESR	Erythrocyte Sedimentation Rate
CRP	C-Reactive Protein
sTR	Soluble transferrin receptor
WHO	World Health Organization
PCDAI	Pediatric Crohn's Disease Activity Index
PUCAI	Pediatric Ulcerative Colitis Activity Index
BMI	Body Mass Index
TBI	Total body iron
TI	Terminal ileum

Abstract:

Objectives

Anemia is the most frequent extra-intestinal finding in Inflammatory Bowel Disease. The aim of this study is to determine the prevalence and types of anemia in pediatric patients with Inflammatory Bowel Disease at diagnosis and at approximately one year follow up.

Methods:

This is a retrospective chart review of patients diagnosed with Inflammatory Bowel Disease from 2005 to 2012, ages 1-18 years. Patients who had hemoglobin, hematocrit, mean corpuscular volume and iron indices obtained at the time of diagnosis and at approximately one year follow up were included in the study. The prevalence of anemia at the beginning and the end of the study was recorded. Using the soluble transferrin receptor index the type of anemia was determined.

Results:

At diagnosis, 67.31% of patients were anemic. Overall, 28.85% of patients had either Iron deficiency anemia or a combination of Iron deficiency anemia and anemia of chronic disease, while 38.46% had anemia of chronic disease alone. At follow up 20.51% were anemic. 15.38% had either iron deficiency anemia or a combination of iron deficiency anemia and anemia of chronic disease; 5.13% had anemia of chronic disease alone. The pattern of anemia and response to therapy differed among the inflammatory Bowel disease phenotypes

Conclusion:

Anemia is frequent in Inflammatory Bowel Disease. The prevalence was higher in Crohn's Disease. At one year, the prevalence of anemia decreased significantly, but persisted. Anemia of Chronic Disease predominated in CD. Iron Deficiency Anemia continued to be present in CD and UC. .

Key Words: Inflammatory Bowel Disease, Crohn's Disease, Ulcerative Colitis, Indeterminate Colitis, Anemia, Iron Deficiency Anemia, Anemia of Chronic Disease.

What is known:

•Anemia frequently complicates Inflammatory Bowel Disease and adversely effects patients' quality of life.

•Anemia can be caused by iron deficiency or due to chronic disease inhibiting erythropoiesis. In either case, it proves difficult to treat.

What is new:

•Anemia is common and persists even after a year of treatment in many patients with Inflammatory Bowel Disease.

•Prevalence of anemia is greater in Crohn's Disease compared to Ulcerative Colitis or Indeterminate Colitis.

•Anemia of Chronic Disease is characteristic of Crohn's Disease while Iron Deficiency Anemia is more a feature of Ulcerative Colitis.

Introduction:

The prevalence of Inflammatory Bowel Disease (IBD) is increasing in both adult and pediatric populations (1) (2). The prevalence of IBD in the US is 0.6% (1) making it one of the most common chronic diseases of childhood. The world-wide spread of IBD parallels industrialization. The risk of IBD seems to be associated with change in environment and diet that may alter the microbiome leading to IBD in genetically susceptible individuals (1). Anemia is the most common extra-intestinal manifestation of IBD and can adversely affect quality of life (3, 4). The two types of anemia associated with IBD are Iron Deficiency Anemia (IDA) and Anemia of Chronic Disease (ACD). These anemias can occur separately or can be present simultaneously.

Iron Deficiency Anemia: The reason for IDA in IBD is multifactorial. The most obvious reason is gastrointestinal (GI) blood loss; however, sloughing of cells that line the GI tract, malabsorption and decrease in oral iron due to poor appetite can also contribute to IDA in IBD.

Anemia of Chronic Disease: ACD frequently accompanies IBD, but is not specific to IBD, occurring in many other chronic inflammatory processes. In ACD hepcidin activity is increased resulting in a decrease in red blood cell production at the level of the erythron and a decrease in iron absorption via the small intestine. In many ways ACD mimics IDA including microcytosis and decreased reticulocyte count. Many indices of iron status are also acute phase reactants, making interpretation of test results problematic in the face of concurrent inflammation. The fact that IDA and ACD often occur together further complicates the diagnosis and planning the treatment. (5)

The aim of this study is to examine the prevalence and types of anemia in pediatric patients with IBD at diagnosis and at approximately one year follow up.

