Review Article

Association Between Polycystic Ovary Syndrome and Nonalcoholic Fatty Liver Disease: A Systematic Review

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ABSTRACT

Polycystic ovary syndrome (PCOS) is a common endocrine and metabolic disorder affecting women of reproductive age, increasingly recognized for its systemic metabolic implications. Nonalcoholic fatty liver disease (NAFLD), recently redefined as metabolic dysfunction-associated steatotic liver disease (MASLD), shares overlapping pathophysiological mechanisms with PCOS, including insulin resistance, hyperandrogenism, and chronic inflammation. This systematic review aimed to comprehensively evaluate the association between PCOS and NAFLD/MASLD, synthesize prevalence estimates and risk factors, and assess liver-related outcomes in this high-risk population. A comprehensive literature search was performed across PubMed/MEDLINE, Scopus, Web of Science, Cochrane Library, and Google Scholar for studies published between 2021 and 2026. Two reviewers independently performed screening, data extraction, and quality assessment using the Newcastle-Ottawa Scale and Joanna Briggs Institute checklist. A narrative synthesis was conducted due to substantial heterogeneity. Sixteen studies (15 independent datasets) were included. NAFLD prevalence in women with PCOS ranged from 19.1% to 77%, exceeding 50% in only some clinical cohorts. Hyperandrogenism and insulin resistance were identified as independent risk factors in several, mostly small, studies. Among women with NAFLD, PCOS was associated with 22% higher odds of NASH. While PCOS patients developed MASLD at younger ages, fibrosis severity appeared more favorable after confounder adjustment. The evidence suggests a higher NAFLD burden in women with PCOS, particularly with hyperandrogenism, insulin resistance, and obesity, but certainty is low (heterogeneous diagnostics, few control groups, cross-sectional designs). Targeted evaluation of high-risk women merits consideration.

Keyword: Polycystic ovary syndrome; Nonalcoholic fatty liver disease; MASLD; Hyperandrogenism; Insulin resistance.

Introduction

According to several diagnostic criteria used globally, PCOS is the most common endocrine and metabolic disorder among women of reproductive age, affecting 9.2% to 12.1% of the population [1, 2]. The syndrome is clinically and morphologically heterogeneous and involves a combination of oligoovulation, hyperandrogenism, and polycystic ovarian morphology, the Rotterdam criteria 2003 being the most widely used diagnostic tool worldwide [2].

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In addition to its reproductive symptoms, PCOS has become a well-established systemic metabolic disorder which has significant long-term health consequences such as insulin resistance, obesity, dyslipidemia, as well as a higher risk of cardiovascular disease and type 2 diabetes [3]. The metabolic disturbances are not only epiphenomena but are key to the syndrome's pathophysiology, resulting in a vicious cycle of endocrine and metabolic dysfunction that affects more than just the reproductive axis. Nonalcoholic fatty liver disease (NAFLD), the most prevalent chronic liver disease worldwide, is now known as metabolic dysfunction-associated steatotic liver disease (MASLD) [4]. The illness encompasses a range of liver conditions, such as simple steatosis, nonalcoholic steatohepatitis (NASH), fibrosis/cirrhosis, and hepatocellular cancer [4]. The prevalence of NAFLD/MASLD has grown with obesity, but it is now known that this condition can manifest in non-obese people, especially when a metabolic abnormality is present [4]. The area of PCOS and NAFLD is a critical and largely overlooked clinical area. Both disorders have common pathophysiological processes, including insulin resistance, hyperandrogenism, visceral obesity, and chronic low-grade inflammation [3, 5]. Both PCOS and NAFLD share insulin resistance, which promotes hepatic de novo lipogenesis and reduces fatty acid oxidation; hyperinsulinemia also increases ovarian production of androgen, which could contribute to the severity of disease in both organs in a metabolic-endocrine vicious circle [3, 5]. Hyperandrogenism has emerged as an independent risk factor for hepatic steatosis, and mechanistic work has shown that androgen excess directly affects hepatocyte metabolism (and indirectly affects adipose tissue function), resulting in increased accumulation of hepatic lipid [5, 6]. Although there is evident biological plausibility, the linkage between PCOS and NAFLD has not been consistently identified in clinical practice, and screening guidelines are still conflicting. The Endocrine Society clinical practice guidelines suggest that NAFLD is a possible complication in women with PCOS and do not support routine screening; however, studies on the yield of screening and the benefit of treatment are low and inconclusive [7]. The burden of NAFLD/MASLD in the context of PCOS has not been clearly established and has been highly variable by diagnostic criteria, imaging techniques and populations studied [8]. This systematic review aims to synthesize the prevalence and risk factors for NAFLD/MASLD in these high-risk individuals, examine liver-related outcomes in PCOS patients, and summarize the available information addressing the association between PCOS and NAFLD/MASLD.

