HIV AND LIVER

Liver disease in one of the leading causes of non-Aids related death
HIV AND LIVER

IAS 2019

217 Abstracts on the Liver
10 Sessions on the Liver
19 Presentation

The Uniqueness of the liver function in HIV Patients
- Liver Inflammation in HIV
- Fatty Liver Disease
- HIV-HBV Infection
- HIV and Liver Cancer diagnose
Causes of Liver Disease in HIV South Africa - Biopsy results

Table 2. Primary histopathology and laboratory findings for HIV-infected patients.

<table>
<thead>
<tr>
<th></th>
<th>Granulomatous disease / TB Median (IQR)</th>
<th>Cholangiopathy Median (IQR)</th>
<th>Steatosis Median (IQR)</th>
<th>Malignancy Median (IQR)</th>
<th>Fibrosis/cirrhosis Median (IQR)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>n (%)</td>
<td>28 (26)</td>
<td>11 (10)</td>
<td>23 (21)</td>
<td>11 (10)</td>
<td>7 (6)</td>
<td></td>
</tr>
<tr>
<td>CD4 count (cells/mm³)</td>
<td>135 (69, 226)</td>
<td>105 (60, 494)</td>
<td>176 (91, 253)</td>
<td>225 (89, 362)</td>
<td>365 (306, 513)</td>
<td>0.5</td>
</tr>
<tr>
<td>ALT (U/L)</td>
<td>57 (45, 112)</td>
<td>31 (20, 63)</td>
<td>50 (41, 76)</td>
<td>194</td>
<td>63 (43, 113)</td>
<td>0.5</td>
</tr>
<tr>
<td>AST (U/L)</td>
<td>105 (72, 260)</td>
<td>47 (10, 59)</td>
<td>112 (101, 122)</td>
<td>271</td>
<td>98 (52, 155)</td>
<td>0.4</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/L)</td>
<td>382 (219, 713)</td>
<td>462 (328, 1757)</td>
<td>275 (260, 359)</td>
<td>1913</td>
<td>200 (187, 307)</td>
<td>0.03</td>
</tr>
<tr>
<td>Total bilirubin (µmol/L)</td>
<td>28 (13, 40)</td>
<td>11 (5, 20)</td>
<td>18 (12, 33)</td>
<td>96</td>
<td>24 (19, 105)</td>
<td>0.5</td>
</tr>
</tbody>
</table>

*Patients with malignancy lacked most laboratory data

doi:10.1371/journal.pone.0117813.t002

malignancy were as follows: metastatic adenocarcinoma, 13 (39%); hepatocellular carcinoma, 11 (20%); lymphoma, 11 (20%); and other, 12 (21%). Specific testing for viral hepatitis was not completed on any of the patients diagnosed with hepatocellular carcinoma.

A number of additional findings were reported. For example, three patients had biopsy findings consistent with chronic hepatitis B, one with chronic hepatitis C, and one with *Schistosoma* infection. Specific serology was available for only one of the patients, one with biopsy findings consistent with chronic hepatitis B and serology positive for HBsAg. Three additional patients had a secondary finding of iron overload, two of whom had primary diagnoses of granulomas consistent with TB and one had a primary diagnosis of cirrhosis without a clear etiology.
Causes of Liver Disease in HIV South Africa-Biopsy results

Liver Disease and HIV/HBV-MORTALITY – HAART ERA

HIV, HCV, HBV, & TB prevalence by region
**Figure 1.** End-stage liver disease (ESLD) incidence rates and 95% confidence intervals by viral hepatitis coinfection...
The uniqueness of the physiology of the Liver is disturbed in various disease process.

