A Silent Gut disease with a loud Hepatic voice

"A clinical case"

Supervised by:

Dr: Hussam Aldeen Al shaikh

Presented by:

Dr:Ahmad Helal

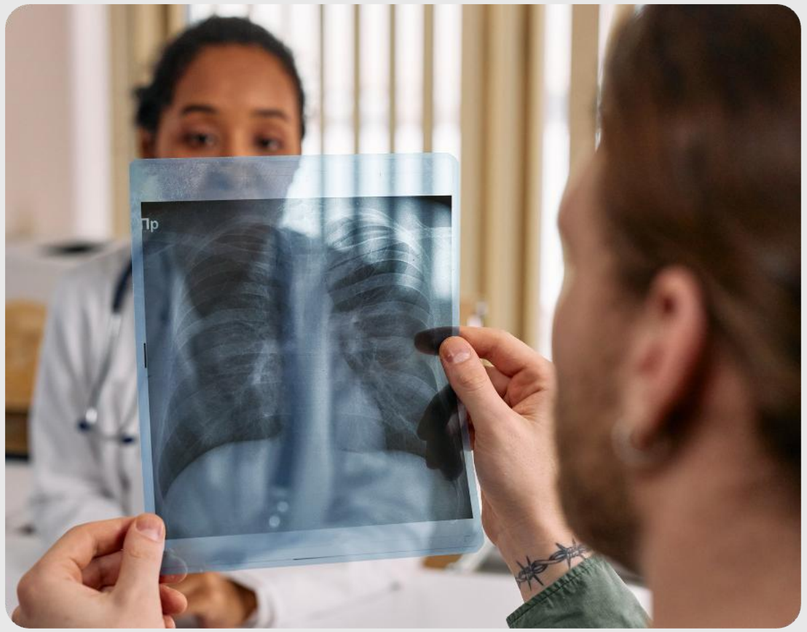
Dr: Mohammed Abdulrahman

Overview

- Case presentation
- Clinical examination
- Investigations
- Discussion
- Management

Case Presentation:

- A 49-year-old female presents with generalized fatigue, weakness.
- Non specific abdominal pain, weight loss, and abdominal bloating.
- She has a history of unexplained iron deficiency anemia.
- Over the past several months, she has developed persistent diarrhea



Clinical examination

The Examination Was Unremarkable Except For Conjunctival Pallor.

Abdominal Examination Revealed Palpable Splenomegaly.

No Tender Points Were Noted.

Interventional History:

Upper Gastrointestinal Endoscopy Was Performed And Showed Normal Results??.

The Vital Sign:

Blood Pressure: 90\70 mm Hg

HR:100 bpm

Spo2:98%

Initial Laboratory Test

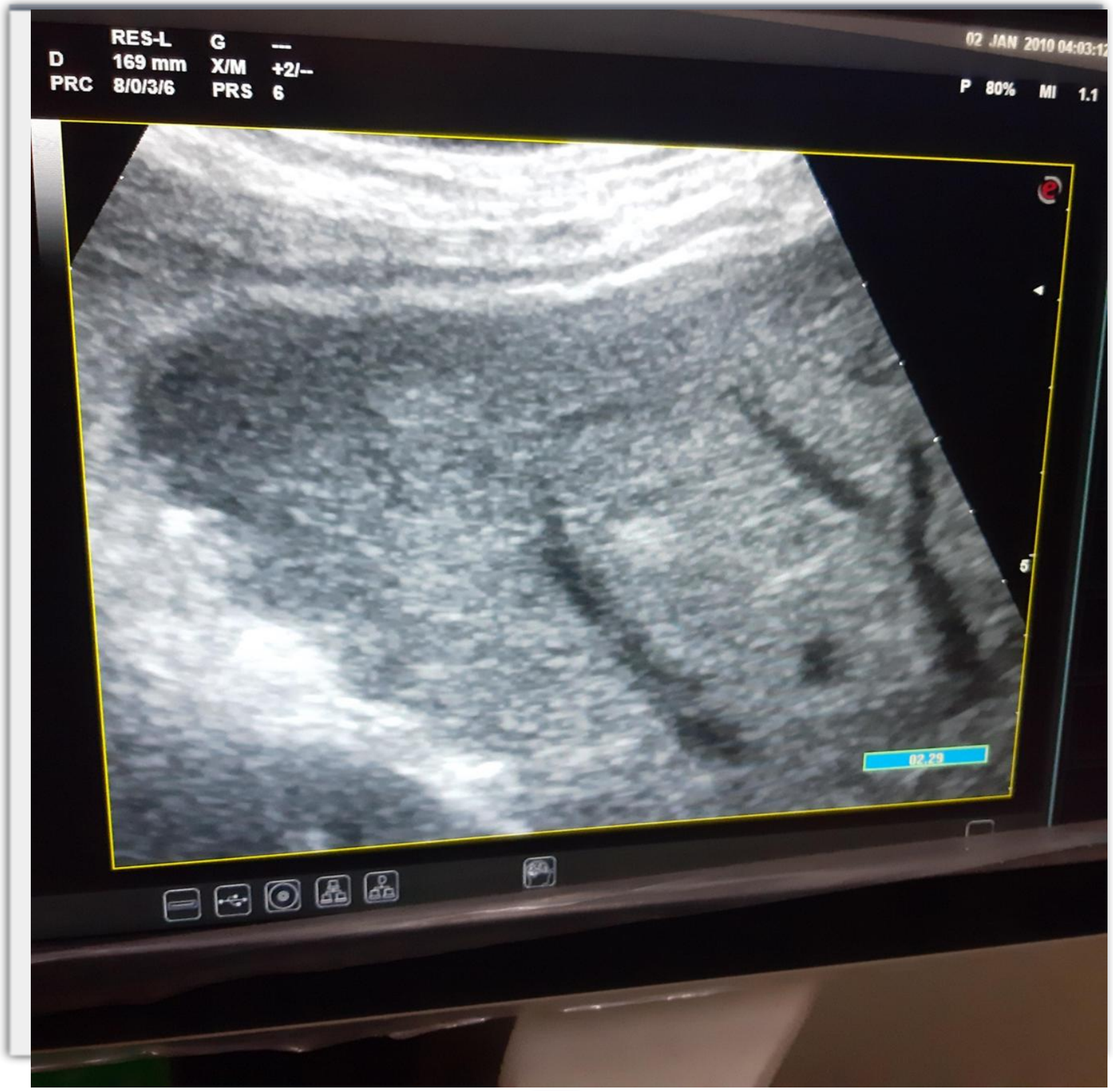
TEST	RESULT
Hemoglobin (Hb)	8 g\dl
MCV	71 FL
Platelets	67000
Whight Blood Count (WBC)	3500
TSH	2.6
TIBC	564
Ferritin	12

Abdominal pain+ IDA + WIGHT LOSS+ Diarrhea

NORMAL TSH

pancytopnea + splenomegaly

ASK FOR ABDOMIAL ULTRASOUND


RES-L
169 mm
8/0/3/6
G
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6
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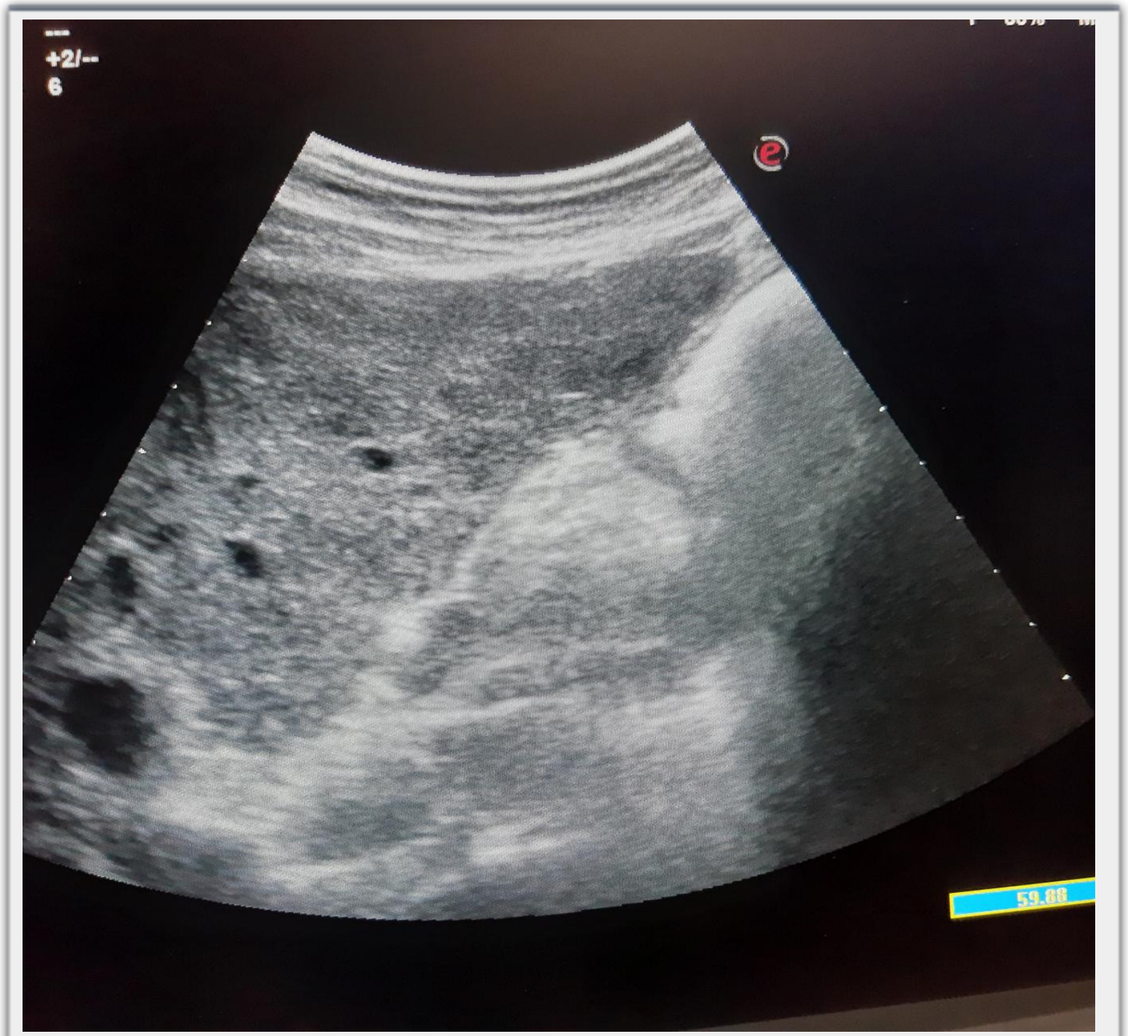
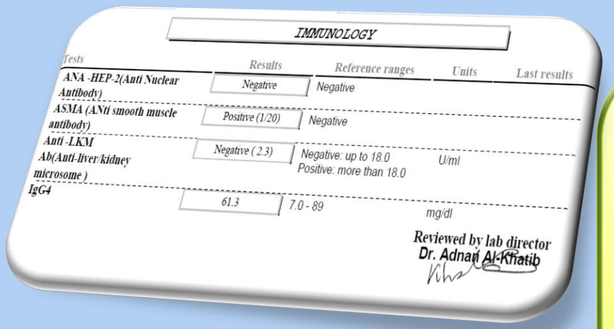
P 80%
MI 1.1
02.29


+2/--
6
59.88



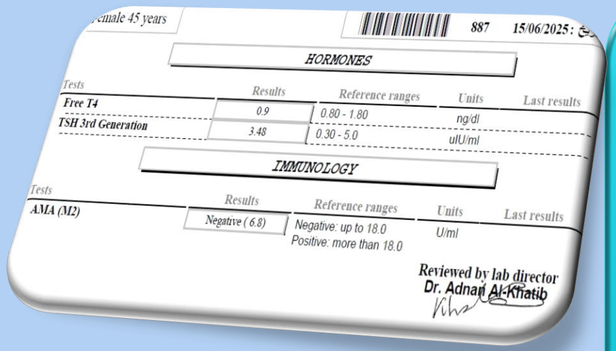
IMMUNOLOGY

Tests	Results	Reference ranges	Units	Last results
ANA -HEP-2(Anti Nuclear Antibody)	Negative	Negative		
ASMA (ANti smooth muscle antibody)	Positive (1/20)	Negative		
Anti -LKM Ab(Anti-liver/kidney microsome)	Negative (2.3)	Negative: up to 18.0 Positive: more than 18.0	U/ml	
IgG4	61.3	7.0 - 89	mg/dl	

Reviewed by lab director
Dr. Adnan Al-Khatib

Hepatic profile

- ALT:25U\L
- AST:34U\L
- INR:1,7
- PT:40%
- Albumin: 2.9 g\dl



...emale 45 years

887 15/06/2025:

HORMONES

Tests	Results	Reference ranges	Units	Last results
Free T4	0.9	0.80 - 1.80	ng/dl	
TSH 3rd Generation	3.48	0.30 - 5.0	uIU/ml	

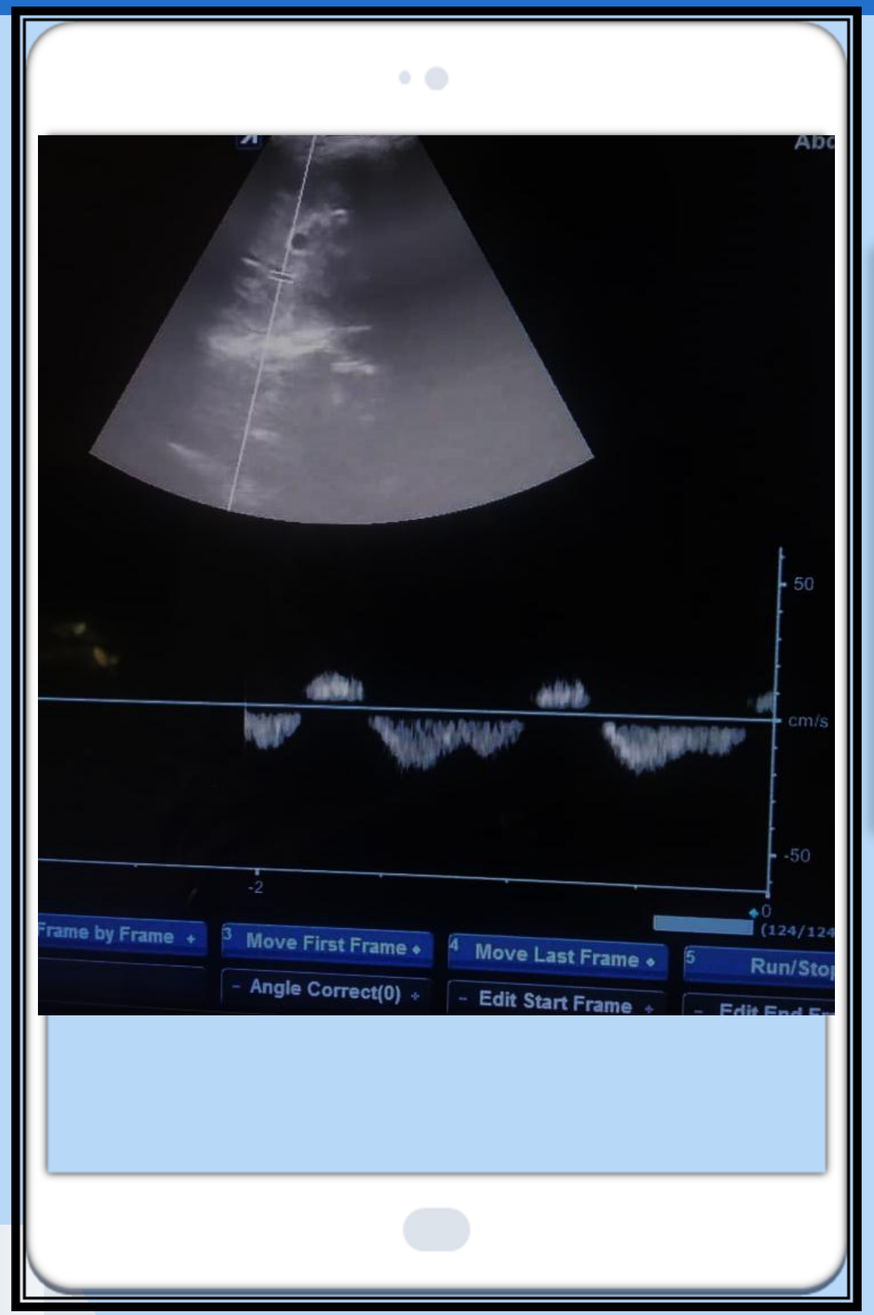
IMMUNOLOGY

Tests	Results	Reference ranges	Units	Last results
AMA (M2)	Negative (6.8)	Negative: up to 18.0 Positive: more than 18.0	U/ml	