Methods

The systematic review used the standard systematic review methodology and followed the guidance of the PRISMA 2020 reporting checklist. The review's objective was to find original research that examined the connection between nonalcoholic fatty liver disease (NAFLD) and polycystic ovarian syndrome (PCOS), as well as the consequences of this link, including MAFLD, MASLD, NASH, liver steatosis, liver fibrosis, liver stiffness, and controlled attenuation parameter. Search Strategy: A thorough search was carried out in Google Scholar, PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library. The search covered studies published between September 2021 and September 2026. Combinations of "polycystic ovary syndrome" OR "PCOS" with "nonalcoholic fatty liver disease" OR "NAFLD" OR "MAFLD" OR "MASLD" OR "NASH" OR "hepatic steatosis" OR "liver fibrosis" OR "liver stiffness" OR "controlled attenuation parameter" were part of the search strategy. Boolean operators AND/OR were applied, and only English-language studies were included. Hand searching was also done to find any additional relevant studies in reference lists of included articles to include all relevant articles in the searched databases. Searching process was undertaken in October 2026. Eligibility Criteria: Studies were chosen based on whether they were original observational studies (case-control, cross-sectional, or cohort designs) or secondary analyses of a randomized controlled trial; included women or adolescents with PCOS; and reported at least one liver-related outcome. Liver-related outcomes included a confirmed or validated surrogate diagnosis of NAFLD, MAFLD, MASLD, or NASH; hepatic steatosis; liver fibrosis; liver stiffness; or controlled attenuation parameter (CAP). Isolated elevation of liver enzymes (ALT/AST) was not considered a sufficient liver-related outcome for inclusion. Excluded were literature reviews, editorials, case reports, conference abstracts (without complete data), and studies lacking a liver-related outcome. Studies and records that were not published in English were also excluded. When reported, ALT/AST were extracted as secondary biochemical variables but were not used to define NAFLD/MASLD or to determine eligibility. NAFLD, MAFLD, and MASLD were extracted as defined by each study; the combined term NAFLD/MASLD is