Methods:

The study was approved by the Children and Youth Institutional Review Board, University at Buffalo, Women and Children's Hospital. It is a retrospective chart review of patients diagnosed with IBD from 2005 to 2012, ages 1-18 years. Patients who had hemoglobin (Hb), hematocrit (Hct), mean corpuscular volume (MCV) and iron indices obtained at the time of diagnosis and at approximately one year follow up were included in the study. A total of 213 charts were reviewed and 153 patients were found to fit these criteria. In this study group the following data was sought: albumin, serum iron, ferritin, total iron binding capacity (TIBC), transferrin, soluble transferrin receptor (sTR), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). World Health Organization's (WHO) hemoglobin thresholds were used to define anemia (6). The sTR/log ferritin index (sTR Index) was employed to differentiate the type of anemia, IDA from ACD (7, 8). This index can differentiate IDA from ACD; however, it cannot separate IDA from the combination of IDA/ACD. IDA or IDA/ACD were considered to be present if the sTR index value was greater than 1.03. An sTR index of less than 1.03 was taken to be indicative of

the presence of ACD. Total body iron was calculated as follows: Total Body Iron=-[log(sTR/Ferritin)-2.8229]/0.1207. (9) The Pediatric Crohn's Disease Activity Index (PCDAI) and the Pediatric Ulcerative Colitis Activity Index (PUCAI) were recorded at diagnosis and at follow up.

All of the statistical analyses were performed by a professional statistician using the SAS System (SAS Institute, Cary, NC). Results are expressed as mean values + standard deviation. A p-value of 0.05 or less was taken to indicate a significant difference.

Results:

The characteristics of the study subjects are given in Table 1. The prevalence of IBD in males and females was not found to be statistically different. Crohn's disease occurred more frequently than UC or IC. The severity of disease for most IBD patients was moderate or less. The severity improved at the time of follow up. The majority of the patients received iron therapy, most frequently oral therapy.

Table 2 lists the parameters at baseline and at follow-up. All parameters improved except serum ferritin and serum soluble transferrin receptor; those two values remained unchanged. The calculated value for total body iron did not change.

The portion of patients with abnormal hematologic parameters decreased significantly from diagnosis to follow up for most values recorded (Figure 1). Percent of patients with abnormal serum iron and abnormal ferritin levels appeared to decrease but did not reach statistical significance. Percent with abnormal sTR did not decrease.

The proportion of patients who were anemic by WHO criteria decreased significantly from diagnosis to follow up. This was true for the entire IBD cohort and for the IBD phenotypes CD and UC. Anemia decreased among the patients with IC, however, this group represented only 4 patients precluding meaningful statistical analysis (Figure 2a). Figure 2b shows the percent of patients with IDA or IDA/ACD initially and at one year follow up. There were significant decreases in these categories of anemia for IBD and for CD as well as UC. Figure 2c shows the percent of patients with ACD alone (without IDA). For the group as whole and for CD there was a dramatic decrease in this category of anemia. For UC at follow up none of the patients had ACD alone. There were insufficient numbers of patients with IC to compare.

We created a correlation table (Supplemental Table 1, Supplemental Digital Content, http://links.lww.com/MPG/B366) crossing all of the parameters we measured at diagnosis. Some interesting correlations were apparent. Albumin correlated with most measures of anemia as well as with nutritional parameters (weight, BMI and BMI-z) and measure of clinical indices of disease severity (ESR, CRP, PDCAI and PCUAI). Interestingly clinical severity scores (PDCAI and PUCAI) were poor predictors of anemia.

Discussion:

In the management of IBD patients with anemia it is important and helpful to determine the type of anemia as treatment options depend on the type of anemia. In IBD, IDA and ACD frequently occur simultaneously, complicating interpretation and treatment.