Reviewed by lab director
Dr. Adnan Al-Khatib

Causal Investigation

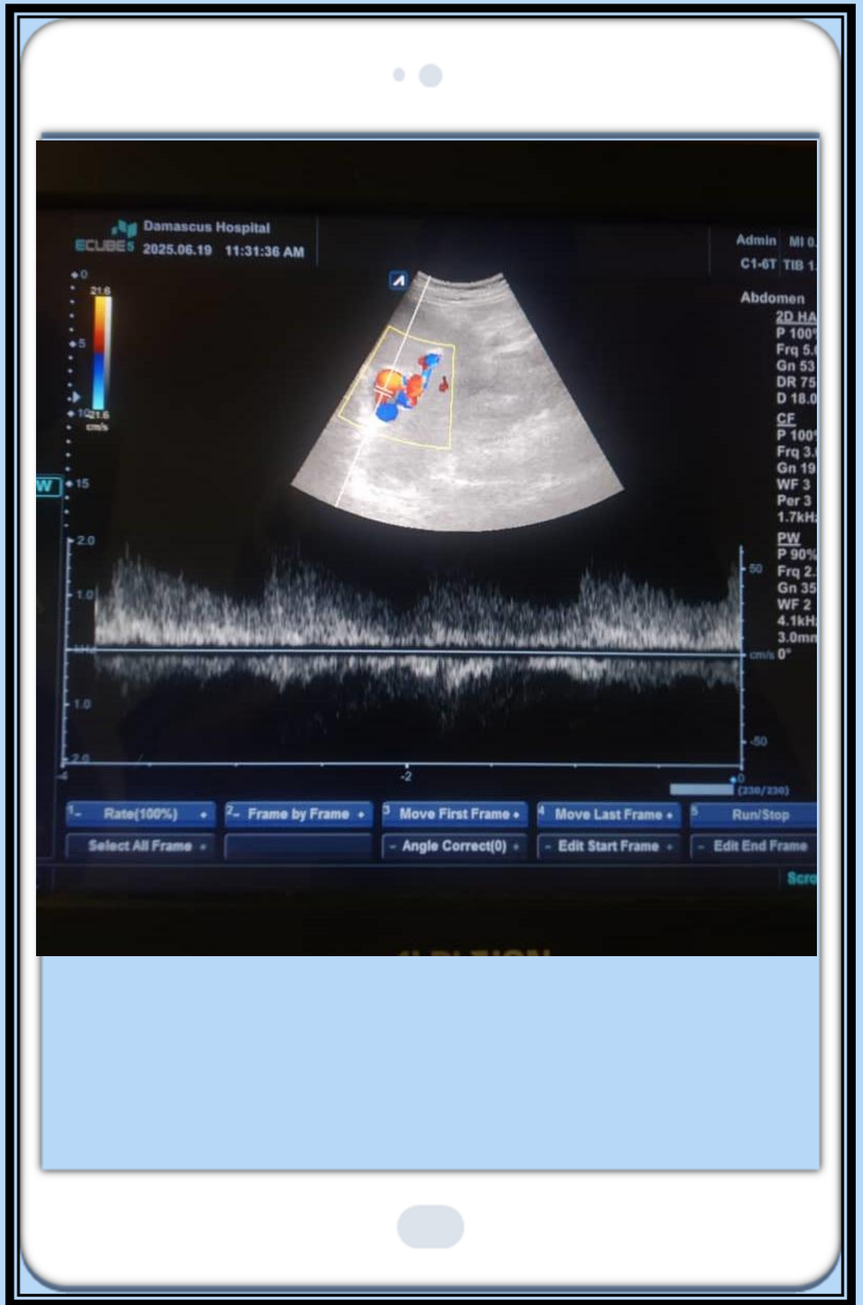
- HBS Ag,Anti HBc,Anti HCV
- Hemochromatosis,wellson’disease
- a-alpha-1 anti trypsin
- AIH(ANA,ASMA,Antil LKM-1,IGg4)
- AMA.M2

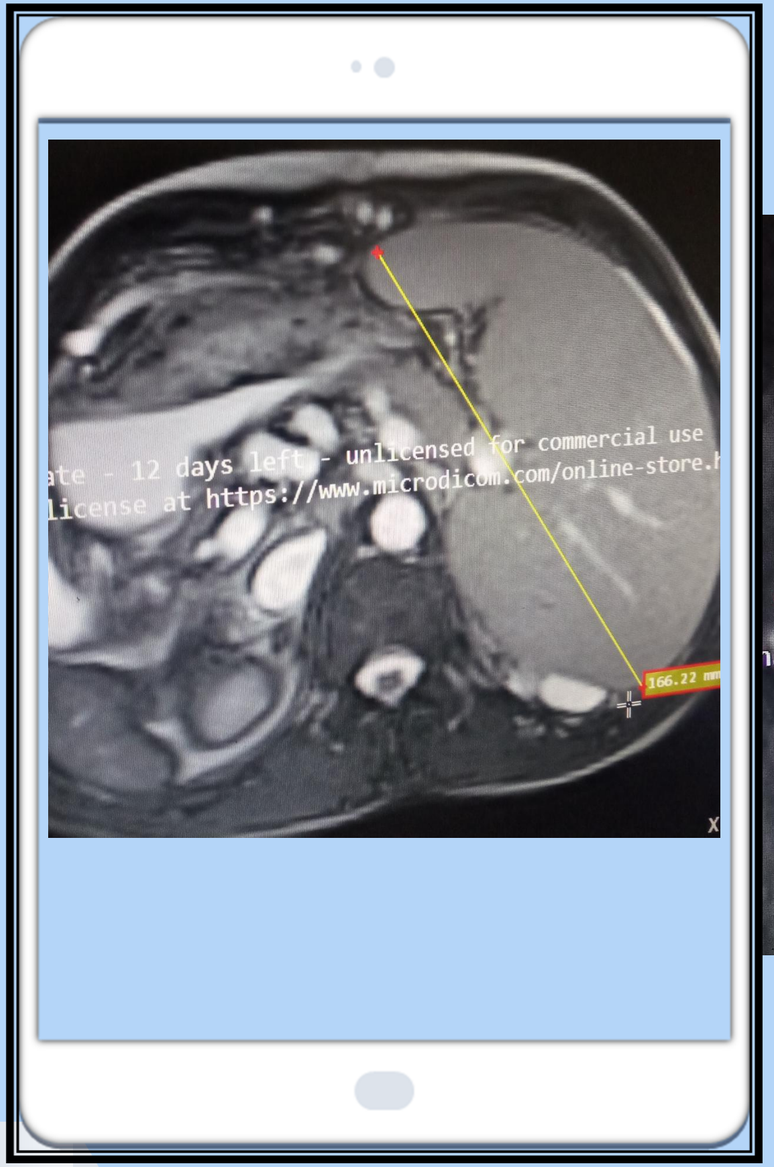


المرارة: طبيعية الجدار دون استسقاء أو حصيات.

الطحال: ضخامة طحالية ١٦ سم طبيعي الشكل والتجانس خال من الآفات الصدوية.

- **الأوردة الكبدية:** تبدو طبيعية الارتسام (ثلاث أوردة) ذات شكل موجة طبيعي.
- **وريد الباب:** قطره ضمن الطبيعي وفرعه الرئيسي الأيسر تبدو سالكة اللمعة طبيعية القطر مع اتجاه جريان طبيعي وشكل موجة وسرعات طبيعية ، الفرع الرئيسي الأيمن مستدق بسرعة ٦ سم /ثا قد يكون في سياق خثار سابق للربط مع السوابق ، علامات دوران جانبي بسرة الكبد وعود تغفي للوريد السري .
- **الوريد الطحالي:** طبيعي القطر مع اتجاه جريان طبيعي وشكل موجة وسرعة جريان طبيعية ، مع علامات لدوران جانبي بسرة الطحال ، وتوسع تموجة وريدية شريانية يقيس ٢.٢ × ١.٥ سم للربط مع الطبقي المحوري لنفي تشوه وعائي .
- **الشريان الكبدي:** سالك اللمعة، مع شكل موجة وسرعة جريان طبيعية.

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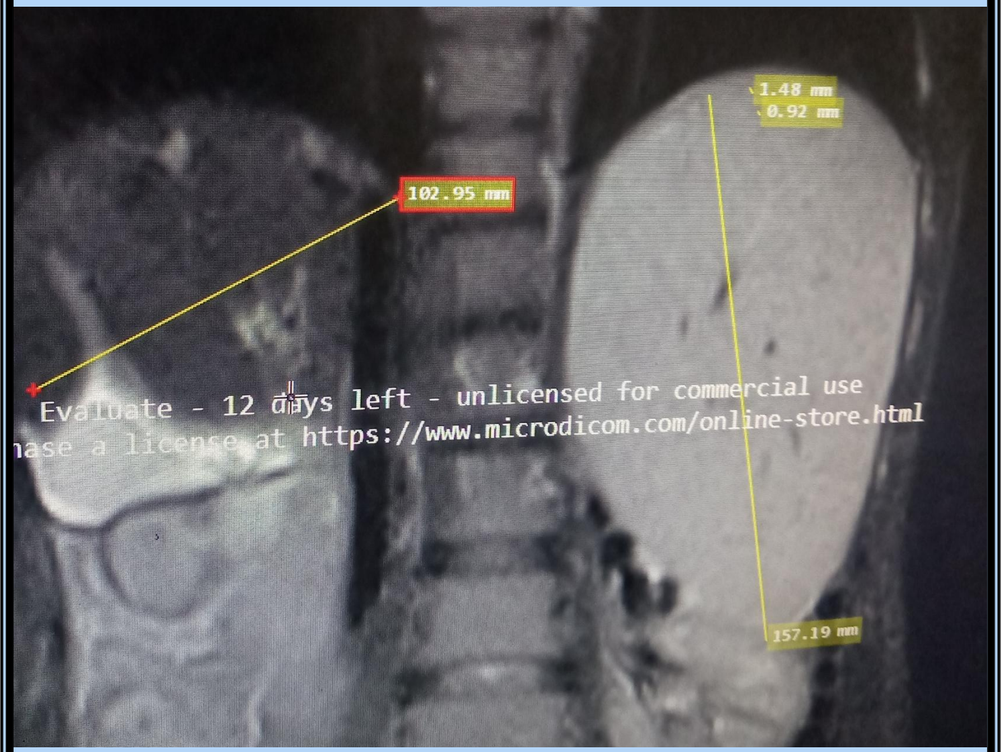
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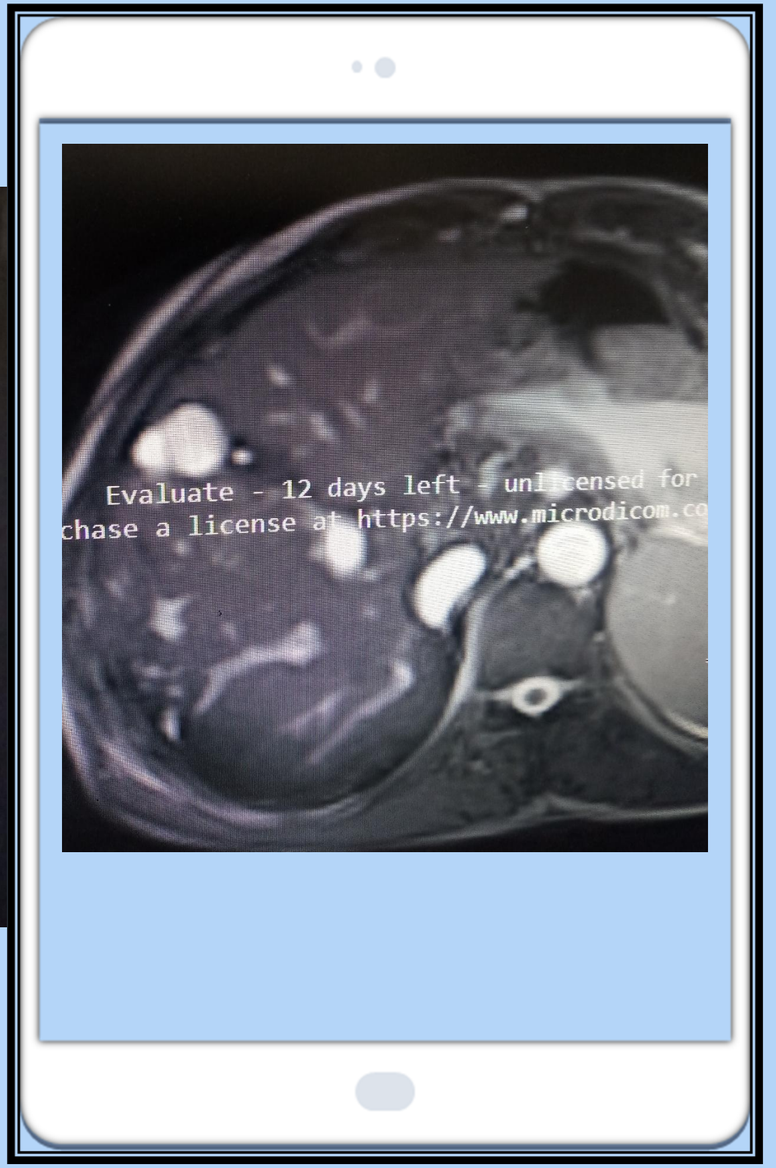
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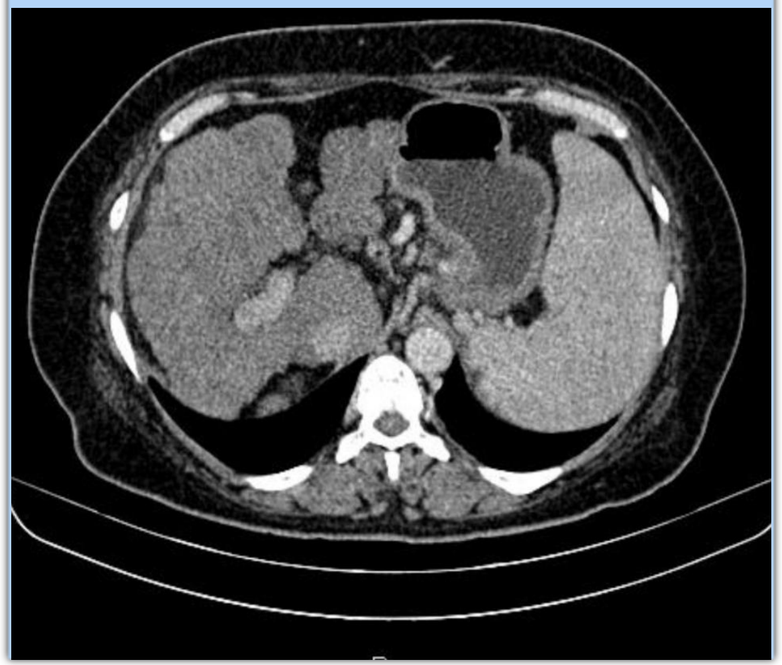
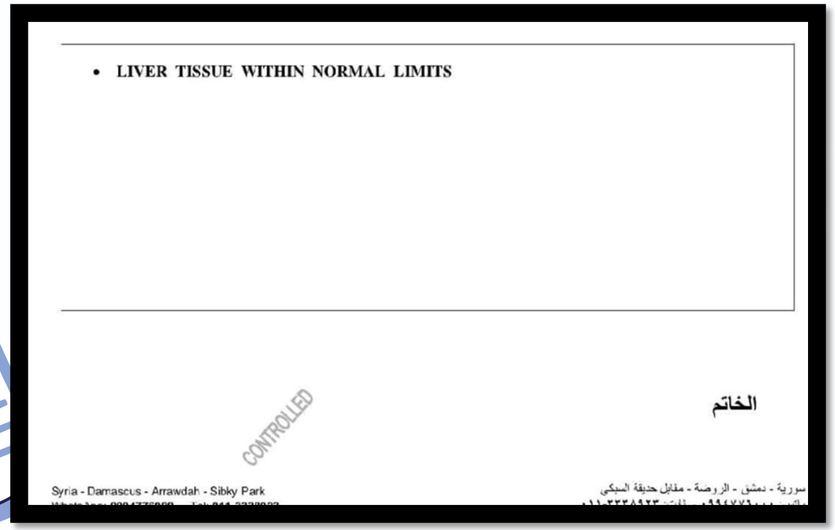


