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CASE REPORT OPEN ACCESS

Autoimmune hepatitis in a young female Nigerian presenting in advanced cirrhosis: A case report

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Abstract
Background: Autoimmune hepatitis is an immune-mediated, chronic inflammation of the liver that can progress to cirrhosis and hepatocellular carcinoma if untreated. It is a rare cause of chronic liver disease in Nigeria.
Case Presentation: Our patient is a 32-year-old lady who presented with a nine-year history of intermittent abdominal swelling and recurrent jaundice. There were peripheral stigmata of chronic liver disease. Abdominal CT scan showed hepatomegaly with multiple ill-defined heterogenous lesions and nodular hepatic margins, splenomegaly and ascites. Anti-Nuclear Antibody was positive, and there was hypergammaglobulinaemia. Liver biopsy showed histologic features that were consistent with Autoimmune Hepatitis. Upper gastrointestinal endoscopy revealed columns of grade 3 oesophageal varices, and she had variceal band ligation therapy done. Based on the International AIH Group revised criteria, she had a score of 8, thus fulfilling the criteria for the diagnosis of definite type 1 AIH. She was commenced on tab prednisolone alongside other liver supportive therapies with improvement in her clinical condition and biochemical profile.
Conclusion: Autoimmune hepatitis is an uncommon cause of chronic liver disease in Nigeria; a high index of suspicion would enable early diagnosis and prompt treatment with immunosuppressants in order to prevent the development of advanced liver disease.

Keywords: Advanced, Autoimmune Hepatitis, Chronic Liver Disease, Liver Cirrhosis, Nigeria

Background

Autoimmune hepatitis (AIH) is an immune-mediated, chronic necroinflammation of the liver, which can potentially progress to cirrhosis and hepatocellular carcinoma if left untreated early (1, 2).

Autoimmune hepatitis occurs in all ethnic groups, and the prevalence differs according to the region (3). The prevalence of AIH in Europe is estimated at 15-25/100,000 population and ranges between 3 and 8/100,000 persons in Asia, while it is highest in Alaska, North America, at 42.9/100,000 persons (2, 3). There is a paucity of epidemiological data on AIH in Africa (3).

Autoimmune hepatitis is classified into type 1 and type 2 based on the pattern of autoantibodies in the patient's serum (1, 2). Type 1 is mainly found in adults and predominantly among females, while

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type 2 is mainly found in children (1, 2). Type 1 AIH, the most common type, is identified by the presence of antinuclear antibody and/or anti-smooth muscle antibody. Type 2 is defined by the presence of anti-liver/kidney microsomal antibody type 1 (2, 4). Occasionally, other atypical types of auto-antibodies may be found in type 1 AIH, such as anti-actin, anti-soluble liver antigen and anti-pANCA (2, 4). Similarly, in type 2 AIH, other atypical auto-antibody types may also be found, such as anti-liver cytosol type 1 and anti-liver/kidney microsomal antibody type 3 (2, 4).

The diagnosis of AIH is based on a combination of suggestive biochemical, immunological and histological features as well as exclusion of other causes of liver diseases such as viral hepatitis (2, 4). The histologic hallmark of AIH is interface hepatitis (1, 4). Other histologic findings include lymphoplasmacytic inflammatory infiltrate, hepatocyte rosettes, and centrilobular necrosis (1, 4). Treatment of AIH is with the use of immunosuppressants (2).

Autoimmune hepatitis is a rare cause of chronic liver disease in Nigeria, and only a very few cases of AIH have been reported in the literature in Nigeria (5, 6, 7, 8). We therefore report a case of AIH in a young lady from south-western Nigeria presenting in advanced cirrhosis. This report is intended to increase the awareness of AIH among clinicians and the need to have a high index of suspicion of the disease, especially when the aetiology of liver disease in a patient is uncertain. This will lead to timely diagnosis and treatment, and therefore reduce the morbidity and mortality associated with this disease among our patients.

