Association between Nonalcoholic Fatty Liver Disease and Type 1 Diabetes Mellitus

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ABSTRACT

Background: Nonalcoholic fatty liver disease (NAFLD) is increasingly recognized in type 1 diabetes mellitus (T1DM), challenging the traditional view of T1DM as pure insulin deficiency.

Objective: To synthesize evidence on NAFLD prevalence, risk factors, diagnostic approaches, and clinical implications in T1DM.

Methods: Systematic review of PubMed, Embase, Scopus, and Cochrane Library (2021–2026). Included studies enrolling T1DM patients with NAFLD assessed by transient elastography, MRI, ultrasound, or non-invasive scores. Ten studies (1,765 participants) met criteria; quality assessed via JBI checklist.

Results: NAFLD prevalence in adults with T1DM ranged 11.6–43% (largest cohort: 28.5%); fibrosis prevalence 0% (pediatric) to 11% (adults). Central obesity was the strongest risk factor (OR 3.1–4.13), followed by higher BMI, insulin resistance markers (eGDR, VAI, TG/HDL-C), poor glycemic control, and longer diabetes duration. Notably, 59–72% of patients with NAFLD/fibrosis had normal ALT, making liver enzymes inadequate for screening. Non-invasive tests (FIB-4, CAP) showed utility for risk stratification. Intensive lifestyle intervention improved NAFLD markers and reduced insulin requirements in obese T1DM patients.

Conclusion: NAFLD affects ~20–30% of adults with T1DM, with a significant subset having advanced fibrosis. Routine screening is warranted in at-risk individuals—particularly those with obesity, central adiposity, insulin resistance, or poor glycemic control—regardless of normal ALT. Future research should explore NAFLD natural history, pharmacotherapy, and establish evidence-based screening guidelines.

Keywords: Nonalcoholic Fatty Liver Disease; Type 1 Diabetes Mellitus; Hepatic Steatosis; Liver Fibrosis; Transient Elastography; Insulin Resistance.

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INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) is now the most common chronic liver disease worldwide, with around a quarter of the global adult population being affected and the prevalence further increasing in the background of the increasing epidemics of obesity and metabolic syndrome. NAFLD is the accumulation of hepatic fat in the absence of significant alcohol intake or other secondary causes of liver disease in hepatocytes ranging from simple hepatic steatosis to nonalcoholic steatohepatitis (NASH) which can lead to fibrosis, cirrhosis, hepatocellular carcinoma and end-stage liver disease [1]. Beyond the liver, the significance of NAFLD is now recognized to be a multisystem disease that has been shown to be independently associated with an increased risk of atherosclerotic cardiovascular disease, chronic kidney disease, heart failure, and all-cause mortality. In fact, cardiovascular disease contributes to the mortality rate in patients with NAFLD of at least 40%, and there is a significant extrahepatic burden of the condition [2].

The link between NAFLD and type 2 diabetes mellitus (T2DM) is bidirectional and strong, with insulin resistance being one of the key pathophysiological links. The pooled prevalence of NAFLD was estimated to be 65.33% (95% confidence interval, CI: 62.35%–68.18%) in the previous studies, and the prevalence among patients with T2DM was confirmed to be 65.33% (95% CI: 62.35%–68.18%) in this case. In addition, most patients with NAFLD also suffer from T2DM, with a high proportion of patients with advanced fibrosis [3]. The prevalence of NAFLD in patients with T2DM is between 50% and 70% in all continents except in Africa, where a low prevalence of 30.4% has been reported. All of these pathways are linked to insulin resistance and play a central role in the development of this strong association, including the promotion of the de novo lipogenesis in the liver, the inhibition of fatty acid oxidation in the mitochondria and the stimulation of lipolysis in the adipose tissue, all contributing to the accumulation of intrahepatic triglycerides. For this reason, current clinical practice guidelines suggest that screening for NAFLD should be routinely offered to people with T2DM as there is a high prevalence and a risk of progressive liver disease [1,4].

Type 1 diabetes mellitus (T1DM) has long been regarded as an absolute insulin deficiency in which autoimmune destruction of the pancreas beta-cells is responsible for the condition and, therefore, T1DM has been traditionally not regarded as a high risk population for NAFLD. There is however increasing evidence to challenge this paradigm, highlighting that the clinical and metabolic profile of T1DM is much more complex than previously recognised [5].

The increasing rates of obesity in people with T1DM and the awareness that insulin resistance is a common finding in many people with T1DM, especially those with poor glycemic control, central adiposity, and/or a family history of T2DM, have led to a re-evaluation of the risk of NAFLD in this population. In addition, as weight gain has been linked to the use of intensive insulin therapy, this could also increase the likelihood of patients with T1DM developing hepatic steatosis [5,6].

There is a need to identify risk factors in T1DM for targeted screening and prevention of NAFLD. There was a consistent association between the presence of NAFLD and obesity, central adiposity, insulin resistance (estimated glucose disposal rate, visceral adiposity index, triglyceride to high-density lipoprotein cholesterol ratio), poor glycemic control, and longer duration of diabetes in patients with T1DM (based on emerging evidence) [5,7].

In addition, the value of non-invasive techniques such as transient elastography (with measurement of controlled attenuation parameter), of Fibrosis-4 index and other serum biomarkers have been investigated in this population with interesting results for risk stratification and exclusion of advanced fibrosis. Considering the high prevalence of NAFLD in T1DM and its potential to develop into advanced liver disease and cardiovascular complications, there is strong need for a detailed assessment of the epidemiology, risk factors and ideal screening programs of NAFLD in T1DM patients [6,8].

Thus, the purpose of this systematic review is to synthesize the available evidence of the association between NAFLD and T1DM, focusing on prevalence, risk factors, and clinical approaches to diagnosis and implications for clinical care and future research.

METHODOLOGY

This systematic review was done following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [9].

