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A CASE REPORT ON AUTOIMMUNE HEPATITIS

Jahnavi G.*[1], H. P. Sahana[2], Sumangala V.[3] and Dr. RLN Murthy[4]

[1,2]Interns, Pharm D, TVM College of Pharmacy, Ballari.

[3]Vth Pharm D Student, TVM College of Pharmacy, Ballari.

[4]Assistant Professor, TVM College of Pharmacy, Ballari.

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***Corresponding Author**

Jahnavi G.

Interns, Pharm D, TVM College of Pharmacy, Ballari.

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ABSTRACT

Introduction: Autoimmune hepatitis is defined as rare disease of unknown etiology characterized by un-resolving inflammation of the liver with presence of interface hepatitis, hyper gamma globulinemia and auto antibodies. AIH age spectrum is broad, from small infants to adults>60 years old, it has been described in both sexes (more common in women), all races, and ethnicities. **Case Presentation:** A 29 years women admitted to the female medical ward of general medicine in tertiary care hospital, with the chief complaints of blackish discoloration of stools since one day which was foul smelly and watery and bleeding from nose one episode since the day of morning. Her past history revealed that she was a known case of autoimmune hepatitis-chronic liver disease on regular medication. On examination her BP was 80/50mm of Hg, Pulse rate was 90bpm and SPO2 was 95%@RA. External examination revealed the presence of icterus, hence physician advised for tests like Complete blood count, Liver function tests, Kidney function tests, Biochemistry, Serum electrolytes, Retic count, Peripheral smear only, Vitamin B12, Serum folic acid, HCV & HBS AG- Rapid, HIV I & II, Abdomen-Pelvic Sonography, MRI CHOLANGIOPANCREATICOGRAPHY, liver-LIA for IgG antibodies, LDH test, Upper GI Endoscopy, Sputum culture and sensitivity test, Ceruloplasmin, Immunoglobulin IgG serum test, Copper- 24hours urine test, Autoimmune serology panel, Anti-Nuclear Antibody test by Indirect Immunofluorescence Assay (IFA), ANA(Anti-Nuclear Antibody) Pattern Interpretation panel for the further diagnosis. **Conclusion:** Autoimmune hepatitis is a immunosuppressed disease and more likely to be affected to the patients of liver

disease associated with immunodeficiency. Early diagnosis and preventive measures should be undertaken to prevent the development of disease.

KEYWORDS: Human immunodeficiency virus (HIV), Lactate dehydrogenase (LDH), Hepatitis c virus (HCV), Antinuclear antibody (ANA).

INTRODUCTION

Autoimmune hepatitis is defined as a rare disease of unknown etiology characterized by unresolving inflammation of the liver, the presence of interface hepatitis, hyper gamma globulinemia and auto antibodies.[1] AIH age spectrum is broad, from small infants to adults over 60 years old, it has been detected in both sex(more common in women),all races, and ethnicities.[2] Origin of this disease is presumed to be a loss of immunologic tolerance against hepatocytes induced by environmental factors in genetically predisposed people, possibly through “molecular mimicry”.

Clinical manifestations for autoimmune hepatitis are expressed in three ways: (a) Acute onset, insidious onset, asymptomatic onset. Among these, acute onset is most frequent pattern around worldwide including children and adults. This acute onset is present with transaminase concentrations at least 5 to 10 times the upper limit of normal, often with jaundice and sometimes with prolonged international normalized ratio. (b) An insidious onset is characterized by non specific symptoms such as fatigue, arthralgias, malaise, amenorrhea. (c) In asymptomatic onset: The patient does not present liver related signs and symptoms and is assessed when altered liver function tests have emerged accidentally or when other medical conditions are being investigated, particularly extrahepatic autoimmune disorders such as thyroid disease, celiac disease and rheumatologic conditions.[3]

Studies on the natural progression of autoimmune hepatitis prior to the advent of autoimmune suppressive therapy indicate that up to 40% of individuals with severe AIH may succumb to the disease within 6 months of diagnosis if no treatment is provided. According to published research, autoimmune hepatitis occurs at an annual incidence rate of approximately 1 to 2 cases per 100000 people, with a prevalence ranging from 10- 20 cases per 100000 individual.[4] Earlier, This disease was known as lupoid hepatitis, plasma cell hepatitis or autoimmune chronic active hepatitis. On the basis of serum autoantibodies, AIH can be classified into three subtypes, they are

1. Type 1 autoimmune hepatitis(positive antibody): it is characterized by the presence of antinuclear antibody(ANA), anti-smooth muscle antibody(SMA), or both and constitutes 80% of AIH cases.
2. Type 2 autoimmune hepatitis (positive antibodies): it is characterized by the presence of anti-liver kidney microsomal(LKM) 1 and /or anti-LKM3 and /or anti-liver cytosol 1(LC1) antibodies.
3. Autoantibody negative autoimmune hepatitis: lack positive ANA, ASMA, LKM-1 etc, antibody panels but presents with clinical features of autoimmune hepatitis that resolve with standard treatment.[5]