used when studies did not distinguish them. Studies restricted to patients with NAFLD/MASLD (Maldonado 2022; Devas 2026) were used for severity-related outcomes only, not for estimating prevalence or the strength of association with PCOS. Patel-Sanchez 2023, which allowed ALT-based criteria in addition to imaging or biopsy, was retained but considered at higher risk of outcome misclassification. Feng 2025 and Shi 2025 are secondary analyses of the same PCOSAct trial and were treated as one dataset when interpreting prevalence. Study Selection: The online systematic review screening software Rayyan was used to manage all retrieved records [9]. Two reviewers independently assessed the abstracts and titles based on the eligibility requirements. After that, the full text of any potentially qualifying records was retrieved and independently assessed. When disagreements arose during the screening of titles, abstracts, or entire texts, a third party was consulted. Data Extraction: A subset of the included studies was used to develop and pilot-test the data extraction tool. Two reviewers independently retrieved data on study characteristics (author, year, country, setting), study design, sample size, population type, mean age, BMI, PCOS diagnosis criteria, presence of a control group, and liver/NAFLD assessment method. Other data collected included additional metabolic and hormonal variables, objectives, main findings, effect estimates, confidence intervals, and p-values. Discrepancies were resolved by consensus or by consulting a third reviewer. Characteristics not reported in the available report were recorded as not mentioned (NM); for several studies, extraction relied on abstract-level information. Quality Assessment: Depending on the study design, validated tools were employed to assess the methodological quality of the included studies. Cohort and case-control studies were evaluated using the Newcastle-Ottawa Scale (NOS), which assigns ratings in three categories: selection, comparability, and outcome/exposure [10]. The cross-sectional studies were evaluated using the Joanna Briggs Institute's (JBI) Critical Appraisal Checklist for Analytical Cross-Sectional Studies [11]. Two reviewers separately assessed the quality, and disagreements were resolved through discussion. Studies were categorized as having a low, moderate, or high overall risk of bias based on the NOS star rating and the proportion of JBI items judged adequate; only the overall rating is reported (Table 2). Data Synthesis: Because of substantial heterogeneity in study design, NAFLD definition, diagnostic modality, and population, no meta-analysis was performed; instead, a narrative synthesis was conducted. Data were summarized in two tables; one table showed demographic and sample characteristics (Table 1) and the second table showed objectives, variables, outcomes, and main findings (Table 2). Results were presented thematically, according to the prevalence of findings, metabolic and hormonal risk factors, hyperandrogenism, hepatic fibrosis severity, type 2 diabetes, obesity, and insulin resistance. The synthesis was organized around consistent directions of association and clinically meaningful effect estimates. Prevalence estimates were stratified descriptively by diagnostic method, and controlled comparisons were summarized separately from single-group prevalence estimates. Because no meta-analysis was performed, publication bias could not be assessed, and the certainty of evidence was not formally graded.

Results

Of 286 records identified, 144 duplicates were removed, and 142 records were screened; 76 were excluded and 66 reports were sought for retrieval, of which 32 could not be retrieved. Of 34 full-text reports assessed, 18 were excluded (wrong outcome, n = 9; wrong population, n = 7; conference abstract, n = 2), leaving 16 studies from 15 independent datasets (Figure 1). The 16 included studies are presented (Table 1) and had the following demographic and methodological features. Many studies are of the cross-sectional or case-control type with some large population-based studies (a national survey [12], a hospital discharge database [13], and a provincial health-database case-control study [14]). The sample sizes are highly variable ranging from small, single center studies with fewer than 100 patients [15, 16, 17, 18, 19] to large national database studies with tens to hundreds of thousands of participants [13, 14]. The majority of studies included adult women, but two included adolescents specifically [20, 21] and these studies contributed to the data available for this young group. The most frequently used criteria for diagnosis of PCOS were the Rotterdam criteria [15, 16, 20, 22] while a few studies did not mention their criteria in the abstract [18, 19, 21, 23, 24]. The tools used to measure liver health are strikingly varied, such as serum-based markers like the Liver Fat Score [22], imaging tools like ultrasonography [16, 17], transient elastography [19, 20, 23, 25] or even administrative diagnostic codes from national health databases [13, 14]. This will be important in interpreting synthesized findings, because of the heterogeneity in diagnostic tools and study populations. The objectives, key variables, and