Search Strategy

Four major electronic databases (PubMed/MEDLINE, Embase, Scopus and the Cochrane Library) were searched systematically. A search was conducted to find all related studies published during the last 5 years (2021-2026) and to capture the latest and most recent information on the subject. The review team and an experienced medical librarian participated in the development of the search strategy to ensure the strategy was as sensitive and specific as possible. Various combinations of the following Medical Subject Headings (MeSH) and keywords were used: ("Nonalcoholic Fatty Liver Disease" OR "NAFLD" OR "Fatty Liver" OR "Hepatic Steatosis" OR "Metabolic Dysfunction-Associated Steatotic Liver Disease" OR "MASLD") AND ("Type 1 Diabetes Mellitus" OR "Type 1 Diabetes" OR "T1D" OR "T1DM"

OR "Insulin-Dependent Diabetes Mellitus" OR "IDDM") AND ("Prevalence" OR "Epidemiology" OR "Risk Factors" OR "Fibrosis" OR "Transient Elastography" OR "Controlled Attenuation Parameter" OR "CAP" OR "Liver Stiffness Measurement" OR "LSM" OR "Magnetic Resonance Imaging" OR "MRI" OR "Noninvasive Tests"). Studies on human beings published in English were included and no efforts were made to limit the search geographically or according to the type of setting. Search terms were combined using boolean operators (AND, OR) and truncation was used as needed to find variations in documentation.

Eligibility Criteria

To be eligible for inclusion in this systematic review, studies had to adhere to the following predefined criteria using the PICOS framework (Population, Intervention/Exposure, Comparator, Outcome, Study Design). Studies were included in the analysis for the population that included patients with a definite diagnosis of type 1 diabetes mellitus. Diagnosis was accepted as per criteria of the original study authors, usually clinical criteria, islet autoantibodies and/or C-peptide levels were included. Studies that included only patients with type 2 diabetes mellitus (T2DM) were excluded, to ensure the specificity of the review question. Studies that evaluated the presence of NAFLD in T1D patients were included in the intervention/exposure group. The diagnosis of NAFLD was accepted by using various modalities including transient elastography with controlled attenuation parameter (CAP), magnetic resonance imaging-proton density fat fraction (MRI-PDFF), magnetic resonance elastography (MRE), ultrasound, computed tomography, or the use of validated non-invasive scoring systems such as the Hepatic Steatosis Index (HSI), Fatty Liver Index (FLI) and Fibrosis-4 (FIB-4) score. Studies with or without a comparator group were included for the comparator; studies comparing patients with T1D to healthy controls or other populations were included to give context to the prevalence estimates. The primary aim was to find the prevalence of NAFLD in patients with T1D for the outcomes. Secondary outcomes were the presence of liver fibrosis, risk factors for NAFLD, glycemic parameters and their correlation with NAFLD, insulin resistance markers and their correlation with NAFLD, body composition measures and their correlation with NAFLD, diagnostic relevance of non-invasive tests and clinical implications for screening and management.

Several criteria were used to filter studies from this review. Studies that included only patients with type 2 diabetes mellitus (T2DM) were excluded. Studies that did not report original data, that is, narrative reviews, editorials, commentaries, letters, opinion articles were not included unless they reported original data or provided significant confirming evidence. Case reports and case series of less than five were excluded because of small number of subjects in the series for generalizability. Extra-English publications, animal studies and in vitro studies were not included. Studies with no full text were not included. Further, studies in which NAFLD assessment methods were poorly defined, or data were insufficient to extract were excluded.

Study Selection and Screening

The process of study selection was done in line with PRISMA guidelines [9] and was carried out in 2 stages, both of which are performed with Rayyan, which is a web-based systematic review software that can be used to facilitate collaborative screening and data extraction. Rayyan was selected as it aims to facilitate the screening process, minimize screening errors, and provide collaboration tools for team-based research with blinding to reduce bias in the selection of studies. The identified electronic database records from all the searches were exported to Rayyan, and duplicates were automatically identified and removed from the electronic database using Rayyan duplicate detection algorithm, and checked by hand to make sure of their accuracy. Titles and abstracts were initially screened by two independent reviewers after deduplication in line with the pre-defined eligibility criteria. At this stage studies that it was clearly evident from the title and abstract that they were irrelevant were discarded. To reduce bias the reviewers' decision on each paper was made blind.

The full texts of all studies that met the criteria for the title and abstract screening were then retrieved and thoroughly evaluated in detail by the same two independent reviewers. Documentation and reporting of the reasons for exclusion at this stage was performed according to PRISMA guidelines. The full-text screening included detailed screening of each study against all the eligibility criteria with special focus on study design, characteristics of the population, NAFLD diagnosis methods, and reporting of relevant outcomes. When there was any difference of opinion, it was settled by discussion and consensus, the third reviewer serving as the arbitrator if needed. The process of selecting studies was reported using a PRISMA flow diagram which outlines the number of records retrieved, screened, assessed for eligibility and included in the systematic review.

Data extraction and management.

Two reviewers independently extracted the data, and used a standardized data extraction form that was developed specifically for this review and piloted on a subsample of three studies for consistency and comprehensiveness. The data extraction form was developed to systematically and reproducibly capture all the information required. When disagreements occurred between reviewers during the

data extraction process, they were discussed and consensus reached, and the third reviewer was referred to when this was not achieved. Data extracted were synthesized and analyzed in the form of full summary tables. The extraction of data was done in MS EXCEL which is organized and helps to analyze data later.

The following types of information were asked for on the data extraction form: general study characteristics (lead author, year of publication, journal, country, study design, study period, and setting); participant characteristics (sample size, age, sex distribution, diabetes duration, BMI, glycemic control, measured by HbA1c, and other relevant clinical parameters); NAFLD assessment methodology (diagnostic method, cutoff values for steatosis and fibrosis, and prevalence of NAFLD and fibrosis); and outcome data (identified risk factors, measures of association, such as odds ratios and confidence intervals, glycemic parameters and association with NAFLD, insulin resistance markers, body composition measures, and diagnostic performance of non-invasive tests, as well as clinical implications).

