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The Afro-Egyptian Journal of Infectious and Endemic Diseases (AJIED) is a peer-reviewed journal that publishes clinical, parasitological, microbiological, physiological, biochemical, immunological and pathological studies in the field of infectious, endemic and tropical diseases. The scope of the journal includes also articles of endemic gastroenterology and hepatology. The journal is published quarterly by Endemic and Tropical Medicine Department, Faculty of Medicine, Zagazig University, Zagazig, 44519, Egypt.

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# Study of Auto-Antibodies in Egyptian Non-B, Non-C Chronic Hepatitis Patients

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**Keywords:** auto-antibodies; non-B, non-C; chronic hepatitis; Egyptian.

**Background and study aim:** Autoimmune hepatitis (AIH) is a curable disease that is under studied in our locality in Egypt and it's role in causation of chronic liver disease is not well studied. This work aimed at evaluation of the pattern and clinical importance of an array of auto-antibodies in non-B, non-C chronic hepatitis Egyptian patients and to detect the prevalence and feature of autoimmune liver diseases (AILDs) in our locality and evaluate other causes of chronic non-B, non-C hepatitis in our patients.

**Patients and methods :** Between January 2007 to September 2009, 50 Egyptian patients with non-B non-C chronic hepatitis were enrolled in this study (18 males and 32 females). All patients were subjected to the following, full history taking, through clinical examination, viral markers (HBsAg, Anti-HCV, HBC Ab), liver function tests, serum protein electrophoresis, abdominal ultrasonographic examination, fine needle liver biopsy and histopathology examination and tests for autoimmune antibodies, ANA, ASMA, ALKM-I and AMA and measuring their titer.

## INTRODUCTION

Autoimmune hepatitis (AIH) is a distinct group of acute and chronic necro-inflammatory disorder. Liver-related autoantibodies are crucial for the correct diagnosis and classification of autoimmune liver diseases (AILD), namely AIH types 1 and 2 (AIH-1 and 2), primary biliary cirrhosis (PBC), and the sclerosing cholangitis variants in adults and children. AIH-1 is specified by anti-nuclear antibody (ANA) and smooth muscle antibody (SMA). AIH-2 is specified by antibody

**Results :** Most patients were middle-age females (27.72±12.1) and the most common auto-antibodies detected in patients group were ANA (48%) followed by ASMA (44%), ALKM-1 (24%) and AMA (20%). Out of the 50 patients; 32 patients (64%) were diagnosed as AILDs and 16 patients (32%) were diagnosed as autoimmune hepatitis (AIH), most of them (10 patients) were classified as type I AIH (with ANA and/or ASM), 2 patients (4%) were classified as type II AIH (with LKM-1) and 4 patients (8%) could not be classified on the basis of routine antibodies profile. 16 patients (32%) were diagnosed as overlap syndrome (AIH with cholestatic feature with or without AMA positive sera).

**Conclusion :** The present study concluded that the distribution of autoantibodies in different group of patients revealed the difficulty to endorse the subclassification of patients of AIH depending on autoantibodies profiles. The role of AIH needs more studies in our locality as it's one of the curable liver diseases.

to liver kidney microsomal antigen type-1 (anti-LKM1) and anti-liver cytosol type 1 (anti-LC1). SMA, ANA and anti-LKM antibodies can be present in de-novo AIH following liver transplantation. PBC is specified by antimitochondrial antibodies (AMA) reacting with enzymes of the 2-oxo-acid dehydrogenase complexes (chiefly pyruvate dehydrogenase complex E2 subunit) [1]. Several studies clearly demonstrate that liver cell damage in autoimmune hepatitis (AIH) is mediated by autoimmune

reaction against normal constituents of the hepatocytes[2]. The presence of circulating auto-antibodies is the single most significant finding in AIH, the discovery of auto-antibodies directed against different cellular targets, including endoplasmic reticulum membrane proteins, nuclear antigens and cytosolic antigen has led to a suggested sub-classification of AIH based upon the presence of three specific autoantibody profiles[3].

In Egypt, where hepatitis C infection is the commonest liver disease among Egyptian patients, the other liver disorder like AILD are usually under studied and it's role in causation of chronic liver disease among Egyptians are not well studied and the true prevalence of autoimmune liver disorder in Egypt is not well know.

## PATIENTS AND METHODS

This work was carried out on 50 patients with non-B non-C chronic hepatitis (the patients group) selected out from 795 chronic hepatitis patients attending Tropical Medicine and Microbiology & Immunology Departments, Faculty of Medicine, Zagazig University, Egypt, in the period from January 2007 to September 2009, including 18 males and 32 females attending Outpatient Clinic of Gastroenterology and Hepatology of Tropical Medicine Department. Informed consents were obtained from all patients.

The selected (50) patients with non-B non-C chronic hepatitis (after exclusion of B and C viral hepatitis by the serological methods and exclusion of drug and alcoholic induced hepatitis by history), were subjected to the following :

- A- Clinical examination.
- B- Routine investigations: urine analysis and stool examination, complete blood picture and kidney function tests.
- C- Serum protein electrophoresis : It was done to detect the hypergamma globulinaemia[4].
- D- Abdominal ultrasonography : For assessment of hepatobiliary system, spleen, portal system and kidneys.
- E- Liver biopsy and histopathological examination using sheathed trucut which is a cutting technique for liver biopsy and the specimen was obtained by aspiration.
- F- Serological examination for serum auto-antibodies and titres as follow: 10cc of venous blood were collected from each case, serum was separated, aliquoted and stored at -20°C till it used in the screening for serum auto-antibodies testing for : serum antinuclear

antibodies (ANA), anti-smooth muscle antibodies (ASMA), anti-liver kidney microsomal-1 antibodies (ALKM-1) and antimitochondrial antibodies (AMA).

The serological examination was done by indirect immunofluorescence (IFA) using autoimmune antibody screening test system containing Rat/liver/stomach/kidney substrate (Trinity Biotech PLC, Bray, county Wicklow, Ireland). Titres <1/20 were considered non-significant for each of the studied antibodies.