طبقي محوري للبطن والحوض تاريخ قراءة الطبقي 2025/9/15

- البرانشيم الرئوي بالقاعدتين طبيعي دون كثافات عقدية أو سنخية أو خلالية مشتبهة.
- لا يوجد انصباب جنب بالقاعدتين.
- الكبد صغير الحجم ، البرانشيم غير متجانس دون كتل صريحة ، لايوجد توسع بالطرق الصفراوية ، وريد الباب طبيعي مع عودة الجريان بمستوى الرباط المدور ، الأوردة فوق الكبد طبيعية .
- المرارة جدرها رقيقة منتظمة دون وجود حصيات متكلسة .
- البنكرياس قياساته طبيعية ، البرانشيم متجانس، الحواف منتظمة.
- الطحال يبدي ضخامة متجانسة (17×9×15سم) مع توسعات وريدية بسرة الطحال حول الكلية اليسرى خاصة بالقسم العلوي منها ومن الناحية الشرسوفية بشكل خفيف أسفل المري .
- الكليتان طبيعيتان بالقياسات و سماكة القشر و انتظام الحواف ، دون استسقاء أو حصيات صريحة
- المثانة جدرها رقيقة منتظمة ، غير محصاة ،بدون رتوج أو بوليبات .
- الأوعية الكبيرة خلف البريتوان طبيعية .
- لا يوجد ضخامات عقدية حرقفية أو خلف البريتوان .
- كمية قليلة من سائل حر بين العرى الحوضية .
- العرى المعوية تبدو طبيعية من حيث قطر اللمعة و انتظام الجدر.
- تبدلات تنكسية عظمية دون علامات انحلالية أو تصلبية مشتبهة.

Liver Biopsy Challenge



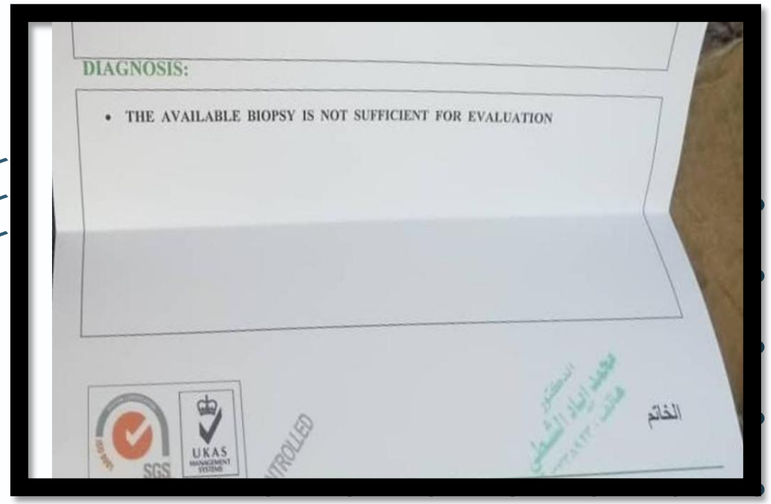
- LIVER TISSUE WITHIN NORMAL LIMITS

الخاتم

CONTROLLED

Syria - Damascus - Arrawdah - Sibky Park

سورية - دمشق - الروضة - مقابل حديقة السبكي



DIAGNOSIS:

- THE AVAILABLE BIOPSY IS NOT SUFFICIENT FOR EVALUATION

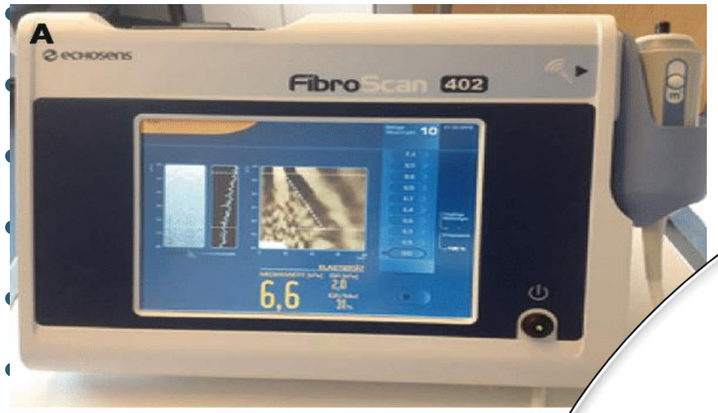
الخاتم

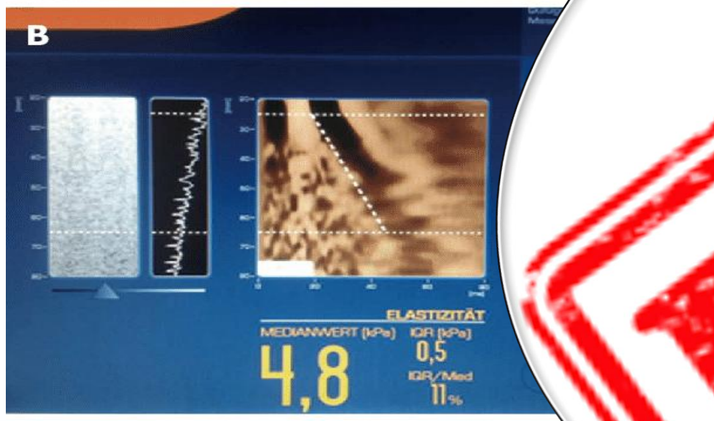
SGS

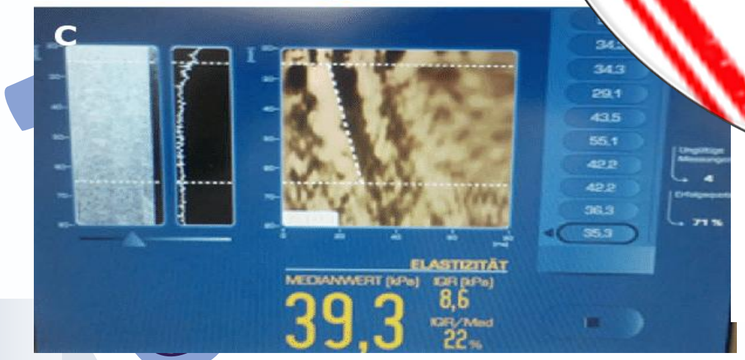
UKAS MANAGEMENT SYSTEMS

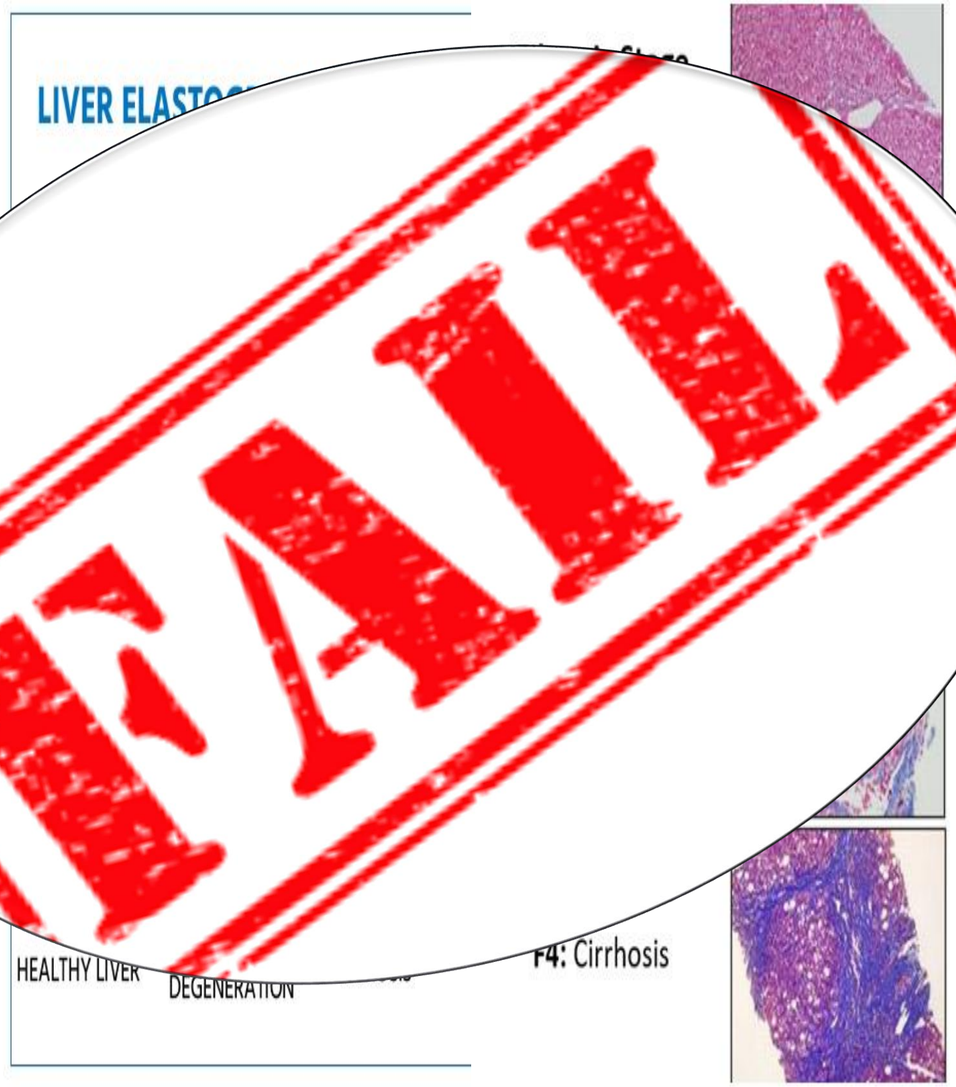
CONTROLLED

Fibroscan,Elastography challenging

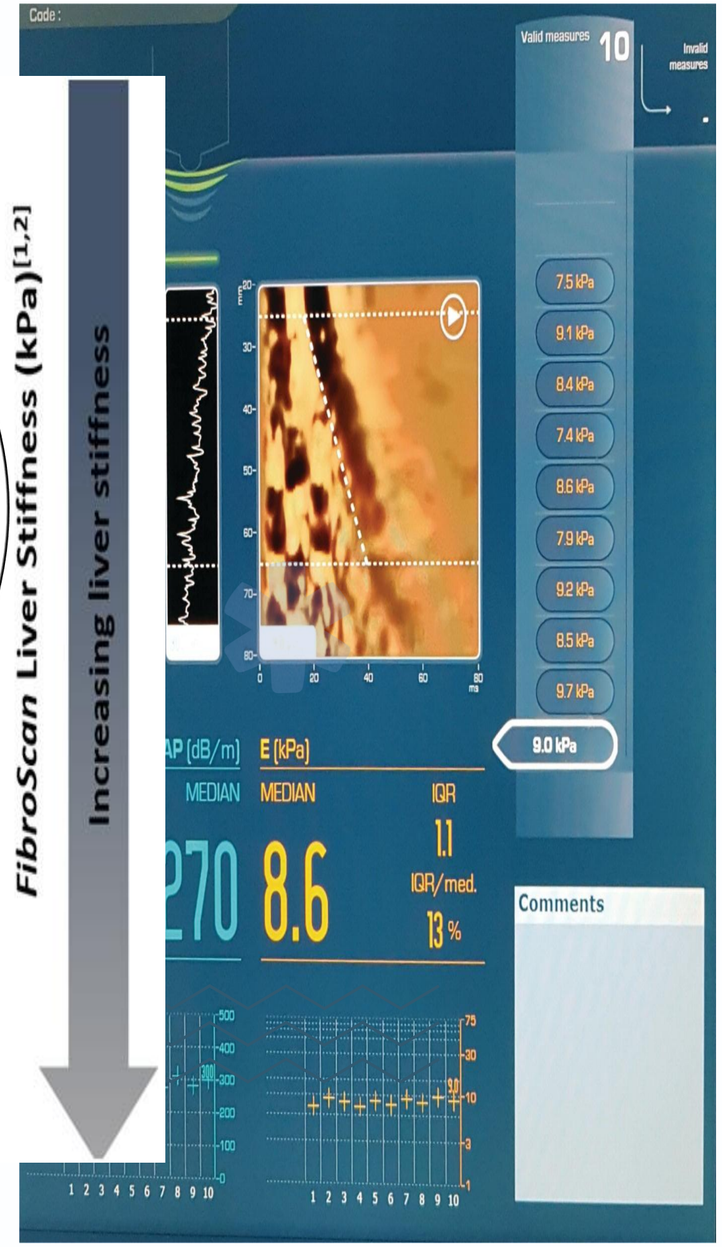








FAIL



$$\text{FIB-4} = \frac{\text{Age (years)} \times \text{AST (U/L)}}{\text{Platelet Count } (10^9/\text{L}) \times \sqrt{\text{ALT (U/L)}}}$$

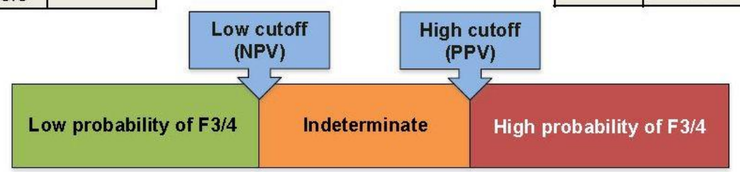
Parameter
Age
AST
ALT
Platelet count
BMI
Albumin
Impaired fasting glucose/diabetes?