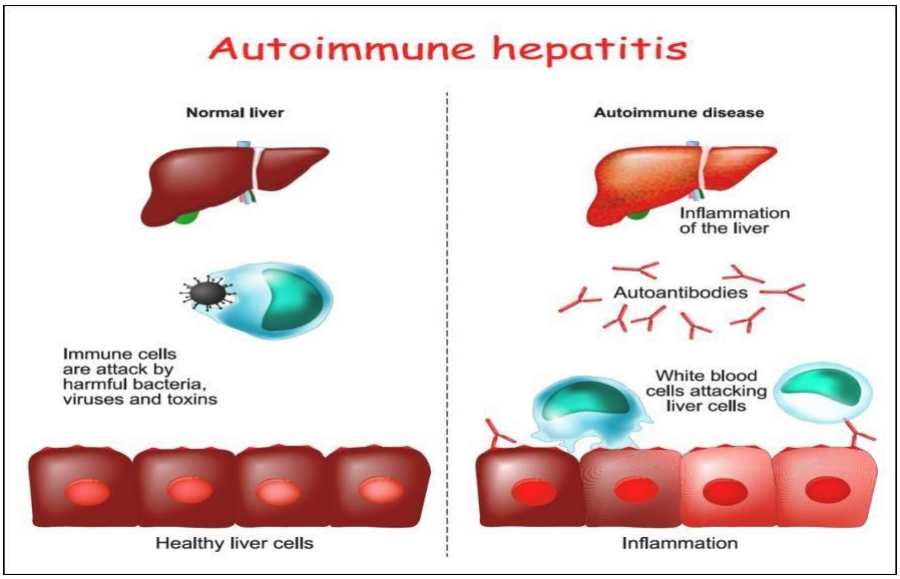

Figure 1: This picture shows difference between normal liver and autoimmune hepatitis liver.

The internal autoimmune hepatitis group created a standardized scoring system to aid in clinical diagnosis for population based studies; however, it has limited usefulness for diagnosing individual cases. A more streamlined scoring system method was later introduced for clinical practice, which considers autoantibody titers, total IgG concentration, liver histology, and the exclusion of viral hepatitis. Diagnosis of autoimmune hepatitis is best achieved with a combination of clinical, laboratory and histological findings after excluding other etiological factors(viral, metabolic). Requirements for histological examination necessitates a liver biopsy, typically performed with a needle by the percutaneous route, to provide liver tissue.[6] The Cornerstone of autoimmune hepatitis treatment is corticosteroids, either used alone or in combination with azathioprine. However, clinical experience has led to the exploration of other therapeutic options, such as calcineurin inhibitors, mycophenolate

mofetil and budesonide, which have shown potential as both initial and rescue treatments in certain patient populations.[7] Complications of autoimmune hepatitis are hyper viscosity syndrome, hepatocellular carcinoma etc.[8]

CASE REPORT

A 29 years women admitted to the female medical ward of general medicine in tertiary care hospital, with the chief complaints of blackish discoloration of stools since one day which was foul smelly and watery and bleeding from nose one episode since the day of morning. Her past history revealed that she was a known case of autoimmune hepatitis-chronic liver disease on regular medication. On examination her BP was 80/50mm of Hg, Pulse rate was 90bpm and SPO_2 was 95%@RA. External examination revealed the presence of icterus, hence physician advised for tests like Complete blood count, Liver function tests, Kidney function tests, Biochemistry, Serum electrolytes, Retic count, Peripheral smear only, Vitamin B12, Serum folic acid, HCV & HBS AG- Rapid, HIV I & II, Abdomen - Pelvic Sonography, MRI CHOLANGIOPANCREATICOGRAPHY, liver-LIA for IgG antibodies, LDH test, Upper GI Endoscopy, Sputum culture and sensitivity test, Ceruloplasmin, Immunoglobulin IgG serum test, Copper- 24hours urine test, Autoimmune serology panel, Anti-Nuclear Antibody test by Indirect Immunofluorescence Assay (IFA), ANA(Anti-Nuclear Antibody) Pattern Interpretation panel for the further diagnosis.

Table 1: Laboratory Parameters.