Iron is present in all mammalian cells and is of essential importance not only for oxygen transport but also for many non-hematological functions (10). In IBD iron deficiency is the result of reduced iron uptake from the enterocyte and chronic blood loss from the gastrointestinal tract. Ongoing daily losses of >10 ml of blood will result in iron deficiency (10).

Anemia of chronic disease is due to inflammatory cytokine-mediated mechanisms thought to be due, at least in part, to hepcidin. Hepcidin is a circulating peptide, which plays a major role in iron homeostasis. Hepcidin reduces the quantity of circulating iron by preventing its exit from the cells, especially from enterocytes and macrophages. Hepcidin expression is controlled by iron and inflammation. The pro-inflammatory cytokines induce the hepcidin gene. In states of inflammation, high hepcidin levels lead to inhibition of iron release from enterocytesdecreased iron levels in the circulation, and decreased utilization of iron at the level of the erythron (11).

Different parameters have been used to evaluate anemia in IBD, such as hemoglobin, ferritin, serum iron, total iron-binding capacity (TIBC), and transferrin saturation (12). Outside the context of IBD, IDA and ACD are differentiated by using iron indices (13, 14). However, these tests are affected by chronic inflammatory disease. Ferritin levels are typically reduced in IDA, but may be increased by the chronic inflammation. When both iron deficiency and the anemia of chronic disease are present, many of the laboratory measures of iron status become unreliable (8).

Hemoglobin: When IDA accounted for most cases of anemia in children, “anemia” and “IDA” were roughly synonymous, and a simple measurement of Hb concentration was sufficient to make a presumptive diagnosis of anemia attributable to iron deficiency. Particularly in industrialized nations, the prevalence of iron deficiency and IDA has decreased, and other causes of anemia, such as hemolytic anemias, anemia of chronic disease, and anemia attributable to other nutrient deficiencies have become proportionately more common (15). Based on body iron measurements, however, only 20% of women with presumptive iron deficiency anemia had total body iron deficiency as defined by a total body iron deficit greater than 4 mg/kg (9).

Serum iron: Serum iron is not an accurate tool to study the iron deficiency anemia. Iron levels change rapidly and serum iron show extensive fluctuations. Serum iron turns over many times daily (11). In our study, iron levels did not correlate with clinical activity but did correlate with the degree of anemia (Supplemental Table 1, Supplemental Digital Content, http://links.lww.com/MPG/B366). There was no correlation between serum iron level and total body iron perhaps reflecting the fact that serum iron represents only a fraction of the total body iron.

Total Body Iron: Calculations of total body iron using the formula: Total Body Iron=-[log (sTR/Ferritin)-2.8229]/0.1207, is not dependent on hemoglobin and thus measures iron status rather than anemia. Approximately half of the total body iron is circulating as serum iron or as hemoglobin, the remainder is found in myoglobin, heme- and non-heme enzymes and in storage forms, ferritin and hemosiderin. Thus, small changes in serum iron may not be reflected in changes in total body iron. The total body iron level did not correlate with gender, terminal ileum (TI) involvement, type of IBD or the severity of the disease (Supplemental Table 1, Supplemental Digital Content, http://links.lww.com/MPG/B366). It also did not correlate with the hemoglobin level.

Serum ferritin is the most accessible measure of storage iron, and a subnormal concentration is indicative iron deficiency. Ferritin, however, is also an acute-phase reactant, and in the context of IBD, a normal or high value does not exclude iron deficiency. In our study, most patients had normal ferritin levels at the time of the diagnosis (72%). Ferritin levels were found to be lower in UC, and higher in CD (p- value <0.05) (Table 2), possibly due to more blood loss in UC and relatively more inflammation in CD. In this study, ferritin levels did not correlate with the hemoglobin levels, serum iron levels or disease activity, but did correlate with inflammatory markers (Supplemental Table 1, Supplemental Digital Content, http://links.lww.com/MPG/B366), sTR, and total body iron. Overall, ferritin alone is not a useful marker to estimate the type of anemia in IBD patients.

