Journal for Research in Applied Sciences and Biotechnology

www.jrasb.com

ISSN: 2583-4053

Volume-3 Issue-3 || June 2024 || PP. 9-12

https://doi.org/10.55544/jrasb.3.3.2

Acute Presentation of Autoimmune Hepatitis: Case Report

Jhumur Ghosh[1] and Jaya Ghosh[2]

[1]Associate Professor, Department of Hepatology, MH Samorita Hospital and Medical College, Tejgaon, Dhaka-1208, BANGLADESH.

[2]Clinical Trial Coordinator, Radiation Oncology Department, Tata Medical Center, Newtown, Rajarhat, Kolkata, INDIA.

[1]Corresponding Author: ghosh95jaya@gmail.com

ORCiD

https://orcid.org/0009-0001-4449-0705

www.jrasb.com || Vol. 3 No. 3 (2024): June Issue

Received: 14-05-2024	**Revised:** 19-05-2024	**Accepted:** 23-05-2024

ABSTRACT

This case report describes the clinical presentation, diagnostic evaluation, and treatment outcome of a 38-year-old female patient presenting with yellow discoloration of eyes and urine, along with associated symptoms such as nausea, poor appetite, abdominal pain, extreme fatigue, and mild joint pain. The patient had a history of amenorrhea for three months and no family history of autoimmune disease or drug-induced liver injury. Upon examination, the patient exhibited deep icterus and mild tender hepatomegaly, but without signs of acute liver failure. Laboratory investigations revealed elevated liver function markers and positive autoimmune antibodies, including antinuclear antibody (ANA) and anti-smooth muscle antibody (ASMA), while ruling out other possible etiologies such as viral hepatitis, Wilson's disease, and hemochromatosis. Imaging studies showed features of acute hepatitis, and liver biopsy could not be performed due to prolonged prothrombin time. The patient was diagnosed with probable autoimmune hepatitis (AIH) and initiated on a treatment regimen consisting of prednisolone 40 mg daily, which was gradually tapered over time and added azathioprine 100 mg daily. The patient demonstrated significant improvement in liver function tests with this treatment approach. However, she discontinued treatment on her own accord but continued to have normal liver function during subsequent follow-up visits. During the consultation, the patient and healthcare provider engaged in a comprehensive discussion concerning the significance of sustained treatment over an extended period and the inherent possibility of relapse.

Keywords- Autoimmune hepatitis, Prednisolone, Azathioprine, Autoantibody.

I. INTRODUCTION

Autoimmune hepatitis (AIH) stands as a persistent liver disorder distinguished by immune-driven inflammation and damage to hepatocytes, the primary cells of the liver. It predominantly affects women and is associated with various clinical manifestations, ranging from asymptomatic elevation of liver enzymes to acute liver failure[1]. Timely identification and prompt initiation of immunosuppressive treatment play a pivotal role in effectively managing autoimmune hepatitis and averting the progression of liver damage. This particular case report emphasizes the patient's clinical manifestation, diagnostic assessment, and treatment outcome, shedding light on the intricacies of probable autoimmune hepatitis.

II. CASE PRESENTATION

A 38-year-old female presented with yellow discoloration of the eyes and urine, accompanied by symptoms such as nausea, poor appetite, abdominal pain, extreme fatigue, and mild joint pain persisting for 15 days. The patient had a history of amenorrhea for the past three months but denied any family history of

ISSN: 2583-4053

Volume-3 Issue-3 || June 2024 || PP. 9-12

https://doi.org/10.55544/jrasb.3.3.2

autoimmune disease or exposure to drugs known to cause liver injury. Upon physical examination, notable findings included profound jaundice and slight tender hepatomegaly. Laboratory tests unveiled elevated levels of bilirubin (24.1 mg/dl), liver enzymes (SGPT-1223 U/L, SGOT-853 U/L), and prolonged prothrombin time (26 second, INR-2.2), emphasizing the presence of liver dysfunction and highlighting the significance of these diagnostic markers. The patient's serum albumin was decreased, and serum IgG level was elevated (25.8 gm/L). Screening for hepatotropic viral infections yielded negative results. Autoimmune profile analysis demonstrated positive ANA and ASMA, while other autoantibodies associated with autoimmune hepatitis were negative. Imaging studies revealed features of acute hepatitis with no intrahepatic or extrahepatic biliary obstruction, and liver biopsy was precluded due to the patient's prolonged prothrombin time.