Normal Liver Functions
1. Bile Production
2. Drug Metabolism
3. Bilirubin Metabolism
4. Thyroid Hormone physiology
5. Synthetic functions of plasma protein clotting factors
   albumin globulins
6. Immunologic Functions
Liver Pathophysiology in HIV

Discrete Mechanisms of Liver Injury
Oxidative stress
Mitochondria Injury
Lipotoxicity
Immune Mediated Injury
Toxic Metabolite Accumulation
Gut Microbial Translocation
Systemic Inflammation
Senescence
Nodular Regenerative Hyperplasia.
Liver Pathophysiology in HIV Patients

- Poorly controlled HIV Mono Infection is an independent risk factor for fibrosis.
- HIV patients on HAART with good viral suppression are prone to common causes of liver injury.
  - Viral Hepatitis A, B, C
  - Non Alcoholic fatty liver disease
  - Ageing
  - HAART Related toxicity
  - Alcoholic liver disease
Mitochondrial injury occurs by increased stress on the Endoplasmic Reticulum. This ‘ER’ stress is also a pathway for NAFLD, Viral Hepatitis, and Alcohol related liver injury.
Mitochondrial Injury: ARV Drugs

NRTI – cause increased lipid content of cell membranes

Protease Inhibitors (PI) PI mediated decrease in sarcoplasmic/ER calcium.

PI Effect is potentiated by alcohol use.
NAFLD Lipotoxicity of the liver in HIV Patients

Occurs due to FFA Accumulation

FFA Accumulation leads to increased reactive oxidative stress and ER stress. Finally this leads to Fibrosis.

NAFLD occurs in 30-40% patients with HIV

Development of NAFLD in HIV occurs due to

Medications e.g. Protease Inhibitors
- Dyslipidemia
- FFA Composition
- Insulin Resistance
- Increase body fat composition
- Increased Inflammatory Markers
Background

Evolution of non-alcoholic fatty liver disease
HIV infection is an independent risk factor for liver steatosis: A study in HIV mono-infected patients compared to uninfected paired controls and associated risk factors

Antonio Pacheco, Hugo Perazzo, Sandra Cardoso, Maria-de-Jesus Fonseca, Rosane Griep, Paulo Lotufo, Isabela Bensenor, Jose Mill, Rodrigo Moreira, Ronaldo Moreira, Ruth Friedman, Marilia Santini-Oliveira, Valdilea G Veloso, Dora Chor, Beatriz Grinsztejn

Oswaldo Cruz Foundation (FIOCRUZ), Rio de Janeiro
University of São Paulo (USP)
Federal University of Espírito Santo (UFES)

Abstract number: THAB0205
Session title: HIV and the liver
July 26h, 2018
## The burden of liver steatosis in HIV-infected patients

<table>
<thead>
<tr>
<th>Authors</th>
<th>Country</th>
<th>N</th>
<th>Gold standard</th>
<th>Prevalence</th>
<th>Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morse <em>et al</em></td>
<td>USA</td>
<td>62</td>
<td>Liver biopsy</td>
<td>73%</td>
<td>-</td>
</tr>
<tr>
<td>Mohr <em>et al</em></td>
<td>Germany</td>
<td>341</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Lombardi <em>et al</em></td>
<td>UK</td>
<td>125</td>
<td>US</td>
<td>55%</td>
<td>Male sex, age, HOMA-IR, GGT</td>
</tr>
<tr>
<td>Liu <em>et al</em></td>
<td>China</td>
<td>80</td>
<td>MRI</td>
<td>29%</td>
<td>Triglycerides</td>
</tr>
<tr>
<td>Macias <em>et al</em></td>
<td>Spain</td>
<td>326</td>
<td>CAP</td>
<td>37%</td>
<td>-</td>
</tr>
<tr>
<td>Sebastiani <em>et al</em></td>
<td>Canada</td>
<td>538</td>
<td>CAP</td>
<td>36%</td>
<td>BMI, triglycerides</td>
</tr>
<tr>
<td>Lemoine <em>et al</em></td>
<td>France</td>
<td>405</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Perazzo <em>et al</em></td>
<td>Brazil</td>
<td>395</td>
<td>CAP</td>
<td>35%</td>
<td>MS, cumulative use of D-drugs</td>
</tr>
</tbody>
</table>