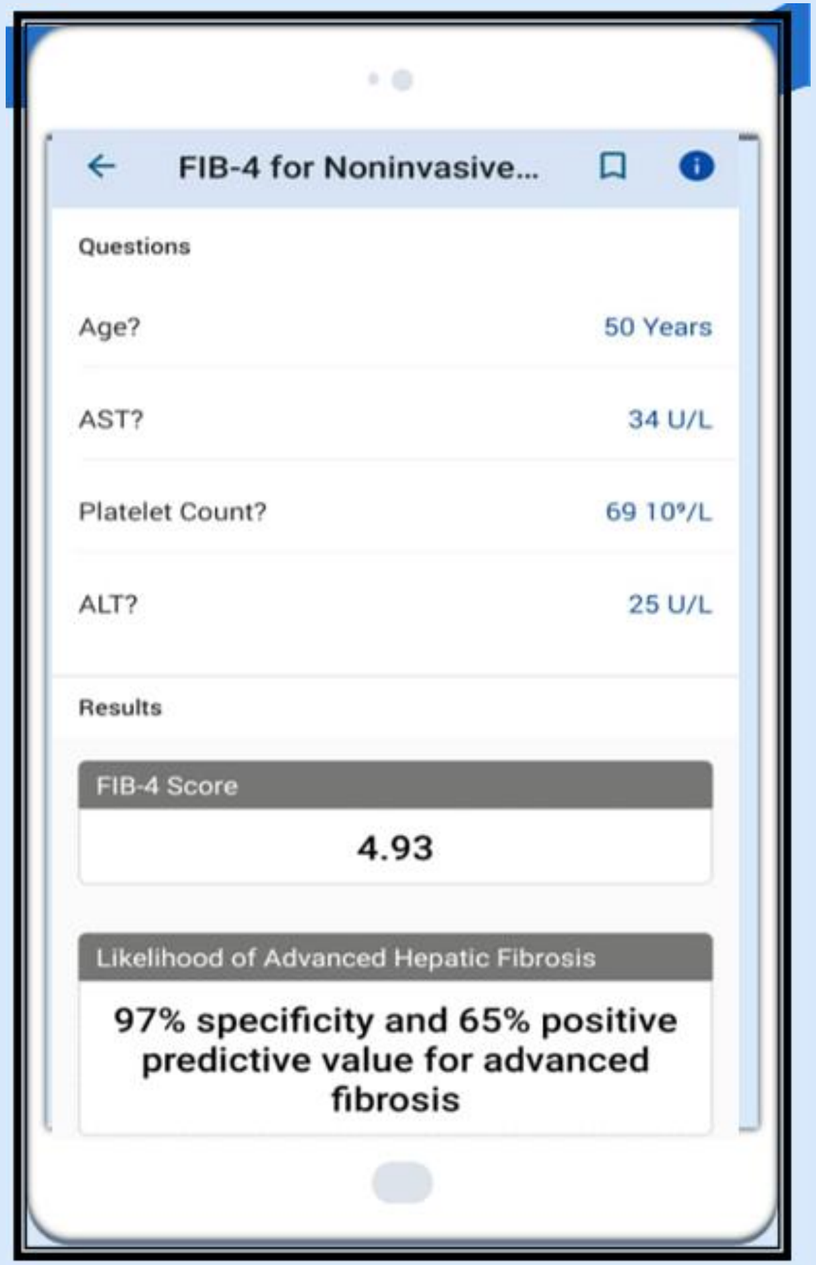
NAFLD fibrosis score

NAFLD fibrosis score[1]	Effect	NPV or PPV, %
<-1.455	Rules out fibrosis	88 to 93
>0.676	Predicts fibrosis	82 to 90

FIB-4 Score

FIB-4 Score [2,3]	Effect	NPV or PPV, %
<1.3	Rules out fibrosis	95
>3.25	Predicts fibrosis	75





Viral
Hepatitis B
Hepatitis C
Hepatitis D

Fatty liver diseases
Alcoholic liver disease
Non-alcoholic
Fatty liver disease

Autoimmune
Autoimmune hepatitis
Primary biliary cirrhosis
Primary sclerosing cholangiitis
IgG4 cholangiopathy

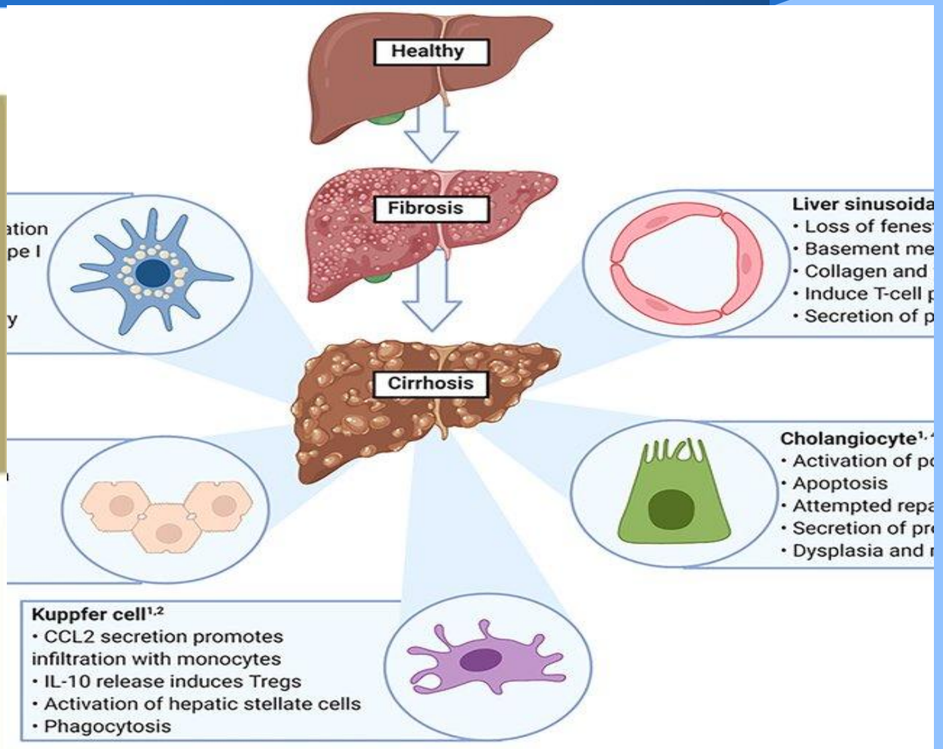
Rare causes
Medications
Porphyria

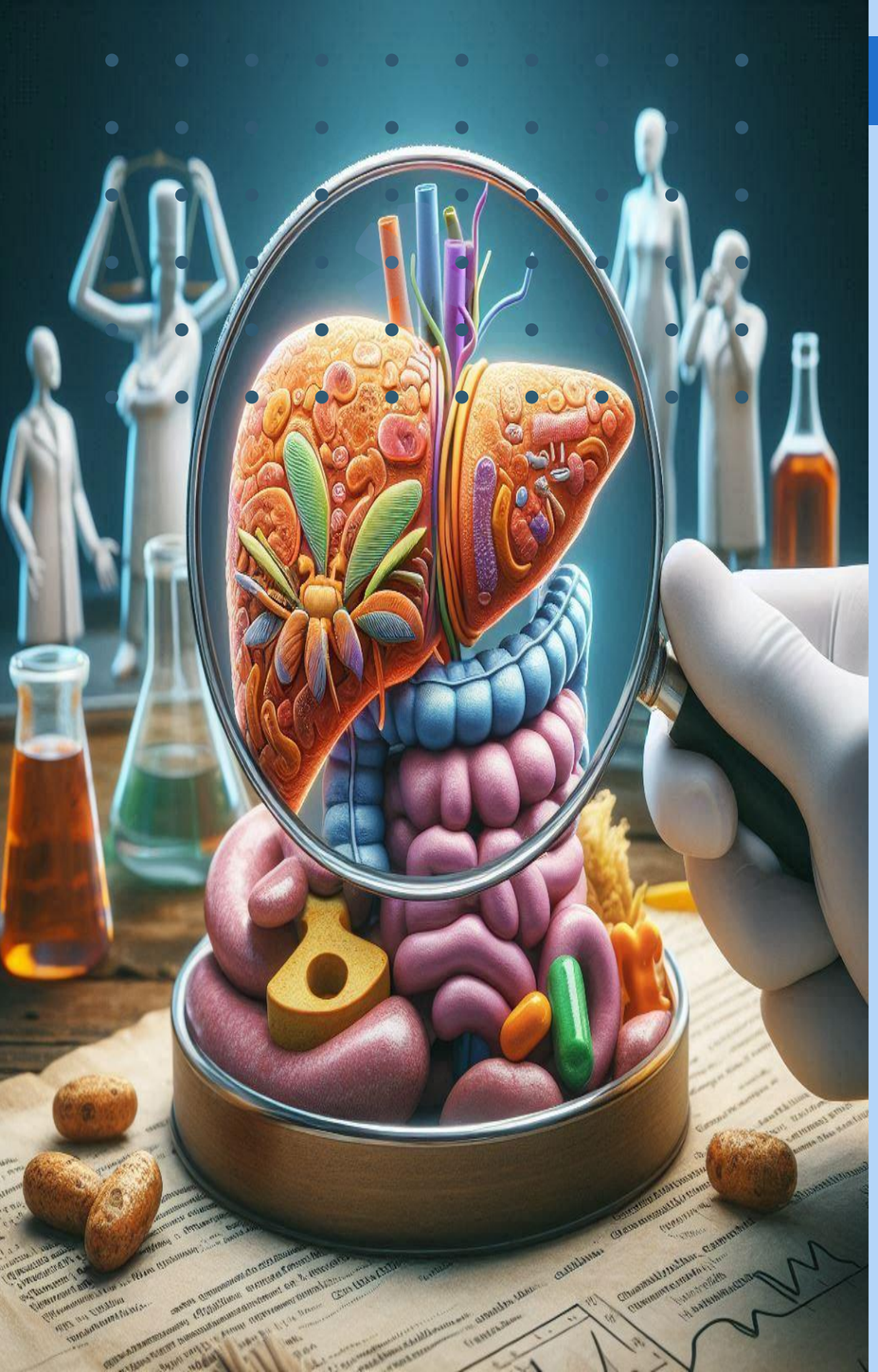
Liver cirrhosis

Chronic biliary disease
Recurrent bacterial cholangitis
Bile duct stenosis

Storage diseases
Hemochromatosis
Wilson disease
Alpha-1-antitrypsin deficiency

Cardiovaskular
Budd-Chiari syndrome
Right-heart failure
Osler disease





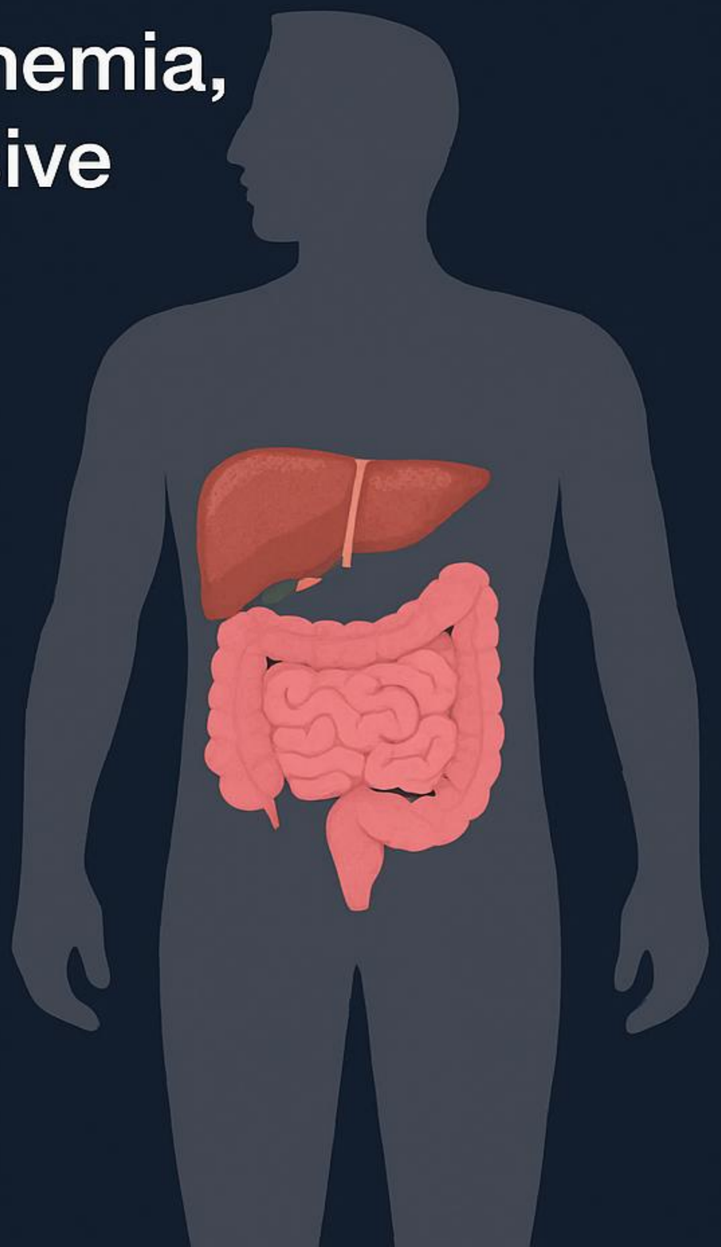
The Turning Point

“From confusion to clarity”

Unexplained iron-deficiency anemia, chronic diarrhea, and progressive weight loss...