main findings of the included studies are summarized (Table 2). Most studies aimed to investigate the connection between PCOS and liver-related outcomes, especially NAFLD, whereas Feng, Shi, and Vine evaluated NAFLD among several metabolic or health outcomes [14, 26, 27]. The reported prevalence of NAFLD in women with PCOS ranged from 19.1% [21] to 77% [16]; it exceeded 50% in some clinical samples [17, 19, 23] but was lower (32.9–46.6%) in population-based or multicentre samples [12, 22, 24]. Metabolic problems, including insulin resistance (IR) as measured by the HOMA-IR and hyperandrogenemia as evidenced by high testosterone and free androgen index (FAI), have been commonly associated with NAFLD [15, 19, 22, 23, 25]. Total testosterone has been demonstrated to be an independent risk factor for NAFLD in obese PCOS patients [19]. The data also reveal the potential value of the different predictive indices, such as the TyG index, which showed a significant association with the prevalence of NAFLD with an AUC of 0.705 for its prediction [26]. Furthermore, a large study of women with NAFLD found that PCOS was independently associated with 22% higher odds of nonalcoholic steatohepatitis (NASH), a more severe form of NAFLD [13]. In contrast, after adjustment for age and BMI, a single-centre retrospective study of 885 patients with MASLD indicated that although patients with PCOS were diagnosed with MASLD at a younger age, they had a more favorable fibrosis profile [25]. Prevalence also varied with the diagnostic method: 53.5% [17] to 77% [16] by ultrasonography (19.1% when ALT-based criteria were combined with imaging or biopsy [21]), 72.4% [23] to 75.6% [19] by transient elastography or CAP, 33.6% by the Liver Fat Score [22], and 32.9% [12] to 46.6% [24] when criteria were not reported. Among studies with non-PCOS comparison groups, NAFLD or hepatic steatosis was more frequent in PCOS in four studies (33.6% vs 4.2% [22]; 75.61% vs 45.00% [19]; higher steatosis prevalence [18]; 2–3-fold higher prevalence [14]), liver stiffness was higher in one [15], and the odds of NASH among women with NAFLD were 22% higher (adjusted OR 1.22, 95% CI 1.05–1.42) [13]. In contrast, no overall difference in steatosis was found in adolescents [20, 21] or in PCOS compared with pre-PCOS women (32.9% vs 33.1%) [12]. The methodological quality and overall risk-of-bias rating of each included study are shown in the last column of (Table 2). To account for the range of study designs, two validated tools were employed: the Joanna Briggs Institute (JBI) Critical Appraisal Checklist for analytical cross-sectional studies and the Newcastle-Ottawa Scale (NOS) for cohort and case-control studies. Three criteria are used to evaluate each study: selection, comparability, and outcome/exposure. The overall score is limited to nine stars, with studies that receive more than seven stars being considered to be low risk of bias [10]. Eight criteria are assessed by the JBI checklist, including control of confounding factors, validity of exposure and outcome measurement, and clarity of inclusion criteria [11]. The methodological quality of the included studies varied. Although several studies were hindered by inadequate control for confounding factors, cohort and case-control studies were generally of moderate to high quality, with good selection of non-exposed groups and exposure ascertainment. A common limitation of cross-sectional designs is the difficulty of controlling for confounders, although they are valuable for estimating prevalence. Several small case-control and cross-sectional studies were rated at low risk of bias because they used validated measurements, but their small samples and, for some, the lack of a non-PCOS control group limit precision and inference; studies relying on administrative databases were rated moderate because of the risk of outcome misclassification.

Discussion

The included studies suggest a clinically relevant association between PCOS and NAFLD, with a prevalence of 32.9% to 77% reported in several studies [12, 16, 19, 23]. These results resemble those of the largest meta-analysis to date, which reported a pooled NAFLD prevalence of 43% (95% CI 35–52%) in women with PCOS [8]. A systematic review and meta-analysis of 36 studies showed that PCOS was associated with an increased risk of NAFLD (OR = 2.93, 95% CI 2.38–3.62, $I^2 = 83.7\%$, $p < 0.001$), even after sensitivity analysis [28]. A 2025 narrative review reported a 2–2.5-fold higher incidence of MASLD among women with PCOS compared with age- and BMI-matched women without PCOS [29]. Our findings are directionally consistent with recent large-scale syntheses; however, heterogeneity in design, diagnostic methods, and populations (the country was not reported in several studies) limits generalizability. An important finding of our analysis is the frequent identification of hyperandrogenism as an independent risk factor for NAFLD in PCOS. Higher levels of androgen (testosterone, free androgen index (FAI), and dehydroepiandrosterone sulfate (DHEAS)) were linked to increased liver steatosis and liver damage,