**Impact of HIV infection for development of steatosis?**
Aims

To evaluate the prevalence and factors associated with liver steatosis in HIV mono-infected patients compared to uninfected subjects paired for confounding factors
Factors associated with liver steatosis in HIV-infected patients (n=649)

Prevalence of steatosis = 35%

<table>
<thead>
<tr>
<th>Factor</th>
<th>No steatosis (FLI &lt; 60)</th>
<th>Steatosis (FLI ≥ 60)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female sex</td>
<td>180 (43%)</td>
<td>93 (40%)</td>
<td>0.542</td>
</tr>
<tr>
<td>Age, years</td>
<td>42 (34 - 50)</td>
<td>46 (40 - 52)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Black/Pardo ethnicity</td>
<td>105 (25%)</td>
<td>44 (19%)</td>
<td>0.096</td>
</tr>
<tr>
<td>Education level &gt; 8 years of study</td>
<td>225 (54%)</td>
<td>114 (49%)</td>
<td>0.312</td>
</tr>
<tr>
<td><strong>Metabolic features</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI, Kg/m²</td>
<td>22.8 (20.9 - 24.9)</td>
<td>28.4 (25.7 - 31.9)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>81 (76 - 87)</td>
<td>97 (92 - 105)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Type-2 diabetes</td>
<td>73 (18%)</td>
<td>94 (41%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>133 (32%)</td>
<td>102 (46%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>97 (23%)</td>
<td>105 (46%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Metabolic syndrome</td>
<td>56 (13%)</td>
<td>156 (68%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Poor clinical management</td>
<td>162 (39%)</td>
<td>123 (54%)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td><strong>HIV history</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CD4 count, cells</td>
<td>529 (352 - 708)</td>
<td>586 (408 - 830)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Undetectable HIV viral load (&lt; 50copies/mm3)</td>
<td>257 (70%)</td>
<td>164 (77%)</td>
<td>0.057</td>
</tr>
<tr>
<td>Nadir CD4 count</td>
<td>226 (104 - 317)</td>
<td>194 (85 - 305)</td>
<td>0.142</td>
</tr>
<tr>
<td>c-ART</td>
<td>369 (88%)</td>
<td>207 (90%)</td>
<td>0.700</td>
</tr>
<tr>
<td>Duration of c-ART, years</td>
<td>3.3 (0.5 - 9.8)</td>
<td>4.8 (1.7 - 11)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Current NNRTI treatment</td>
<td>265 (63%)</td>
<td>159 (69%)</td>
<td>0.191</td>
</tr>
<tr>
<td>Current PI treatment</td>
<td>208 (50%)</td>
<td>132 (57%)</td>
<td>0.085</td>
</tr>
</tbody>
</table>

Data expressed as n (%) or median [IQR]
Presence of steatosis in HIV and non-HIV individuals paired by the nearest neighbor propensity score with a caliper of 0.05

<table>
<thead>
<tr>
<th>HIV infection</th>
<th>OR (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2.1 (1.49-2.95)</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Logistic regression-based scores were used for matching and balance between groups was checked with usual procedures.
Tuberculosis and Hepatic Steatosis Are Prevalent Liver Pathology Findings among HIV-Infected Patients in South Africa

Christopher J. Hoffmann¹*, Jennifer D. Hoffmann¹, Caroline Kensler², Martin van der Watt³, Tanvrie Omar⁴, Richard E. Chaisson¹, Neil A. Martinson⁵, Ebrahim Varia⁵

¹ Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, United States of America, ² University of Pittsburgh School of Medicine, Pittsburgh, United States of America, ³ Perinatal HIV Research Unit, University of the Witwatersrand, Johannesburg, South Africa, ⁴ Department of Anatomical Pathology, University of the Witwatersrand and National Health Laboratory Service, Johannesburg, South Africa, ⁵ Department of Medicine, Klerksdorp Tshepong Hospital Complex and University of the Witwatersrand, Klerksdorp, South Africa

