

Hepatic Encephalopathy in Patient with Lupus

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Citation: Mahdi AS, Nasra K. Al Adhoubi (2018) Hepatic Encephalopathy in Patient with Lupus. Ann Case Rep: ACRT-160. DOI: 10.29011/2574-7754/100060

Received Date: 14 February, 2018; **Accepted Date:** 05 March, 2018; **Published Date:** 15 March, 2018

Abstract

Liver dysfunction in the presence of Systemic Lupus Erythematosus (SLE) can be caused by many factors including drug-induced, SLE itself, fatty liver, Autoimmune Hepatitis (AIH), primary biliary cirrhosis, cholangitis, alcohol or viral hepatitis. However, Lupus hepatitis and autoimmune hepatitis are two distinct immunological conditions involving the liver, which can have similar clinical, laboratory and systemic presentations, leading to difficulties in diagnosis [1,2].

Keywords: Autoimmune Hepatitis, Hepatic Encephalopathy, SLE

Case Report

A 35 years old Omani lady who is known to have SLE with history of Lupus nephritis and pregnancy-induced hypertension maintained on Azathioprine and Hydroxychloroquine, presented to ER at Royal hospital (Muscat, Oman) with history of deep jaundice of the sclera and skin and loose motion not associated with nausea, vomiting or abdominal pain and cola like urine for 4 days' duration. Her blood pressure was 147/90 mmHg, heart rate: 90/min, respiratory rate: 19/min and oxygen saturation: 100% at room air. Her clinical examination was unremarkable.

Her initial workup revealed normal complete blood count and renal function test. She had raised Liver enzymes with ALT: 1676 [IU]/L (Normal 0-40), AST: 1713 [IU]/L (Normal 0-35), GGT: 59 [IU]/L (Normal 10-35), Alkaline phosphatase: 220 [IU]/L, Lactate dehydrogenase: 375 [IU]/L, Total bilirubin: 189 umol/L (Normal 0-20) and Conjugated bilirubin: 278 umol/L (Normal 0-8), Prothrombin time: 20.8s (normal 9-11.6), Activated partial thromboplastin time: 70s (Normal 27-39), fibrinogen: 1.60 g/L (Normal 1.5-4.2), Thrombin time : 21.70s (Normal 12-16). Her ESR and CRP were normal. Her viral screening showed a negative viral Hepatitis, CMV and EBV serology and PCR. Her complements C₃: 47 mg/L (Normal 150-530), C₄: 47 mg/L (Normal 820-1930). Her antimitochondrial antibodies and lupus anticoagulant were positive. B₂ glycoprotein, Anticardiolipin antibodies, immunoglobulins, anti-liver/Kidney Microsome

antibodies and anti-smooth muscle antibodies were negative as well. Alfa-1 Antitrypsin and Ceruloplasmin were negative.

Repeated anti dsDNA was Positive. Her liver MRI showed no evidence of hepatic vein thrombosis with no liver or biliary abnormalities apart from acute hepatitis picture. She declined liver biopsy. The patient was also taking curcumin daily as part of traditional therapy and she was advised to stop it. She was managed with high dose of Prednisolone and all other medications were hold. After few days she developed signs of hepatic encephalopathy with confusion and flapping tremor, ammonia level of 176 umol/L (normal 18-70), for which she was managed with (Furosemide, Spironolactone and Lactulose). A liver transplant was arranged for her and as she was pulsed with methylprednisolone infusion for 3 days. Following the pulse, her clinical condition had improved remarkably and her liver enzymes went back to normal on subsequent follow-up in the clinic. Her repeated liver ultrasound showed some evidence of cirrhosis on one-year follow-up. But she remained asymptomatic with normal liver function tests after 5 years' follow-up. She was maintained on mycophenolate to control her disease.

Discussion

Liver dysfunction has been documented in 59.7% of SLE patients, up to 30.9% caused by drugs and 28.5% caused by SLE itself. It tends to be mild except in autoimmune hepatitis which is relatively severe [2]. Immunosuppressive medications including Methotrexate, Hydroxychloroquine and Azathioprine were all reported to cause hepatic dysfunction.

Historically, when hepatic abnormalities were the most prominent feature during the first visit, the patient was more likely to receive an incorrect diagnosis or be diagnosed with SLE late. Only 46.7% were identified as SLE within a week of presentation of an abnormal liver function while 46.7% patients were not correctly diagnosed until more than 2 weeks to 4 months [3]. Usually, complications of portal hypertension, cirrhosis, and hepatic encephalopathy are rare manifestations of SLE unless a coexistent liver disease is present [4].