Additional tests were performed to special group of patients :

- 1- Serum ceruloplasmin, copper in serum and 24-hour urinary copper excretion to patients with suspicious of Wilson's disease.
- 2- L-E cells and ds-DNA to patients with suspicious of SLE, by latex agglutination.
- 3- Renal biopsy for patients with history suspecting glomerulonephritis.
- 4-  $\alpha$ -1-antitrypsin level and serum iron concentration and ferritin concentration (Eletech Co., Germany) for cryptogenic chronic hepatitis patients.

### Statistical Analysis :

Data were collected, checked, entered and statistically analyzed using Epi-Info version 6.0 software computer package. Data were expressed as mean  $\pm$  SD for quantitative variables, number and percentage for qualitative ones.

## RESULTS

Fifty Egyptian patients with non-B non-C chronic hepatitis were selected out of 795 chronic hepatitis patients. The mean age of this patients was  $39.72 \pm 12.12$  years, range (15-60 years). They were 32 female (64%) and 18 male (36%).

As a regard for laboratory findings all patients (100%) had elevated ALT (>41 u/L) and AST (>38 u/L) levels but 36 patients (72%) of them had elevated ALT more than 3 folds and 42 patients (84%) of them had elevated AST more than 3 folds. Total bilirubin was elevated more than 2 mg/dl in 44 patients (88%), direct bilirubin increased in 42 patients (84%). Hypoalbuminemia (<3.4 g/dl) was detected in 18 patients (36%), 2 cases of them were associated with albuminuria. Hypergammaglobulinaemia ( $\gamma$ -globuline >1.5 g/dl) was detected in 36 patients (72%), also prothrombin time was prolonged (>14 sec) in 28 patients (56%).

As a regard for histopathological findings by liver biopsy; all the studied patients (100%) showed



inflammatory reactions with chronic inflammatory cells predominate in portal tracts mostly of lymphocyte in 42 patients (84%), followed by plasma cells in 24 patients (48%) and finally P.N.Ls in 12 patients (24%). Piecemeal necrosis (interface hepatitis) was reported in 32 patients (64%), fibrosis was recorded in 36 patients (72%). As regard the liver parenchyma, disturbed architecture was seen in 12 patients (24%), hyperplastic kuppfer cells in 14 patients (28%), and cirrhotic nodules in 10 patients (20%). The liver cells showed different pathological changes, as ballooning in 16 patients (32%), moderate to marked steatosis in 14 patients (28%), hydropic changes and ground glass appearance in 4 patients (8%). On the other hand, findings suggestive cholestasis, bile duct proliferation and ductopenia were encountered among 18 patients (36%), 10 patients (20%) and 10 patients (20%) respectively (Table 1 & Figs. 1,2,3).

The auto-antibodies studied in our group of patients showed that 24 patients (48%) had ANA with statistically significant difference ( $P=0.004$ ) regarding other auto-antibodies. Also, 22 patients (44%) had ASM auto-antibodies, 14 patients (63.4%) of them showed titer 1/40 with high statistically significant difference ( $P<0.001$ ). As regard ALKM-1 auto-antibodies, it was reported in 12 patients (24%), 6 patients (50%) of them showed titer 1/40 with high statistically significant difference ( $P<0.001$ ). On the other hand, 10 patients (20%) had AMA at titer 1/20 with high statistically significant difference ( $P<0.001$ ) when compared with other auto-antibodies (Table 2).

As a regard for the suspected etiological diagnosis, 16 patients (32%) out of 50 patients were diagnosed as AIH, most of them (10 patients "20%") was of type I. While type II AIH were diagnosed in 2 patients (4%) only, while 4 patients (8%) with unclassified AIH were also reported, and 16 patients (32%) were diagnosed as overlap syndrome.

On the other hand, other chronic liver disorders such as Wilson's disease and glycogen storage disease were the least reported suspected diagnosis as each of them was encountered among 2 patients (4%). Also 8 patients (16%) were diagnosed as a cases of cryptogenic chronic hepatitis and 6 patients (12%) were diagnosed as non-alcoholic steato-hepatitis (NASH) (Table 3).

AMA positive overlap syndrome was more common in patients with age group 20-40 years (8

out of 22 patients) (36.4%). While only 4 out of 14 patients (28.6%) were less than 20 years had overlap syndrome with AMA negative. All patients with NASH were of age group 20-40 years. Also ANA and ALKM-1 were more common in the same age group; 12 patients (50%) and 8 patients (66.7%) respectively. Ten female patients out of 32 (31.25%) had AIH type I which is of statistically significant difference ( $P=0.02$ ). On the other hand, 6 male patients out of 18 (33.3%) had cryptogenic chronic hepatitis with statistically significant difference ( $P=0.035$ ). A part from these difference, there was no statistically significant difference between sex distribution and diagnosis. Auto-antibodies are more common in female than in male patients. All patients with AMA positive overlap syndrome had hyperbilirubinemia and increased alkaline phosphatase more than 3 folds that consistent with cholestatic pattern with statistically significant difference ( $P=0.01$ ). There is no statistically significant difference between the studied auto-antibodies and the level of serum bilirubin, serum albumin, gamma globulin, transaminase, alkaline phosphatase or prothrombin time and I.N.R (Table 4). There was high statistically significant difference in the differentiation of the cells in portal tract among our group of patients ( $P<0.001$ ). All patients with overlap syndrome with AMA positive had cholestasis with high statistically significant difference ( $P<0.001$ ). Eight patients (80%) of them had ductopenia with high statistically significant difference ( $P<0.001$ ). On the other hand, bile duct proliferation was reported among 4 patients out of 6 with overlap syndrome with AMA negative with statistically significant difference ( $P=0.004$ ). Twenty patients (90.9%) of the patients who were positive for ASM autoantibody had lymphocytes in portal tracts ( $P=0.02$ ). However, 6 (50%) of ALKM-1 positive cases had P.N.Ls in liver biopsy with statistically significant difference ( $P=0.02$ ). As regard bile ducts and cholestatic features, all patients with AMA positive showed features of cholestasis (Figs. 1,2,3,4).

Both types of AIH have similar course and outcome. In contrast to the unclassified type of AIH which had more advanced clinical manifestation, as 50% of them had shrunken liver and 50% had history of hematemesis and melena. Detection of AMA in AIH might identify a subset of patients at risk of developing hepatic/cholestatic syndrome.