*choffmann@jhmi.edu

Abstract

Liver disease epidemiology in sub-Saharan Africa has shifted as a result of HIV and the increased use of antiretroviral therapy leading to a need for updated data on common causes of liver disease. We retrospectively reviewed records from all hospitalized patients who had liver biopsy at a single hospital in South Africa from 2001 to 2009 and compared diagnosis, demographics, and outcomes of patients with and without HIV infection.
Immune Mediated Injury

The Primary Cells involved are the Kupfer cell and the HSC (Hepatic Stellate Cells). HBV, HCV, HIV affect liver via these Mechanism. HBV + HCV may cause immune mediated injury as IRIS (Immune Reconstitution Inflammatory Syndrome) leading to Hepatitis Flares, and Liver decompensation.
Figure 1. Categorization of hepatic fibrosis by human immunodeficiency virus (HIV) and viral hepatitis disease...
HIV Monoinfection Is Associated With Increased Aspartate Aminotransferase-to-Platelet Ratio Index, a Surrogate Marker for Hepatic Fibrosis

Jennifer C. Price,1 Eric C. Seaberg,2 Sheila Badri,3 Mallory D. Witt,4 Kristin D’Acunto,5 and Chloe L. Thio6

1Department of Medicine, Division of Gastroenterology and Hepatology, Johns Hopkins School of Medicine, 2Division of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland; 3Department of Medicine, Division of Infectious Diseases, Hospital of Cook County, Rush University Medical Center, Ruth M. Rothstein CORE Center, Chicago, Illinois; 4Department of Medicine, David Geffen School of Medicine at UCLA, Los Angeles Biomedical Research Institute at Harbor–UCLA, Torrance, California; 5Department of Infectious Diseases and Microbiology, University of Pittsburgh Graduate School of Public Health, Pennsylvania; and 6Department of Medicine, Division of Infectious Diseases, Johns Hopkins School of Medicine, Baltimore, Maryland

Background. Although liver disease commonly causes morbidity and mortality among human immunodeficiency virus (HIV)–infected individuals, data are limited on its prevalence in HIV monoinfection. We used the aspartate aminotransferase-to-platelet ratio index (APRI) as a surrogate marker of hepatic fibrosis to characterize liver disease in the Multicenter AIDS Cohort Study.

Methods. Men were categorized based on their HIV and viral hepatitis status: uninfected (n = 1170), HIV monoinfected (n = 509), viral hepatitis monoinfected (n = 74), and HIV–viral hepatitis coinfected (n = 66).

Results. The median APRI in the HIV-monoinfected group was similar to that in the hepatitis-monoinfected group (0.42 vs 0.43; P > .05), higher than in the uninfected group (0.42 vs 0.27; P < .001) but lower than in the coinfected group (0.42 vs 1.0; P < .001). On multivariable analysis, HIV infection (1.39-fold increase [FI]; P < .001), viral hepatitis infection (1.52-FI; P < .001), and the interaction between HIV and viral hepatitis infections were independently associated with a higher APRI (1.57-FI; P < .001). Among the HIV-infected men, viral hepatitis coinfecion (2.34-FI; P < .001), HIV RNA ≥100 000 copies/mL (1.26-FI; P = .007), and CD4 count ≤200 cells/mL (1.23-FI; P = .022) were independently associated with a higher APRI.