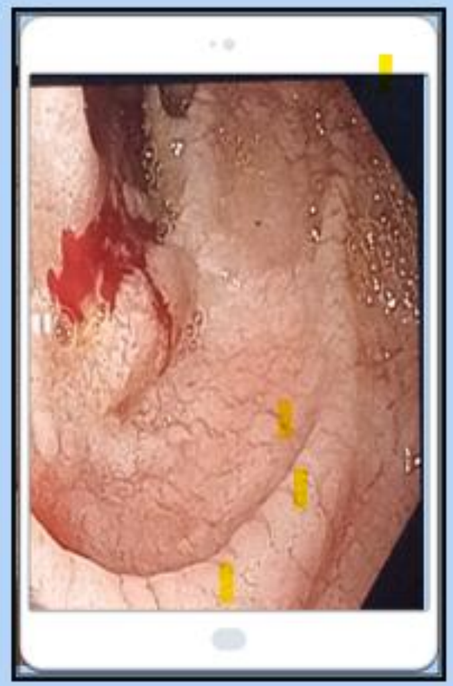
IMMUNOCHEMISTRY

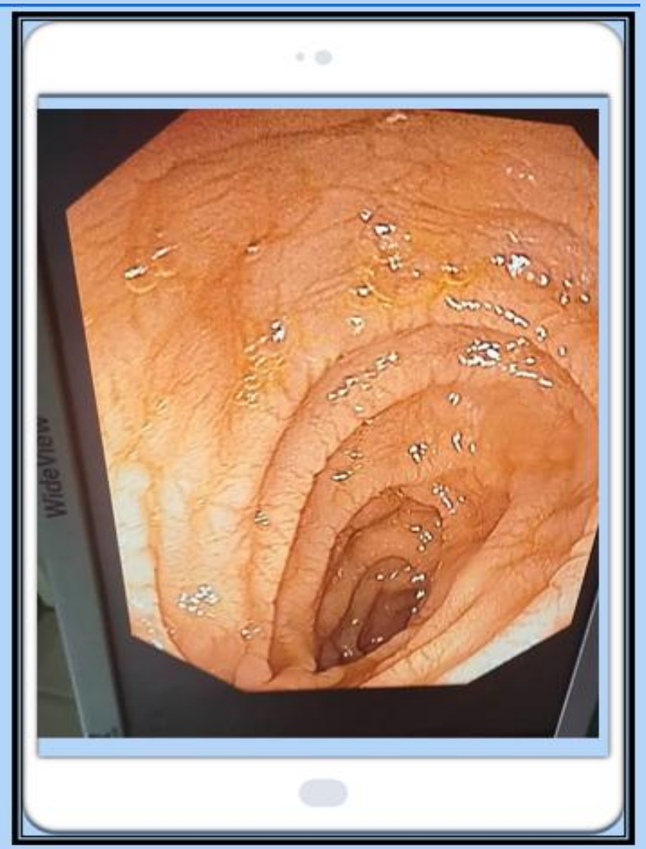
Tests	Results	Reference Range	Units	Last Test
Immunoglobulin A, IgA	371	70-400	mg/dl	
Transglutaminase IgA Ab	Pos : 550	Negative Less Than 20 Positive More Than 20	U/L	

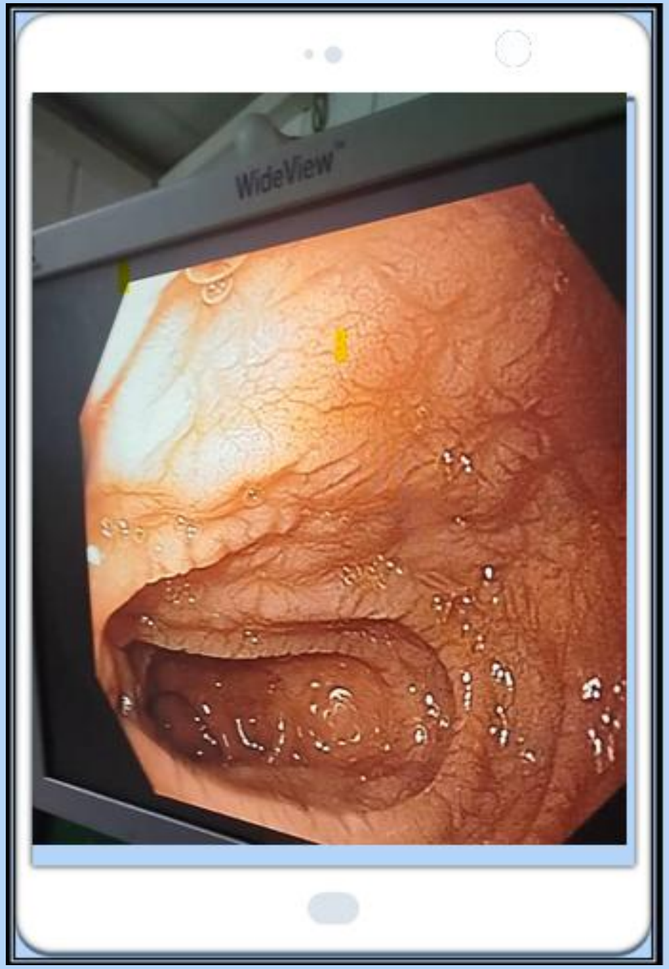


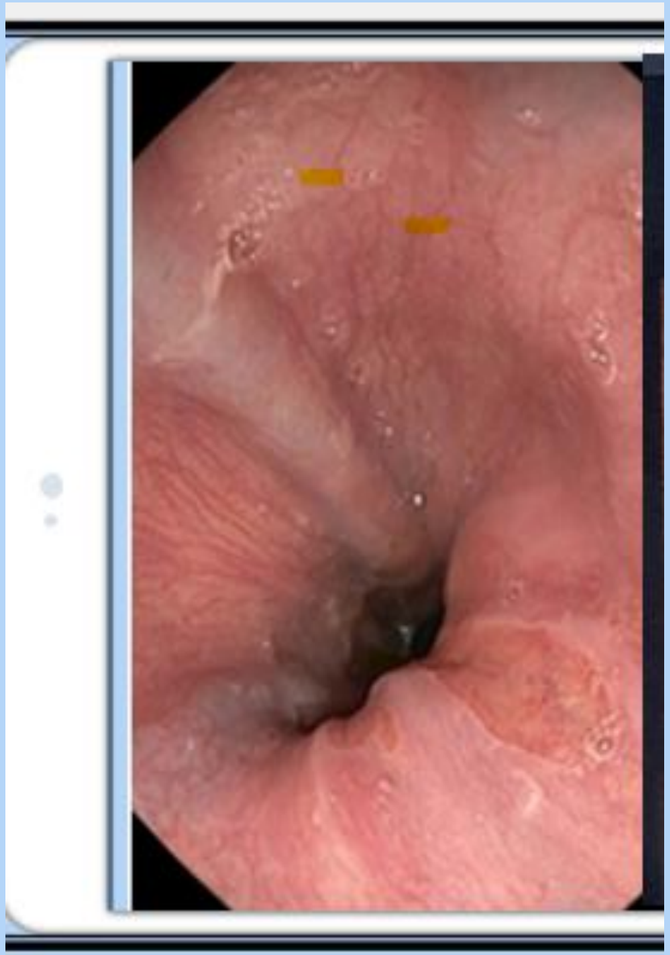
Are we facing the silent monster in the gut?

Serologic and Endoscopic Findings

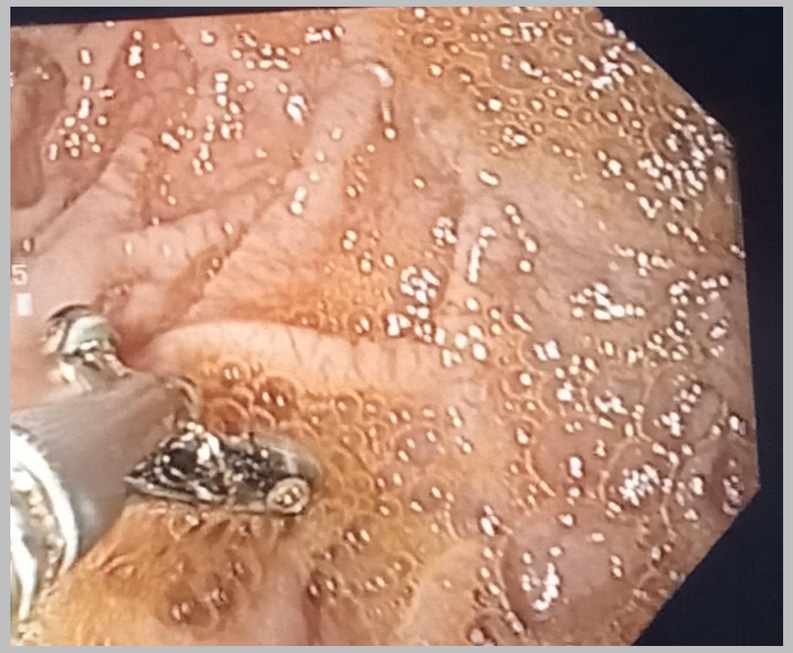


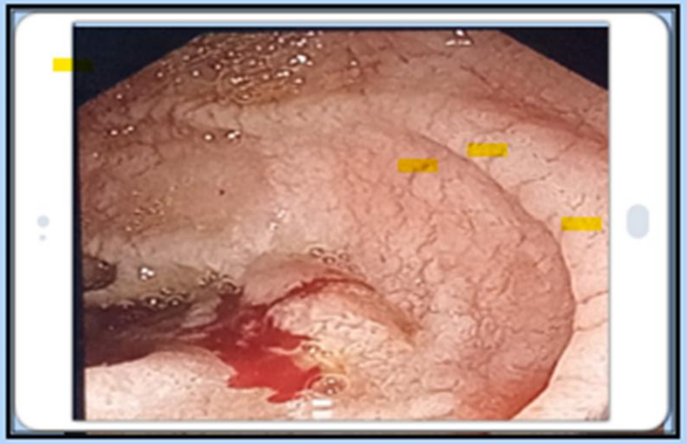




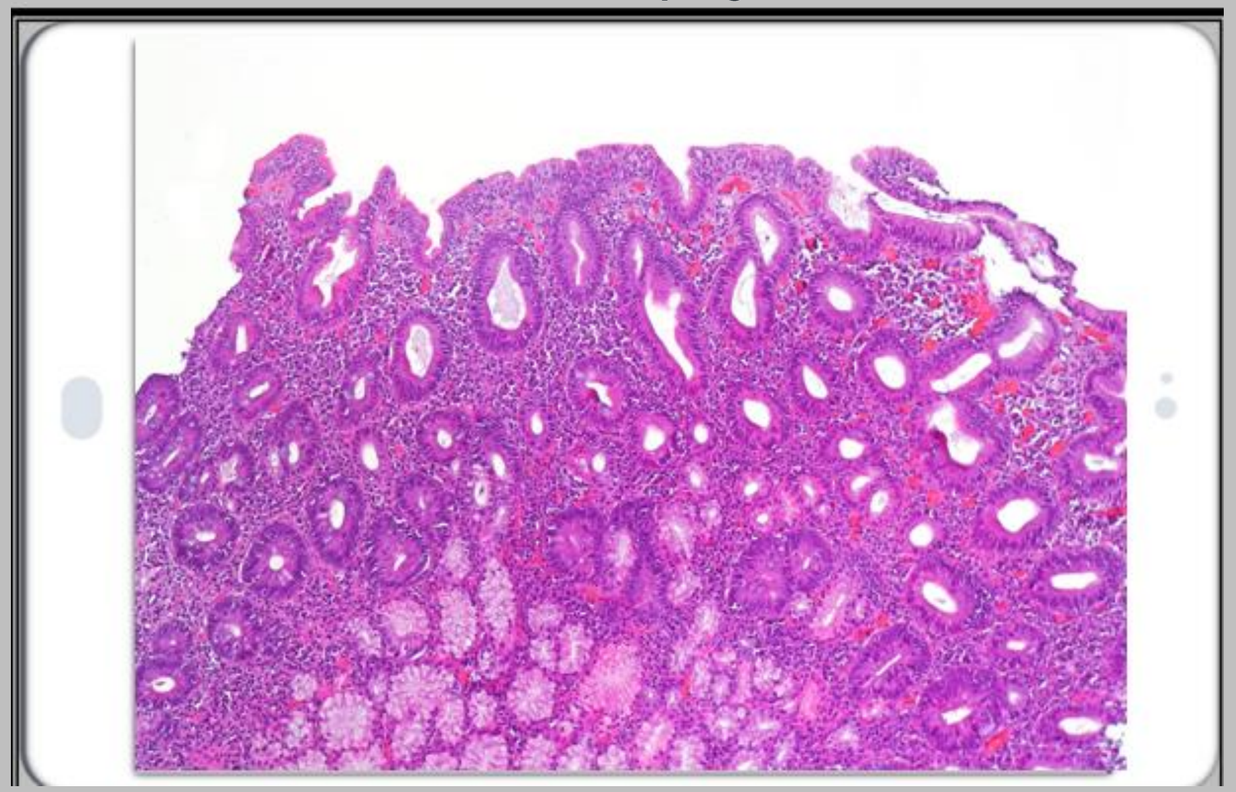


Diagnosis





- Celiac disease
- Partial Villous atrophy :**Marsh 3A**



Celiac disease as a rare cause for cryptogenic cirrhosis

basis of its positive response to gluten-free diet (GFD).

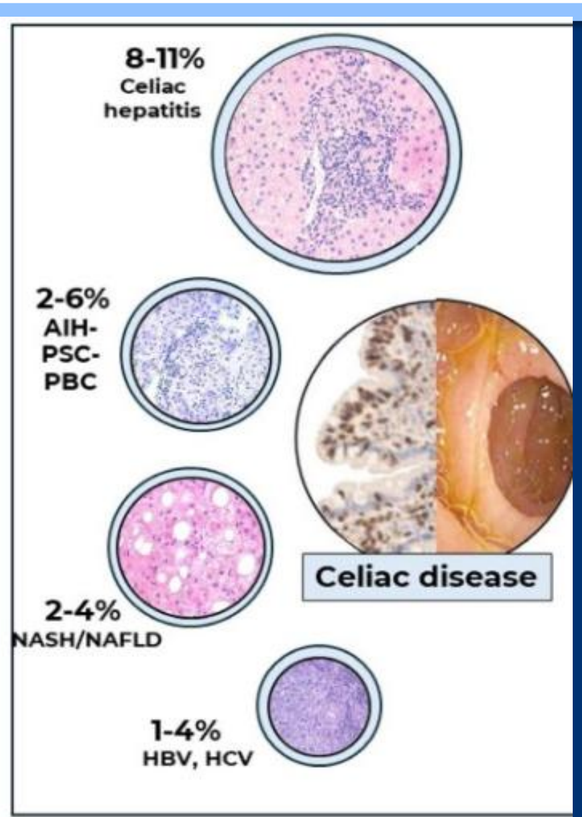
Cryptogenic liver disorders

1. Mild liver damage (gluten-induced hepatitis)

The first report of gluten-induced hepatitis, published in The Journal of Pediatric Gastroenterology and Nutrition in 1986, was the case of a young girl with persistent cryptogenic elevation of serum aminotransferase levels and mild inflammation of the portal tract (26). A diagnosis of CD, suggested in this case by a high titer of antireticulin antibody, was confirmed by duodenal biopsy. Bardella *et al.* performed a similar study and found that 13 (9%) of 140 screened patients tested positive for anti-