Based on the clinical presentation, laboratory findings, and exclusion of alternative etiologies, the patient was diagnosed with probable autoimmune hepatitis. Treatment was initiated with prednisolone 40 mg daily in June 2022, and the dosage was gradually tapered every two weeks. Once prednisolone reached a dose of 10 mg, azathioprine 100 mg was added to the treatment regimen. The patient also received insulin for her pre-existing diabetes mellitus, along with supportive care. Liver function tests normalized with this treatment approach, and the patient was advised to continue therapy for at least 24 months after achieving normal liver function. However, the patient discontinued treatment on her own accord in February 2023. During subsequent follow-up visits, conducted at three-month intervals, liver function tests remained within normal.

Table 1: Blood and Imaging investigations of patient.

Investigation	**18/06/2022**	**07/09/2022**	**22/02/223**	**15/6/2023**
Hb (gm/dl)	15	14	14.2	13.0
TC (/cmm)	11000	10360	7640	6500
N (%)	67	64	54	67
PC (/cmm)	250000	213000	172000	234000
PBF	Non-specific	-	-	-
SGPT (U/L)	1223	125	22	20
SGOT (U/L)	856	28	30	18
ALP (U/L)	179	127	78	69
GGT (U/L)	164	-	56	-
S.Bilirubin (mg/dl)	25.1	2.4	1.1	0.8
Direct (mg/dl)	10.3	-	-	-
Indirect (mg/dl)	14.8	-	-	-
PT(Second)	26.0	16	13	12
INR	2.27	1.4	1.1	1.0
S. Albumin (gm/l)	34	38	36	37
Creatinine (mg/dl)	0.8	0.7	0.6	0.8
FBS (mmol/l)	17.2	5.2	4.9	4.5
2HABF(mmol/l)	22.3	13.5	5.8	5.8
HbA1c (%)	8.8	7.2	6.5	6.2
S. Sodium (mmol/l)	141	143	-	-
S. Potasium (mmol/l)	3.9	4.2	-	-
S. lipase (U/L)	73	-	-	-
CRP (mg/l)	6.6	-	-	-
TSH (uIU/ml)	0.66	-	-	-
S. Copper (ug/dl)	39.75	-	-	-
S. Ceruloplasmin (mg/dl)	25	-	-	-
S. Ferritin (ng/ml)	438	-	-	-
S. IgG (N-6.0-16.0 gm/L)	25.8	-	-	-
USG W/A	Acute hepatitis	Normal	-	Normal
MRCP	No IH & EH biliary obstruction	-	-	-

Table 2: Autoimmune profile of patient.

Autoantibody	**Interpretations**
Anti-nuclear antibody (ANA)	Positive (Fine speckled)
Anti-smooth muscle antibody (ASMA)	Positive
Anti-ds-DNA	Negative

ISSN: 2583-4053

Volume-3 Issue-3 || June 2024 || PP. 9-12

https://doi.org/10.55544/jrasb.3.3.2

Anti mitochondrial antibody (AMA-M2)	Negative
Speckled Protein 100 (SP 100)	Negative
Liver Kidney microsomal antibody type1(LKM1)	Negative
Glycoprotein-210 (gp 210)	Negative
Liver Cytosolic antigen type-1 (LC1)	Negative
Soluble liver antigen (SLA)	Negative

Table 3: Viral profile of patient.

Viral profile	Comments
AntiHAVIgM	Negative
AntiHEVIgM	Negative
AntiHBcIgM	Negative
HBsAg	Negative
AntiHBcTotal	Negative
AntiHCV	Negative

III. DISCUSSION

AIH is diagnosed through the identification of abnormal histological features (interface hepatitis), clinical and laboratory findings (elevated AST and ALT levels), elevated serum IgG concentration, and the presence of characteristic autoantibodies[2]. There is no specific diagnostic marker for AIH, so diagnosis relies on the presence of distinctive features and the exclusion of other similar conditions such as viral hepatitis, drug-induced liver injury, Wilson's disease, and hereditary hemochromatosis[3].

AIH is classified into two types based on specific autoantibodies. Type 1 is characterized by the presence of antinuclear antibodies (ANA) and/or smooth muscle antibodies (SMA)/anti-actin antibodies. Type 2 is identified by the presence of antibodies to liver kidney microsome type 1 (anti-LKM1), usually without ANA and SMA. Up to 20% of AIH patients do not exhibit ANA, SMA, or LKM1 autoantibodies, despite showing other characteristic features. This condition is known as seronegative AIH[4]. A liver biopsy with compatible histological findings is necessary for confirming the diagnosis of AIH. The histological hallmark is interface hepatitis, often accompanied by plasma cell infiltration (in 66% of cases) and lobular hepatitis (in 47% of cases). Centrilobular necrosis can be found in 29% of patients, with or without cirrhosis. Emperiopolesis is observed in 65% of patients, and hepatocyte rosettes are present in 33% of AIH patients[5].