Reports of 6 SLE cases by Beissel C. et al showed that all patients had coexisting of autoimmune hepatitis in their medical history. Remission of acute hepatitis was achieved in all patients after the initiation of immunosuppressive therapy [5].

Lupus hepatitis has been documented as one of the indices indicating SLE activity. Positive intense deposit of complement Iq in the liver may be a characteristic immunopathological feature of lupus hepatitis. A low serum alanine transaminase levels at diagnosis and high doses of prednisone were associated to the resolution of Lupus Hepatitis [6,7]. A study done by Khalifa et.al showed that one out of 12 patients with liver abnormalities died due to hepatic encephalopathy [8].

An autoimmune liver disease may occur during the course of SLE. Due to biochemical similarities between AIH and SLE, AIH could be considered very probable by using both International Autoimmune Hepatitis Group (IAIHG) scoring system and simplified criteria [9].

Curcumin has shown anti-inflammatory, anti-oxidant, antifungal, antibacterial and anticancer activities. Initiation of curcumin as treatment caused hepatic recovery within 2 weeks in hepatic injury induced by CCl₄ and apoptosis. [10,11]. This may explain partly, as a contributing factor, the patient clinical deterioration following the withdrawing of curcumin. But the progression of the disease remains the main explanation confirmed by the good response to the pulse of methylprednisolone. We are in need of large-scale, well-organized studies in order to identify the rule of this plant product and its anti-inflammatory effects, before drowning any scientific conclusion.

Conclusion

Diagnosing liver disease in SLE patients is always challenging, a definite diagnosis should be confirmed by patient

history and clinical examination, blood investigations, and liver biopsy. Drug-induced hepatic dysfunction remains the commonest cause but other differentials like Lupus hepatitis and autoimmune hepatitis should always be kept in mind.

References

1. Adiga A, and Nugent K. Lupus (2017) Hepatitis and A autoimmune Hepatitis (Lipoid Hepatitis). Review article. Am J Med Sci. 353: 329-335.
2. Takahashi A, Abe K, Saito R, Iwadata H, Okai Ket al. (2013) Liver dysfunction in patients with systemic lupus erythematosus. InternMed.52: 1461-1465.
3. Gao Y, Li W, Meng QH, Liu HX, Zhu YK (2016) Analysis of 15 patients with abnormal liver function as the first systemic lupus erythematosus symptom. ActaGastroenterology Belg.79: 441-446.
4. Chowdhary VR, Crowson CS, Poterucha JJ, Moder KG (2008) Liver involvement in systemic lupus erythematosus: case review of 40 patients.J Rheumatol.35: 2159-64.
5. Beisel C, Weiler-Normann C, Teufel A, Lohse AW (2014) Association of autoimmune hepatitis and systemic lupus erythematosus: a case series and review of the literature.Reviewarticle. World J Gastroenterol.20: 12662-12667.
6. Zheng RH, Wang JH, Wang SB, Chen J, Guan WM, et al. (2013) Clinical and immunopathological features of patients with lupus hepatitis. Chin Med J (Engl). 126:260-266.
7. Piga M, Vacca A, Porru G, Cauli A, Mathieu A (2010) Liver involvement in systemic lupus erythematosus: incidence, clinical course and outcome of lupus hepatitis.ClinExpRheumatol. 28:504-510.
8. Khalifa M, Benjazia E, Rezgui A, Ghannouchi N, Alaoua A, et al. (2011) Lupus hepatitis: a case series of 12 patients.Rev Med Interne. 32: 347-349.
9. Efe C, Purnak T, Ozaslan E, Ozbalkan Z, Karaaslan Y, Altiparmak E, et al. (2011) Autoimmune liver disease in patients with systemic lupus erythematosus: a retrospective analysis of 147 cases. Scand J Gastroenterol.46: 732-737.
10. Rivera-Espinoza Y and Muriel P (2009) Pharmacological actions of curcumin in liver diseases or damage.Reviewarticle. Liver Int. 29:1457-1466.
11. Priya S and Sudhakaran PR (2008) Curcumin-induced recovery from hepatic injury involves induction of apoptosis of activated hepatic stellate cells. Indian J BiochemBiophys.45: 317-325.