INFLAMMATORY BOWEL DISEASE (ULCERATIVE COLITIS) CO-EXISTING WITH COELIAC DISEASE

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Abstract:
A 24 year old male presented with weakness and fatigue. On investigation, he was found to have microcytic hypochromic anemia and later diagnosed as having celiac disease both by serology and duodenal histology. Eighteen months later he developed chronic large bowel type of diarrhea which was found to be due to ulcerative colitis. This case is being reported to highlight the co-existence of celiac disease and ulcerative colitis, an uncommon occurrence.

Keyword:
ulcerative colitis, coeliac disease

CASE DETAILS:
Mr V, a 28 year old computer operator from Rajasthan was well till 2008, when he developed symptoms of easy fatigue and generalized weakness, for which evaluation revealed a hemoglobin of 5.6 gm%, which was microcytic hypochromic. On further evaluation was found to be positive for the anti-transglutaminase (TTG) antibody. Upper GI scopy showed attenuation of duodenal folds with minimal nodularity. Duodenal histology which shows partial villous atrophy with intra-epithelial lymphocytes.

Duodenal histology revealed partial flattening of villi and non-specific inflammation of the interglandular areas. He was initiated on gluten-free diet and iron supplements. He subsequently was asymptomatic.

For the past two years has developed chronic diarrhoea which was intermittent and watery to semisolid in consistency. Initially thought to be due to celiac disease, a repeat duodenal biopsy revealed flattening...
of villi with crypt hyperplasia and lamina propria showed increased lympho-plasmocytic infiltration. He was on a strict gluten free diet. For the past one year, he had loose stools mixed with blood, and mucus and he had tenesmus. There was associated intermittent low grade fever, once a month. For the above symptoms, he was subjected to a colonoscopy which showed the entire colonic mucosa to have superficial ulceration, loss of vascular pattern, loss of haustration with diffuse erythema and friability.

Segmental colonic mucosal biopsies showed distortion and reduction in the number of crypts. Cryptitis and crypt abscesses were seen. The lamina propria showed a mixture of acute and chronic inflammatory cells. There was non specific ileitis with mild inflammation. No granulomas were seen. The villi were normal. The findings were in favor of acute over chronic ulcerative colitis.

He was subsequently started on T.Mesacol 1.2 gm bd and then required frequent doses of tapering steroids. The diarrhea persisted and thus had to be started on T.Azoran 50 mg once daily which was stepped up to 100 mg once daily later.

Despite being on a strict gluten free diet with persistence of symptoms, he was referred here for further evaluation and management. Here on evaluation, his hemoglobin was 11.8 gm %. The inflammatory markers were found to be elevated (ESR, CRP). Anti-TTG antibody was positive. The stool culture/routine/special stains as well as the clostridium assay were negative. The serum immunoglobulin profile was normal.

He was also found to be vitamin B12 deficient. The pernicious anemia antibody screen was found to be negative. On imaging - contrast enhanced computer tomography, there was diffuse thickening of the bowel with maintained wall stratification and positive water-halo sign (fig 2) involving the descending colon and rectosigmoid. The above mentioned features were representative of ulcerative pancolitis with active inflammation in the left colon.

Cytomegalovirus superinfection (CMV) of the colon was looked for. The blood for CMV polymerase chain reaction (PCR) was 94498 copies/ml. Colonoscopy done showed edematous mucosa of the cecum transverse and descending colon with loss of vascular pattern and haustations. The mucosa of the rectum and colon showed multiple ulcers, friability, granularity with loss of vascular pattern. Segmental biopsies along with tissue for CMV PCR were taken. The colonic histology showed mild chronic pancolitis with architectural alteration and mild activity, segmental colorectal mucosal biopsies with mild CMV infection (cytopathic changes). Oesophagogastroduodenoscopy was a normal study. The duodenal biopsy (2nd part) showed duodenal mucosa with minimal eosinophilia. Mild chronic gastritis with focal activity and regenerative changes. The fundal biopsy did not reveal any abnormality.
He was then initiated on gancyclovir after stopping the azathioprine. He experienced symptoms of proctitis for which dexamethasone enema was given. While on treatment had developed low total counts. The bone marrow showed a mildly hypocellular marrow with adequate trilineage differentiation. It reverted with one dose of granulocyte-monocyte colony stimulating factors and by withholding gancyclovir temporarily. There was gradual improvement in stool frequency with no blood. Blood Cytomegalovirus polymerase chain reaction was repeated after a total of 21 days of gancyclovir and was negative. He was thereafter discharged in a stable condition.

**REVIEW OF LITERATURE**

**IBD and COELIAC disease:** There is a suggestion in the literature that IBD (ulcerative colitis > Crohn’s disease) may be more common both in patients with coeliac disease, and in their first degree relatives. The prevalence of coeliac disease in patients with IBD does not appear to be increased compared with the general population (1-2%). This observation was noted in a large multi-centre Italian study which observed lower rates of coeliac disease (0.5%) It is strongly hereditary (monozygotic concordance 70%, compared with 35% in Crohn’s disease). HLA plays a major role; DQ2 carriage is the most important genetic determinant of disease risk (almost 90% of patients are DQ2 positive). One study published looked at the prevalence of coeliac disease in IBD and the prevalence of IBD in coeliac disease. It was found that the prevalence of IBD in coeliac disease was increased 10-fold than compared with that in controls (odds
While the prevalence of celiac disease in IBD was comparable with that in controls (odds ratio 1.02, 95% CI, 0.24-4.29, p=1.0). Of the confirmed celiac disease genes, PTPN2, ICOSLG, REL and KIF21B are associated with IBD, BACH2, TAGAP, IL18RAP, ZMIZ1, and ZFP36L1 with Crohn’s disease, and IL2 / IL21 with ulcerative colitis.

**BIBLIOGRAPHY:**