Transferrin and Total Iron Binding Capacity: Serum transferrin (sTF) and total iron binding capacity (TIBC) are reciprocal measurement. Transferrin is synthesized in the liver and mediates the transfer of iron between the enterocytes and the erythron. Serum TF generally carries about one third of its iron binding capacity as a mixture of apo-, mono- and di-ferric forms. Serum TF levels go up in iron depletion and decrease as in iron overload. TIBC is a rough a measure of the amount of iron it takes to saturate sTF. Because TIBC measures non-specific binding to other proteins, TIBC often overestimated the binding capacity. Serum TF is the more accurate of the two, nevertheless, our data showed that both values increased significantly with treatment.

Soluble Transferrin Receptor (sTR) and Soluble Transferrin Receptor Index (sTR-index): Serum sTR is an indicator of iron deficiency and is unaffected by chronic disease and inflammation (16). The transferrin receptor is a protein expressed in cells that require iron, and the soluble form is elevated in serum in cases of iron deficiency (17). Serum ferritin levels reflect iron stores while sTR levels reflect the degree of availability of iron for cells. The sTR Index, developed by Skikne et al, represents the relationship between two variables influenced by iron deficiency, sTR and ferritin (18). In this study we used the sTR index to differentiate the ACD from IDA. At the time of the diagnosis, IDA or (IDA +ACD) prevalence was 28%. The ACD prevalence was 38%. In our study, sTR correlates with PCDAI, not with PUCAI score, possibly due to the degree of inflammation in CD compared to UC. There was no correlation with inflammatory markers and there is no difference in the sTR level between the type of IBD or the TI

involvement (Supplemental Table 1, Supplemental Digital Content, http://links.lww.com/MPG/B366).

Intuitively, degree of anemia and clinical indices of disease severity should correlate. In our study there is no such correlation (Supplemental table 1, Supplemental Digital Content, http://links.lww.com/MPG/B366). This suggests that clinical indices of disease severity and anemia are not related. A possible explanation is that cytokines can lead to subclinical anemia before the laboratory signs and clinical symptoms of anemia are apparent. Alternatively, the indices of clinical indices of disease severity (PUCAI and PCDAI) may be inaccurate. Supporting the last of these possibilities is our finding that inflammatory markers (ESR and CRP) are consistent with the degree of anemia. Among all parameters examined, albumin is the single factor that correlates with most of the iron indices.

We found that the degree of anemia is consistent with inflammatory markers, not with clinical measures of disease activity. Anemia may exist even with mild inflammatory disease. Perhaps because activation of the inflammatory cascade is enough to cause anemia in the absence of the clinical picture of severe IBD. In states of inflammation, high hepcidin levels block iron release from enterocytes and decrease iron availability. This might explain our finding of lack of correlation between total body iron and the severity of the clinical activity.

Conclusion:

Anemia is frequently encountered in both Crohn's disease and Ulcerative Colitis. Measures of clinical indices of disease severity, PDCAI and PUCAI, were poor predictors of anemia. In CD and UC anemia due to IDA and ADC occur, either singly or together. The anemia of IBD proves difficult but not impossible to treat. The anemia in CD and UC do not follow the same pattern, perhaps pointing to differing pathogenesis.

References

1 Kaplan GG, Ng SC Understanding and Preventing the Global Increase of Inflammatory Bowel Disease. Gastroenterology 2017;152(2):313-21 e2.

2 Benchimol EI, Fortinsky KJ, Gozdyra P, et al. Epidemiology of pediatric inflammatory bowel disease: a systematic review of international trends. Inflamm Bowel Dis 2011;17(1):423-39.

3 Pizzi LT, Weston CM, Goldfarb NI, et al. Impact of chronic conditions on quality of life in patients with inflammatory bowel disease. Inflamm Bowel Dis 2006;12(1):47-52.