Quality Assessment and Risk of Bias

Two reviewers (R1 and R2) independently scored the methodological quality and risk of bias of the included studies according to the Joanna Briggs Institute (JBI) Critical Appraisal Checklist for Analytical Cross-Sectional Studies [10]. The tool was chosen as being the most suitable for use in this review as most studies included in the review used a cross sectional design. The JBI checklist includes eight questions that assess the methodological domains of a clearly defined inclusion criteria (Q1), detailed description of study subjects and setting (Q2), valid and reliable measurement of exposure (Q3), valid and reliable measurement of outcome (Q4), identification of confounding factors (Q5), management of confounding factors (Q6), valid and reliable outcome measurement (Q7), and appropriate statistical analysis (Q8).

The information reported in the included study was used to rate each item on the checklist as ‘Yes’ (low risk of bias), ‘No’ (high risk of bias), ‘Unclear’ (insufficient information to make a rating), or ‘Not Applicable'. Studies were graded depending on the number of ‘Yes' answers: studies with 7 or more ‘Yes' answers were graded as low risk of bias and studies with 5 to 6 ‘Yes' answers were graded as moderate risk of bias, and studies with less than 5 ‘Yes' answers were graded as high risk of bias. If no agreement was reached between the two or more reviewers, the third reviewer (R3) was available for arbitration and was asked to reach a consensus in case of any disagreement. The findings of the quality assessment were summarized in a table and the overall risk of bias of each study was taken into account in interpreting the findings of the review. Although the number of studies precluded formal meta-analytic sensitivity analysis, sensitivity analyses were planned to further explore the influence of moderate and high risk of bias studies on the overall conclusions. Quality assessment was undertaken with sensitivity to the facts that there might be publication bias, selection bias, and measurement bias and it influenced the recommendations and conclusions.

Data collection, synthesis and analysis.

In view of the wide variation among the studies included in the review with respect to study design, population characteristics, the way NAFLD was assessed and how its outcome was reported, a narrative synthesis approach was used instead of formal meta-analysis. Narrative synthesis was carried out using guidelines provided by the Cochrane Handbook for Systematic Reviews of Interventions and the Synthesis Without Meta-analysis (SWiM) reporting guidelines. The synthesis was organized to highlight the most important themes related to the review question, such as NAFLD prevalence in T1D, risk factors and associated factors, approaches to diagnosis and non-invasive tests, and implications for clinical practice. Qualitative assessment of heterogeneity was conducted to determine whether there were differences in study populations, methods, or outcome definitions among included studies.

Studies for the narrative synthesis were broken down into three categories: studies reporting prevalence of NAFLD and fibrosis, studies defining risk factors/associations, and studies assessing diagnostic tools and interventions. The data were summarized descriptively with ranges, medians and means, as appropriate. The results of the studies included in each theme were synthesized, focusing on consistency or lack of consistency in results across studies.

RESULTS

The systematic literature search initially identified 216 records across databases, of which 114 duplicates were removed, leaving 102 records for screening. After title and abstract screening, 39 records were excluded, and 63 reports were sought for retrieval. However, 38 full-text reports could not be accessed, leaving 25 reports assessed for eligibility. Of these, 15 were excluded due to wrong outcomes (n=11), wrong population (n=3), or being conference abstracts only (n=1), ultimately resulting in 10 studies included in the final systematic review.