Case Presentation

A 32-year-old single lady from south-western Nigeria who was referred to the Gastroenterology specialist clinic from the General Out-Patient clinic of our hospital with a nine-year history of intermittent abdominal swelling, which became persistent and progressive in the last two years, and there is also associated nine years history of recurrent jaundice. She has unexplained fatigue, reduced appetite and has lost some weight. No swelling in any other part of her body, no right upper quadrant abdominal pain or differential swelling, no pain in any part of the abdomen, no passage of dark-coloured urine, no pruritus, no passage of pale stool or bulky, difficult to flush stool and no undigested food particles in the stool. No dyspnoea on mild exertion, no orthopnoea, no paroxysmal nocturnal dyspnoea, no cough, no palpitations and no pedal swelling. No nocturia, no passage of frothy urine, no facial puffiness and no reduction in urine output.

No use of alcohol, tobacco, steroids, NSAIDS, herbs or illicit drugs/substance abuse. No sharing of sharps or intravenous drug abuse, no prior blood transfusion, no indiscriminate sexual intercourse or multiple sexual partners. No intake of mouldy food substances or exposure to aflatoxin. No joint pains or bone pains, no skin rash, no recurrent fever, no menstrual irregularity. No use of alcohol. No family history of liver disease, autoimmune disease or malignancy.

She has intermittent episodes of bleeding gums and easy bruising, with no change in personality or sleep pattern. She was admitted and managed in the emergency room of another hospital on two occasions for upper GI bleeding within the preceding nine months before her presentation to our facility, but she was not properly investigated. Gynaecological history was not remarkable, and review of systems was not contributory. She is not known to have systemic arterial hypertension, diabetes mellitus or sickle cell disease, and she has no known allergy.

She was icteric, had grade 3 digital clubbing, palmer erythema, loss of tenar and hypotenar eminences, Dupuytren's contracture, Terry's nails and bilateral pitting pedal oedema but no significant peripheral lymph node enlargement and no asterixis. Her abdomen was asymmetrically distended with right upper quadrant preponderance, para-umbilical hernia, and there were prominent visible distended anterior abdominal wall veins draining cephalad. There's firm, nodular, non-tender hepatomegaly (span=20cm), splenomegaly (firm, non-nodular and palpable by 8cm below the left coastal margin), moderate ascites and normoactive bowel sounds. Digital rectal examination was essentially normal. Her pulse rate was 92/min, blood pressure was 120/90mmHg, jugular venous pressure was not raised, and heart sounds were S1 and S2 only. Other systemic examinations were not remarkable. Her haemoglobin genotype is AA, and her Blood group is A (Rhesus positive). The results of her laboratory tests are as shown in Table 1.

Table 1: Investigation results of the patient.

Investigation	Baseline Result	9 months on treatment	Reference Interval
Liver Function Test			
Conjugated Bilirubin	38µmol/l	19µmol/l	< 17µmol/l
Unconjugated Bilirubin	18.9µmol/l	8.2µmol/l	< 5.1µmol/l
Aspartate Aminotransaminase (AST)	77iu/l	24iu/l	15-40 iu/l
Alanine Aminotransaminase (ALT)	37iu/l	13iu/l	13-34 iu/l
Alkaline Phosphatase (ALP)	553iu/l	172iu/l	< 240 iu/l
Protein	81g/l	78g/l	60-80 g/l
Albumin	27g/l	32g/l	35-50 g/l
Globulin	54g/l	46g/l	18-30 g/l
Albumin/Globulin Ratio	0.5	0.7	1.5-2.5
Full Blood Count			
Haemoglobin Concentration	12.5g/dl	13.7g/dl	11.5 - 16.5g/dl
White Blood Count	4.2 x 10^9/L	5.1 x 10^9/L	4.0 - 11.0 x 10^9/L
Platelet Count	51 x 10^9/L	98 x 10^9/L	150 - 450 x 10^9/L
Electrolytes, Urea & Creatinine			
Sodium (Na^+)	140mmol/l	142mmol/l	135-145mmol/l
Potassium (K^+)	3.7mmol/l	3.9mmol/l	3.5-5.0mmol/l
Chloride (Cl^-)	102mmol/l	105mmol/l	90-110mmol/l
Bicarbonate (HCO_3^-)	20mmol/l	23mmol/l	22-30mmol/l
Urea	2.0mmol/l	2.9mmol/l	2.5-6.5mmol/l
Creatinine	89µmol/l	78µmol/l	64-115µmol/l
Egfr	77.3ml/min/1.73m^2	90ml/min/1.73m^2	90-120ml/min
Prothrombin Time (PT)	22.5secs	16secs	12-14secs
International Normalised Ratio (INR)	1.8	1.3	0.9-1.1
Erythrocyte Sedimentation Rate (ESR)	58mm/hr	32mm/hr	1-12mm/hr
Alpha Fetoprotein (AFP)	4.5ng/ml	4.3ng/ml	< 5ng/ml
Hepatitis B Panel			
HBsAg	Negative		
HBsAb	Negative		
HBeAg	Negative		
HBe Ab	Negative		
HBcAb(Total)	Negative		
Anti-HCV	Negative		
HIV-1 & HIV-2	Non-reactive		
Serum Protein Electrophoresis	Elevated gamma fraction		
Anti-Nuclear Antibody	Positive (1:160)		< 1:80 (Negative)
Anti-Smooth Muscle Antibody	Negative		
Anti-Mitochondrial Antibody	Negative		
Anti-Smith Antibody	Negative		