**Table (1) :** Histopathological findings among the studied groups of patients.

	N	%
<b>Portal tracts :</b>		
Thickened	4	8.0
Main cells : Lymphocytes	42	84.0
Plasma cells	24	48.0
Polymorphonuclear leucocytes (P.N.Ls)	12	24.0
Fibrosis	36	72.0
Interface hepatitis	32	64.0
<b>Parenchyma :</b>		
Disturbed architecture	12	24.0
Hyperplastic kuppfer cells	14	28.0
M.N cells and lymphocytes	6	12.0
Fibrosis	14	28.0
Cirrhotic nodules	10	20.0
Collapse	4	8.0
<b>Hepatocytes :</b>		
Ballooning	16	32.0
Steatosis	14	28.0
Hydropic changes	4	8.0
Ground glass cells	4	8.0
Round bland nuclei	2	4.0
<b>Cholestatic features and bile ducts:</b>		
Deposite of orcein +ve copper and protein granules	2	4.0
Peripheral xanthomatous changes and pseudoglandular pattern	2	4.0
Cholestasis	18	36.0
Proliferation of bile ducts	10	20.0
Ductopenia	10	20.0

**Table (2) :** Titre of auto-antibodies among the studied groups of patients

Titre of auto-antibodies	Total No.		1/20 +		1/40 ++		1/80 +++		X <sup>2</sup>	P
	No	%	No	%	No	%	No	%		
ANA	24	48.0	16	66.7	6	25.0	2	8.3	3.03	>0.05 NS
ASMA	22	44.0	8	36.4	14	63.4	0	0.0	10.05	<0.01 Sig.*
ALKM-1	12	24.0	2	16.7	6	50	4	33.3	14.07	<0.001 HS**
AMA	10	20.0	10	100	0	0.0	0	0.0	10.42	<0.001 HS**
X <sup>2</sup>	13.19		19.41		14.68		12.06			
P	0.004 Sig.*		<0.001 HS**		<0.001 HS**		<0.01 Sig.*			

**Table (3) :** Classification of the patients according to etiological diagnosis

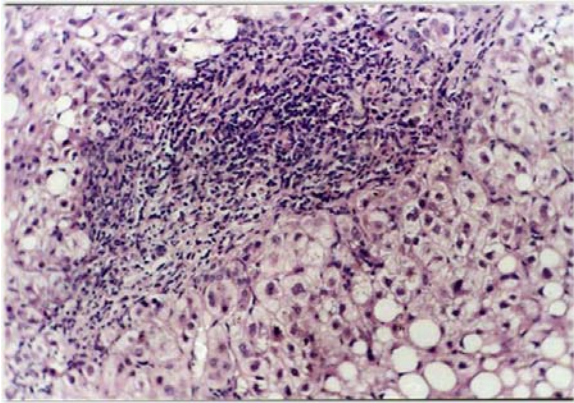
Diagnosis	Total No	%
<b>Autoimmune hepatitis :</b>	<b>16</b>	<b>32.0</b>
AIH type I	10	20.0
AIH type II	2	4.0
Unclassified AIH	4	8.0
<b>Overlap syndrome :</b>	<b>16</b>	<b>32.0</b>
AIH with AMA +ve	10	20.0
AIH with AMA -ve	6	12.0
<b>Cryptogenic chronic hepatitis</b>	<b>8</b>	<b>16.0</b>
<b>Wilson's disease</b>	<b>2</b>	<b>4.0</b>
<b>Non-alcoholic steatohepatitis (NASH)</b>	<b>6</b>	<b>12.0</b>
<b>Glycogen storage disease</b>	<b>2</b>	<b>4.0</b>

**Table (4) :** Relationship between liver function tests and auto-antibodies among the studied groups of patients

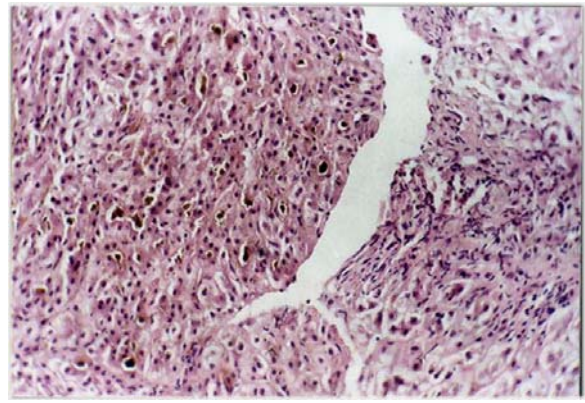
Parameter of liver function test	N	ANA N = 24		ASM N = 22		ALKM-1 N = 12		AMA N = 10		X <sup>2</sup>	P
		N	%	N	%	N	%	N	%		
Hyperbilirubinaemia (jaundice)	44	24	54.5	22	50	12	27.3	10	22.7	0.0	1.0 NS
- Cholestatic	22	16	72.7	14	63.6	8	36.4	10	45.45	4.95	0.17 NS
- Non-cholestatic	22	8	36.4	8	36.36	4	18.18	-	0	4.95	0.17 NS
Hypoalbuminaemia	18	10	55.55	10	55.55	4	22.2	2	11.1	2.14	0.54 NS
Hypergammaglobuline.	36	24	66.7	22	61.1	12	33.3	10	27.8	0.0	1.0 NS
ALT >3 folds	36	16	44.4	18	50	10	27.8	8	22.2	2.02	0.56 NS
AST >3 folds	42	22	52.4	20	47.6	10	23.8	10	23.8	1.89	0.59 NS
↑ Alkaline phosphatase	28	18	64.3	16	57.14	10	35.7	10	35.7	3.59	0.3 NS
Prolonged prothrombin time	28	14	50	16	57.14	8	28.6	6	21.4	1.16	0.76 NS