Conclusions. HIV and viral hepatitis are independently associated with an increased APRI. Further studies are needed to understand the biological basis for the association between HIV and liver disease.
Table 4. Factors Associated With Difference in ln(APRI), Among All HIV-Positive Participants

<table>
<thead>
<tr>
<th>Factor</th>
<th>Unadjusted</th>
<th>Adjusted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fold Change in APRI</td>
<td>P Value</td>
</tr>
<tr>
<td></td>
<td>(95% CI)</td>
<td></td>
</tr>
<tr>
<td>Hepatitis status</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uninfected</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>HBV or HCV infected</td>
<td>2.32 (1.95–2.76)</td>
<td>.001</td>
</tr>
<tr>
<td>White race</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>0.86 (.75–.99)</td>
<td>.036</td>
</tr>
<tr>
<td>Yes</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>CD4 count ≤200 cells/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>1.37 (1.16–1.61)</td>
<td>.001</td>
</tr>
<tr>
<td>Viral load ≤100,000 copies/mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>1.37 (1.16–1.61)</td>
<td>.001</td>
</tr>
<tr>
<td>Current ART use</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No therapy</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Monotherapy</td>
<td>1.08 (.94–1.25)</td>
<td>.273</td>
</tr>
<tr>
<td>Combination therapy</td>
<td>1.24 (1.04–1.47)</td>
<td>.015</td>
</tr>
<tr>
<td>Current trimethoprim/sulfamethoxazole use</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>1.37 (1.16–1.61)</td>
<td>.001</td>
</tr>
<tr>
<td>Current fluconazole</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Yes</td>
<td>1.37 (1.16–1.61)</td>
<td>.001</td>
</tr>
</tbody>
</table>

Abbreviations: APRI, aspartate aminotransferase-to-platelet ratio index; ART, antiretroviral therapy; CI, confidence interval; HBV, hepatitis B virus; HCV, hepatitis C virus; HIV, human immunodeficiency virus type 1; ln, natural logarithm.

*a* Model also adjusts for age, heavy alcohol use, body mass index, and year of Multicenter AIDS Cohort Study visit.

Injury and fibrosis. The HIV envelope protein gp120 may also translocate have also been implicated in promoting hepatic
Cytotoxicity

HIV and HCV have direct Cytopathic effects on the liver.

These effects are enhanced in HIV – HCV Co-Infection.

HIV/HCV CoInfection causes fibrosing cholestatic Hepatitis (FCH)
MicroRNAs in liver disease

Gyongyi Szabo and Shashi Bala

Abstract

Small, noncoding microRNAs (miRNAs) regulate diverse biological functions in the liver and increasing evidence suggests that they have a role in liver pathology. This Review summarizes advances in the field of miRNAs in liver diseases, inflammation and cirrhosis. MicroRNA-122, the most abundant miRNA in hepatocytes, has well-defined roles in HCV replication and liver injury, and supports the idea that liver-specific miRNAs are potential targets for the development of new therapeutic strategies.
Cytotoxicity

Hepatocytes

ROS
↑ miR-217
↓ SIRT1
↓ miR-199a
Endothelial cell

ROS
↑ miR-34a
↓ miR-122
? Injury

Steatosis

Release of danger molecules
Amplification of inflammation

 ↑ ET1 and HIF-1α

miR-155

Kupffer cell

TLR4

↑ TNF

TNFR

LPS

Increased intestinal permeability

Epithelial cells

Alcohol
Circulating microRNAs in HIV patients reveal specific signatures for liver damage

Miguel Angel Martinez¹, Sandra Franco¹, Daniela Buccione², Beatriz Mothe¹, Lidia Ruiz¹, Maria Nevot¹, Ana Jordan-Paiz¹, Raquel Pluvinet², Susanna Aussó², Rosa M. Morillas², Laura Sumoy², Cristina Tural²

¹IrsiCaixa Institute for AIDS Research, ²Hospital Germans Trias i Pujol, Badalona, Spain

Poster # 639

This study was funded by the Spanish Instituto de Salud Carlos III (PI14/00195)
Cytotoxicity

HIV and HCV have direct cytopathic effects on the liver. These effects are enhanced in HIV–HCV co-infection. HIV/HCV co-infection causes fibrosing cholestatic hepatitis (FCH).