2. Severe liver damage

Severe histological diseases, including chronic hepatitis, severe fibrosis, and cirrhosis have been reported in adults and children (14, 25, 31). CD was detected in some patients with severe liver damage of unknown origin, and surprisingly, clinical improvement in the liver condition was noted when the patients consumed a GFD (33-35). The prevalence of CD in patients with chronic liver disease is higher than in the general population. Lindgren et al. reported that in 327 patients with chronic liver disease, the prevalence of CD was 1.5%, which is 15 times higher than that in the general population (36). In a Finnish study, CD was reported in 4 adult patients



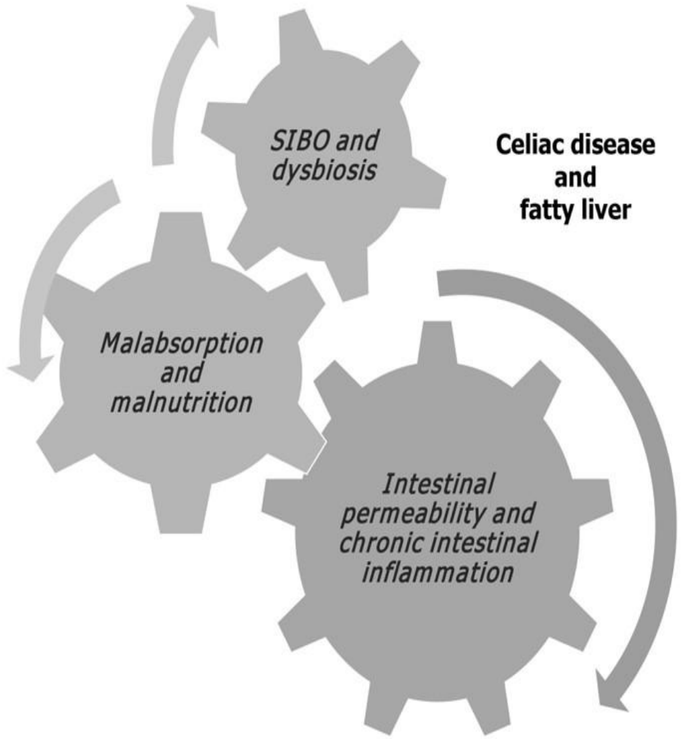


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Figure 1 Pathophysiological mechanisms associated with fatty liver in patients with celiac dis

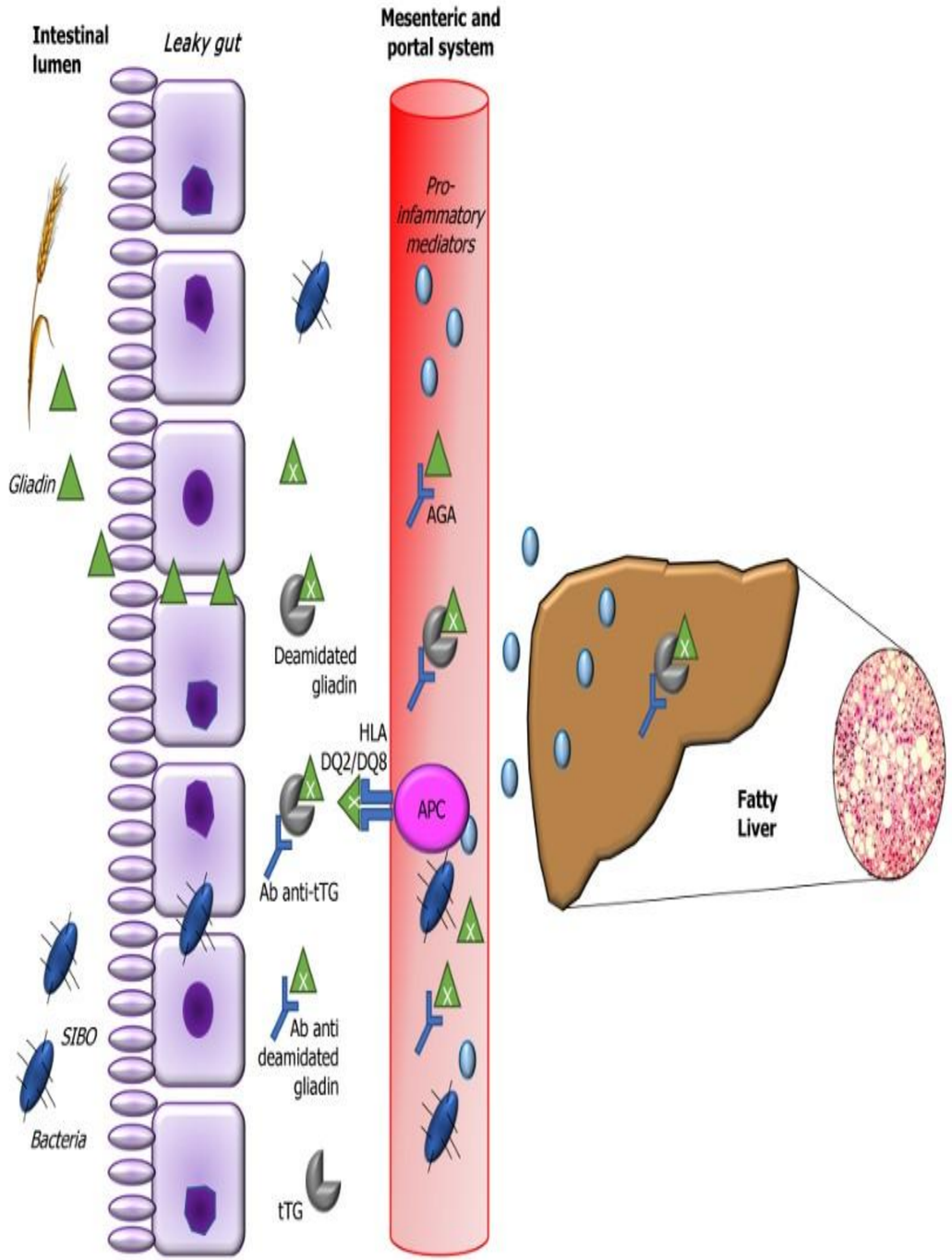

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Table 1. Histological Findings in the Livers of Patients with Celiac Disease

Nonspecific abnormalities (most common)
Periportal inflammation
Increased number of Kupffer cells
Mononuclear infiltration on the portal triad
Steatosis
Microvesicular and macrovesicular
Fibrosis
Cirrhosis

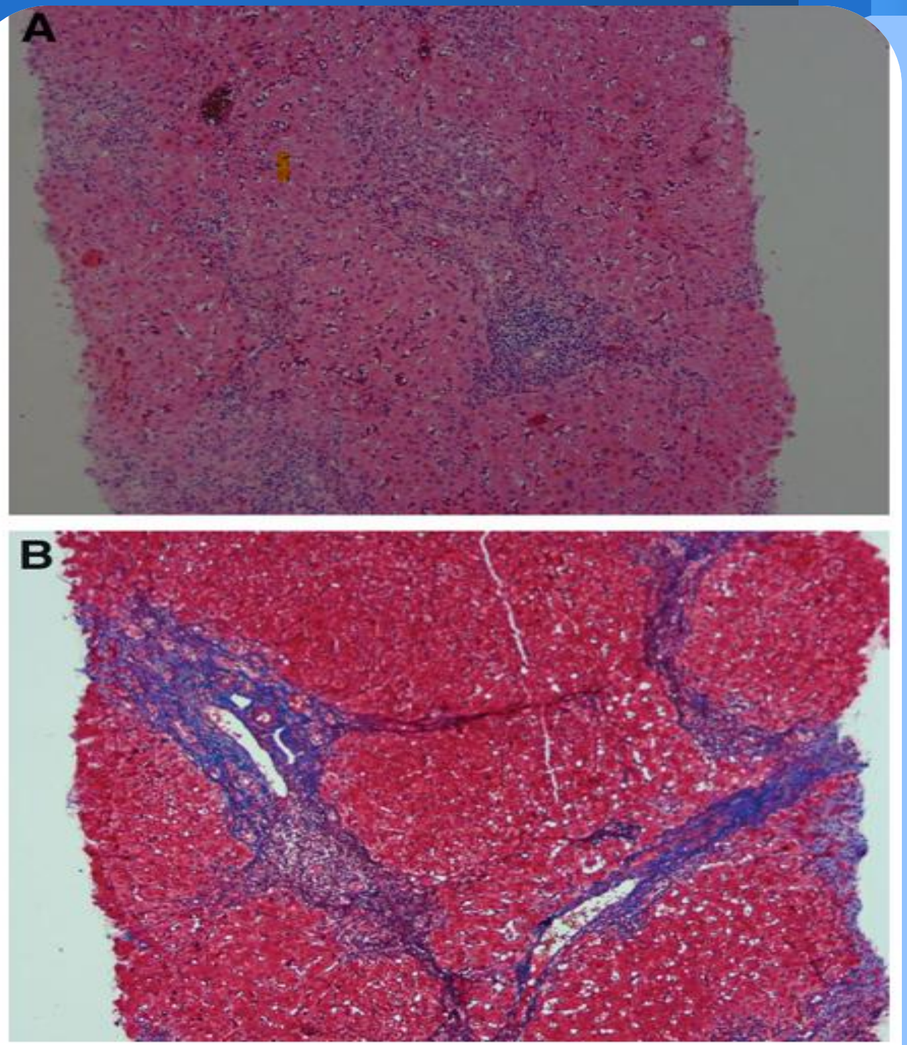

Fig. 2. Findings of liver injury associated with celiac disease on liver-biopsy specimens. (A) Nonspecific changes (mononuclear infiltration of the portal triad) in a patient with celiac disease and elevated aminotransferases (hematoxylin and eosin, ×200). (B) Extensive fibrosis in a patient with celiac disease and severe liver injury (Masson's trichrome, ×200).

RESULTS:

Of 6,871 articles screened, 20 articles were included finally in 3 meta-analyses for cryptogenic cirrhosis, all-cause cirrhosis, and cryptogenic hypertransaminasemia. For the all-cause hypertransaminasemia group, a qualitative review of 4 studies was conducted instead of a meta-analysis due to significant differences in studies. The pooled prevalence (95% confidence interval) of biopsy-confirmed CeD in cryptogenic cirrhosis was 4.6% (2.2%–7.5%) while the pooled prevalence of biopsy-confirmed CeD in all-cause cirrhosis was 0.8% (0%–3.4%). The pooled prevalence of biopsy-confirmed CeD in cryptogenic hypertransaminasemia was 5.7% (3.2%–8.8%).

DISCUSSION:

Nearly 1 in 20 patients each with cryptogenic cirrhosis and cryptogenic hypertransaminasemia have CeD; hence, they should both be considered high-risk groups for CeD. While the prevalence of CeD in those with all-cause cirrhosis is similar to that in general population, it may be worth screening them for CeD because liver pathology has the potential for reversal in them.

Meta-Analysis › Am J Gastroenterol. 2023 May 1;118(5):820-832.

doi: 10.14309/ajg.0000000000002123. Epub 2022 Dec 23.

Prevalence of Celiac Disease in Patients With Liver Diseases: A Systematic Review and Meta-Analyses

Shakira Yoosuf [1,2], Prashant Singh [3], Ashank Khaitan [1], Tor A Strand [4], Vineet Ahuja [1], Govind K Makharia [1]

Affiliations + expand

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ARTICLE: LIVER

Prevalence of Celiac Disease in Patients With Liver Diseases: A Systematic Review and Meta-Analyses

Yoosuf, Shakira MD[1,2]; Singh, Prashant MD[3]; Khaitan, Ashank MBBS[1]; Strand, Tor A. MD[4]; Ahuja, Vineet MD, DM[1]; Makharia, Govind K. MD, DM, DNB[1]

Author Information

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Follow up

PATHOLOGY REPORT

Liver; needle biopsy:
- LIVER TISSUE CORES SHOW PRESERVED ARCHITECTURE WITH MODERATE STEATOSIS.
- PORTAL AREAS APPEAR UNREMARKABLE WITH NO INFLAMMATION OR FIBROSIS.
- NO PLASMA CELLS SEEN.
- NO GRANULOMA OR NEOPLASTIC CHANGES IDENTIFIED.

GROSS DESCRIPTION

Labeled liver, needle biopsy, consists of 2 tissue cores measuring 0.5 and 1.2 cm. The specimen is entirely embedded for histologic examination.

Ghiath Hamed, MD

Ghiath Hamed, M.D.
Seinor Pathologist

Results Report

AUTOIMMUNE MARKERS

Tests	Results	Reference range	Units	Last Result
Anti Transglutaminase IgA (TTG IgA)	16.32	Negative: Less than 20 Positive: More than 20	U/L	