**Table (5) :** Distribution of histopathological findings in the studied groups

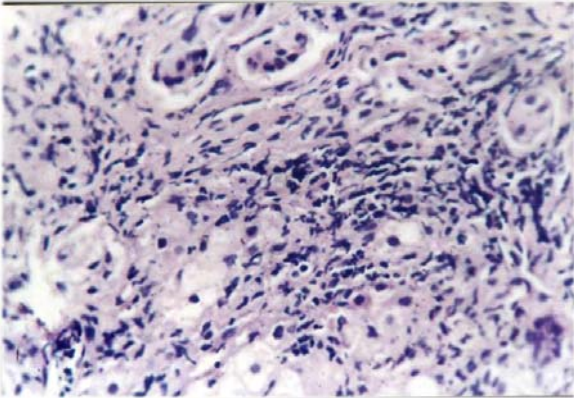
Histopathological findings	N	Autoimmune hepatitis						Overlap syndrome				Cryptogenic Ch. hepatitis		Wilson's disease		NASH		Glycogen storage disease		X <sup>2</sup>	P	
		Type I N=10		Type II N=2		Type ? N=4		AMA +ve N=10		AMA -ve N=6		N=2		N=6		N=2						
		N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%			
<b>Portal tracts</b>																						
Thickened	4	-	-	-	-	-	-	-	4	66.7	-	-	-	-	-	-	-	-	-	-	31.8	<0.001HS**
Main cells :																						
Lymphocytes	42	10	100	-	-	4	100	6	60	6	100	8	100	2	100	6	100	2	100	27.27	<0.001HS**	
Plasma cells	24	4	40	2	100	4	100	8	80	6	100	-	-	-	-	-	-	-	-	33.9	<0.001HS**	
PNLs	12	-	-	2	100	-	-	4	40	-	-	-	-	-	-	6	100	-	-	36.8	<0.001HS**	
Fibrosis	36	6	60	2	100	4	100	10	100	6	100	8	100	-	-	-	-	-	-	38.1	<0.001HS**	
Interface hepatitis	32	8	80	2	100	4	100	6	60	4	66.7	6	75	2	100	-	-	-	-	20.34	0.009 Sig.*	
<b>Parenchyma</b>																						
Disturbed architecture	12	2	20			2	50	2	20	-	-	4	50	2	100	-	-	-	-	16.01	0.04 Sig.*	
Hyperplastic kuppfer cells	14	2	20			2	50	4	40	2	33.3	4	50	-	-	-	-	-	-	8.66	0.32 NS	
MN cells and lymphocytes	6	2	20			-	-	2	20	-	-	-	-	2	100	-	-	-	-	19.7	0.01 Sig.*	
Fibrosis	14	2	20			4	100	4	40	-	-	2	25	2	100	-	-	-	-	22.7	0.003 Sig.*	
Cirrhotic nodules	10	4	40			2	50	-	-	-	-	2	25	2	100	-	-	-	-	19.38	0.01 Sig.*	
Collapse	4	-	-			-	-	-	-	-	-	2	25	2	100	-	-	-	-	29.6	0.001 HS**	
<b>Hepatocytes</b>																						
Ballooning	16	4	40					6	60	4	66.7	2	25	-	-	-	-	-	-	14.92	0.06 NS	
Steatosis	14	8	80					-	-	-	-	-	-	-	6	100	-	-	-	42.06	0.001 HS**	
Hydropic changes	4	-	-					-	-	-	-	4	50	-	-	-	-	-	-	17.26	0.02 Sig.*	
Ground glass cells	4	-	-					-	-	-	-	4	50	-	-	-	-	-	-	22.8	0.003 Sig.*	
Round bland nuclei	2	-	-					-	-	-	-	-	-	-	-	-	-	2	100	50	<0.001HS**	
<b>Cholestatic features and bile ducts :</b>																						
Deposition of orcein +ve copper & protein granules	2	-	-											2	100					50	<0.001HS**	
Peripheral xanthomatous changes and pseudoglandular pattern	2	-	-					2	20	-	-									8.3	0.4 NS	
Cholestasis	18	-	-					10	100	6	100	2	25							43.4	<0.001HS**	
Ductopenia	10	-	-					8	80	2	33.3	-	-							43.4	<0.001HS**	
Proliferation of bile ducts	10	-	-					2	20	4	66.7	2	25	2	100					22.29	0.004 Sig.*	



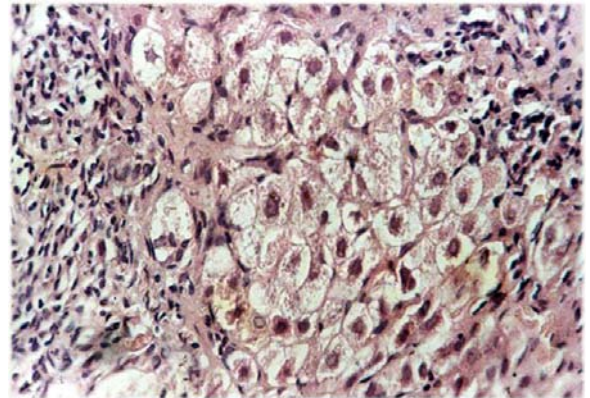
**Fig. (1) :** Hepatocyte showing marked ballooning and marked steatosis, there is marked portal lymphocytic and polymorphonuclear leucocyte infiltration with moderate piecemeal necrosis (autoimmune hepatitis) (H & E x 200)



**Fig. (2) :** Liver tissue showing disturbed architecture with hydropic changes. Portal tract showing moderate lymphocytic infiltration with moderate piecemeal necrosis and ductopenia and marked cholestasis (overlap syndrome AMA +ve) (H & E x 200)



**Fig. (3):** Hepatocytes showing moderate ballooning, the portal areas revealed marked lymphocytes infiltration with piecemeal necrosis and proliferation of bile ducts (overlap syndrome AMA –ve) (H & E x 400)



**Fig. (4) :** Moderate ballooning of hepatocytes, there are ground glass cells, the portal areas revealed chronic inflammatory cells mainly lymphocytes with marked fibrosis and proliferation of bile ducts (cryptogenic chronic hepatitis) (H & E x 400)

## DISCUSSION

AIH has been defined as "an unresolving, predominantly periportal hepatitis, usually with hypergammaglobulinemia and tissue auto-antibodies, which is responsive to immunosuppressive therapy in most cases." It is a relatively rare disorder, with a preponderance of female patients, that can present at any age (although onset in most cases is after 40 years of age)[5].

