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A rare case of Hepatic Sinusoidal obstruction syndrome due to a drug use

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ABSTRACT

Here, we report a case of hepatic sinusoidal obstruction syndrome(HSOS) of a 30 years old female patient with Ulcerative colitis(UC) who was being treated by mesalazine.

The patient was admitted to the emergency unit by harsh and cramping abdominal pain, she had increased AST-ALT-Bilirubine and GGT levels in the blood laboratory analysis. In the Portal venous Doppler US; Splenomegaly and coarse granular appereance in liver parenchymal echogenecity were observed. In the abdominal CT, Splenomegaly was confirmed.

In the Dynamic Abdominal MRI, pathognomonic liver finding which was the patchy contrast-enhancing reticular appereance, was visualized and the exact diagnosis was handled by true-cut biopsy.

The aid of Dynamic Abdominal MRI to the early diagnosis of HSOS was also demonstrated in this case.

Keywords: Hepatic; Veno-occlusive; Diseases; MRI; Mesalazine

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INTRODUCTION

Hepatic sinusoidal obstruction syndrome(HSOS) was previously named as hepatic veno-occlusive disease, but however it was discovered that HSOS was appeared due to the injury of the centrilobuler endothelium of the liver (zone 3 liver parenchyma). [1-5] In the etiology of HSOS; Hematopoetic transplantations, chemotherapy, alchol, toxins, radiotherapy, liver transplantations and etc. were believed to be responsible. [2-4]

We report a rare case of hepatic sinusoidal obstruction syndrome of a 30 years old female patient with Ulcerative colitis (UC).

CASE REPORT

30 years old female was diagnosed as UC in an outer center in 2014 and mesalazine was started as the first step-treatment. In 2015/10, she was admitted to the emergency unit with cram- ping abdominal pain and with a pancreatitis attack.

She was referred to the Radiology unit, Magnetic Resonance Cholangiopancreatography (MRCP) was normal, in CT; A diffuse mini-mally increased pancreatic dimensions were observed.

Colonoscopy was performed after Radiological procedures and up to the mid-part of Transverse colon, whole colon segments were analysed in the Colonoscopy. Ulcerations with exudas, granuler avascular mucosal paterns were observed and rectal biopsies were also taken. In the hystopathology, surface epithelium and cryptic patern was protected with a little decrease in goblet mucus cells. No cryptitis, crypt abcess, or chronic inflammations were dis- covered.

Steroid was applied to the patient and after remission, mesatazine (Pentasa) was started. Pentasa was relieved after 2 months due to a violent UC attack, IV Ferrum was acquired in this attack and then mesalazine was re-applied to the patient for cure. Steroids were stopped due to a myopathy and liver function tests were normal initially.

She used mesalazine for about 20 months continuously, in the routine laboratory control at May 2017; ALT/AST: 32/48, ALP/GGT: 178/188 and total bilirubine was 2.72. 2 months later, July 2017; ALT/AST: 27/41, ALP/GGT: 160/76 and total bilirubine was 2.92 (0.92 direct biln). WBC:3600, HB:13 g/dl, Platelet: 182.000. IG G4 and autoimmune markers were all negative.

In the Portal venous Doppler Ultrasonography (US); Splenomegaly (162 mm spleen lenght) and coarse granular echogenecity in liver parenchyma were observed. Portal venous diameter was normal with hepatopedal venous flow. No ascites in the abdomen was seen.

MRCP was performed due to the suspicion of sclerosing cholangitis, Gall bladder and extrahepatic biliary system were normally visualized, minimally dilated central intra-hepatic bile ducts were seenIn the T2W and T1W in and out of phase images, heterogeneous intensities in the liver parenchyma were demonstrated.

In the Dynamic Abdominal CT; Cranio-caudal length of spleen was 165 mm, portal venous diameter was 13 mm (Within normal range), dilated splenic vein at the hilum of spleen with 10 mm size, were visualized and calculated.Patchy, geographic lineated areas without any significant mass appereance, were shown in the liver at early and late venous phases (Figure 1).

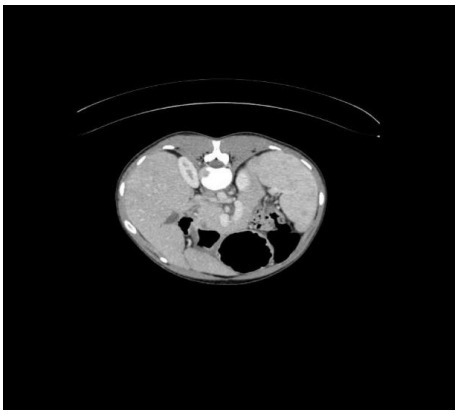

Figure 1: Delineated, nodular intensities in the right lobe of liver at venous phase of Abdominal CT, Axial image.