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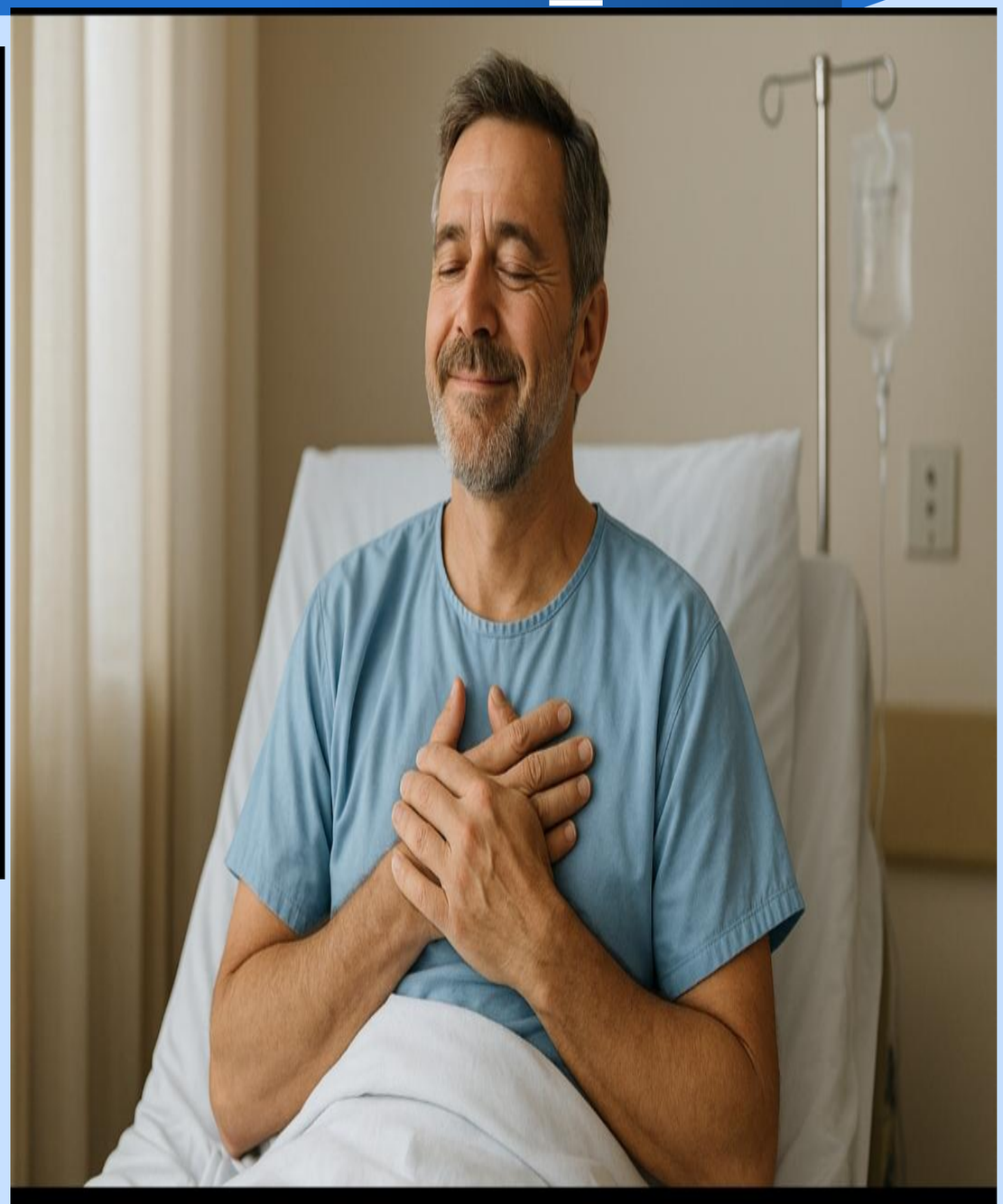
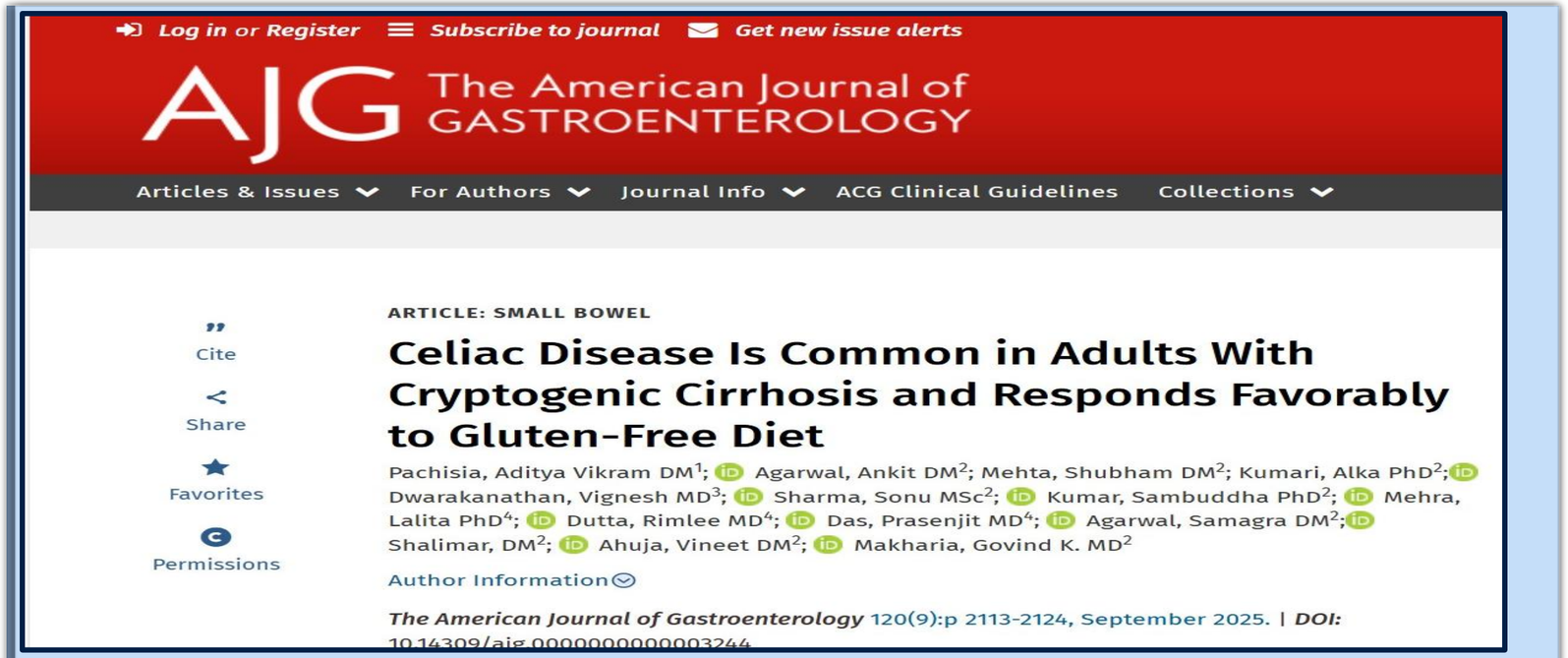




RESULTS:

Of 232 patients with cryptogenic cirrhosis, 14 had high anti-tTG Ab (16.9 ± 10.5 fold rise), with 9 antiendomysial antibody–positive and 11 (4.7%) biopsy-proven CeD. IgA/anti-tTG Ab colocalization was demonstrated in 7/8 liver and 10/11 duodenal biopsies. Patients with cryptogenic cirrhosis with definite CeD (n = 11) and matched cohort without CeD (n = 44) were similar at baseline (age: 31.3 ± 7.7 vs 31.8 ± 9.3 years; 5 [45.5%] vs 15 [34.1%] females; MELDNa 9 [interquartile-range: 8–15.5] vs 12 [9–15]; CTP 7 [6–7.5] vs 6 [5.75–7]). Patients with CeD on GFD improved significantly on follow-up compared with those without CeD (follow-up MELDNa: 9 [7.5–10.5] vs 18.5 [12-20]; $P = 0.001$ and follow-up CTP: 5 [5-5] vs 8 [7–9]; $P < 0.001$) with less frequent further decompensations and similar mortality (9.1% vs 18.2%; $P = 0.67$).

Celiac disease is common in adults with cryptogenic cirrhosis and responds favourably to gluten free diet

300 patients with cryptogenic chronic liver disease screened

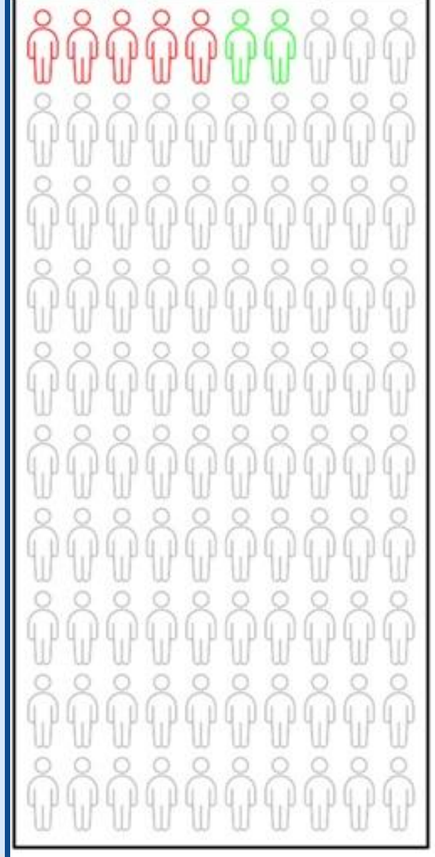
232 patients with confirmed cryptogenic cirrhosis worked up in detail

216 IgA TTG negative
44 matched controls selected

5 Potential celiac disease
IgA TTG > 2 ULN; duodenal biopsy normal

11 Definite celiac disease
IgA TTG > 2 ULN; duodenal biopsy modified Marsh grade 2 or more

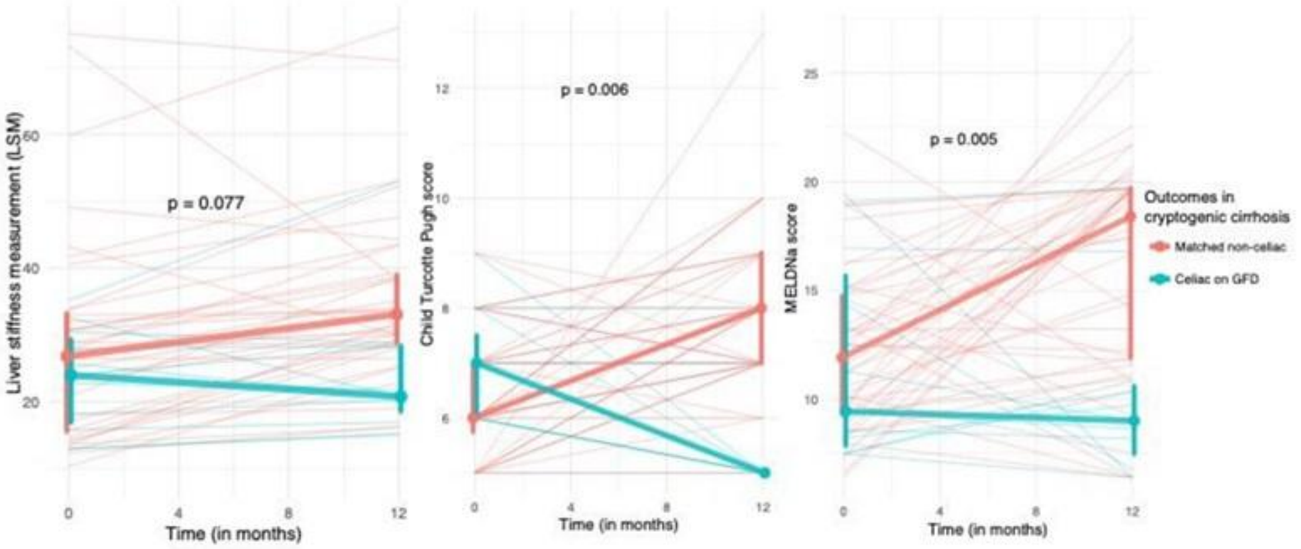
Definite/ potential celiac disease in 100 cryptogenic cirrhosis patients



Conclusions

5 in 100 (prevalance 4.7%, 95% CI = 2.4% - 8.3%) have definite celiac disease (biopsy proven)
7 in 100 (prevalence 6.9% , 95% CI = 4.0-10.9%) have seropositive celiac disease

Liver related outcomes in patients with cryptogenic cirrhosis and definite celiac disease (treated with gluten-free diet + standard of care) compared to a matched cohort without celiac disease (treated with standard of care alone)



Pachisia et al, 2024, doi: 10.14309/ajg.0000000000003244

RESULTS

Of 232 patients with cryptogenic cirrhosis

14 had high anti-tTG Ab (16.9 ± 10.5 fold rise)

9 were antiendomysial antibody-positive

COMPARISON

Patients with celiac disease	VS	Cohort without celiac disease
Age		31.1 vs
MELD		9.1.3
CTP		7 v 6

GLUTEN-FREE DIET

Improvement

MELD	12 → 8.5
CTP	7 → 5

Fewer decompestions

astroenterology aga

patients with severe liver failure. **Methods:** Four patients with untreated celiac disease and severe liver disease are described. Further, the occurrence of celiac disease was studied in 185 adults with previous liver transplantation using serum immunoglobulin A endomysial and tissue transglutaminase antibodies in screening. **Results:** Of the 4 patients with severe liver disease and celiac disease, 1 had congenital liver fibrosis, 1 had massive hepatic steatosis, and 2 had progressive hepatitis without apparent origin. Three were even remitted for consideration of liver transplantation. Hepatic dysfunction reversed in all cases when a gluten-free diet was adopted. In the transplantation group, 8 patients (4.3%) had celiac disease. Six cases were detected before the operation: 3 had primary biliary cirrhosis, 1 had autoimmune hepatitis, 1 had primary sclerosing cholangitis, and 1 had congenital liver fibrosis. Only 1 patient had maintained a long-term strict gluten-free diet. Screening found 2 cases of celiac disease, 1 with autoimmune hepatitis and 1 with secondary sclerosing cholangitis. **Conclusions:** The possible presence of celiac disease should be investigated in patients with severe liver disease. Dietary treatment may prevent progression to hepatic failure, even in cases in which liver transplantation is considered.

- Celiac + GFD → Improved liver function↓ Liver stiffness (p = 0.077)
- ↓ Child–Turcotte–Pugh score (p = 0.006)
- ↓ MELD score (p = 0.005)

Table 1. Findings Before and After the Introduction of a Gluten-Free Diet in Patients With Severe Liver Failure Subsequently Found to Have Celiac Disease

	Patient 1		Patient 2		Patient 3		Patient 4	
	Before GFD	After GFD	Before GFD	After GFD	Before GFD	After GFD	Before GFD	After GFD
General condition	Poor	Improved	Poor	Improved	Poor	Improved	Poor	Improved
Jaundice	+++	0	+	±	0	0	0	0
Ascites	+++	0	+++	0	+++	0	+++	0
INR (0.9–1.2)	3.0	1.3	1.5–1.1	1.0	2.1–1.1	1.1	1.1	1.3
Albumin, g/L (>40 g/L)	18	41	16	38	12	44	29	37
Bilirubin, μmol/L (<20 μmol/L)	>500	25	40	31	13	8	25	24
Alkaline phosphatase, U/L (60–275 U/L)	940	735	188	96	358	117	622	835
Alanine aminotransferase, U/L (<50 U/L)	3390	91	57	25	122	18	41	33–50
Liver histology	Acute hepatitis	Improved	Increased fibrosis with bile duct proliferation	ND	50% steatosis	Improved	Early cirrhosis with mild lymphocytic infiltration	Micronodular cirrhosis, chronic hepatitis
HLA type	HLA-DQ2		ND		HLA-DQ2		HLA-DQ2	

GFD, gluten-free diet; INR, international normalized ratio; ND, not done.

Research Article

JOURNAL OF HEPATOLOGY

Increased risk of non-alcoholic fatty liver disease after diagnosis of celiac disease

Norelle R. Reilly[1,2], Benjamin Lebwohl[1,3], Rolf Hultcrantz[4], Peter H.R. Green[1], Jonas F. Ludvigsson[3,5,*]

[1]Celiac Disease Center, Department of Medicine, Columbia University College of Physicians and Surgeons, New York, NY, USA; [2]Department of Pediatrics, Columbia University College of Physicians and Surgeons, New York, NY, USA; [3]Department Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, Sweden; [4]Department of Medicine, Karolinska Institutet, Stockholm, Sweden; [5]Department of Pediatrics, Örebro University Hospital, Örebro University, Örebro, Sweden

Background & Aims: Non-alcoholic fatty liver disease is a common cause of chronic liver disease. Celiac disease alters intestinal permeability and treatment with a gluten-free diet often causes weight gain, but so far there are few reports of non-alcoholic fatty liver disease in patients with celiac disease.
Methods: Population-based cohort study. We compared the risk of non-alcoholic fatty liver disease diagnosed from 1997 to 2009 in individuals with celiac disease (n = 26,816) to matched reference individuals (n = 130,051). Patients with any liver disease prior to celiac disease were excluded, as were individuals with a lifetime diagnosis of alcohol-related disorder to minimize misclassification of non-alcoholic fatty liver disease. Cox regression estimated hazard ratios for non-alcoholic fatty liver disease were determined.
Results: During 246,559 person-years of follow-up, 53 individuals with celiac disease had a diagnosis of non-alcoholic fatty liver disease (21/100,000 person-years). In comparison, we identified 85 reference individuals diagnosed with non-alcoholic fatty liver disease during 1,488,413 person-years (6/100,000 person-years). This corresponded to a hazard ratio of 2.8 (95% CI 2.0–3.8), with the highest risk estimates seen in children (HR = 4.6; 95% CI 2.3–9.1). The risk increase in the first year after celiac disease diagnosis was 13.3 (95% CI 3.5–50.3) but remained significantly elevated even beyond 15 years after the diagnosis of celiac disease (HR = 2.5; 95% CI 1.0–5.9).
Conclusion: Individuals with celiac disease are at increased risk of non-alcoholic fatty liver disease compared to the general population. Excess risks were highest in the first year after celiac disease diagnosis, but persisted through 15 years after diagnosis with celiac disease.