It is one entity of the heterogeneous syndrome of chronic hepatitis, the true prevalence of AIH among the countries in the world are not well known, especially in countries in which viral hepatitis B and C are endemic, and the presence of circulating auto-antibodies is the single most

significant finding in AIH and they are the key for diagnosis[6].

In the present study, measurement of autoantibodies in the sera of AILD patients revealed that 24 patients (48%) were positive for ANA, 22 patients (44%) were positive for ASMA, 12 patients (24%) were positive for LKM-1 antibody and 10 patients (20%) were positive for AMA. This measurements reflected the significant serum levels of autoantibodies. This result is in contrast with the study which was carried out in upper Egypt by El-Sayed et al.[7] who found that LKM-1 autoantibody was the most common autoantibody detected in patients serum (62.5%). This discrepancy is due to the difference in selective criteria, as they studied serum autoantibodies with

inclusion of HCV, HBV and AILD in studied patients.

Classification of patients in the present study according to the etiological diagnosis revealed that, out of the Egyptian 50 patients with non-B, non-C chronic hepatitis 32 (64%) were diagnosed as AILDs, 16 patients (32%) of them were diagnosed as AIH, most of them; 10 patients (62.5%) were classified as type I AIH, with positive ANA and/or ASMA, 2 patients (12.5%) were classified as type 2 AIH, with positive ALKM-1, but 4 patients (25%) could not be classified on the bases of available autoantibodies profiles. Overlap syndrome (hepatitis and cholestatic AILD) with or without AMA was diagnosed in 16 patients (32%), 10 patients of them (62.5%) had AMA positive and 6 patients (37.5%) were AMA negative. So, the prevalence of AILDs in our area was about 40.2/100000, half of them were of AIH, mostly of AIH type I and the other half was of overlap syndrome mostly with AMA positive sera. In comparison with prevalence of AILDs among various countries in the world, it was 1.9/100.000 in North America[8], 16.9/100.000 in Europe[9], 14/100.000, 5.6%, 2% in India[10].

Most AIH type I patients were of age around forty years, and patients with unclassified AIH were in peri-pubertally 50% and another 50% were between the fourth and sixth decades. This finding is in concordance with other study[11], they reported that AIH is disease of young especially women. On the other hand, Mc Farlane[5] reported that most cases of AIH reported in two peaks of onset: peripubertally and between fourth and sixth decades of life. However the point of controversy is the age of AIH type II patients, which is predominantly affects the children[11,12], while in the present study, patients were adults, this in agreement with Czaja et al.[13].

In the present study, biochemical liver tests (LFT) typically showed a "hepatitic" (with or without cholestatic) pattern of abnormalities, as serum aminotransferase activity and bilirubin concentrations varied widely from very mildly abnormal to more than 50 fold rise the upper normal limits in all patients with AILDs and cryptogenic chronic hepatitis, but the hyperbilirubinemia is notably cholestatic in all patients (100%) with overlap syndromes, as it was associated with alkaline phosphatase elevation more than 3 time the upper normal limit. This observation is in coincidence with many other studies[14,15,16], they classified overlap syndrome as an

"intermediate level between cholestatic form of AIH or hepatic form of cholestatic syndromes".

The hypergammaglobulinemia, which is the most reliable laboratory parameter in AILDs, was increased in this study in all patients with AILDs and much higher value were observed in type I AIH with average 2.5 g/dl, when compared to other types of AILDs, this observation also recorded by Porta and Squires [16,17].

There was no statistically significant difference between the degree of liver function impairment and the presence of specific auto-antibody, indicating that these auto-antibodies should be used for diagnostic purpose only and they do not correlate with disease severity and activity and also similar to that as reported by Luxon[18].

In the present study, histopathological examination of AIH group showed that, interface hepatitis was the main feature, the inflammatory activity within and around the portal tracts predominated over the lobular changes with aggregation of lymphocytes, plasma cells and polymorphonuclear cells in the portal tract or periportal area. This periportal inflammatory cell infiltration present without bile duct damage. This was coincides with that of other study[19]. As regard overlap syndrome, mixed cholestatic and hepatocellular features were found in them which supported the clinical diagnosis of overlap syndrome. The histologic features in overlap group comprised of cholestasis in 100% of patients, ductopenia in 10 patients (62.5%), proliferation of bile ducts in 6 patients (37.5%) and peripheral xanthomatous changes and pseudoglandular pattern in 2 patients (12.5%) which coincidence with others[19,20].

In the present study, measurement of numbers and titres of each autoantibody in each group of studied patients revealed that, although ANA was found in large number of patients with overlap syndrome (AMA positive and AMA negative) in 10 patients (100%) and 4 patients (66.7%) respectively, it was detected in low titre (1/20) in all of them, compared to its presence in high titre in patients with AIH especially in AIH type I. ALKM-1 was detected in high titre in 100% of patients with AIH type 2 (1/80), also it was found in AMA positive patients and it was not present in AMA negative patients. However, AMA reactivity may be false because of confusion with ALKM-1 by indirect immuno-fluorescence as proposed[21].

In this work, distribution of auto-antibodies among each group of studied patients arised question

about 4 patients who cannot be classified according to auto-antibody profiles, as 2 of these patients had (ANA, ASM and LKM-1) and the other two had (ASM and LKM-1). Also, among 6 AMA positive overlap syndrome patients, 4 of them had 4 autoantibodies (ANA, ASM, LKM-1 and AMA) and the other 2 patients showed 3 autoanti-bodies (ANA, LKM-1 and AMA).

So, although the autoantibodies serve mainly as a marker of disease and key for diagnosis, but there is still a long way to go, as many authors considering the problems with definition of AIH and relation to stage of disease, overlap and/or coexistence among autoimmune diseases, and distinction of possible subtypes, suggested the necessity of further characterization of autoantibodies relevant for AIH and a better pathophysiological understanding will allow subclassification on the basis of etiopathogenesis[22]. So, as the classification of patients according to auto-antibody profiles and/or different immuno-genetic markers is a controversial area with some authorities recommending various subdivisions of AIH along these lines[23,24].

So characterization of other auto-antibodies in the future rather than the conventional auto-antibodies may facilitate reclassification of patients from cryptogenic chronic hepatitis to AIH.