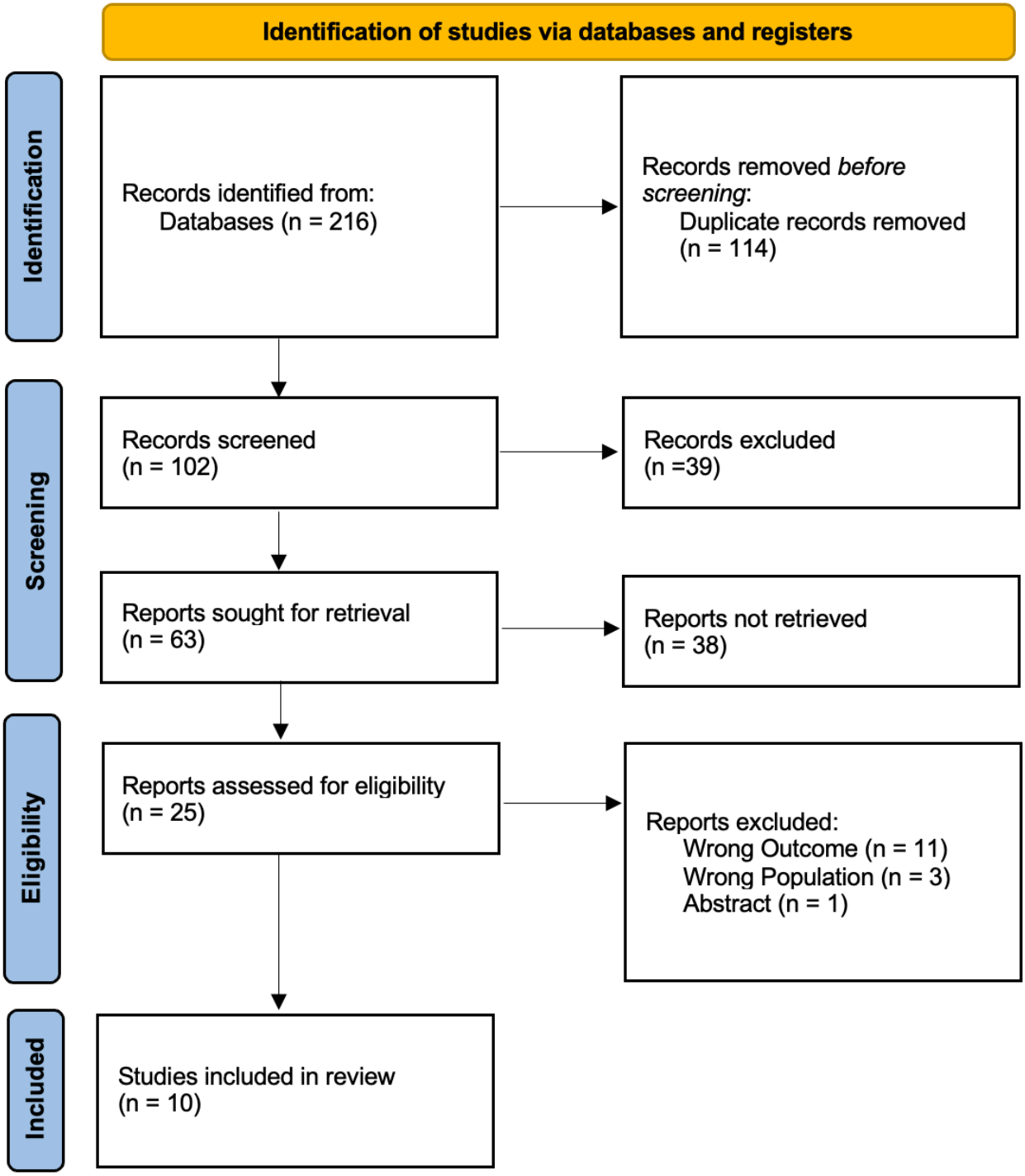

Figure 1: PRISMA Flow Diagram of Study Selection Process.

Table 1 summarizes the demographic and methodological features of the 10 studies reviewed. The largest number of adults with type 1 diabetes (T1D) included in this review was from the USA (n = 537) [11]. The research done by **Dhaver *et al.*** [12] **and Tomah *et al.*** [13] both from USA included 200 and 285 adult participants respectively. For the pediatric population, **Zeiad *et al.***[14] included 18 children aged 7-20 years, while **Sae-wong *et al.*** [18] included 50 children and young adults from Thailand. The remaining studies were conducted in adults with the following numbers of participants: **Tomah S. *et al.*** [15] (88 participants in the intervention group and 88 controls); **Grzelka-Woźniak *et al.*** [16] (151 participants); **Ubrangala *et al.*** [17] (85 participants); **Parente *et al.*** [19] (121 participants); and **de Vries *et al.*** [20] (150 participants). Except for **Tomah S. *et al.*** [15] who used a retrospective matched-cohort design, all of the studies used a cross-sectional design.

Studies included different populations with ages ranging from mean age of 16.9 years in the pediatric studies [18] to the largest adult study with a mean age of 49 years [11]. The percentage of females in the studies varied from 45% [20] to 65% [15] and the majority of studies reported a percentage of around 55% females. The length of diabetes consistently was reported as a median of 6.5 years [18] to 29 years [11]. There was significant variation in BMI, from 25 kg/m² [20] to 35.3 kg/m² [15] in the obese group. Some studies reported glycemic control (HbA1c) levels to be 7.1% [11,13] to 8.2% [15]. Most studies used transient elastography (TE) and the controlled attenuation parameter (CAP) measurement as methods to assess NAFLD, with cut-off values ranging from 238 dB/m [16] to 274 dB/m [11,12,13]. **Saewong *et al.*** [18] **and Parente *et al.*** [19] used more advanced imaging techniques, magnetic resonance imaging-proton density fat fraction (MRI-PDFF) and magnetic resonance elastography (MRE), respectively, whereas **Tomah S. *et al.*** [15] used the non-invasive Hepatic Steatosis Index (HSI) as a surrogate marker. The reported prevalence of NAFLD varied from study to study, from 10% in the pediatric MRI study [18] to 43% in the Polish study [16] with most studies conducted in adults showing prevalence of 27% to 34%. Four studies [11,13,14,17] reported prevalence of liver fibrosis, assessed via liver stiffness measurement (LSM), ranging from 0% in the study by **Sae-wong *et al.*** in the children group [18] to 11% in the study by **Tomah *et al.*** [13]**.**

The results of each of the studies included in the analysis are summarized in Table 2, which includes the main objectives, main results, risk factors identified, and clinical implication of each study. Despite normal liver enzymes, there was a significant burden of NAFLD in the T1D population in all studies, with **Salah *et al.*** (2023) [11] reporting that 4.1% of the population had ≥F2 fibrosis, suggesting nonalcoholic steatohepatitis (NASH). **Dhaver *et al.*** 2023 [12] presented novel findings, showing that glucose variability (coefficient of variation) was associated with hepatic steatosis in lean patients (BMI <25 kg/m²) only; this indicates that a different pathophysiological mechanism exists for this group. The authors of Tomah ***et al.*** [13] pointed out that alanine aminotransferase (ALT) was not suitable for screening, as 72% of the patients with NAFLD and 59% of the patients with fibrosis had normal ALT levels. In the pediatric study by **Sae-wong *et al.*** [18] liver fibrosis was not detected in their cohort, indicating that fibrosis could be a later event in the disease process.

Consistently, the various studies identified that there were certain factors and associations that were related to risk. The most common risk factors found were higher BMI and central obesity – measured as waist circumference or waist-to-height ratio (WHtR) [11,13,15,17-20]. Obesity (BMI >30 kg/m2) had an odds ratio of 3.1 and large waist circumference (>102 cm in men and >88 cm in women) had an odds ratio of 4.0 for hepatic steatosis, respectively, according to **Salah *et al.*** [11]. **Tomah and others** [13] reported that odds ratio for central obesity was 4.13 for predicting NAFLD independently. In the study by **Grzelka-Woźniak *et al.*** [16] insulin resistance markers such as estimated glucose disposal rate (eGDR), visceral adiposity index (VAI) and triglyceride to high-density lipoprotein cholesterol ratio (TG/HDL-C) were independently associated with the presence of NAFLD. However, **Parente *et al.*** [19] found that the specific visceral fat was associated with NAFL and a WHtR threshold of 0.5 had the highest sensitivity (86%) for detecting NAFL. These results were replicated by de **Vries *et al.*** [20] who found that waist circumference had an area under the curve (AUC) of 0.80, significantly larger than BMI ($p<0.001$). The clinical implications from these studies highlight the need for targeted screening in high-risk patients, especially those with obesity, central adiposity, greater diabetes duration and insulin resistance. Furthermore, a number of studies [11,13,17] have suggested that ALT is not sufficient to screen, while non-invasive markers like FIB-4 could be used to rule out fibrosis. In obese patients with T1D, intensive lifestyle intervention could positively impact the markers of NAFLD and insulin requirements, according to **Tomah S. *et al.*** [15].