Keywords: Autoimmune; Steatohepatitis; Gluten; NASH; NAFLD; Celiac disease.
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* Corresponding author. Address: Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, 171 77 Stockholm, Sweden. Tel.: +46 (0) 8 5248 2356; fax: +46 (0) 8 31 49 75.
E-mail address: jonasludvigsson@yahoo.com (J.F. Ludvigsson).
Abbreviations: CD, celiac disease; NAFLD, non-alcoholic fatty liver disease; NASH, non-alcoholic steatohepatitis; SIBO, small intestinal bacterial overgrowth; VA, villous atrophy; ICD, international classification of disease (codes); CI, confidence Interval; HR, hazard ratio; OR, odds ratio; BMI, body mass index.

Introduction

Celiac disease (CD) is associated with both acute and chronic liver diseases, especially autoimmune liver disease [1,2]. Non-alcoholic fatty liver disease (NAFLD) is the most common cause of chronic liver disease in children and adolescents in Western nations [3], it is estimated to be present in 20% of the population [4], and some individuals may progress to fibrosis and cirrhosis [4,5]. Yet, data establishing whether individuals with CD may be at risk of NAFLD are nascent [1,6]. Metabolic risks as well as those altering intestinal permeability found in CD can also be potential triggers for NAFLD.

Obesity and metabolic syndromes are accepted as major accessory complications of NAFLD [7], though not all patients with NAFLD are obese [8]. A high-fat diet and sedentary lifestyle are adaptable risks, modifications to these can yield improvements in NAFLD [9], and recovery after weight loss may be related to reduced insulin resistance [10]. Many adults and children with CD are overweight or obese at diagnosis, or become overweight after treatment [11,12], consequently increasing the risk to develop NAFLD. Children [13] and adults [14] with CD may have increased cardiovascular risks which overlap with those associated with NAFLD.

The gut-liver axis via the portal system has been implicated as a potential route of inflammatory cytokines, which may trigger the onset of non-alcoholic steatohepatitis (NASH). Levels of endotoxin (lipopolysaccharide), derived from intestinal Gram-negative microbiota, are elevated in the sera of adults [15] and children [16] with NAFLD, suggesting these individuals have increased intestinal permeability. Small intestinal bacterial overgrowth (SIBO) is more common among patients with NAFLD than healthy individuals [17] and is associated with higher TNF-α levels, independent of increases in gut permeability markers [18]. Individuals with CD have altered intestinal permeability due to the disturbance of mucosal integrity induced by gluten and the associated inflammatory response [19]. Additionally,

Background & Aims: Non-alcoholic fatty liver disease is a common cause of chronic liver disease. Celiac disease alters intestinal permeability and treatment with a gluten-free diet often causes weight gain, but so far there are few reports of non-alcoholic fatty liver disease in patients with celiac disease.
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Conclusion: Individuals with celiac disease are at increased risk of non-alcoholic fatty liver disease compared to the general population. Excess risks were highest in the first year after celiac disease diagnosis, but persisted through 15 years after diagnosis with celiac disease.

Long-term risk of chronic liver disease in patients with celiac disease: a nationwide population-based, sibling-controlled cohort study

Jialu Yao,[a,*] Jiangwei Sun,[a] Fahim Ebrahimi,[a,b] David Bergman,[a] Peter H. R. Green,[c,d] Hannes Hagström,[e,f] Benjamin Lebwohl,[c,d,g] Daniel A. Leffler,[h,i] and Jonas F. Ludvigsson[a,d,j]

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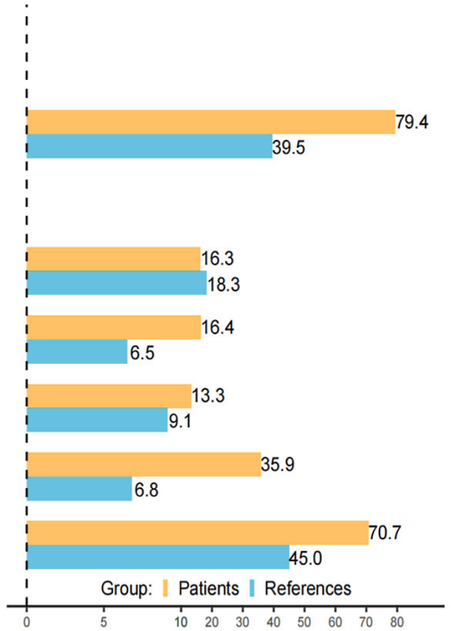
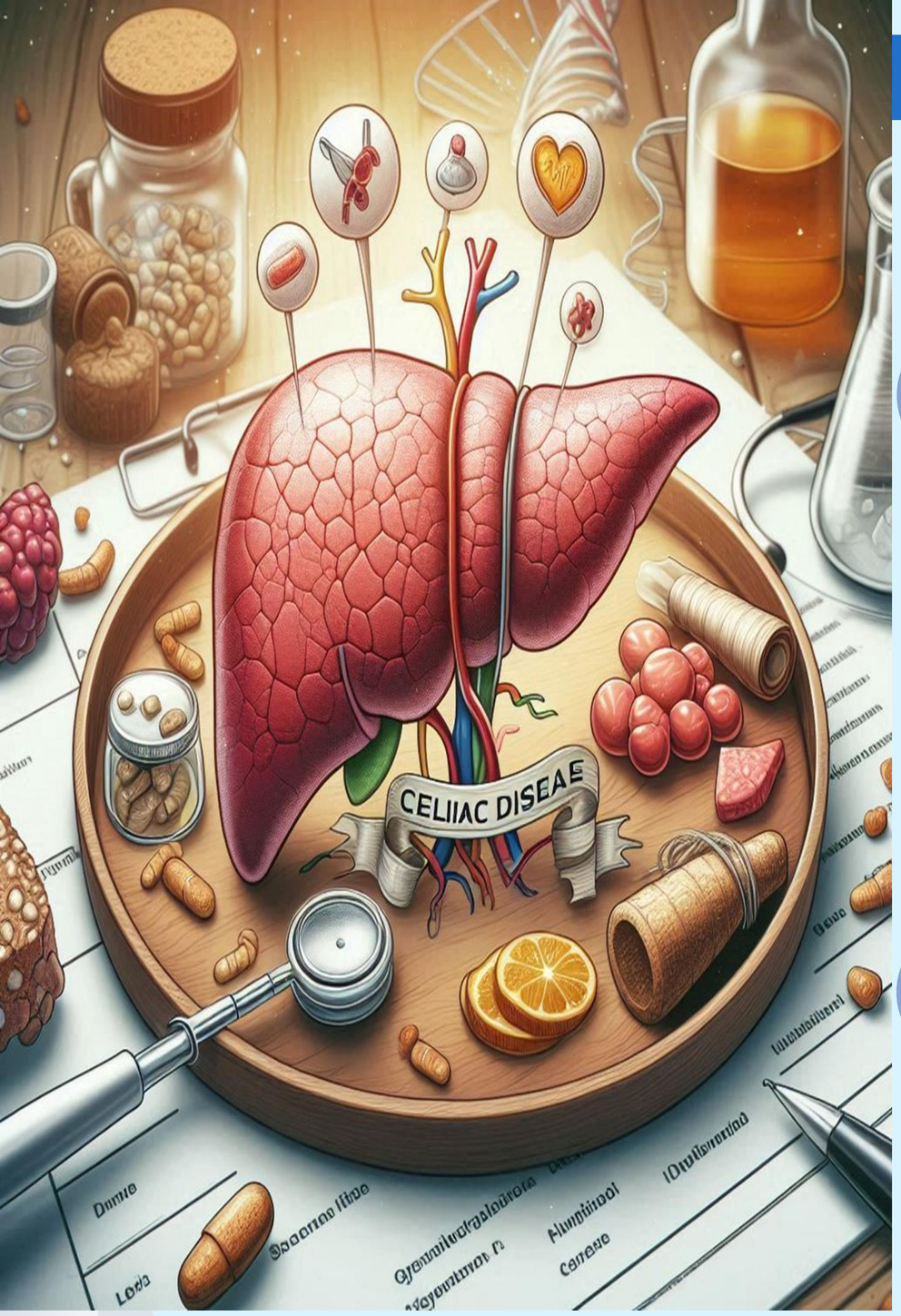
	No. of events in patients	No. of events in references	Incidence rate (IR) per 100,000 person-years	IR difference (95%CI) per 100,000 person-years	Hazard ratio (95%CI), Model 2
Primary outcome					
Any CLD	649	1571	79.4 / 39.5	40·0 (33·6 to 46·4)	2·01 (1·82 to 2·22)
Secondary outcomes					
Viral hepatitis	134	731	16.3 / 18.3	-2·0 (-5·1 to 1·1)	0·89 (0·73 to 1·10)
MASLD	131	254	16.4 / 6.5	9·9 (7·0 to 12·8)	2·54 (2·03 to 3·18)
Alcohol-related liver disease	109	363	13.3 / 9.1	4·2 (1·5 to 6·8)	1·51 (1·20 to 1·90)
Autoimmune liver disease	294	271	35.9 / 6.8	29·1 (24·9 to 33·3)	4·86 (4·06 to 5·82)
MALO	580	1795	70.7 / 45.0	25·7 (19·6 to 31·8)	1·54 (1·40 to 1·70)



Incident chronic liver disease (CLD) and major adverse liver outcomes (MALO) in patients with celiac disease compared with their matched reference individuals

In conclusion, CeD is associated with a two-fold increased relative risk of any CLD (including autoimmune liver disease, MASLD, and alcohol-related liver disease) for ≥25 years after diagnosis. High-risk populations for any CLD included individuals with a history of autoimmune or metabolic-related diseases. Clinicians should be vigilant to signs of progressive liver disease such as elevated liver enzymes in patients with CeD to prevent the long-term risk of developing MALO.

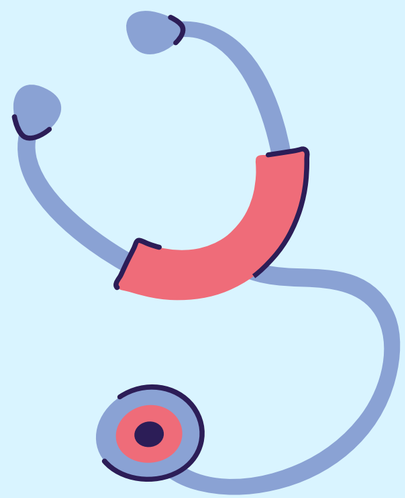


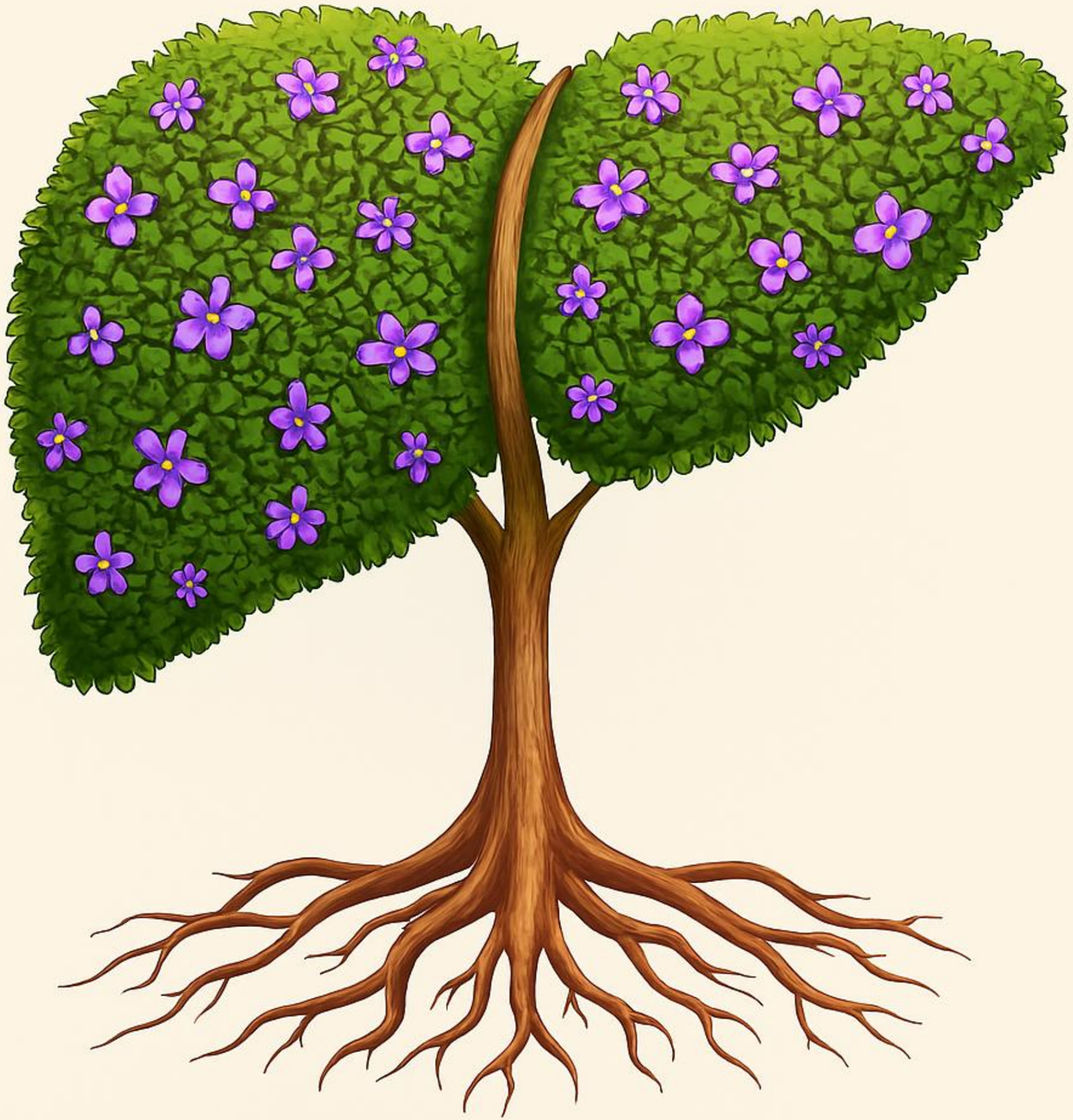
Take-home Messages

1. Celiac disease may silently manifest as liver cirrhosis.

2. Always test for celiac antibodies in unexplained hepatic dysfunction.

3. Gluten-free diet can significantly improve liver function.





The liver sometimes speaks for the silent intestine

CARE Your Liver

Thank You