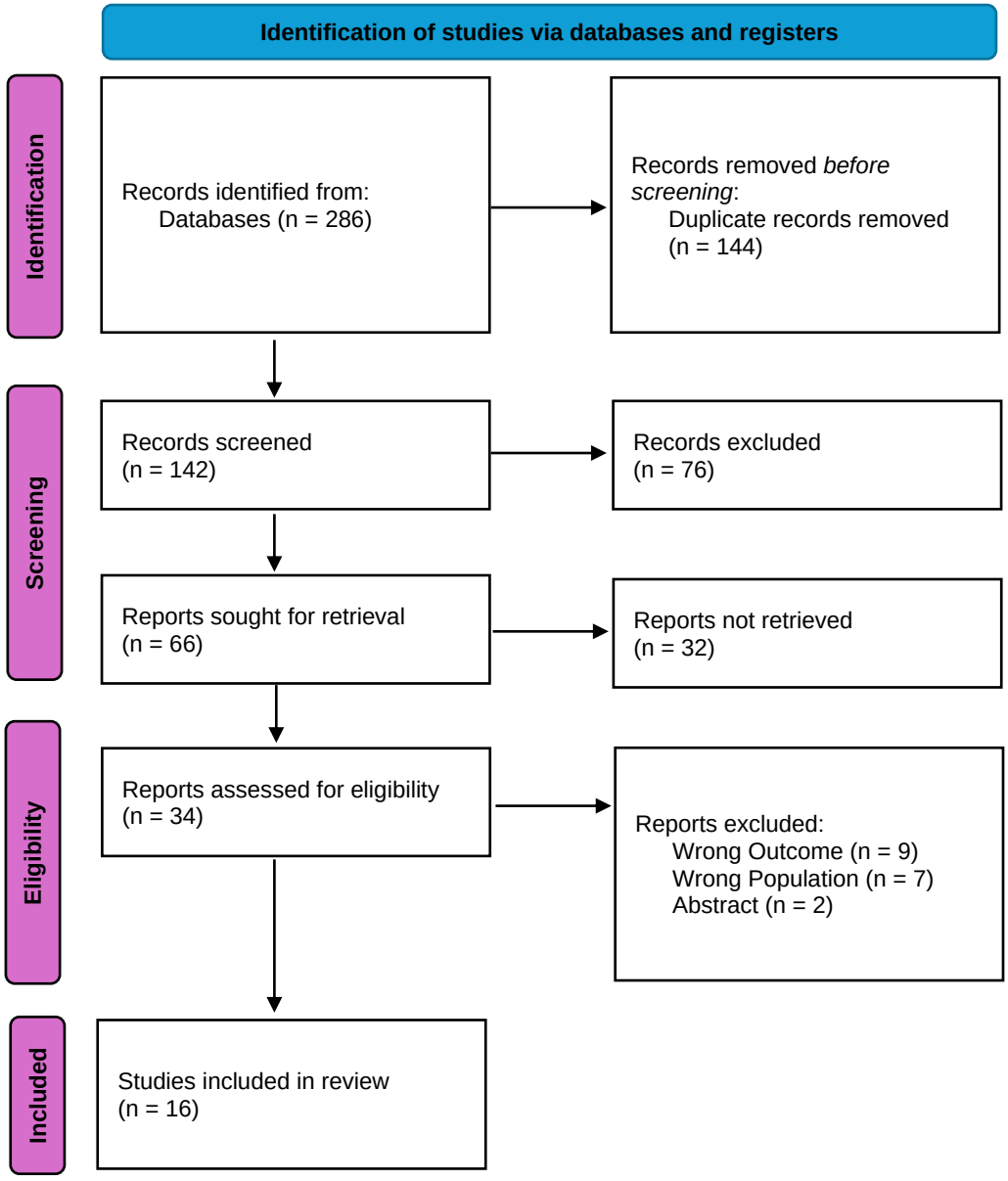

Figure 1: PRISMA flow diagram of study selection.

Table 1: Demographic and sample characteristics of included studies.

