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Original Research Paper

Gastroenterology

A CASE OF ULCRATIVE COLITIS WITH AUTOIMMUNE HEPATITIS

Dr. A. S. K Manoj Postgraduate Resident, Dept of General Medicine, PIMS, Udaipur.

Dr. N. K. Gupta Professor, Dept of General Medicine, PIMS, Udaipur.

ABSTRACT

Abnormal liver biochemical tests are present in up to 30% of patients with inflammatory bowel disease (IBD), and therefore become a diagnostic challenge. Liver and biliary tract diseases are common extraintestinal manifestations for both Crohn's disease and ulcerative colitis (UC), and typically do not correlate with intestinal activity[1],in which most common hepatobiliary manifestation of IBD is Primary sclerosing cholangitis (PSC), andmore prevalent in UC. Approximately 5% of patientswith UC develop PSC, with the prevalence reaching upto 90%. Whereas less common disordersinclude **autoimmune hepatitis/PSC overlap syndrome**,IgG4-associated cholangiopathy, primary biliary cirrhosis, hepatic amyloidosis, granulomatous hepatitis, cholelithiasis, portal vein thrombosis, liver abscess, andnon-alcoholic fatty liver disease[1]. We are presenting a case of 28yr old male who came to opd with c/c of bloodtinged diarrhoea since 1.5 yrs, on initial investigations, it was observed that his LFT was deranged, on further evaluation;CECT abdomen showed Diffuse parenchymal liver disease, a liver elastography of liver was performed which showed mean values of 12 kPa;Cirrhosis was diagnosed,Initially he was treated symptomatically, latercolono scopy was performed to find out the cause for chronic diarrhoea which led to the diagnosis of ulcerative colitis.

KEYWORDS :

CASE SCENARIO :-

A 28 yr old male came to the opd with a chief complaint of Diarrhoea, and blood in stool since past 6 months, The pt who was apparently well before 1.5 years started complaining of frequent passing of stools (diarrhoea), 10-15 times a day, characteristically black to brownish in colour, semisolid to watery in consistency , contains blood and mucous,non-foul smelling, non-floating, non-greasyin nature which was aggravated on food intake , The ptgave a history of similar complaints in past 5 yrs (3-5 episodes a day) excluding the presence of blood in stools; for which he took treatment for 4-5 months as advised by the local physician which gave him temporary relief, although the pain still persisted. Furthermore, He started taking anon and off ayurvedic preparation for 5-6 months containingamla and giloyfor recurrent ulcers in mouth, the pthad right-sided pleural effusion 2 yrs back for which he had undergone pleural fluid tapping,which revealed raised ADA with proteins in it. Henceforth, suspecting it as a case of TB- related pleural effusion, was started on AKT.A past c/o pain in throatwas there which on examination by an ENT specialist was diagnosed as a Peri-Tonsillar Abscess that was drained under aseptic precautions followed by the treatment with antibiotics.

Pt's initial vital data BP-116/70 mmHg, PR- 82 /min, Spo_2-98 %, RR-20 /min

INITIAL INVESTIGATION :-

CBC – RBC-4.67 l/ul, HB-12.6gm%, HCT-39.0%, MCV-83.5FL, MCH-27pg.MCHC-32.3g/dl, RDW-15.9%, WBC-10,000/cumm, NEUTROPHIL-66%, LYMPHOCYTES-29%, MONOCYTES-04%, EOSINOPHILS- 1%, BASOPHILS-00%. PLATELET COUNTS-316thousand/cumm, ESR-88 1st hr mm.

LFT – BILIRUBIN- TOTAL-0.6 mg/dl,DIRECT-0.3mg/dl, INDIRECT-0.3mg/dl, SGOT-76.2 U/L, SGPT- 54.1 U/L, ALKALINE PHOSPHATASE- 223.5 u/l, TOTAL PROTIEN- 88.0 g/dl, ALBUMIN- 3.5 g/dl, GLOBULIN- 4.5 g/dl, A/G RATIO- 0.8.

RFT AND ELECTROLYTES – SODIUM-138.7mEq/L, POTASSIUM- 4.3mEq/L, BLOOD UREA – 29.4mg/dl, S.CREATNINE- 0.9mg/dl, URIC ACID- 5.0mg/dl, CRP-37.1 mg per L.

OTHER– PT-13.6, INR-1.18, IL-6 – 16.7,

ELASTOGRAPHY OF LIVER SONOGRAPHY – Mean A.R.F.I values was taken overall mean values of 12 kPa, Suggestive of cACLD but needs further evaluation

CECT WHOLE ABDOMEN– Diffuse parenchymal liver disease with dilated portal and splenic vein possibly changes of cirrhosis with portal hypertension, circumferential enhancing mural wall thickening involving rectum, rectosigmoid junction, sigmoid and descending colon upto the splenic flexure – suggest possibility of inflammatory bowel disease (? ULCERATIVE COLITIS).

