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The Metabolic Pandemic: Non-Alcoholic Fatty Liver Disease

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ABSTRACT

Recently re-named Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD), Non-Alcoholic Fatty Liver Disease (NAFLD) has risen to the level of a global Metabolic Pandemic with an estimated prevalence in the world of 25 to 30%. This disease is a progressive continuum of hepatic steatosis, starting with a simple hepatic steatosis and progressing to Non-Alcoholic Steatohepatitis (NASH) and cirrhosis, as well as hepatocellular carcinoma.

A complex, multiplexed, multiple-hit hypothesis pathogenesis results in convergent convergences of insulin resistance, lipotoxicity, mitochondrial oxidative stress, and gut-derived endotoxemia to initiate chronic inflammation. The disease exhibits a clinical paradox in the Indian subcontinent referred to as Lean NAFLD in which affected individuals develop massive hepatic pathology, but they have a normal Body Mass Index (BMI). The high visceral adiposity and genetic populations, namely, PNPLA3 polymorphism, are largely involved in this phenotype.

The synthesis of the world molecular developments and certain Indian epidemiological findings is reviewed and it assesses the current change of invasive biopsy to Non-Invasive Tests (NITs) which includes the FIB-4 Index and Transient Elastography. Therapeutic approaches are examined, including how dual PPAR agonists such as Saroglitazar have come to light and how the so-called Epi-therapeutics could be used to undo maladaptive epigenetic signatures. We end by concluding that this silent crisis needs a multi-systemic approach, focusing on the Gut-Liver-Heart axis, to reduce the long-term cardiovascular and hepatic burden associated with its impact.

KEYWORDS: *Metabolic Dysfunction, MASLD, NAFLD, NASH, Pathophysiology.*

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INTRODUCTION

Previously viewed as a harmless side effect of excessive calorie intake, Non-Alcoholic Fatty Liver Disease (NAFLD) has since turned into the most widespread chronic liver disease worldwide with an incidence of about 25 to 30% of the total world population. It is a continuum of liver redness that starts with easy steatosis (or the buildup of triglycerides in the hepatocytes) and progresses to Non-Alcoholic Steatohepatitis (NASH), fibrosis, cirrhosis, and even Hepatocellular Carcinoma (HCC). According to Younossi et al.[1], the spread of NAFLD all over the world is comparable to the epidemics of obesity and Type 2 Diabetes (T2DM), hence the saying that it is the Metabolic Pandemic of the 21st century.

The NAFLD pathophysiology is complicated and is generally explained by a multiple-hit hypothesis. It is proposed in this model that a first hit of lipid accumulating sensitizes the liver, but the subsequent hits (e.g., oxidative stress, mitochondrial dysfunction, pro-inflammatory cytokines and gut dysbiosis) are what leads to the advancement to severe liver injury. In biochemical terms, the main cause is insulin resistance that alters peripheral lipolysis and rises the flux of free fatty acids (FFAs) to the liver. As noted by Loomba and Sanyal[2], it is this metabolic excess that causes cellular stress responses, such as endoplasmic reticulum (ER) stress and activation of the inflammasome, which coordinates the switch between simple fat to active inflammation.

The pandemic has a distinct and a paradoxical profile in the Indian context. Although the prevalence of generalized obesity is low, compared to western cohorts, the incidence of "Lean NAFLD" is high in the Indian population. People in India tend to develop fatty liver at lower Body Mass Indices (BMIs) because they have increased visceral adiposity and unique genetic predisposition (3). The PNPLA3 (patatin-like phospholipase domain-containing protein[3] risk allele is especially notable with respect to the Indian subcontinent where Eslam et al.[4], posit that genetic variants in conjunction with high carbohydrate diets hasten the onset of the disease in an Indian patient.

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The Indian healthcare system is becoming strained. In urban India, Saraya et al.[5], have highlighted that NAFLD is currently a major cause of cryptogenic cirrhosis and liver transplantation. Also, NAFLD has a close relationship with cardiovascular disease (CVD), so that in the case of a large number of Indian patients, the liver is only the canary in the coal mine of systemic vascular failure. This is no longer a mere academic quest in the field of pathology, it is a clinical crisis to find out the molecular drivers of this condition.

This review is a synthesis of the world developments in the molecular knowledge of NAFLD in conjunction with particular epidemiological and clinical data in India. Exploring how simple steatosis is transformed into NASH and the importance of the gut-liver axis, we seek to overlay the existing state of diagnostic and treatment of this metabolic.