4 Wells CW, Lewis S, Barton JR, et al. Effects of changes in hemoglobin level on quality of life and cognitive function in inflammatory bowel disease patients. Inflamm Bowel Dis 2006;12(2):123-30.

5 Cullis J Anaemia of chronic disease. Clin Med (Lond) 2013;13(2):193-6.

6 Iron Deficiency Anemia: Prevention, Assessment, Prevention, and Control, A guide for programme managers. In: W. H. Orgainzation ed.; 2001.

7 Global surveillance and control of hepatitis C. Journal of Viral Hepatology 1999;6(1):35-47.

8 Punnonen K, Irjala K, Rajamaki A Serum transferrin receptor and its ratio to serum ferritin in the diagnosis of iron deficiency. Blood 1997;89(3):1052-7.

9 Cook JD, Flowers CH, Skikne BS The quantitative assessment of body iron. Blood 2003;101(9):3359-64.

10 Cronin CC, Shanahan F Anemia in patients with chronic inflammatory bowel disease. Am J Gastroenterol 2001;96(8):2296-8.

11 Weiss G, Gasche C Pathogenesis and treatment of anemia in inflammatory bowel disease. Haematologica 2010;95(2):175-8.

12 Oustamanolakis P, Koutroubakis IE, Kouroumalis EA Diagnosing anemia in inflammatory bowel disease: beyond the established markers. J Crohns Colitis 2011;5(5):381-91.

13 Baer AN, Dessypris EN, Krantz SB The pathogenesis of anemia in rheumatoid arthritis: a clinical and laboratory analysis. Semin Arthritis Rheum 1990;19(4):209-23.

14 Ferguson BJ, Skikne BS, Simpson KM, et al. Serum transferrin receptor distinguishes the anemia of chronic disease from iron deficiency anemia. J Lab Clin Med 1992;119(4):385-90.

15 Baker RD, Greer FR, Committee on Nutrition American Academy of P Diagnosis and prevention of iron deficiency and iron-deficiency anemia in infants and young children (0-3 years of age). Pediatrics 2010;126(5):1040-50.

16 Cook JD, Skikne BS, Baynes RD Serum transferrin receptor. Annu Rev Med 1993;44(63-74.

17 Baillie FJ, Morrison AE, Fergus I Soluble transferrin receptor: a discriminating assay for iron deficiency. Clin Lab Haematol 2003;25(6):353-7.

18 Skikne BS, Punnonen K, Caldron PH, et al. Improved differential diagnosis of anemia of chronic disease and iron deficiency anemia: a prospective multicenter evaluation of soluble transferrin receptor and the sTfR/log ferritin index. Am J Hematol 2011;86(11):923-7.

Figure legends

Figure 1: Percent of patients with abnormal values at diagnosis and at follow up. Significance was determined using number of patients and T-test. P<0.05 was taken to indicate a significance and is denote with *. MCV= mean corpuscular volume. TIBC= total iron binding capacity. sTR= serum transferrin receptor.

