

Incidental Detection of Non-Alcoholic Fatty Liver Disease on Ultrasound: Clinical Significance, Gaps in Practice, and Management Strategies with A Focus on Saudi Arabia

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Abstract

Original Research Article

Non-alcoholic fatty liver disease (NAFLD) represents one of the most common chronic liver disorders globally and has become increasingly recognised as a multisystem condition strongly associated with obesity, insulin resistance, type 2 diabetes and cardiovascular disease. Abdominal ultrasound remains the most widely used imaging modality for identifying hepatic steatosis, with many cases detected incidentally during investigations for unrelated complaints. Although these findings provide a potential opportunity for early identification of metabolic risk, incidental NAFLD is frequently overlooked or labelled as clinically insignificant. Evidence suggests that limited awareness, incomplete metabolic assessment and lack of structured counselling are common following incidental detection. This omission has important implications because NAFLD can progress to non-alcoholic steatohepatitis, fibrosis, cirrhosis and hepatocellular carcinoma, and is associated with increased cardiovascular morbidity and mortality. Early intervention focusing on weight reduction, dietary modification and physical activity can improve hepatic fat, slow progression and reduce cardiometabolic risk. However, adoption of evidence-based management strategies remains inconsistent, particularly in primary care where NAFLD is often first recognised. These challenges are amplified in settings with high metabolic risk such as Saudi Arabia, where obesity and diabetes are highly prevalent. Improving outcomes requires a multifaceted strategy that includes enhanced clinician training, standardised reporting practices, integration of risk stratification tools, improved access to lifestyle support and public health initiatives. Recognising incidental ultrasound findings as a marker of systemic metabolic dysfunction rather than a benign radiological observation is central to enabling timely intervention. Addressing clinical and system-level barriers may reduce the long-term burden associated with NAFLD and improve metabolic health outcomes in high-risk populations such as those in Saudi Arabia.

Keywords: Non-alcoholic fatty liver disease; hepatic steatosis; ultrasound; metabolic syndrome; obesity; Saudi Arabia; primary care.

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INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as a major public health concern, affecting up to one-quarter of adults globally (Younossi *et al.*, 2016). Historically perceived as a benign hepatic condition, NAFLD is now recognised as a multisystem disorder strongly associated with insulin resistance, obesity, metabolic syndrome and cardiovascular disease (Byrne and Targher, 2015). Disease progression varies substantially, with some individuals remaining stable over years, while others develop non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis or

hepatocellular carcinoma (Loomba *et al.*, 2021). Fibrosis severity is the strongest predictor of morbidity and mortality, highlighting the importance of early identification and risk stratification.

Abdominal ultrasound is widely used in clinical practice and is frequently requested to evaluate abdominal pain, suspected gallbladder disease or abnormal liver enzyme results. Hepatic steatosis is often reported incidentally, meaning the imaging was undertaken for reasons unrelated to liver disease. Despite its clinical relevance, incidental NAFLD is frequently ignored or regarded as a minor finding, particularly when

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liver biochemistry is normal (Dyson *et al.*, 2014). Such clinical inertia represents a major missed opportunity because even early-stage steatosis has metabolic implications, and lifestyle modification can improve outcomes (Romero-Gómez *et al.*, 2017).

The global rise of NAFLD reflects wider trends in sedentary behaviour, energy-dense diets and increasing prevalence of obesity and diabetes. The Middle East has some of the highest reported prevalence rates, influenced by lifestyle transitions and genetic predisposition (Alsheikh *et al.*, 2020). In Saudi Arabia, studies suggest NAFLD prevalence ranges from 16% to more than 40%, with substantially higher burden among individuals with diabetes and obesity (Alnezi *et al.*, 2023). Importantly, NAFLD is increasingly identified in younger age groups, potentially leading to earlier onset of complications.

Despite its significance, NAFLD is under-recognised within clinical practice, particularly when detected incidentally. Barriers include limited awareness of disease progression, uncertainty regarding appropriate investigations and lack of confidence in lifestyle counselling. These challenges are amplified in healthcare systems with fragmented services, limited access to specialists and cultural barriers to behaviour change. Understanding the implications of incidental NAFLD and recognising opportunities for early intervention is essential to reducing long-term disease burden.

The aim of this narrative review is to examine the epidemiology, pathophysiology and clinical consequences of NAFLD, explore the role of ultrasound in detection, identify gaps in current practice and discuss management strategies with specific attention to challenges and opportunities in Saudi Arabia.