Pathophysiology and the Multiple-Hit Hypothesis

Progression of a normal liver to one full of inflammation and fibrosis is a multifactorial process rather than a linear progression of metabolic, genetic and environmental factors. It has been proposed that the older and better-supported "two-hit hypothesis" (steatosis (first hit) and oxidative stress (second hit)) was the main pathway however modern science has embraced the "multiple-hit hypothesis" as a consideration to the concomitant and parallel processes to promote disease progression.

Lipid Overload and De Novo Lipogenesis (DNL): The NAFLD is characterized by the deposition of intrahepatic triglycerides (IHTG) greater than 5 percent liver weight. This is seen when lipids coming to the liver are more than what can be oxidized or exported by the liver. The main contributors to this lipid pool are peripheral lipolysis of insulin-resistant adipose tissue (~60%), dietary intake (~15%), and De Novo Lipogenesis (DNL) (~25%).

During a state of insulin resistance, hormone-sensitive lipase (HSL) is no longer suppressed in the adipose tissue resulting in a continuous release of non-esterified fatty acids (NEFAs) into the liver. Hyperinsulinemia at the same time causes transcription factors such as SREBP-1c and ChREBP to be activated, leading to an increase in the enzymes of DNL. DNL is markedly increased in NAFLD patients in comparison with healthy controls even in the fasting state, which is why it is a large metabolic engine of steatosis.[7]

Mitochondrial Dysfunction and Oxidative Stress: When the liver gets flooded with fatty acids it tries to counter this by raising the amount of the process of beta-oxidation. This chronic upregulation however causes an electron leak in the mitochondrial respiratory chain producing Reactive Oxygen Species (ROS) including superoxide anions and hydrogen peroxide (H2O2).

Only when the synthesis of ROS exceeds the defense to the antioxidants (e.g., glutathione, superoxide dismutase), there is oxidative stress. Sanyal et al.,[8] have shown that mitochondrial structural abnormalities, including mega-mitochondria, and loss of cristae are very common in patients with NASH. The condition is also exacerbated by the presence of lipotoxic intermediates such as ceramides and diacylglycerols (DAGs) that disrupts insulin signaling and induces cellular apoptosis.

The Inflammasome and Kupffer Cell Play a Role: Innate immune system activation is seen to mark the progression of simple steatosis to Non-Alcoholic Steatohepatitis (NASH). Lipotoxic products, along with ROS, are Damage-Associated Molecular Patterns (DAMPs), a signal perceived by the NLRP3 inflammasome in hepatocytes and resident macrophages (Kupffer cells).

According to Tilg and Moschen,[9] these cytokines attract neutrophils, activate Hepatic Stellate Cells (HSCs), which are the main agents of fibrosis. Chronic inflammatory milieu is also marked by the increased levels of circulating TNF-alpha and IL-6 that worsen the systemic insulin resistance, entering into the vicious metabolic cycle.

Gut-Liver Axis and Endotoxemia: The liver is the initial barrier to the blood flow that originated in the gastrointestinal tract and thus, the gastrointestinal tract-liver axis is one of the most important aspects of the multiple-hit model. Changes in the gut microbiota (dysbiosis) as a result of a diet can raise intestinal permeability, also known as a leaky gut.

This enables the translocation of Lipopolysaccharides (LPS) endotoxins of cell walls of Gram-negative bacteria, into the portal circulation. Zhu et al.[10] discovered that the gut microbiome signature of NAFLD patients is unique, with an abundance of ethanol-producing bacteria. At the liver, LPS interacts with Toll-Like Receptor 4 (TLR4) on Kupffer cells, which results in a strong pro-inflammatory response via the NFkB pathway. This gut-produced hit is more and more accepted as a distinguishing factor between individuals who stay with a stage of mere steatosis and those who develop into NASH.

Genetic Predisposition and Indian situation: Environmental factors take the centre stage; however, genetics dictate the liver injury threshold. I148M polymorphism of the PNPLA3 gene is the greatest genetic variation. This form compromises the breakdown of triglyceride in lipid droplets, which build up. The occurrence of this PNPLA3 variant is very high in the Indian context. Chalasani et al.[11] have noted that steatosis grades increase faster in Indian patients at lower BMIs which has been associated with PNPLA3 as well as the TM6SF2 gene. Moreover, a study by Saraswat et al.,[12] at SGPGIMS Lucknow indicates that a large amount of omega-6 fatty acids and refined carbohydrates in the Indian diet is a particular nutritional hit that combines with these genetic variants to promote fibrogenesis.