Abdominal CT scan showed hepatomegaly with multiple ill-defined heterogenous lesions and nodular hepatic margins, splenomegaly and ascites (Figure 1). These features were consistent with advanced liver cirrhosis with portal hypertension.



Figure 1: Abdominal Computerised Tomography scan showing hepatomegaly with multiple ill-defined heterogenous lesions and nodular hepatic margins, splenomegaly and ascites

She had a liver biopsy done after correcting her coagulopathy, and the liver biopsy histology showed effacement of the lobular architecture due to the presence of vascularized fibrous septae and loss of central-portal relationship. There were foci of emperiopolesis, hepatocyte rosetting and multinucleated giant hepatocytes. There was lymphoplasmacytic infiltration of the intervening connective tissue, and no evidence of malignancy was seen (Figure 2). The histologic features were consistent with AIH.

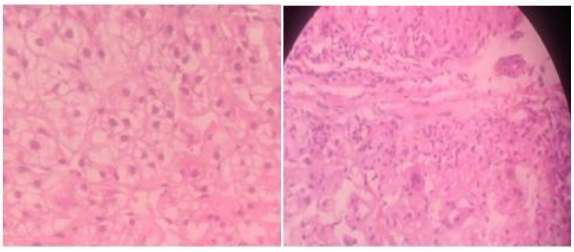


Figure 2: Histologic sections of liver biopsy showing features that are consistent with Autoimmune Hepatitis

Upper GI endoscopy revealed about 4 to 5 columns of Grade 3 oesophageal varices with red whale sign and features of portal hypertensive gastropathy were seen, but there were no gastric varices (Figure 3). She had an endoscopic variceal band ligation (EVBL) therapy done and subsequently had 3 additional sessions of EVBL done at 3 weekly intervals.

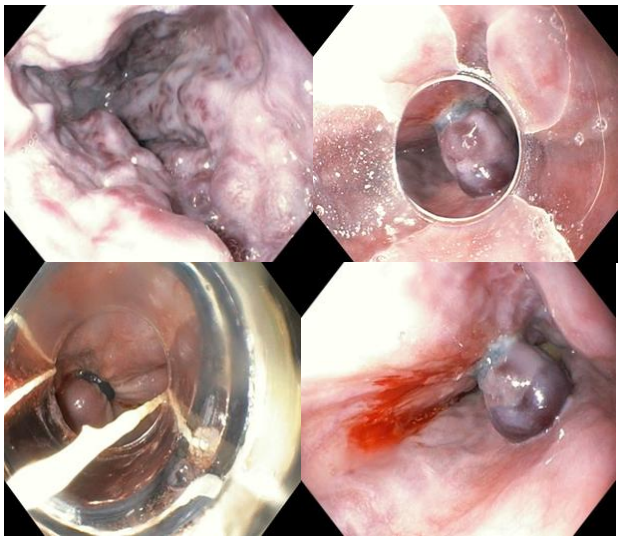


Figure 3: Endoscopic images of the patient showing Endoscopic Variceal Band Ligation (EVBL) therapy performed for extensive Grade 3 oesophageal varices.