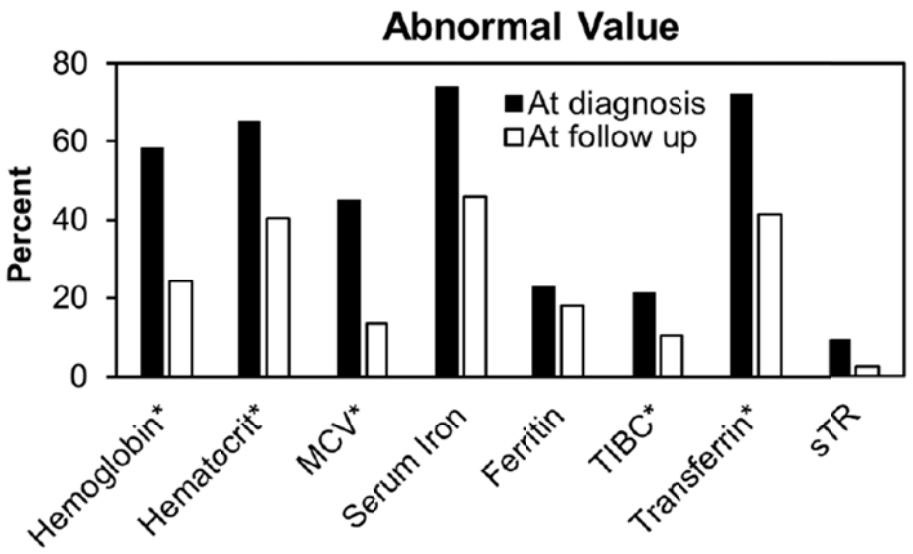


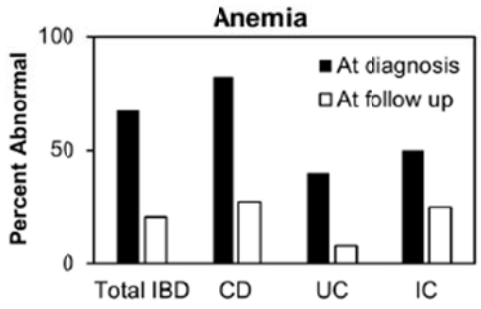
Figure 2: 2a shows the percent of patients who were anemic by WHO standards at diagnosis and at follow up. IBD=inflammatory bowel disease. CD=crohn's disease. UC=ulcerative colitis. IC=indeterminate colitis.

2b shows percent of patients with IDA+IDA/ACD at diagnosis and at follow up. IBD=inflammatory bowel disease. IBD=inflammatory bowel disease. CD=crohn's disease. UC=ulcerative colitis. IC=indeterminate colitis.

2c shows percent of patients with ACD (without IDA) at diagnosis and at follow up. . IBD=inflammatory bowel disease. CD=crohn's disease. UC=ulcerative colitis.

Figure 2. Anemia at diagnosis and follow up

(a)



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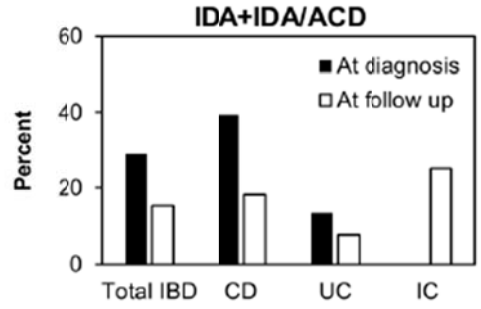

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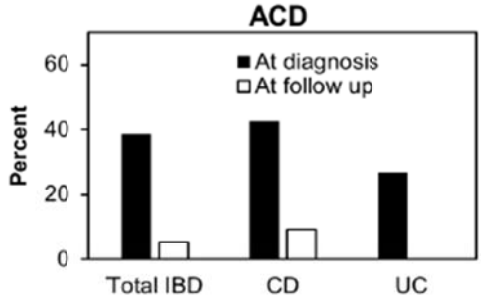

IBD = inflammatory bowel disease, CD = Crohns disease, UC = ulcerative colitis, IC = indeterminate colitis, IDA = iron deficiency anemia, ACD = anemia of chronic disease.

Table 1: Patient Characteristics

Gender (N=153)	
Male	86 (56.2%)
Female	67 (43.8%)
Diagnosis (N=153)	
CD	92 (60.1%)
UC	49 (23.0%)
IC	12 (06.5%)
PCDAI (initial) (N=87)	
Quiescent	22 (25.2%)
Mild	51 (58.6%)
Moderate to severe	14 (16.1%)
PCDAI (follow up) (N=73)	
Quiescent	64 (87.7%)
Mild	7 (9.6%)
Moderate to severe	2 (2.7%)
PUCAI (Initial) (N=35)	
Remission	4 (11.4%)
Mild	15 (42.9%)
Moderate	13 (37.1%)
Severe	3 (8.6%)
PUCAI (follow up) (N=27)	
Remission	15 (55.6%)
Mild	9 (33.3%)
Moderate	3 (11.1%)
Severe	0
Iron Therapy (N=143)	
Iron therapy	97 (67.8%)
No iron therapy	46 (32.2%)
Type of iron therapy (N=143)	
PO only	88 (61.5%)
IV Dextran	5 (3.5%)
IV Venofer	4 (2.8%)
No iron therapy	46 (32.2%)

CD=crohn's disease. UC=ulcerative colitis. IC=indeterminate colitis. PCDAI=pediatric crohn's disease activity index. PUCAI=pediatric ulcerative colitis activity index. PO=oral treatment. IV=intravenous treatment.