Table 3 summarizes the results of the risk of bias assessment of the ten studies that met the inclusion criteria, which was performed using the Joanna Briggs Institute (JBI) Critical Appraisal Checklist for Analytical Cross-Sectional Studies. In general, there is a good methodological quality of the evidence base for the association between NAFLD and T1D, and most studies were well conducted and provided reliable data to support the findings of this review**.**

Table 1: Demographic and Study Characteristics of Included Studies

Study	Location	Study Design	Sample Size	Population	Age (years)	Female (%)	Diabetes Duration (years)	BMI (kg/m²)	HbA1c (%)	NAFLD Assessment Method	NAFLD Prevalence	Fibrosis Prevalence
Salah *et al.* 2023 [11]	USA	Cross-sectional	537	Adults with T1D	49 ± 16	55	29 ± 15.6	27.6 ± 5.5	7.1 ± 1.0	TE (CAP ≥274 dB/m)	28.5% (95% CI 24.6-32.6)	8.4% (LSM ≥7 kPa)
Dhaver *et al.* 2023 [12]	USA	Cross-sectional	200	Adults with T1D	46.9 ± 16.5	55.5	27.4 ± 15.7	26.9 ± 5.0	NR	TE (CAP ≥274 dB/m)	NR (CAP correlated with A1C)	NR (LSM correlated with BMI)
Tomah *et al.* 2022 [13]	USA	Cross-sectional	285	Adults with T1D	48 ± 16	55	28 ± 15	27 ± 4.9	7.1 ± 1.1	TE (CAP ≥274 dB/m)	29.3% (95% CI 24-35)	11% (LSM ≥7 kPa)
Zeiad *et al.* 2022 [14]	USA	Cross-sectional (pilot)	18	Children with T1D (age 7-20)	7-20 (range)	NR	≥5 years	NR	NR	TE (CAP)	~19%	NR
Tomah S. *et al.* 2023 [15]	USA	Retrospective matched-cohort	88 ILI / 88 SC	Adults with T1D and obesity	43 ± 12	65	22 ± 9	35.3 ± 4.9	8.2 ± 0.9	HSI (noninvasive surrogate)	100% at baseline (by selection)	NR
Grzelka-Woźniak *et al.* 2023 [16]	Poland	Cross-sectional	151	Adults with T1D	40 (IQR 33-47)	60.9	19 (IQR 13-21)	NR	7.5 (IQR 6.8-8.3)	TE (CAP ≥238 dB/m)	43% (n=65)	NR
Ubrangala *et al.* 2026 [17]	India	Cross-sectional	85	Adults with T1D	NR	NR	NR	NR	NR	TE (CAP ≥248 dB/m or LSM ≥7 kPa)	27% (n=23)	9.5% (n=8)
Sae-wong *et al.* 2021 [18]	Thailand	Cross-sectional	50	Children and young adults with T1D	16.9 (IQR 13.6-20)	56	6.5 (IQR 4-11)	NR	NR	MRI-PDFF + MRE	10% (n=5)	0%
Parente *et al.* 2021 [19]	Finland	Cross-sectional	121	Adults with T1D	38.5 (IQR 33-43.7)	52.1	21.2 (IQR 17.9-28.4)	NR	NR	MRI	11.6% (n=14)	NR
de Vries *et al.* 2022 [20]	Netherlands	Cross-sectional	150	Adults with T1D	47 ± 14	45	25 ± 14	25 (median)	NR	TE (CAP ≥248 dB/m)	34% (n=51)	NR

ILI, intensive lifestyle intervention; SC, standard care; TE, transient elastography; CAP, controlled attenuation parameter; LSM, liver stiffness measurement; HSI, Hepatic Steatosis Index; MRI-PDFF, magnetic resonance imaging-proton density fat fraction; MRE, magnetic resonance elastography; NR, not reported; IQR, interquartile range.

Table 2: Study Objectives, Key Findings, and Identified Risk Factors/Associations