Clinical Presentation, Diagnosis, and Lean NAFLD Paradox

The Non-Alcoholic Fatty Liver Disease (NAFLD) nowadays advanced to clinical nomenclature level of MASLD (Metabolic Dysfunction-Associated Steatotic Liver Disease) is frequently characterized as a path with no voice. In most of the patients, the illness does not manifest itself with pain or jaundice until it is on the threshold of the advanced cirrhosis or malignancy. In this section, the clinical face of this metabolic pandemic and its diagnostic change to non-invasive precision are discussed.

The Silent Sentinel: clinical presentation: In its initial phases (allow simple steatosis), NAFLD is asymptomatic. Symptoms are usually non-specific when they appear, chronic fatigue, malaise, dull pain in the right upper quadrant caused by the distension of Glisson capsule by an enlarging, lipid-laden liver. This exhaustion is probably based on a biochemical premise in which mitochondrial depletion may cause the systemic depletion of ATP due to undermining of the electron transport chain.

Since the disease becomes NASH (then MASH - Metabolic Dysfunction-Associated Steatotic Hepatitis), the signs of portal hypertension and hepatic decompensation can be observed. Nevertheless, the worst thing about the silent manifestation is that when a NAFLD patient first has a clinical event, it is often not hepatologic-related, but cardiovascular, as myocardial infarction or stroke, which is why the liver is considered as a sentinel organ of systemic metabolic failure.

The lean NAFLD Paradox: The South Asian Mirage: The issue of Lean NAFLD is one of the most important in the field of hepatology in modern days. Western paradigms tend to associate fatty liver with high Body Mass Index (BMI), but a significant percentage of the Indian and South Asian population in general, develop this condition even though they are considered lean by Western standards (BMI < 25 kg/m2).

This metabolic illusion arises due to the fact that such individuals tend to have a lot of visceral adiposity-fat that is located by the inner organs and not the skin. Anstee et al. (13), have established that a more pro-inflammatory adipose tissue phenotype and a higher occurrence of the PNPLA3 risk allele are common in Lean NAFLD patients. Duseja (14), has believed that the cut-off of overweight has to be redefined in the Indian clinical setting, where the metabolic threshold of liver damage in South Asians is very low in comparison with Caucasians.

Diagnostic Evolution: More than the Liver Biopsy: The liver biopsy has been used as the past gold standard of diagnosis. Non-Invasive Tests (NITs) have emerged however due to its invasive nature, potential complications and a high sampling error.

- **Biochemical Scores and Biomarkers:** Next-generation scoring systems that employ easily accessible laboratory data have gained preference among clinicians. The FIB-4 (Fibrosis-4) index is a very common tool in excluding the advanced fibrosis. The computation is done as follows: FIB-4=Platelets (109/L) ×ALT (U/L) Age (years) ×AST (U/L).

As Newsome et al. (15), noted, an FIB-4 score of less than 1.30 has a high negative predictive value, essentially enable clinicians to reassure their patients without the need of invasive measures. Other emerging biomarkers are Cytokeratin-18 (CK-18) fragments which is a molecular footprint of an apoptotic hepatocyte.

- **Imaging and Elastography:** The development of imaging technology has transformed the process of seeing fat and stiffness. Vibration- Controlled Transient Elastography (VCTE) or the Fibroscan is a procedure, which uses the velocity of a shear wave that traverses the liver to measure it.

  i. Controlled Attenuation Parameter (CAP): Assesses steatosis (fat content).

  ii. Liver Stiffness Measurement (LSM): This is expressed in kilopascals (kPa) which is a proxy of fibrosis.

According to Siddiqui et al., (16), Magnetic Resonance Elastography (MRE), is the most precise non-invasive instrument of fibrosis staging at the moment but the cost is still a challenge in most Indian clinical centers.

Indian Clinical Challenge: Screening and awareness: India has a problem of poorly screened diagnostics coupled with the lack of routine screening. Sanyal,[17] has indicated that a significant number of patients are merely diagnosed accidentally during abdominal ultrasounds when they are being treated of other issues. Even more, Hepatitis B and alcohol-related liver disease is very active in India, which can easily lead to the so-called dual-etiology liver damage and prevent the isolation of the metabolic part. According to the studies published by the Indian National Association to Study the Liver (INASL), and reported by Saraswat,[18] NAFLD is highly correlated with the metabolic syndrome, and thus its diagnosis should stimulate a thorough cardiovascular and kidney screening. The result is a shift of the focus on treatment of an isolated organ to a multi-systemic metabolic crisis, which is treated using this humanic approach to science.