SL. NO	TESTS	PARAMETERS	RESULTS ON ADMISSION	REFERENCE RANGE
1.	COMPLETE BLOOD COUNT	Hemoglobin	**7.0**	12.5-16gm%
		Total WBC Count	**12280**	4000-11000 cells/cumm
		Red blood cells	**2.71**	4.5- 5.5million/cumm
		Platelets	**1.04**	1.5- 4.5lakh/cumm
		Neutrophils	**85**	40-70%
		Packed cell volume	**22.7**	35-46%
		Lymphocytes	**10**	20-40%
		Mean platelet volume	**11.4**	7-11 fL

		Mean corpuscular hemoglobin	**25.9**	27-34 pg
		Mean corpuscular hemoglobin concentration	**30.9**	31-36 %
		RDW-CV	**18.7**	11.5-14.5 %
		PDW-CV	**18.5**	10-18%
		ESR	**22**	0-5 mm/hr
2.	LIVER FUNCTION TESTS	Albumin	**2.0**	3.2-5.4g/dl
		Globulin	**3.7**	2.5-3g/dl
		A/G ratio	**0.5**	1.2-1.5
		Total bilirubin	**19.8**	0.2-1.2mg/dl
		Total protein	**5.7**	5-8.3 g/dl
		Conjugated bilirubin	**11.7**	0.1-0.4mg/dl
		Unconjugated bilirubin	**8.1**	0.2-0.7mg/dl
		Aspartate transaminase	**101**	0-40 IU/L
		Alkaline phosphate	**1297**	20-140U/L
3.	RENAL FUNCTION TESTS	Serum urea	**50**	15-45 mg/dl
4.	SERUM ELECTROLYTES	Sodium	**138**	136-146mEq/l
		Potassium	**3.0**	3.48-5mEq/l
		Chloride	**105**	96-106mEq/l
5.	BIOCHEMISTRY	Random blood sugar	**60**	70-140 mg/dl

- HBSAG Rapid – Negative
- HCV - Negative
- HIV I & II - Negative
- Abdomen-pelvic sonography – Diffusion liver disease, Splenomegaly.
- MRI CHOLANGIO-PANCREATICOGRAPHY – Marked splenomegaly. Mild hepatomegaly.
- Upper GI Endoscopy - Esophageal candidiasis : Non erosive gastritis : mild PHG (Portal hypertensive gastropathy).

- Sputum culture and sensitivity test - Gram stain : 10-12 pus cells/LPF and 6-8 Epithelial cells/LPF seen.

OCCASIONAL GRAM NEGATIVE BACILLI SEEN.

Sputum Culture and antibiotic sensitivity report:

Organism isolated: (Pseudomonas aeruginosa).

- Resistant to: Ceftriaxone
  Doxycycline
  Ampicillin
  Tetracycline

- LDH - 725 U/L (225-450 U/L)
- Retic count – 2% (0.2-2%)
- Vitamin B12 – 267pg/ml (211-911pg/ml)
- Serum folic acid – 1276 (2.5-20 ng/ml)
- Peripheral smear only - Dimorphic anemia
- DIRECT COOMB'S TEST (Gel method) - POSITIVE
- Serum ferritin - >2000 (11-307 ng/mL)
- CRP (QUANTITATIVE) - 55.1mg/L (0-6 mg/L)
- INR - 1.1 (0.8-1.1)
- APTT - 41.7 seconds (30-40 seconds)
- PT - 13.4 seconds (11-16 seconds)
- Liver-LIA (Line Immunoassay) - 1.00 (Negative)
- CERULOPLASMIN, SERUM - 21.90 mg/dL (20-60 mg/dL)
- IMMUNOGLOBULIN IgG, SERUM - 2057.40 mg/dL (700-1600 mg/dl)
- COPPER, 24-HOUR URINE (ICPMS) -

Copper, 24-hour urine: 31.55 ug/L (2-80 ug/L)

Copper, 24-hour urine: 47.33 ug/day (3-50 ug/day)

Total urine volume: 1500 mL/day (600-1600 mL/day)

- Autoimmune Serology Panel - dsDNA (Double-stranded DNA Antibody) : Negative

Nucleosomes: Positive

Histones: Equivocal

Other autoantibodies: Negative

- Anti-Nuclear Antibody (ANA) Test by Indirect Immunofluorescence Assay (IFA)

Observed value: Weak Positive

Pattern: Spindle Fiber and cytoplasmic speckled

Grade: +

Estimated Titer: 1:100

Medical Remarks: Suggested ANA Profile (A0435) to rule out NuMA- associated autoimmunity.

- ANA (Antinuclear Antibody test) -
- Homogeneous pattern → Systemic Lupus Erythematosus (SLE), Rheumatoid Arthritis (RA)
- Speckled pattern → SLE, Sjo gren's syndrome, Mixed Connective Tissue Disease (MCTD)
- Dense Fine Speckled (DFS) → Usually seen in healthy individuals but also in some autoimmune conditions
- Nucleolar pattern → Systemic Sclerosis (Scleroderma), Polymyositis
- Cytoplasmic speckled → Autoimmune Hepatitis, Myositis

Based on the External and laboratory examinations, final diagnosis was confirmed as **AUTOIMMUNE HEPATITIS** presented with Malena and GI bleed.