Dynamic Abdominal MRI was performed afterwards in order to specify these suspicious areas in the liver. In the Diffusion-weighted MR (DWI) images, there was patchy areas which presnted restriction of diffusion in the right lobe anterior and posterior segments. These areas revealed heterogeneous intensities with lower Apparent Diffusion coefficient (ADC) values in ADC mapping (Figure 2a-b).

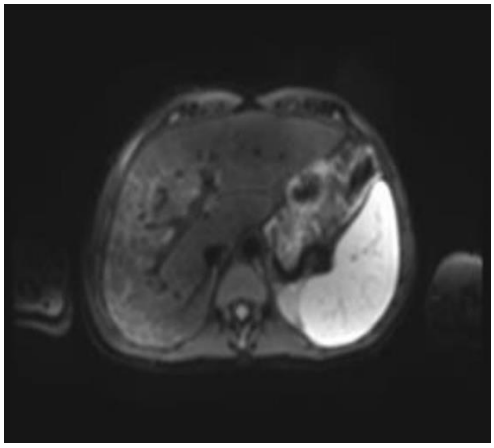

Figure 2a

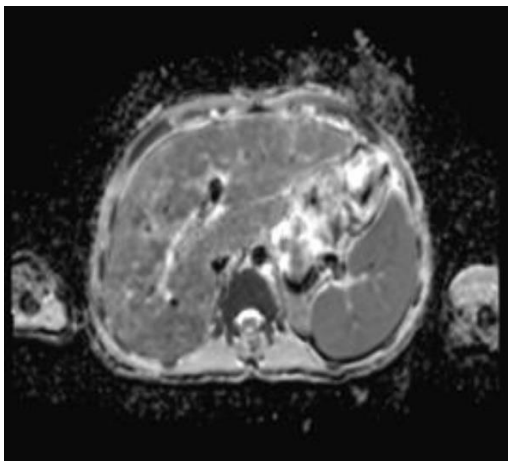

Figure 2a-b: Patchy geographic areas in the right lobe of liver, showing diffusion restrictions with low ADC values.

In T1W images, punctuated heterogeneous intensities and in T2W images, reticular patterns were visualized in the liver parenchyma (Figure 3).

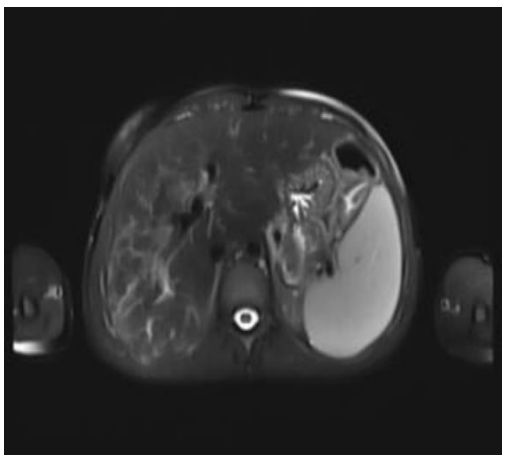

Figure 3: Heterogeneous reticular high intensity changes in the right lobe of liver at T2W axial image.

In the dynamic contrast-enhanced images; Patchy style contrast enhanced areas were demonstrated in the liver at late portal venous phase (Figure 4a-b).