Background & Aim

- Elevated liver enzymes are common in patients infected with HIV-1, even in the absence of co-infection with hepatotropic viruses or alcohol abuse.
- HIV-1-induced inflammation and/or long-term antiretroviral drug toxicity may contribute to the evolution of liver disease.
- Cellular micro(mi)RNAs are potential disease biomarkers and therapeutic targets.
- **Study aim**: To investigate circulating plasma miRNAs among HIV-1 infected patients and their association with liver injury.
- **Study approach**: Large-scale deep sequencing analyses of small RNA expression on plasma samples from 144 patients infected with HIV-1 that had elevated levels of alanine aminotransferase (ALT), focal nodular hyperplasia or HCV co-infections. Healthy donors and HCV mono-infected individuals were also explored. This approach allowed us to identify 1425 different mature miRNAs in the study samples.
HIV and HCV have direct cytopathic effects on the liver. These effects are enhanced in HIV-HCV co-infection. HIV/HCV co-infection causes fibrosing cholestatic hepatitis (FCH).

**Results**

- Compared to healthy donors, patients with HIV-1 or HCV mono-infections showed significantly altered (fold change >2, adjusted p <0.05) expression of 25 and 70 miRNAs, respectively.
Accumulation of Toxic Metabolites

Medications used in HIV patients
Ketoconazole – Inhibit Cytochrome P450
Erythromycin – Inhibit Cytochrome P450
Isoniazid – direct Cytotoxicity
Cotrimoxazole – Hypersensitivity reactions
Acyclovir – Hypersensitivity reaction
Polypharmacy – Potentiate Hepatotoxicity of other drugs
Alcohol – Increase Hepatotoxicity by Protease Inhibitors
Gut Microbial Translocation

Hepatic Inflammation occurs due to high levels of bacterial lipopolysaccharides (LPS)
- Recruitment of Kupffer cell and HSC
- Systemic Immune responses are enhanced.

Acute HIV Infection directly targets gut Lymphocyte tissue. Break down of gut Epithelium and increased apoptosis of Epithetical cell leading to ‘Leakage’ Gut barrier dysfunction Persists even after HAART.

NAFLD, Alcoholic liver disease both cause increase gut permeability.
Systemic Inflammation

Occurs in NAFLD, HCV, HIV, HBV
Senescence

It is a process of cellular ageing.

It Ageing in chronic HIV Infection affects liver, kidney, bones, non Aids cancer and is also associated with Diabetes.
Nodular Regenerative Hyperplasia

Rare condition
Diffuse transformation of liver parenchyma into micronodules
No intervening fibrosis
Causes on Cirrhotic Portal Hypertension
Liver function is preserved
It is due to endothelial damage by Gut Bacterial translocation causing stenosis, and Portal HT.
Approach to Clinical Management

HISTORY

Careful history is important

- Duration
- Alcohol use
- Type of ARV
- TB Medication
- Toxins
Approach to Clinical Management

EXAMINATION

• Grade liver disease e.g portal Hypertension Hepatic Encephalopathy
• Check presence of Co-Morbidities
• Stigmata of Malignancy, Alcoholism Hemochromatosis Wilson’s Disease Lipodystrophy,
Approach to Clinical Management

LABOTRATOTY

- Consider cost
- Consider outcome of each Test
- Basic Tests: LFT, HBV markers, HCV and HAV serology, coagulation, Kidney and FBC, alpha fetoprotein
- Ultrasound
- Expensive Tests: e.g., NAT, LiverBx; maybe for specialist evaluation
A Tiered Testing Service with Test Format Menu and Staff Competencies

NAT: Nucleic acid tests; Lab-NAT: laboratory-based; POC-NAT: at point-of-care; CLIA: chemiluminescence immunoassay; ECL: electrochemiluminescence immunoassay; EIA: enzyme immunoassay; RDT: rapid diagnostic test