Table 2: Treatment Chart.

SL. NO	NAME OF THE DOSE	MEDICATIONS	ROUTE	FREQUENCY	DURATION
1.	Inj. Ceftazidime	1gm	IV	1-0-1	D1-D10
2.	Inj. Pantoprazole	40mg	IV	1-0-0	D1-D10
3.	Inj. Vitamin-K	10mg	IV	1-0-0	D1-D3
4.	Inj. Tranexamic acid	500mg	IV	1-0-1	D1-D10
5.	Tab. Ursodeoxycholic acid	300mg	PO	1-0-1	D1-D4
6.	Syrup. Lactulose	10ml	PO	1-1-1	D1-D10
7.	Inj. 25% dextrose	100ml	IV	1-1-1	D1-D10
8.	1Pint PRBC		IV		D3
9.	Inj. Levetiracetam	500mg	IV	1-0-1	D1-D10
10.	Tab. Prednisolone	16mg	PO	1-0-1	D1
11.	Inj. Multivitamin + NS	100ml NS	IV		D4-D5
12.	Inj. Noradrenaline in 100ml NS		IV	1-0-1	D9-D10

Blood transfusion of packed red cells on day 3 of admission to normalize the decreased hemoglobin and red blood cells.

The patient was treated with above medications.

Table 3: DISCHARGE MEDICATION.

SL. NO	NAME OF THE DOSE	MEDICATION	ROUTE	FREQUENCY
1.	Tab. Urso deoxy cholic acid	300mg	PO	1-0-1
2.	Tab. Fluconazole	150mg	PO	1-0-0 ~1week
3.	Tab. Levofloxacin	750mg	PO	1-0-0
4.	Tab. Pantoprazole	40mg	PO	1-0-0
5.	Tab. Prednisolone	10mg	PO	1-0-0 ~1week
6.	Tab. Rifaximin	550mg	PO	1-1-0
7.	Tab. Vitamin B Complex		PO	0-0-1
8.	Tab. Acetylcysteine	600mg	PO	1-1-1

DISCUSSION

Autoimmune hepatitis is a chronic inflammation of the liver due to an immune attack against hepatocytes. The etiology of autoimmune hepatitis remains unknown though risk factors have been identified.

The pathogenesis mainly depends of following factors:

Genetic predisposition

Genetic variations includes variation in the Human leucocytes Antigen [HLA] gene HLA-DR3 and HLADR4 haplotypes.

Deletions in the C4A gene, final resulting in AIH.

Immune tolerance

In AIH, T cells mistakenly recognize hepatocyte cells as foreign bodies and initiates both humoral and cell mediated immunity.

Environmental factors: such as Bacteria viruses, drugs and poisons can also triggers autoimmune responses.

Cellular immune mechanism in AIH

Cytotoxic T cells cause effect by two ways

Degranulation pathway: Leading to rapid target cell death (CD8 CTL)

Fas mediated pathway: Fas ligand on CTL binds with target cell fas antigen, inducing apoptosis of target cells (CD4CTL)

Finally resulting in hepatocellular injury and uncontrolled inflammation.[9,10]

As per physicians, various lab tests including autoimmune serology pannel were performed, in addition biopsy, sonography and MRI scanning were also performed, based on which it was final diagnosed as AUTOIMMUNE HEPATITIS presented with Malena and GI bleed. The primary treatment with corticosteroids, prednisolone of 16mg BD was given to treat the AIH, Inj. Ceftazidime 1 g was given to treat nosocomial infection, Inj. Pantoprazole 40mg was given to treat GI irritation, Inj. Vitamin-K 10mg was given to treat increase clotting factors, Inj. Tranexamic acid 500mg, Tab. Ursodeoxycholic acid 300mg was given to treat abnormal liver conditions, Syp. Lactulose 10ml was given to prevent the further complications, Inj. 25% dextrose 100ml was given to treat hypoglycemia and fluid imbalance, 1Pint PRBC to treat decreased hb levels ,Inj. Levetiracetam 500mg was given to treat headache, Inj. Multivitamin + NS100ml Ns to treat vitamin deficiency, Inj. Noradrenaline in 100ml NS to treat low blood pressure.

CONCLUSION

Autoimmune hepatitis is a immunosuppressed disease and more likely to affect to the patients of liver disease associated with immunodeficiency. Early diagnosis and preventive measures may help to prevent further deterioration of liver. Life style modification can contribute to restore the liver functions. The primary treatment options include immunosuppressant therapy like azathioprine, corticosteroids. Drugs such as prednisolone can be given in dose tapering manner. This approach could result in improved outcomes and lower mortality rates under such conditions.

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