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**Funding:** Non

**Ethical approval:** Approved.

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# Current Status of Schistosomiasis in Egypt: Parasitologic and Endoscopic Study in Sharqia Governorate

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**Keywords:**

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**Background and study aim:** Schistosomiasis was endemic in Egypt since the ancient times. It was traditionally the most important public health problem. This study aimed to evaluate the current status of schistosomiasis in Sharkia governorate, Egypt.

**Patients and methods:** Over 5 years (2005-2010), schistosome eggs were sought by sedimentation techniques in stool and urine samples of 30,000 outpatient persons attending the Tenth of Ramadan Charity Hospital, Tenth of Ramadan City, Sharqia Governorate, Egypt and in histologically processed rectal biopsy samples from patients with negative coproscopy. These samples were obtained by lower endoscopy from 250 patients out of them.

**Results:** Eggs of *Schistosoma haematobium* were encountered in urine samples of 6 persons (0.02%) of the 30,000 outpatients. Eggs of *Schistosoma mansoni* were encountered in stool samples of 99 persons (0.33%) of the 30,000 outpatients. Eggs were also found in 12 persons (4.8%) (2 with living *Schistosoma mansoni* eggs, 1 with dead *Schistosoma haematobium* eggs and 9 with dead *Schistosoma mansoni* eggs) out of the 250 patients contributing to rectal biopsy samples.

**Conclusion:** The present findings revealed a decrease in the prevalence of schistosomiasis that may be explained by the current policy of schistosomiasis control in Egypt.

## INTRODUCTION

Schistosomiasis is one of the most widespread parasitic infections of man [1]. Egypt is a cradle of civilization, but has been plagued by schistosomiasis since at least the Middle Kingdom period (1,500 BC)[2]. It was traditionally the most important public health problem[3,4]. This study aimed to evaluate the current status of schistosomiasis in Egypt.

## PATIENTS AND METHODS

Over 5 years (2005-2010), schistosome eggs were sought by sedimentation technique in stool and urine samples of 30,000 outpatient persons attending the Tenth of Ramadan Charity Hospital, Tenth of Ramadan City, Sharqia Governorate, Egypt.

Tenth of Ramadan City is an industrial urban large city in Sharkia

Governorate but most of it's inhabitants are immigrants of other Egyptian Governorates endemic with schistosomiasis.

250 patients with negative coproscopy were examined by lower endoscopy using Olympus CF160AL colonoscope (Olympus Company Ltd, Japan). Rectal biopsy samples were obtained and examined microscopically. Informed consents were obtained from all patients.

**Sedimentation technique of urine:** 50 ml urine samples were left to sediment spontaneously. Small drop of the sediment was examined microscopically.

**Sedimentation technique of stool:** The technique standardized by Hoffman et al.[5] involves the filtration of saline-feces mixture through a metal sieve to remove larger

fecal residues and to allow the remainder to sediment spontaneously. A sample of the sedimented material is then examined between a slide and cover slip.

## RESULTS

Eggs of *Schistosoma haematobium* were encountered in urine samples of 6 ones (0.02%) of

the 30,000 outpatients. Eggs of *Schistosoma mansoni* were encountered in stool samples of 99 ones (0.33%) of the 30,000 outpatients.

Eggs were also found in 12 ones (4.8%) (2 with living *Schistosoma mansoni* eggs, 1 with dead *Schistosoma haematobium* eggs and 9 with dead *Schistosoma mansoni* eggs) out of the 250 patients contributing to rectal biopsy samples.

**Table (1) :** Results.

	Patients with <i>S. mansoni</i> eggs				Patients with <i>S. haematobium</i> eggs			
	Living		Dead		Living		Dead	
	N	%	n	%	n	%	n	%
Stool samples n = 30,000	99	0.33	-	-	-	-	-	-
Urine samples n = 30,000	-	-	-	-	6	0.02	-	-
Rectal biopsy samples n = 250	2	0.8	9	3.6	-	-	1	0.4



**Figure (1):** 3 dead *Schistosoma haematobium* eggs in rectal biopsy samples

## DISCUSSION

In this study; eggs of *Schistosoma mansoni* were encountered in 0.33% of stool samples and eggs of *Schistosoma haematobium* were encountered in 0.02% of urine samples. *Schistosoma* eggs were also found in 4.8% of patients contributing to rectal biopsy samples. In agreement with our results; Rabello [6] who concluded that a single rectal biopsy resulted in the

diagnosis of more individuals than a single fecal examination by Kato/Katz method.

In 1937; Scott [7] reported the results of parasitologic examination performed on 2 million patients seen between 1932 and 1934 in government treatment centers, and on results of examination of stool and urine samples collected from 40,000 subjects. He found that 60% of the rural population in north and



east of the Nile delta were infected with *S. haematobium* and about the same proportion was infected with *S. mansoni*. In the south central delta, the prevalence of *S. haematobium* was 60%, but only 6% had *S. mansoni*. *S. mansoni* was not found in Upper Egypt and *S. haematobium* was found in 60% of patients in areas under perennial irrigation. In areas under basin irrigation, only 5% were infected by *S. haematobium*.

An increase in the prevalence of *S. mansoni* infections with their snails and decrease in prevalence of *S. haematobium* infections with their snails were noticed after construction of the High Dam in the 1960s because of reclamation of the land, conversion of annual flooding into perennial irrigation and other ecological factors as changes in the water-flow pattern [8]. In 1977, El Alamy and Cline [9] found that the prevalence of *S. mansoni* infection was 40.5%, and the prevalence of *S. haematobium* infection was 27% in Qalyub region in the south of Nile Delta. In 1979; Abdel-Wahab et al.[10] found that the prevalence of *Schistosoma mansoni* infection had increased from 3.2% to 73%, whereas *S. haematobium* infection which had very common in 1935 (74%), had almost disappeared (2.2%) in the same village in the Nile delta surveyed by Scott in 1935.

In 1982, King et al.[11] found that the prevalence of *Schistosoma haematobium* in six rural villages of the Qena governorate was 37.1%.