Therapeutic Strategies: From Lifestyle to Emerging Epi-therapeutics: Lifestyle to Emerging Epi-therapeutics Approach to Non-Alcoholic Fatty Liver Disease (NAFLD) management is changing towards a multi-pronged, proactive intervention approach, and is no longer a watchful waiting response. Due to the heterogeneity of the disease, the treatment should be focused on the liver-specific pathology and simultaneous management of the systemic

metabolic dysfunction that is propelling the disease. The first objective is permanent loss of 7 to 10% of total body weight, proven to treat NASH and even reversing fibrosis.

- **Nutritional Biochemistry:** The international gold standard is the Mediterranean diet, which has a large percentage of monounsaturated fatty acids (MUFAs) and polyphenols. Nevertheless, in Indian culture, attention should be paid to decreasing the number of glycemic loads of refined carbohydrates (e.g., polished rice, maida). The modified Indian Diet that has been promoted by Misra et al.[19] highlights high-fiber grains and cancelling trans-fats with more healthy oils such as mustard or olive oil.
- **Exercise and Epigenetics:** As explained in our above analysis, exercise is a strong epigenetic builder. Kistler et al.[20] discovered that regular aerobic exercises decrease hepatic fat regardless of weight loss by suppressing SREBP-1c, the master of lipid metabolism de novo regulator.

Pharmacotherapy: Targeting the Metabolic Engine: When lifestyle changes are insufficient, or when advanced fibrosis is present, pharmacological intervention becomes necessary.

- **PPAR Agonists and the Indian Innovation:** Peroxisome Proliferator-Activated Receptors (PPARs) are nuclear receptors that regulate lipid metabolism and insulin sensitivity.

i. **Pioglitazone (PPAR-gamma):** A global staple that improves insulin sensitivity and has shown significant efficacy in resolving NASH in diabetic patients.

ii. **Saroglitazar (Dual PPAR\alpha/gamma):** This is a landmark in Indian pharmaceutical research. Developed by Zydus Cadila, Saroglitazar was the first drug in the world specifically approved for the treatment of NASH. Bhatt et al.,[21] demonstrated that this dual agonist effectively reduces hepatic fat, triglycerides, and improves glycemic control, making it a tailored solution for the Indian metabolic phenotype.

The GLP-1 and SGLT2 Revolution

The emergence of Glucagon-like Peptide-1 (GLP-1) receptor agonists (e.g., Semaglutide) and Sodium-Glucose Cotransporter-2 (SGLT2) inhibitors has bridged the gap between diabetes management and hepatology. Newsome et al.[22] reported in a landmark trial that Semaglutide led to NASH resolution in over 50% of participants. These drugs act by slowing gastric emptying and promoting a caloric deficit, while also exerting direct anti-inflammatory effects on the liver.

Emerging "Epi-therapeutics" The latest trend in NAFLD management is referred to as the Epi-therapeutics category of drugs that combine the effects of reducing the caloric deficit through slowing gastric emptying, and applying direct anti-inflammatory effects on the liver. As the marks of epigenetics are reversible, drugs that are able to inhibit the writers and erasers of them are highly being studied.

- DNMT and HDAC Inhibitors: Inhibitors of DNA Methyltransferase (DNMT) are also being investigated as tools to re-activate silenced metabolic genes.[23] have proposed that the activation of histone deacetylases (HDACs) may be inhibited by targeting them, which would lead to the cessation of fibrosis development through the suppression of the activation of Hepatic Stellate Cells (HSCs).
- miRNA-based Therapies: miR-122 plays a key role in preserving the hepatocyte phenotype. To recap the normal RNA repertoire of the liver, therapeutic copies or inhibitors (antagomirs) are under development.[24] demonstrated that the silencing of miR-122 leads to a significant decrease in hepatic fatty acid synthesis and the alleviation of steatosis.

Integrative Medicine and the Indian Perspective: In India, there is a huge push in combining AYUSH (Ayurveda, Yoga, Unani, Siddha, and Homeopathy) interventions with conventional care.