Study	Primary Objective	Key Findings	Risk Factors / Associations Identified	Clinical Implications
Salah *et al.* 2023 [11]	Determine NAFLD and fibrosis prevalence in T1D	NAFLD in 28.5%; fibrosis in 8.4%; 4.1% had ≥F2 fibrosis; ALT/AST within normal range despite NAFLD	Higher BMI ($p<0.001$), longer diabetes duration ($p<0.01$), higher TDI ($p<0.001$), male gender (OR 2.1); obesity (OR 3.1) and large WC (OR 4.0) increased steatosis risk	Screening advisable regardless of liver enzymes, especially in those with higher BMI, longer duration, or higher insulin dose
Dhaver *et al.* 2023 [12]	Evaluate relationship between glycemic control, body weight, and NAFLD	CAP positively correlated with A1C ($p=0.009$) and BMI ($p<0.001$); NAFLD patients had higher A1C ($p=0.025$); CAP correlated with average glucose ($p=0.029$) and GMI ($p=0.008$)	CAP correlated with glucose variability (CV) only in lean individuals (BMI≤25) ($p=0.031$); LSM correlated with BMI ($p<0.001$) but not A1C or CGM parameters	Evaluate uncontrolled T1D patients for NAFLD, especially those with high BMI and high glucose variability
Tomah *et al.* 2022 [13]	Assess moderate-to-advanced fibrosis in T1D with NAFLD	NAFLD 29.3%; fibrosis 11%; among NAFLD, 13% had ≥F2 fibrosis; 72% with NAFLD and 59% with fibrosis had normal ALT	Large WC (OR 4.13) and BMI>30 (OR 2.4) increased steatosis risk; CGM parameters not associated with steatosis or fibrosis	ALT alone inadequate for screening; central obesity independently predicts NAFLD; screen T1D patients with high BMI or large waistline
Zeiad *et al.* 2022 [14]	Estimate NAFLD prevalence in children with T1D and association with arterial stiffness	NAFLD ~19%; positive correlation between CAP and PWV; higher PWV in steatosis group; negative correlation between IS and both arterial stiffness and steatosis	Decreased insulin sensitivity associated with both arterial stiffness ($R=-0.56$, $p=0.038$) and steatosis ($R=-0.56$, $p=0.063$)	NAFLD common in pediatric T1D and associated with higher arterial stiffness; larger studies needed
Tomah S. *et al.* 2023 [15]	Evaluate impact of ILI on NAFLD markers in obese T1D	ILI group lost 5.6 kg (5.8%) at 12 months; HSI decreased significantly (-2.7, $p=0.01$); high-likelihood NAFLD decreased from 100% to 88.3%	ILI reduced total daily insulin dose (-6.1 vs +1.34 units/day, $p<0.01$)	Short-term ILI improves NAFLD markers and reduces insulin requirements in obese T1D patients
Grzelka-Woźniak *et al.* 2023 [16]	Explore relationship between indirect IR markers and NAFLD in T1D	NAFLD in 43%; NAFLD patients less insulin-sensitive (lower eGDR, higher VAI, higher TG/HDL-C); higher HbA1c in NAFLD group (7.75% vs 7.3%, $p=0.02$)	eGDR (OR 0.86), VAI (OR 1.61), TG/HDL-C ratio (OR 1.88) independently associated with NAFLD after adjustment	NAFLD more likely in individuals with lower insulin sensitivity; indirect IR markers may aid risk stratification
Ubrangala *et al.* 2026 [17]	Evaluate TE and NITs for NAFLD screening in T1D	NAFLD 27% (steatosis 21.4%, fibrosis 9.5%); lean NAFLD in 11 patients (6 with fibrosis); FIB-4 higher in fibrosis (0.69 vs 0.40)	FIB-4 cut-off 0.5 predicted fibrosis (75% sensitivity, 31.6% specificity, NPV 86.2%); LSM correlated with FIB-4	NITs, particularly FIB-4, may aid screening; lower FIB-4 score can help exclude fibrosis
Sae-wong *et al.* 2021 [18]	Determine NAFLD prevalence and risk factors in pediatric/young adult T1D	NAFLD 10% (5/50); no liver fibrosis detected; 4 of 5 NAFLD patients overweight/obese; NAFLD patients had higher ALT and lower HDL-C	High BMI-SDS was sole independent risk factor (OR 5.79, 95% CI 1.04-32.18)	Routine screening not necessary for all; targeted screening recommended for overweight/obese T1D patients
Parente *et al.* 2021 [19]	Explore associations between body fat	NAFL 11.6%; visceral adipose tissue volume ($p=0.03$) and percentage ($p=0.02$)	WHtR cutoff 0.5: sensitivity 86%, specificity 55%; BMI cutoff 26.6:	Visceral adipose tissue associated with NAFL; WHtR may be useful screening tool

Study	Primary Objective	Key Findings	Risk Factors / Associations Identified	Clinical Implications
	distribution and NAFL in T1D	associated with NAFL; gynoid, appendicular, and total fat not associated	sensitivity 79%, specificity 57%; WHtR AUC larger than BMI (p=0.04)	
de Vries *et al.* 2022 [20]	Confirm relationship between body fat distribution and NAFL in T1D	NAFL 34%; central obesity (WHtR≥0.5) in 88.2% of NAFL group vs 44.4% of non-NAFL group	WHtR AUC 0.76; BMI AUC 0.71; optimal WHtR cutoff 0.51 (sensitivity 86%, specificity 62%); optimal BMI cutoff 25.2 (sensitivity 72%, specificity 67%); WC AUC 0.80 (larger than BMI, p<0.001)	Clinical markers of visceral fat (WHtR, WC) strongly associated with NAFL and may guide need for hepatic imaging

TDI, total daily insulin dose; WC, waist circumference; GMI, Glucose Management Indicator; CV, coefficient of variation; CGM, continuous glucose monitoring; PWV, pulse wave velocity; IS, insulin sensitivity; ILI, intensive lifestyle intervention; HSI, Hepatic Steatosis Index; IR, insulin resistance; eGDR, estimated glucose disposal rate; VAI, visceral adiposity index; TG/HDL-C, triglyceride to high-density lipoprotein cholesterol ratio; NITs, noninvasive tests; NPV, negative predictive value; BMI-SDS, body mass index standard deviation score; NAFL, nonalcoholic fatty liver; WHtR, waist-to-height ratio; AUC, area under the curve.

Table 3: Risk of Bias Assessment of Included Studies

Study	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Overall Risk
Salah *et al.* 2023 [11]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Low
Dhaver *et al.* 2023 [12]	Yes	Yes	Yes	Yes	Yes	Yes	Unclear	Yes	Low
Tomah *et al.* 2022 [13]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Low
Zeiad *et al.* 2022 [14]	Yes	Yes	Yes	Yes	Yes	No	Unclear	Yes	Moderate
Tomah S. *et al.* 2023 [15]	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Moderate
Grzelka-Woźniak *et al.* 2023 [16]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Low
Ubrangala *et al.* 2026 [17]	Yes	Yes	Yes	Yes	Yes	Unclear	Yes	Yes	Low
Sae-wong *et al.* 2021 [18]	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Moderate
Parente *et al.* 2021 [19]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Low
de Vries *et al.* 2022 [20]	Yes	Yes	Yes	Yes	Yes	Unclear	Unclear	Yes	Moderate

DISCUSSION

The prevalence of NAFLD in adults with T1D varied from 11.6% to 43% among the studies included, with the largest study (537 patients) reporting a prevalence of 28.5% [11] and the Polish study (151 patients) reporting the highest prevalence at 43% [16]. The results are similar to the overall epidemiological data. A key systematic review and meta-analysis by **de Vries *et al.*** [21] that included 20 studies found a pooled prevalence of NAFLD in adults with T1D of 19.3% overall, and 22% in an adequately matched sample of the general population, suggesting a prevalence of NAFLD at least as high and, likely, higher in adults with T1D than in matched controls.