HISTOPATHOLOGIAL STUDIES –

MICROSCOPIC: -

A: Sections from Duodenum show distorted mucosa. Lamina propria shows inflammatory cells mainly lymphocytes, neutrophils and eosinophils. Submucosa shows unremarkable brunner's glands.

B: Sections from lleum showed mucosal glands. Fragment ofileal tissue show distorted mucosal glands. At places epithelium of mucosal glands show regenerative changes. Lamina propria show dense chronic inflammatory cell infiltrate mainly lymphocytes, plasma cells. Lymphoid follicles are also identified. Muscular coat and serosa is not identified

C: Sections from cecum/ascending colon showed distorted mucosal glands. At places marked inflammatory cells comprising of neutrophils, lymphocytes, eosinophils and plasma cells are seen upto the submucosa. Mucosal ulceration is also identified at one place. Muscularlayer and serosa is not identified in sections examined.

D: Sections from Transverse descending colon showed distorted mucosal glands. At places mucosa is eroded and extensive inflammatory cell infiltrate mainly neutrophils, lymphocytes, eosinophils are seen forming ulcer. Anotherfragment show mucosal glands shows regenerative changes. Lamina propria shows dense inflammation with mild congestion.

E: Sections from Sigmoid/rectum showed fragment of mucosa showing distorted mucosal glands. Lamina propria shows dense mixed inflammatory cells.

IMPRESSION:- These histological features from sections studied are suggestive of Ulcerative lesion possibility of

Ulcerative Colitis.

F: Sections from Liver biopsy showed multiple white soft tissue pieces show linear tissue comprising of distorted parenchymal architecture. Hepatocytes are showing degenerative changes. At places hepatocytes are exhibiting ballooning degeneration with necrosis. There are marked lymphoplasmacytic infiltrate surrounding the hepatocytes are replaced by fibroblastic proliferation and abundant lymphoplasmacytic infiltrate. Portal tracts are showing lymphoplasmacytic infiltration around them. Periportal fibrosis is also seen. Florid villi duct ecanthosis is also appreciate. At places Rossete junction by hepatocytes is also identified. Endothelial proliferation along with dilated congested blood vessels is seen around the portal tract and also surrounding the hepatocytes. In a focal areas villi pigment is identified in the hepatocytes.

Liver Biopsy GROSS - Received multiple thread like structure measuring 1.0cm in length. Sections from Liver biopsy show multiple white soft tissue pieces show liver tissue comprising of distorted parenchymal architecture. Hepatocytes are showing degenerative changes. At places hepatocytes are exhibiting ballooning degeneration with necrosis. There are marked lymphoplasmacytic infiltrate surrounding the hepatocytes are replaced by fibroblastic proliferation and abundant lymphoplasmacytic infiltrate. Portal tracts are showing lymphoplasmacytic infiltration around them. Periportal fibrosis were also seen. Floroid duct proliferation was also appreciated. At places Rossete formation by hepatocytes is also identified. Endothelial proliferation along with dilated congested blood vessels is seen around the portal tract and also surrounding the hepatocytes. In a focal areas bile pigment is identified in the hepatocytes(**Figure1,2**).

IMPRESSION: -

These histological features are suggestive of Autoimmune Hepatitis with Cirrhotic changes.

Treatment Given Initially :-

he was treated initially symptomatically by giving injectable ciprofloxacin 500mg, injectable metronidazole 500mg, tablet ursodeoxycholic acid 450mg.

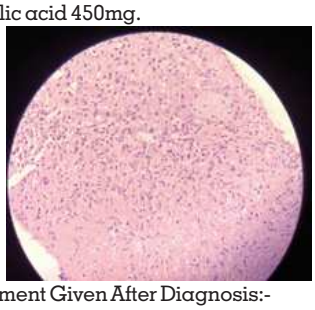

Figure-1 Treatment Given After Diagnosis:-

Pt was started on Steroid's

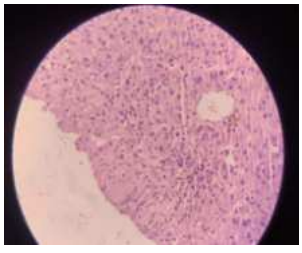

Figure-2

DISCUSSION :-

The mode of onset and progression of symptoms in our patient suggests inflammatory bowel disease (ulcerative colitis) which in-turn led to Autoimmune hepatitis with cirrhotic changes.

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