EPIDEMIOLOGY

Global prevalence estimates suggest that NAFLD affects 24–30% of adults, although rates vary considerably by region due to differences in obesity, diet, ethnicity and socioeconomic status (Riazi *et al.*, 2022). Industrialised nations such as the United States and Europe demonstrate high prevalence linked to sedentary lifestyles and metabolic disorders. In individuals with obesity or diabetes, prevalence exceeds 60%, reflecting the close association between NAFLD and insulin resistance (Wong *et al.*, 2015).

In recent years, increasing prevalence has also been reported in Asian populations, despite lower average BMI, highlighting ethnic susceptibility and the importance of visceral adiposity (Ye *et al.*, 2020). Lean NAFLD, characterised by hepatic steatosis in individuals with normal BMI, remains clinically significant, as these patients may still develop fibrosis and cardiovascular disease (Choi *et al.*, 2021).

NAFLD is also increasingly recognised in children, reflecting global trends in paediatric obesity. Early-onset NAFLD is concerning, as paediatric steatosis has been associated with higher lifetime risk of metabolic disease and advanced fibrosis (Anderson *et al.*, 2015).

MIDDLE EAST AND SAUDI ARABIA

The Middle East has some of the highest global rates of obesity and diabetes, resulting in substantial NAFLD burden (Alsheikh *et al.*, 2020). In Saudi Arabia, lifestyle changes associated with urbanisation and dietary westernisation have contributed to widespread metabolic dysfunction. National studies suggest NAFLD prevalence may range from 16% to 40% in the general population and exceed 50% in individuals with obesity or diabetes (Alnezi *et al.*, 2023; Hussein *et al.*, 2023).

Paediatric NAFLD is increasingly reported, paralleling high rates of adolescent obesity and raising concerns about long-term disease burden (Babatin *et al.*, 2020). Despite high prevalence, awareness remains low, resulting in underdiagnosis and limited early intervention (Alswat and Ahmed, 2020).

The high baseline risk in Saudi Arabia underscores the importance of early detection, particularly when steatosis is identified incidentally on ultrasound.

PATHOPHYSIOLOGY

NAFLD is characterised by excessive hepatic triglyceride accumulation resulting from metabolic and inflammatory processes. Insulin resistance plays a central role by increasing lipolysis, elevating circulating fatty acids and promoting hepatic fat uptake (Bugianesi *et al.*, 2005). Excess glucose and fructose intake stimulate de novo lipogenesis, further enhancing lipid accumulation (Schwarz *et al.*, 2015).

Progression from steatosis to NASH involves oxidative stress, mitochondrial dysfunction and activation of inflammatory pathways. Pro-inflammatory cytokines, including TNF- α and IL-6, induce hepatocyte injury and promote recruitment of immune cells, perpetuating inflammation (Tilg and Moschen, 2010). Adipose tissue dysfunction further contributes to systemic inflammation and lowers protective adiponectin levels (Polyzos *et al.*, 2013).

Activation of hepatic stellate cells leads to fibrosis and architectural remodelling. Fibrosis stage is strongly correlated with mortality risk, regardless of steatosis severity (Angulo *et al.*, 2015). Individuals with diabetes often experience accelerated fibrosis progression, reflecting metabolic stress (Targher *et al.*, 2010).

Genetic susceptibility, including variants in PNPLA3 and TM6SF2, influences disease risk and

severity, and may partly explain regional variation in prevalence (Eslam *et al.*, 2018). Environmental factors, including diet, sedentary lifestyle and exposure to endocrine disruptors, further modulate risk.

Lean NAFLD represents a clinically important phenotype, influenced by visceral adiposity, genetics and metabolic dysfunction, despite normal BMI (Choi *et al.*, 2021).

Understanding these mechanisms underscores the importance of addressing metabolic risk following incidental detection.

Clinical Significance

Although NAFLD may remain asymptomatic for years, the condition is associated with substantial morbidity. Approximately 10–20% of individuals with steatosis progress to NASH, and 15% of those with NASH develop cirrhosis (Loomba *et al.*, 2021; Dulai *et al.*, 2017). Hepatocellular carcinoma may develop in both cirrhotic and non-cirrhotic livers.

Cardiovascular disease is the leading cause of death in NAFLD, exceeding liver-related mortality (Adams *et al.*, 2005). Individuals with NAFLD have higher rates of coronary artery disease, stroke and heart failure, reflecting systemic inflammation, dyslipidaemia and pro-atherogenic factors (Targher *et al.*, 2010).

NAFLD significantly increases risk of type 2 diabetes, independent of BMI, and individuals with diabetes have higher risk of progressive liver disease (Sung *et al.*, 2019). Fatigue and impaired health-related quality of life are common, and psychological distress may exacerbate disease progression (Dan *et al.*, 2007; Sayiner *et al.*, 2016).