More recently, a meta-analysis and systematic review by **Souza *et al.*** [22] identified a total of 23 studies that included 13,006 children with T1D, which yielded a prevalence of metabolic dysfunction associated steatotic liver disease (MASLD) of 22.24% (95% CI, 15.62–30.66). Together, these data clearly demonstrate that NAFLD is a frequent and clinically relevant comorbidity in T1D, and not just a complication of type 2 diabetes as previously believed.

Risk factor profile of the included studies is consistent and offers key insights into the pathogenesis of NAFLD in T1D. In all studies higher body mass index (BMI) and central obesity were the most consistently recognized risk factors [11,13,15,17-20].

Salah *et al.* [11] found that obesity (BMI>30 kg/m2) had an odds ratio of 3.1, and large waist circumference (>102 cm in males, >88 cm in females) an odds ratio of 4.0, for hepatic steatosis.

Central obesity was found to be an independent predictor of NAFLD with an odds ratio of 4.13 [13] and **de Vries *et al.*** [20] showed that the area under curve for waist circumference was larger (0.80) than that of BMI for predicting NAFL. These results are similar to those of Parente ***et al.*** [19] who showed, by means of DEXA, that the volume and percentage of VAT instead of gynoid, appendicular or total adipose tissue was specifically linked to NAFL in T1D.

The association of metabolic syndrome with NAFLD is further supported by **Mertens *et al.*** [23] who used cross-sectional data from 530 adults with T1D to find that metabolic syndrome was significantly associated with NAFLD (OR: 2.35, 95% CI 1.08–5.12, $p = 0.031$). **Lundholm *et al.*** [24] also found that BMI ≥ 25 kg/m² was associated with the steatosis scores, ($p < 0.001$) and that older age (≥ 40 years), hypertension and dyslipidaemia were associated with the steatosis markers, in a group of 447 patients with T1D. The weight of evidence to date is that central obesity is a major risk factor in the pathogenesis of NAFLD in T1D, in a context where the disease was traditionally regarded as a model of pure insulin deficiency.

A few studies included here have identified a relationship between insulin resistance and NAFLD in T1D, which is a key mechanistic pathway. After adjusting for sex, diabetes duration and HbA1c, Grzelka-Woźniak and colleagues [16] found that indirect insulin resistance markers, such as estimated glucose disposal rate (eGDR), visceral adiposity index (VAI) and triglyceride to high-density lipoprotein cholesterol ratio (TG/HDL-C), were independently associated with NAFLD. The patients with NAFLD were significantly less insulin-sensitive (eGDR 8.93 vs 9.94 mg/kg/min, $p = 0.001$) and had higher HbA1c values (7.75% vs 7.3%, $p = 0.02$).

A multi-center cross-sectional study by **Vergani *et al.*** [6] that included 198 adults with T1D who underwent vibration-controlled transient elastography found that insulin resistance (eGDR) was independently correlated with MASLD. Moreover, **Bulum *et al.*** [25] indicated that the increase in ALT, AST, and alkaline phosphatase (ALP) are other indicators of insulin resistance in type 1 diabetes and that people with high levels of these liver enzymes may be affected by liver disease as well as by a multisystemic condition because of the changes in insulin sensitivity.

In fact, the link between insulin resistance and NAFLD in T1D is strengthened by the recent findings of **Mertens *et al.*** [26] who could demonstrate an independent association of elevated liver fat with insulin resistance in T1D, further underscoring the notion that insulin resistance is also an important contributor to hepatic steatosis in T1D. This evidence refutes the long held paradigm that insulin deficiency is the hallmark of T1D and has led to the recent realization that insulin resistance is a major pathophysiological component in a significant proportion of individuals with T1D.

In addition to the obesity and insulin resistance, glycemic control and glucose variability were identified as additional factors associated with NAFLD in T1D. The study by **Dhaver *et al.*** [12] offered new insights by showing that CAP scores were positively correlated with HbA1c ($p = 0.009$), average blood glucose ($p = 0.029$) and Glucose Management Indicator ($p = 0.008$). Interestingly, they discovered that the coefficient of variance, a measure of glucose variability, is associated with hepatic steatosis only in lean patients (BMI ≤ 25 kg/m2) ($p = 0.031$), implying that glycemic swings could be involved in the process of hepatic fat accumulation even without the presence of overweight or obesity. This is an important discovery because it reveals a novel pathogenic pathway in lean patients with T1D that may be overlooked as being low-risk for NAFLD.

This correlation between glycemic control and NAFLD was also seen by **Grzelka-Woźniak *et al.*** [16] who found significantly higher HbA1c in those with NAFLD than in those without (7.75% vs 7.3%, p = 0.02). But **Tomah *et al.*** [13] reported that parameters of continuous glucose monitoring were not correlated with steatosis or fibrosis, implying that the correlation between glycemia and NAFLD could be complex and other factors also may have an effect, such as insulin resistance and adiposity.

This was investigated further in the multicenter study by **Vergani *et al.*** [6] which studied the relationship between continuous glucose monitoring parameters and MASLD in adults with T1D, which also highlighted the link between glycemic parameters and hepatic steatosis.

Liver fibrosis is the main driver of liver related morbidity and mortality and therefore an important clinical problem in T1D. Of the studies included, **Salah *et al.*** [11] found that the percentage of their cohort with liver stiffness measurement (LSM) ≥7 kPa (fibrosis) was 8.4%, and those with ≥F2 fibrosis (nonalcoholic steatohepatitis [NASH]) was 4.1%. In another study, **Tomah *et al.*** [13] established a prevalence of fibrosis of 11% and 13% of those with NAFLD had ≥F2 fibrosis. Fibrosis was reported in 9.5 % of their cohort; there were 11 lean NAFLD, of which 6 had fibrosis [17]. In children, **Sae-wong *et al.*** [18] did not find fibrosis with magnetic resonance elastography, indicating fibrosis may occur later during the disease course. The results of this study agree with the meta-analysis by **Souza *et al.*** [22] which showed that significant fibrosis (F2 or greater) and advanced fibrosis (F3 or more) were observed in 13.25% and 5.12% of T1D patients with MASLD, respectively.