Table 2: Initial and follow up characteristics.

	N	Mean	Std Dev	P Value
AGE (years)	153	12.7	3.73	
Weight I (Kg)	153	46.8	20.5	<0.0001
Weight F/U (Kg)	119	52.2	20.6	
BMI z score I	153	-0.3	1.14	<0.0001
BMI z score F/U	116	0.2	1.1	
Hemoglobin I (g/dL)	153	11.5	1.9	<0.0001
Hemoglobin F/U (g/dL)	119	12.7	1.6	
Hematocrit I (%)	153	34.5	5.43	<0.0001
Hematocrit F/U (%)	119	37.8	4.4	
MCV I (fL)	153	78.9	7.7	<0.0001
MCV F/U (fL)	119	85.4	7	
Albumin I (g/dL)	139	3.4	0.77	<0.0001
Albumin F/U (g/dL)	109	4.0	0.5	
Serum Iron I (µg/dL)	150	42.7	49.7	0.0005
Serum Iron F/U (µg/dL)	85	63.1	37.5	
Ferritin I (ng/ml)	126	47.3	51.5	0.35
Ferritin F/U (ng/ml)	67	48.8	55.44	
TIBC I (µg/dL)	146	311.9	74.6	0.002
TIBC F/U (µg/dL)	76	333.9	68.3	
Transferrin I (g/dL)	145	13.0	13.01	0.0002
Transferrin F/U (g/dL)	75	20.1	13.69	
sTR I (nmol/L)	63	1.54	0.66	0.3
sTR F/U (nmol/L)	42	1.66	0.64	
ESR I (mm/hr)	150	30.8	22.1	<0.0001
ESR F/U (mm/hr)	112	18.8	19.55	
CRP I (mg/L)	109	29.9	46.3	<0.0001
CRP F/U (mg/L)	73	8.3	20.8	
Total body iron I (mg/kg)	51	9.9	4.44	0.7083
Total body iron F/U (mg/kg)	39	10.0	4.4	

N=number of subjects. Std Dev=Standard Deviation. I=Initial. F/U=Follow up. BMI=Body Mass Index. MCV=Mean Corpuscular Volume. TIBC=Total Iron Binding Capacity. sTR=Serum Transferrin Receptor. ESR=Erythrocyte Sedimentation Rate. CRP=C-Reactive Protein.

Table 3.

Type of Anemia at diagnosis	Total IBD (n=52)	CD (n=33)	UC (n=15)	IC (n=4)
IDA or (IDA+ACD)	28.85% (n=15)	39.39% (n=13)	13.33% (n=2)	0
ACD	38.46% (n=20)	42.42% (n=14)	26.67% (n=4)	50.00% (n=2)
No Anemia	32.69% (n=17)	18.18% (n=6)	60.00% (n=9)	50.00% (n=2)
Type of Anemia at F/U	Total IBD (n=39)	CD (n=22)	UC (n=13)	IC (n=4)
IDA or (IDA+ACD)	15.38% (n=6)	18.18% (n=4)	7.69% (n=1)	25.00% (n=1)
ACD	5.13% (n=2)	9.09% (n=2)	0	0
No Anemia	79.49% (n=31)	72.73% (n=16)	92.31% (n=12)	75.00% (n=3)

IBD=Inflammatory Bowel Disease. CD=crohn's disease. UC=ulcerative colitis. IC=indeterminate colitis. IDA=Iron Deficiency Anemia. ACD=Anemia of Chronic Disease. F/U=Follow Up