Importantly, ultrasound-detected steatosis is a marker of metabolic dysfunction, even when liver enzymes are normal. However, incidental findings are often overlooked, resulting in missed opportunities for early intervention.

Role of Ultrasound

Ultrasound is widely used due to its affordability, accessibility and safety. Diagnostic performance is high for moderate-to-severe steatosis, but sensitivity declines for mild steatosis or in individuals with obesity (Hernaez *et al.*, 2011). Ultrasound cannot differentiate between steatosis and NASH or reliably assess fibrosis, but it remains valuable for initial detection.

Incidental findings are common due to widespread use of ultrasound for non-hepatic indications. Yet radiology reports may lack detail or fail to quantify severity, limiting clinical response (Patel *et al.*, 2019). Standardised reporting could facilitate improved follow-up.

In resource-limited settings, ultrasound may serve as a practical tool for opportunistic case-finding among high-risk populations.

MANAGEMENT

Lifestyle modification remains first-line treatment. Weight loss of 7–10% is associated with improvement in steatosis, inflammation and fibrosis (Vilar-Gomez *et al.*, 2015). Diets low in saturated fat and refined sugars and high in vegetables and whole grains are recommended, with evidence supporting Mediterranean dietary patterns (Trovato *et al.*, 2018). Exercise improves steatosis independent of weight change (Sullivan *et al.*, 2012).

Screening and treatment of metabolic comorbidities is essential. Statins are safe and effective in NAFLD, despite historical concerns (Athysos *et al.*, 2021). Glycaemic control may improve steatosis but has limited impact on fibrosis (Belfort *et al.*, 2006).

Pharmacotherapy remains reserved for selected individuals. Pioglitazone and GLP-1 receptor agonists show benefit but have side-effect considerations (Cusi, 2009; Armstrong *et al.*, 2016). Vitamin E may improve histology in non-diabetic adults but concerns about long-term safety remain (Sanyal *et al.*, 2010).

Fibrosis risk stratification using FIB-4 or NFS can identify high-risk individuals and reduce unnecessary referrals (Angulo *et al.*, 2007), yet utilisation remains low. Multidisciplinary support improves adherence, but access is variable.

Gaps in Practice

Under-recognition of NAFLD persists despite evidence of harm. Clinicians often regard steatosis as benign when liver enzymes are normal (Guzman *et al.*, 2020). Only a minority of individuals receive metabolic screening or lifestyle counselling following incidental detection (Alhodaib *et al.*, 2021).

Barriers include limited knowledge, unclear guidelines, time constraints and lack of referral pathways. Risk stratification tools are rarely used and radiology reporting may be inconsistent (Byrne *et al.*, 2021).

System-level barriers, including fragmented care and lack of integrated services, further limit effective management (Wallace *et al.*, 2018).

Challenges in Saudi Arabia

Saudi Arabia faces high rates of obesity and diabetes, contributing to substantial NAFLD burden (Memish *et al.*, 2014). Clinician awareness and confidence in managing NAFLD are limited, resulting in inconsistent practice (Sabry *et al.*, 2020). Care is fragmented, and access to multidisciplinary services is variable (Al-Shaikh *et al.*, 2018).

Patient awareness is low, and many individuals do not perceive NAFLD as serious (Alghamdi *et al.*, 2021). Health system priorities have not yet fully incorporated NAFLD, despite increasing burden (Rahman *et al.*, 2021).

Recommendations

Improving NAFLD management requires enhanced clinician education, standardised reporting and structured pathways. Integration of risk stratification tools into electronic records could support timely intervention (Lim *et al.*, 2019). Expanding access to lifestyle services and adopting culturally tailored public health strategies may improve adherence (Ratziu *et al.*, 2016). Integrated care models that link primary care and specialist services may reduce fragmentation (Lazarus *et al.*, 2021). Policy initiatives should prioritise NAFLD within national health strategies due to its long-term burden.

CONCLUSION

NAFLD is a prevalent and clinically significant condition with substantial hepatic and cardiometabolic consequences. Incidental detection on ultrasound presents an opportunity for early identification and intervention; however, this potential is often unrealised. Limited awareness, absence of structured pathways and fragmented services contribute to suboptimal management, especially in settings with high metabolic risk such as Saudi Arabia. Improving clinical outcomes requires enhanced knowledge, standardised approaches, integrated care and culturally tailored behavioural support. Recognising incidental steatosis as a marker of systemic risk is essential to reducing the future burden of disease.

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