Ciardullo *et al.* [27] performed a systematic review and meta-analysis of elevated liver stiffness in diabetes, and estimated that around 5% of patients with T1D have elevated liver stiffness, which was associated with significant or advanced fibrosis. Importantly, several studies [11,13,17] pointed out that liver enzymes are often not elevated in the presence of NAFLD and fibrosis, making liver enzymes alone a poor screening tool. Among NAFLD patients, mean ALT was found to be only 21±11 IU/L by **Salah *et al.*** [11] and 72% of NAFLD patients and 59% of NAFLD patients with fibrosis had ALT levels below the conventional limits by **Tomah *et al.*** [13]. This observation has wide clinical ramifications because if liver enzymes were relied upon, a large number of patients with moderate to severe liver disease would be excluded.

Several of the studies included assessed the diagnostic value of the non-invasive tests for screening for NAFLD in T1D. **Ubrangala *et al.*** [17] showed that the FIB-4 score was significantly elevated in fibrosis (0.69) compared to no fibrosis (0.40), and an FIB-4 cut-off of 0.5 had a sensitivity of 75% and a negative predictive value of 86.2% for fibrosis. The AST/ALT ratio and chemerin was suboptimal for the detection of steatosis, and Enhanced Liver Fibrosis scores were not significantly increased in fibrosis.

Mertens *et al.* [23] performed a thorough prospective analysis of non-invasive tests in 530 adults with T1D and confirmed the results with magnetic resonance spectroscopy in a subpopulation of 132 persons. They discovered that both ultrasound and controlled attenuation parameter had good sensitivity and specificity, with AUROC values of 0.98 (95% CI 0.95–1.00) and 0.85 (95% CI 0.77–0.93) respectively. The fatty liver index was similarly able to achieve an AUROC of 0.83 (95% CI 0.74–0.91); however, the conventional cut-off used to rule in (≥60) was found to have a low level of sensitivity and specificity. Based on these findings, ultrasound screening could be suggested in individuals with T1D and metabolic syndrome, while CAP and FIB-4 could be used as additional tools. Liver scores for steatosis were high among adults with T1D (61% HSI ≥36 and 52% FSI ≥23) and few adult patients had any abdominal imaging (21%), hepatology referral (2%), or liver biopsy (1%), demonstrating that NAFLD is significantly under-diagnosed and under-investigated in clinical settings.

Tomah *et al.* [15] studied the effect of lifestyle intervention on NAFLD in T1D, showing that a 12-month intensive lifestyle intervention led to an improvement in Hepatic Steatosis Index and a reduction in total daily insulin dose in T1D patients with obesity at 1 year. The intervention group was treated with a significant weight loss of 5.6 kg (5.8%, $p < 0.05$) and a significant decrease in HSI from baseline (-2.7 ± 1.1, $p = 0.01$) with no change in the standard care group. The percentage of patients with high likelihood of NAFLD diagnosis decreased from 100% to 88.3% in the intervention group. This study is important because lifestyle changes, which are the mainstay of the therapy for NAFLD in non-T1D individuals, is effective in patients with T1D as well. But there are no data specifically regarding pharmacotherapeutic interventions in T1D and NAFLD. Glucagon-like peptide-1 receptor agonists and sodium-glucose cotransporter-2 inhibitors have been seen to be beneficial in T2D-associated NAFLD [28,29] but their efficacy and safety in T1D with NAFLD have yet to be defined. These agents have the potential to play a role in T1D and this is an area that is being studied.

Furthermore, the association between NAFLD and cardiovascular disease in T1D, as demonstrated by the positive correlation between CAP and pulse wave velocity in T1D children by **Zeiad *et al.*** [14] should be explored. There is an independent association between NAFLD and cardiovascular disease in adult patients with type 1 diabetes [30] and the mechanism of coupling

hepatic steatosis, systemic inflammation and atherosclerosis warrants further investigation.

LIMITATIONS

This review's findings about NAFLD are limited by a number of factors. The range of prevalence rates in adults, from 11.6% to 43%, was large due to the heterogeneity of the studies, such as using different assessment modalities, different cutoff values for CAP and LSM. Majority of the studies were cross-sectionals, which did not allow for causality to be inferred. Another limitation is that reliance on non-invasive tests, rather than liver biopsy as the gold standard, has been a drawback. The sample size in pediatric studies was limited which led to low generalizability. There was also a lack of ethnic and geographic variety, primarily North American and European populations. One limitation for one lifestyle intervention study is its retrospective design, which introduces selection bias and the use of HSI rather than direct imaging is another limitation. A lack of long-term follow-up data restricts understanding of NAFLD's natural history. Also, one of the cited comments was not a complete original research article, and did not give sufficient detail of methodology. Lastly, publication bias in prevalence and associations reported could be due to publication of only studies that had positive findings.

CONCLUSION

This review describes the high prevalence of Non-Alcoholic Fatty Liver Disease (NAFLD), a condition that affects approximately 20-30% of adults with Type 1 Diabetes (T1D) and many of them have a significant extent of fibrosis. There are important risk factors such as obesity, central adiposity, insulin resistance, and glycemic control, which highlight the importance of screening. Using ALT alone to screen for NAFLD is not enough because liver enzymes can be normal in patients with severe liver disease. Non-invasive testing such as the ultrasound and FIB-4 have great potential for risk stratification, however, they need to be validated. There is the possibility that markers of NAFLD will improve with intensive lifestyle interventions, so all at-risk T1D patients should be screened regularly, and not only when liver enzymes are abnormal. Further research is needed to improve the understanding of the natural history, pharmacologic therapies, and outcome of NAFLD to create evidence-based guidelines.

DECLARATIONS

Ethics Approval and Consent to Participate

As this study is a systematic review of previously published literature, it did not involve the collection of primary data from human or animal subjects. Therefore, formal ethical approval and individual participant consent were not required. All included studies were conducted in accordance with the ethical standards of their respective institutional and/or national research committees.

Consent for Publication

Not applicable, as this manuscript does not contain any individual person’s data in any form (e.g., individual details, images, or videos).

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Competing Interests

The authors declare that they have no competing interests.

Authors' Contributions

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