She was placed on tab spironolactone 100mg daily, tab furosemide 40mg daily, tab propranolol 40mg daily, cap Livolin forte® (one cap) twice daily and cap rabeprazole 20mg daily. Based on the diagnosis of AIH, she was commenced on tablet prednisolone 20mg daily, and there was improvement in her clinical symptoms and biochemical profile. She is scheduled for regular clinic visits at the Gastroenterology specialist clinic for follow-up and adjustment of her medications as appropriate.

Discussion

The diagnosis of AIH is simplified using the 2008 revised criteria of the International AIH Group (IAIHG), which has 4 key elements: increased serum IgG levels, presence of serum autoantibodies, liver histology and absence of viral hepatitis markers (4). According to the revised criteria, a score of ≥6 is probable AIH, and a score of ≥7 is definite AIH (4).

Our patient has clinical and biochemical evidence of liver disease. She has positive immunological markers (hypergammaglobulinaemia and positive anti-nuclear antibodies) and liver histological features of AIH. Furthermore, she had no marker of viral hepatitis and no other cause was attributed to her liver disease. Based on the IAIHG revised criteria, her score is 8, thus fulfilling the criteria for the diagnosis of definite type 1 AIH.

The clinical manifestations of AIH follow a fluctuating course, and the presentation varies (1, 2). The index case is a young female Nigerian who was overtly symptomatic for liver disease and presented in decompensated liver cirrhosis (ascites, hypoalbuminaemia, coagulopathy, thrombocytopaenia, hyperbilirubinaemia, upper gastrointestinal bleeding) with Child-Pugh score of 11 (CTP class C), APRI score of 3.8, FIB-4 score of 7.94 and MELD score of 16.

This underscores the typical late presentation of our patients for proper medical evaluation whenever they are unwell. The healthcare-seeking behaviour of Nigerians is poor. This is partly due to prevalent poverty, lack of adequate healthcare insurance and payment for healthcare services from 'out-of-pocket', which most people cannot afford. Therefore, many patients are compelled to present only when their symptoms are persistent, worsening or with severe complications.

In Nigeria, chronic hepatitis B virus infection is the leading cause of chronic liver disease (CLD) (9, 10). Autoimmune hepatitis is a very rare cause of CLD in Nigeria (11, 12). This case report has highlighted the presentation of a rare cause of chronic liver disease in Nigeria. This condition was not diagnosed early due to the late presentation of the patient when she had already become symptomatic of advanced liver disease. Her diagnosis could not be established at a secondary healthcare facility, partly due to poor medical resources when she presented there twice on account of upper gastrointestinal bleeding. The diagnosis was eventually made in our tertiary hospital, although late. If she had presented much earlier, the diagnosis could have been made earlier, and immunosuppressants would have been commenced, and the disease progression prevented.

Conclusion

Autoimmune hepatitis remains a rare cause of chronic liver disease in Nigeria, nevertheless, there is a need to maintain a high index of suspicion for AIH as a cause of CLD of apparently unknown aetiology, especially among young individuals. Autoantibody testing and liver biopsy are crucial for accurate diagnosis. Early diagnosis of AIH and early commencement of immunosuppressive therapy would prevent progression to end-stage liver disease.

List of Abbreviations

AIH: Autoimmune Hepatitis
APRI: Aspartate Transaminase/Platelet Ratio Index
CLD: Chronic Liver Disease
CT: Computerised Tomography
CTP: Child Turcotte Pugh
FIB-4: Fibrosis 4 Index
IAIHG: International Autoimmune Hepatitis Group
IgG: Immunoglobulin G
MELD: Model for End-stage Liver Disease

Declarations

Ethical Approval

Not required and not applicable

Consent to Participate

Not required and not applicable

Consent for Publication

Written informed consent was obtained from the patient for the anonymised information to be published in this article.

Availability of Data and Materials

The datasets used in this study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions
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OOO, OE, OOA, AO and IA conceived the study and were involved in the direct clinical management of the patient. OOO, OE and AO were involved in conducting the investigations and reporting the results. OOO, OE and OOA wrote the first draft of the manuscript. OOO, OE, OOA, AO and IA contributed to the subsequent draft and were involved in the critical revision of the article for important intellectual content. All authors approved the final version of the article to be published. OOO is the guarantor for this paper, accepts full responsibility for the work, had access to the data, and controlled the decision to publish.

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