In the nineties, El-Kohby et al.[4] found that the prevalence of *S. mansoni* and *S. haematobium* in Ismailia governorate was 42.9% and 3.5% respectively, Kafr-El-Sheikh; 39.1% and 2.5%, Gharbia: 37.7% and 2.06%, Monofia: 28.49% and 5.5%, Qalubia: 17.47% and 1.53%, Fayoum: 4.3% and 9.95%, Minya: 1.04% and 8.47%, Assiut: 0.42% and 6.63% and Qena: 0.44% and 7.04%.

In this study, the prevalence of *S. mansoni* and *S. haematobium* depending on stool and urine examination in Tenth of Ramdan Charity Hospital, Tenth of Ramadan city which is an urban city in Sharqia Governorate was 0.33% and 0.02% respectively. In comparison with our study; el-Badawy et al.[12] in 1996 who found that the prevalence of *S. mansoni* and *S. haematobium* depending on stool and urine examination in urban Sharqia Governorate was 6.8% and 0.09%.

In this study, *S. haematobium* eggs were found in rectal biopsy sample of one patient. In agreement with our finding; Abdel-Wahab et al.[13] who found a mild grade of periportal fibrosis, hepatomegaly and splenomegaly in school children with *S. haematobium* infection in a village in the Fayoum.

**Conclusion:** The present findings revealed a decrease in the prevalence of schistosomiasis that may be explained by the current policy of schistosomiasis control in Egypt. Further studies in different localities in Egypt are needed to reflect the actual prevalence of schistosomiasis. Much more strict programs to control schistosomiasis hoping at complete eradication of this disease are recommended.

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# Thrombosis, an Early Presentation and Extensive Spondylosis, a Late Complication of Inflammatory Bowel Disease

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Inflammatory bowel disease (IBD) comprises two major disease entities, ulcerative colitis and Crohn's disease, that may be complicated by a variety of extra-intestinal manifestations. We reported a 55 years old male patient presented by leg thrombosis years before developing florid intestinal manifestations of IBD.

After a detailed history taking and investigations, the patient proved to have ulcerative colitis with extensive spondylosis of the spine and had thrombocytosis. The condition of the patient improved on medical therapy for ulcerative colitis plus anti-platelet drug.

## INTRODUCTION

Inflammatory bowel disease (IBD), comprise two major disease entities: ulcerative colitis (UC) and Crohn's disease (CD). IBD is associated with a variety of extraintestinal disorders, including thromboembolism (TE), arthritis, ankylosing spondylitis, uveitis, pyoderma gangrenosum, and erythema nodosum[1].

## CASE PRESENTATION

A 55 years old male patient presented to the outpatient clinic with dizziness, fatigue, weakness, mild attacks of haematochezia. On detailed history taking the patient gives troublesome since 1996, when he developed severe pain in the left lower limb below the knee and diagnosed as thrombosis, 6 months later the patient developed numbness and tingling in both lower limbs and the patient improved after hospital admission, 2 months later he experienced the same complain, he consulted a neurosurgeon, he was examined by MRI and diagnosed as having L2-3 disc prolapse and operated upon,

when the patient visited our outpatient clinic due to the previous non-specific symptoms he was investigated by CBC that showed HB: 6 gm/dl (microcytic hypochromic), WBCs :18x10<sup>3</sup>/dl (mainly neutrophils), platelets :1066x10<sup>3</sup>/dl, reticulocytic count (R.C): 3%, serum iron : 53.7 ug/dl, serum ferritin: 11.4 ng /ml, bone marrow aspiration was hypercellular with increased megakariocytes series in immature and dwarf forms, while myeloid and erytheroid series were normal, kidney functions were normal, liver functions apart from low albumin level (2.8 gm/dl) were normal, ESR>100, stool analysis showed pus and mucus, urine analysis was normal, US examination revealed mildly enlarged homogenous liver, CT scan detected enlarged para-tracheal lymph nodes. The patient was discharged to be followed and prescribed aspirin 150 mg daily as antiplatelet aggregation drug. On revision the patient was still complaining of small recurrent attacks of haematochezia and still had high platelet count, decision was taken to examine the patient

by colonoscopy, findings revealed interno-external piles, severe inflammation and ulceration seen all over the rectum, descending and transverse colon, multiple biopsies were taken for histopathological examination that revealed microscopic picture of ulcerative colitis (focal surface sloughing, the glands showed goblet cell depletion and reduced mucous secreting activity with early crept abscesses formation with laminal edema and inflammation). The condition of the patient was controlled by blood transfusion, antibiotics, salazopyrine 500 mg 2 tablets/ 8 hours, prednisone 60 mg/day that later tapered gradually over 2 weeks. One month later the patient suddenly stopped salazopyrine and experienced bleeding per rectum and abdominal pain, upon giving the original dose

salazopyrine the condition controlled. Few months later the patient admitted due to exacerbation in the form of severe abdominal pain and distension, dizziness, tingling and numbness of both lower limbs, the condition was controlled by antibiotics, steroids and salazopyrine, CBC showed mild polymorphonuclear leucocytosis ( $11.5 \times 10^3$ ), mild microcytic hypochromic anemia (10.1g/dl), thrombocytosis ( $829 \times 10^3$ ), kidney functions were normal, serum albumin level was low (1.9 gm/dl), to evaluate the numbness of both lower limbs, lumbar X-ray was done and followed by X-ray examination of the cervical spine both showed spondylitic lesions, osteophytes and calcified disc spaces but no soft tissue pathology was noticed.

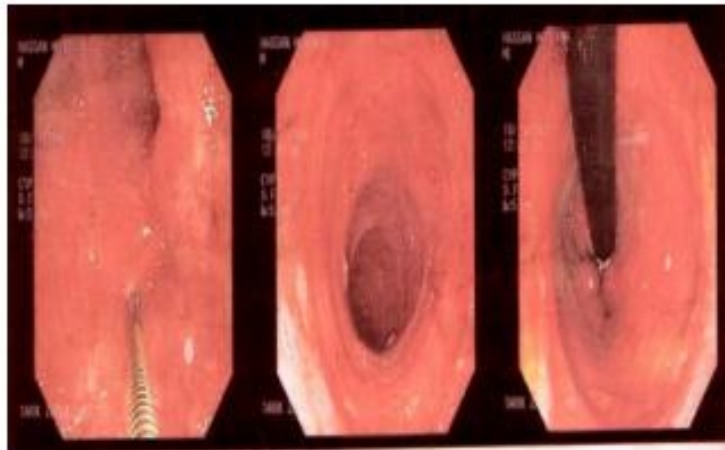


Fig 1: Colonoscopic findings of ulcerative colitis.