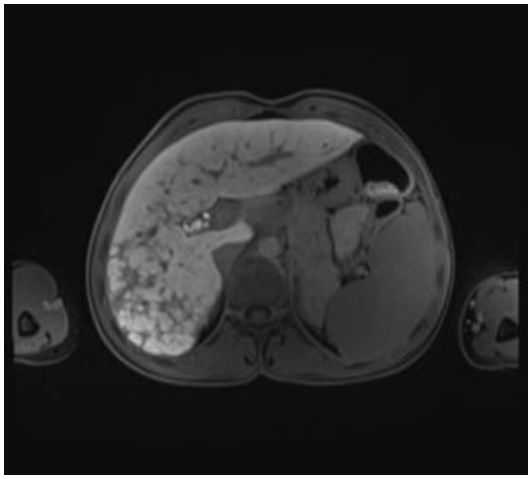

Figure 4a

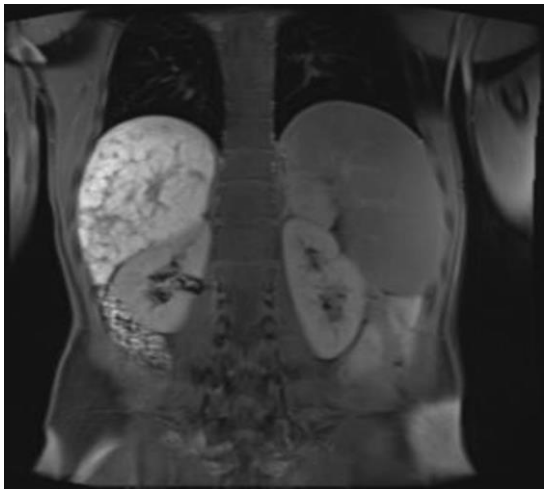

Figure 4a-b:Patchy, usually lineated clumping contrast enhancing areas of right liver lobe at venous phases, Axial and coronal Contrast enhanced MR images.

Non-specific ascites were observed in the Douglas space. All Radiological imaging results were consistent with HSOS?.

True-cut liver biopsy was performed to confirm the diagnosis and the result confirmed the diagnosis of HSOS. In the pathological examination of consecutive slices of biopsy material, Edge of liver parenchyma especially in zone 2 and 3 revealed sinusoidal dilatations, congestion and extravasations of RBC, at the same time atrophy of hepatocytes and regenerative nodular changes at the other parenchymal areas. Inflammation, loss and/or injury of biliary ducts were not observed. Mild bile accumulations in hepatocytes were shown.

In Histochemical analysis; Peri-sinusoidal fibrosis in the dilated sinusoidal areas were present with Masson trikom stain and portal fibrous dilatations were minimal.Nodular degenerative changes were observed wth reticulin stain and no iron deposition was predicted with prussian blue stain. No ductal metaplasia was shown in periportal hepatocytes by CK7 immunohistochemically, no plasma cell was seen with CD 138.

In this case, Extrahepatic cholestatic disease and chronic vascular pathologies were recommended to be evaluated via clinical and radiological findigs due to presence of bile stasis, sinusoidal dilatations and fibrosis, atrophy of hepatocytes.

In January 2018, mesalazine was stopped and up to now, she was under follow-up without any UC attack, without any recurrence and without any progression to the chronic liver disease.

DISCUSSION

HSOS was generally developed after hematopoietic cell transplantations, Chemotherapy against cancer, Radiotherapy especially after whole body radiation, organ transplantations, alchol and oral contraceptive use, radiation injuries. [2-4] Mortality due to HSOS, was ranged between 3-67% due to its severity, early diagnosis and effective therapy were very important to increase the survival rates. [5,6]

In the differential diagnosis of HSOS, Dynamic MRI had an essential role while both US and CT had limited aid in the exact diagnosis. [7-8] Patchy liver parenchymal enhancement was typical in the Dynamic MRI due to sinusoidal/venous obstructions, radiological imaging of HSOS varied with regard to the injury of sinusoidal epithelial cells. [8-11]

Zhoue et al. declared that severity of HSOS was strictly related to the prevelance of the patchy style enhanced liver parenchyma. [8] In the Budd-Chiari syndrome, similar contrast enhancement was revealed but Inferior vena cava and hepatic veins were patent in HSOS. [7-9]

Most colloborating finding to the HSOS, was declared as abdominal ascites in the literature. [8,10,11] We had only non-spesific fluid in the Douglas space in our case. Hepatomegaly was the second related finding, periportal cuffing and thickening of gall bladder wall could also accompany to the HSOS. [8-10] In our case, those findings were not present.

HSOS incidence was very rare due to mesalazine use in the inflammatory bowel syndrome, Holtman et al. predicted a HSOS case, progressed in a Crohn disease who was being treated by azathioprine. [12] Russmann et al. Stated a patient with UC who developed HSOS and hepatocellular carcinoma after azathioprine therapy. [13] In our case, HSOS was developed after mesalazine use for 20 months against UC but she was stable under follow-up care. Brancatelli et al. declared that radiological findings about sinusoidal dilatations could regress in 82+_68 days. [11] but in our patient, neither progression to chronic liver disease nor regression of hepatic findings were observed during 15 months follow-up, she was totally stable during this period without any drug administration.

In conclusion, we report a case of HSOS, developed in a 30 years old patient with Ulcerative colitis (UC) who was being treated by mesalazine about 20 months. Patchy style contrast enhancing areas were demonstrated in the liver at dynamic MRI, diagnosis was confirmed histopathologically and mesalazine use was withdrawn after that. She was stable in the follow-up without any recurrence or progression.

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