- Yoga: Duseja et al.,[25] have pointed out that Yoga-based lifestyle interventions do not only aid in weight loss but also alleviate systemic oxidative stress, and pro-inflammatory cytokines such as TNF-alpha.
- Ayurvedic Compounds: The botanical extracts like Silymarin (Milk Thistle) and Curcumin (Turmeric) are under investigation due to their mechanobiological actions. According to Sanyal,[26] because of its capability to prevent the NFkB pathway, Curcumin is an important natural anti-inflammatory agent in the prevention of steatohepatitis at its initial stages.

Discussion, Potential Problems and Future Research

There is nothing hyperbolic about calling Non-Alcoholic Fatty Liver Disease (NAFLD) a Metabolic Pandemic: it indicates a systemic collapse of human metabolic homeostasis in response to the contemporary environmental stressors. The shift of the biochemical build-up of triglycerides to the pathological devastation of hepatic tissue is a pivotal "tipping point" at which the body adaptive processes turn maladaptive.

DISCUSSION

As has been highlighted in this review, NAFLD is not a common liver condition. Rather, it is a reflection of such systemic problems as insulin resistance, adipose tissue dysfunction, and chronic inflammation. The development of the multiple-hit hypothesis has allowed us to think beyond just a mere fatty liver and has led to it being a multi-organ crisis of the gut, the brain, and the cardiovascular system.

Cardiovascular disease is the main cause of mortality of NAFLD according to the consensus of the world (Younossi (1), and Powell et al.[6] But there is a critical twist to the situation in the Indian context: a Lean NAFLD phenotype. This group does not match the conventional Western obesity-first paradigm,

indicating that among South Asian individuals, genetic susceptibility (PNPLA3 variant) and certain nutritional stress factors (high fructose and refined carbohydrate) can cause progressive liver damage at low body weights.[27]

Critical challenges in the Indian Landscape: Nevertheless, there are various very tough obstacles on the way to the Indian metabolic pandemic:

- **The Diagnostic Gap in Rural India:** Although in urban centers such as Lucknow, Delhi and Mumbai, high end Vibration-Controlled Transient Elastography (VCTE) is available, rural populations, in which most of India is located, have access to outmoded ultrasound systems which are ineffective in detecting early fibrosis.
- **The "Silent" Nature of Progression:** NAFLD has no symptoms until it progresses to cirrhosis, which has led to an enormous gap in public knowledge. When patients receiving an ultrasound report of fatty liver, many perceive this as a non-threatening incidental finding and not a serious metabolic warning.
- **Absence of Universal Treatment Protocols:** India is the first to use Saroglitazar (21) but there are no formalized "Metabolic Clinics to point of care treatment with a combination of hepatologists, cardiologists, and nutritionists.
- **Cultural and Dietary Inertia:** It is hard to change the traditional high-carbohydrate Indian diet. Cultural trend towards festive foods with high levels of trans-fats and sugars serves as a form of continual nutritional shot that undermines lifestyle changes.

Future Directions: Precision Hepatology is the future of NAFLD management as the shift in focus is away from a universal model to a personalized treatment.

- Artificial Intelligence and Screening: Kumar et al.,[28] points out that AI algorithms are under development to examine conventional blood parameters, and identify persons at high risk of fibrosis, even before they set foot in a clinic. This "tele-screening" can transform rural health in India.[29]
- The Microbiome and Transcriptomics: Future treatments are expected to target the Gut-Liver Axis. Studies are now underway into the investigation of fecal microbiota transplants, and tailored probiotics to repair intestinal permeability and the "leaky gut" endotoxemia underlying NASH.
- Genetic Tailoring: Early detection of PNPLA3/TM6SF2 allele carriers, and their subsequent enforcement of lifestyle modifications in early life, may prevent the risk by silencing the genetic risk with epigenetics.[30]

CONCLUSION

The clear-cut metabolic bane of the day is NAFLD. The disorder is characterized by an intersection of the macro-world of the lifestyle and culture with the micro-world of biochemistry and genetics. As the world pandemic keeps expanding, the Indian viewpoint has been a special source of information on the importance of visceral adiposity and genetic disposition. The scientific methodology the one which will help to effectively manage this pandemic - will be the one that will be carried out in such a manner that it will be based on the rigor of molecular pathology, and the compassion of clinical care. With the emphasis on the early non-invasive diagnosis, promotion of particular dietary recalibration, or the Indian pharmacological developments based on Saroglitazar, we will be able to start turning the trend of this silent crisis around. The final aim is to take the liver out of a metabolic storage state, to a state of metabolic vitality so that this silent disease will cease to have the last word in determining human longevity.

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