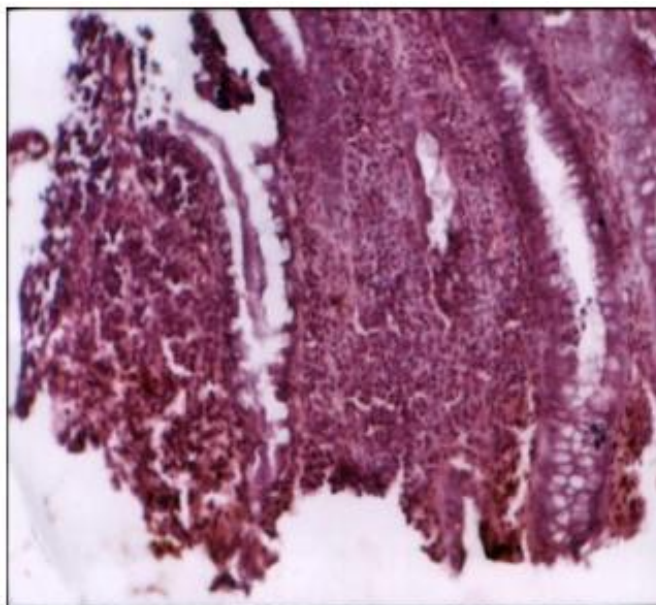


Fig 2: Histopathological findings of UC



Fig 3: X-ray lumbar spine shows extensive spondylosis.

## DISCUSSION

Prevalence of TE in IBD, varies between 1.2% and 6.7% in clinical studies and rising to 39% in postmortem studies. IBD found to be a 3.6 fold higher in the risk of TE compared with controls matched for age and sex. This represents a relevant extra-intestinal complication of IBD, including life threatening pulmonary embolism[2]. The mechanism of enhanced pro-coagulant activity is not understood. Thrombosis in inflammatory bowel disease is important because it occurs in a young population, often in unusual sites, and has a high mortality. The development of thrombosis is related to active inflammatory disease in most patients with Crohn's disease but apparently not in those with ulcerative colitis[3]. In IBD, the platelet count correlates with disease activity, high counts are more likely associated with severe UC than with mild disease[4], this coincide with our case. In our patient thrombocytosis and thrombotic diathesis preceded florid colonic manifestations by a long period of time, this should raise the suspicion of IBD in causes of acquired thrombophilia even in absence of colonic manifestations. Spontaneous platelet aggregation is common with IBD but there is no correlation with disease activity. Patients with IBD have abnormal platelet activity, which may contribute to the inflammatory process[5]. Inflammatory arthropathies are the most common

extraintestinal manifestations in IBD patients with a prevalence ranging between 7% and 25% [6]. Articular and musculoskeletal manifestations are included in the spondyloarthropathies (SpAs) that are a group of seronegative autoimmune related disorders with common characteristics including: ankylosing spondylitis, reactive arthritis, psoriatic arthritis, inflammatory bowel disease, some forms of juvenile arthritis and acute anterior uveitis[7]. Articular involvement (peripheral or axial) can precede, be synchronous or begin afterward the diagnosis of IBD, it is characteristically pauciarticular, asymmetrical, transitory, migrating, prevalently non deforming. This occurred in our patient, where he consulted a neurosurgeon and operated upon for disc prolapse with unsatisfactory improvement. The axial involvement can vary from asymptomatic sacroiliitis to inflammatory lower back pain to ankylosing spondylitis (that occurs in 3% of IBD patients)[6]. Ankylosing spondylitis (AS) affects the vertebral column by progressive ankylosis of the vertebral facet joints and the sacroiliac joint. The prevalence of AS in IBD (1%-6%) is higher than in the general population (0.25%-1%)[8]. Bacteria and gut inflammation seem to play an important role in the pathogenesis of AS. Interestingly, ileocolonoscopy in patients with idiopathic spondylarthropathies reveals ileal inflammation in more than two thirds of patients[9]. The

clinical course of AS in IBD is similar to idiopathic AS, and disease progression leads to increasing immobility of the spine resulting in ankylosis (bamboo spine). Secondary to reduced chest expansion, poor lung expansion with fibrosis and dilatation of the aortic root can occur. AS is associated with peripheral arthritis in about 30% of patients and with uveitis in 25% of patients[10]. Although in our patient mobility disorders were evident, no pulmonary or aortic damage were seen.

In conclusion: Thrombocytosis and thrombosis may precede colonic manifestations and add to the risk of TE in IBD, while extensive spondylosis may complicate IBD and results in marked disability.

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## Video Case: Bilharzial Polyposis

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### COMMENT

A 23 years male Egyptian patient from rural area( Kafr Sakr) in Sharkia Governorate presented with bleeding per rectum for 3 months. Digital rectal examination revealed firm rectal masses. Colonoscopy showed multiple rectal and sigmoid polypoidal masses . Pathological examination of biopsies from these masses found bilharzial ova within chronic inflammatory cells.

## Image case: Aggressive Benign Gastric Ulcer

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### COMMENT

This 48 years old male patient presented with 2 months history of persistent vomiting and epigastric pain and unsatisfactory response to multiple courses of proton pump inhibitors. He had no chronic medical disease nor GIT bleeding. On examination he was pale with epigastric tenderness, otherwise free. Investigations showed mild microcytic hypochromic anemia. Diagnostic upper endoscopy was done and showed GERD with huge active gastric ulcer with marked mucosal congestion and inflammation, necrotic floor and edematous edge, for the first time it was suspected to be malignant (image 1), multiple biopsies were taken. Biopsy result showed moderately active chronic H Pylori with intestinal metaplasia. Treatment included two days of parenteral PPI, then two weeks of triple therapy followed by four weeks of oral omeprazole 40 mg/day. Follow up one month later showed marked improvement (image 2). Thus it could be concluded that apparently aggressive lesions are not always malignant, any suspicious lesion should be biopsied , treatment of H Pylori promotes healing of gastric ulcer and follow up of endoscopic healing is advised.