AIH can present as asymptomatic in approximately 25% - 34% of patients and can have an acute onset (duration less than 30 days) in 25% - 75% of patients[6]. Most individuals with AIH experience chronic nonspecific symptoms such as fatigue, malaise, arthralgia, or amenorrhea[7]. Fatigue is the primary complaint in 85% of patients, and jaundice may be present. Concurrent autoimmune diseases are found in 14% - 44% of AIH patients. Type 1 AIH is commonly associated with autoimmune thyroid disease (10% - 18%), while type 2 AIH is linked to type 1 diabetes, autoimmune thyroid disease, and autoimmune skin disease. Extrahepatic autoimmune diseases are more frequent in women, and the specific types vary among different age groups[7].

Prednisolone is the recommended initial treatment for AIH, followed by the addition of azathioprine after two weeks. The starting dose of prednisolone should range between 0.5 and 1 mg/kg/day. Once the bilirubin levels drop below 6 mg/dl, it is advisable to commence the administration of azathioprine, approximately two weeks after the initiation of steroid therapy[8]. The initial daily dosage of azathioprine ought to be 50 mg, with subsequent adjustments based on the patient's response and the presence of any adverse effects. The ultimate objective is to achieve a maintenance dose of 1-2 mg/kg, while closely monitoring for any signs of toxicity. The duration of treatment should be a minimum of three years or extending for at least 24 months beyond the point of complete normalization of serum transaminase and IgG levels. Considering the high likelihood of relapse (50% - 90%) upon discontinuation of medication, particularly within the initial 12 months post-treatment cessation, prolonged therapy may be contemplated[8]. This particular case report highlights the significance of promptly identifying, correctly diagnosing, and effectively managing of autoimmune hepatitis.

IV. CONCLUSION

The importance of educating patients about the importance of long-term treatment and the possibility of relapse in autoimmune hepatitis is emphasized. It is crucial for patients to understand the significance of regular medical follow-ups and adherence to their prescribed treatment plan in order to prevent the progression of the disease and improve long-term outcomes.

Conflict of interest

Authors have declared no conflict of interest.

ISSN: 2583-4053

Volume-3 Issue-3 || June 2024 || PP. 9-12

https://doi.org/10.55544/jrasb.3.3.2

Acknowledgement

We are very grateful to our patient for giving informed consent to publish this manuscript.

REFERENCES

[1] Czaja AJ. Diagnosis and Management of Autoimmune Hepatitis: Current Status and Future Directions. Gut Liver. 2016 Mar;10(2):177-203. doi: 10.5009/gnl15352. PMID: 26934884; PMCID: PMC4780448.

[2] Manns MP, Czaja AJ, Gorham JD, Krawitt EL, Mieli-Vergani G, Vergani D, et al. Diagnosis and management of autoimmune hepatitis. Hepatology 2010; 51: 2193- 2213.

[3] Alvarez F, Berg PA, Bianchi FB, Bianchi L, Burroughs AK, Cancado EL, et al. International autoimmune hepatitis group report: review of criteria for diagnosis of autoimmune hepatitis. J Hepatol 1999; 31: 929- 938.

[4] Czaja AJ, Manns MP. The validity and importance of subtypes in autoimmune hepatitis: a point of view. Am J Gastroenterol 1995; 90: 1206- 1211.

[5] Balitzer D, Shafizadeh N, Peters MG, Ferrell LD, Alshak N, Kakar S. Autoimmune hepatitis: review of histologic features included in the simplified criteria proposed by the international autoimmune hepatitis group and proposal for new histologic criteria. Mod Pathol 2017; 30: 773- 783.

[6] Kogan J, Safadi R, Ashur Y, Shouval D, Ilan Y. Prognosis of symptomatic versus asymptomatic autoimmune hepatitis: a study of 68 patients. J Clin Gastroenterol 2002; 35: 75- 81.

[7] Bittencourt PL, Farias AQ, Porta G, Cancado EL, Miura I, Pugliese R, et al. Frequency of concurrent autoimmune disorders in patients with autoimmune hepatitis: effect of age, gender, and genetic background. J Clin Gastroenterol 2008; 42: 300- 305.

[8] European Association for the Study of the Liver. EASL Clinical Practice Guidelines: Autoimmune hepatitis. Journal of Hepatology. 2015;63:971-1004. doi:10.1016/j.jhep.2015.06.030.