Study [Ref]	Country/ Setting	Study design	Sample size	Population/sample type	Mean age	BMI	PCOS diagnostic criteria	Control group	NAFLD/ liver assessment	Notes
Gulumsek 2021 [15]	NR	Case-control comparative study using point shear wave elastography	66 (38 PCOS, 28 controls)	Women with PCOS; healthy age- and sex-matched controls	NR (age-matched)	Higher in PCOS vs controls ($P<0.05$); mean NR	Rotterdam	28 healthy age- and sex-matched controls	Liver stiffness by point shear wave elastography; no explicit NAFLD diagnosis	Small sample; BMI differed
Kara 2023 [20]	NR	Cross-sectional case-control study	124 adolescent girls (61 PCOS, 63 controls)	Adolescents aged 12–18 years with PCOS; controls with regular menses >2 years	Range 12–18 years; mean NR	BMI z scores similar; mean NR	Rotterdam	63 controls	USG for hepatic steatosis; VCTE for LSM and CAP	Adolescent population; hyperandrogenemia subgroup
Feng 2025 [26]	China	Secondary analysis of PCOS Act randomized clinical trial	956	Chinese women with PCOS	NR	NR	NR in abstract (PCOS Act trial)	None; within-cohort quartiles	NAFLD by criteria not specified in abstract	Overlaps PCOSAct with Shi 2025 [27]
Devas 2026 [25]	Single center; country NR	Single-center retrospective study; 3 cohorts; propensity score matching	885 (286 PCOS+MASLD, 521 MASLD without PCOS, 78 males with MASLD)	Adults with MASLD	Median 32.5 years PCOS; 54.0 years non-PCOS females; 36.0 years males	Median 37.3 kg/m^2 in PCOS; others NR	NR	Females with MASLD without PCOS; males with MASLD	MASLD; fibrosis by NAFLD Fibrosis Score, FIB-4, TE, MRE, and biopsy when available	Primary outcome was fibrosis stage

Ganie 2024 [12]	India, 5 zones	Cross-sectional study	9824 recruited; 8993 enrolled; PCOS n=1224; pre-PCOS n=492	Women aged 18–40 years	Overall 29.5 ± 6.2 years; screen-positive 28.1 ± 6.4; screen-negative 29.7 ± 6.1	Obesity in 529/1224 (43.2%) PCOS; mean NR	NIH 1990, Rotterdam 2003, AE-PCOS	Screen-negative/ healthy women	NAFLD prevalence 403 (32.9%) in PCOS; pre-PCOS 163 (33.1%); criteria NR	National prevalence and comorbidity study
Guimarães 2023 [16]	Brazil (HEI adapted for Brazilian population); setting NR	Cross-sectional study	87	Patients with PCOS	35.2 ± 5.7 years	NAFLD group 34.9 ± 4.5 vs no NAFLD 30.4 ± 4.9 kg/m²; p=0.001	Rotterdam	PCOS without NAFLD	Abdominal ultrasound for liver steatosis	No non-PCOS control
Eslami 2022 [17]	Iran, Tehran, Arash Women's Hospital	Cross-sectional study	71	Women with PCOS referred to hospital	NR	NAFLD higher than non-NAFLD; cutoff 25.5 kg/m²; mean NR	NR	PCOS without NAFLD	Liver ultrasonography by radiologist	No non-PCOS control
Patel-Sanchez 2023 [21]	NR	Retrospective study	323 female adolescents (PCOS n=161, non-PCOS n=162); overweight n=52, obese n=271	Overweight/obese female adolescents aged 12–18 years, clinically evaluated 2012–2020	Range 12–18 years; mean NR	Overweight/obese	NR	Age-matched non-PCOS controls	NAFLD defined as ALT ≥44 U/L ×2 and/or ≥80 U/L ×1, hepatic steatosis on imaging, or NAFLD on biopsy, in absence of other liver disease	T2DM was a key effect modifier

Özgürlük 2025 [18]	NR	Case-control study	90 (44 PCOS, 46 controls)	PCOS patients and age- and BMI-matched controls	NR (matched)	Matched; mean NR	NR	46 age- and BMI-matched controls	Liver steatosis grading by ultrasound-guided attenuation parameter	Exact prevalence not reported
Hong 2023 [22]	China, 6 centers	Multi-center cross-sectional study	217 PCOS + 72 healthy controls	PCOS by Rotterdam criteria; age-matched healthy volunteers	NR (age-matched)	BMI ≥24 kg/m² subgroup analyzed; mean NR	Rotterdam	72 healthy controls	Liver Fat Score > −0.640 for NAFLD; MAFLD by 2020 definition	NAFLD and MAFLD outcomes
Maldonado 2022 [13]	USA, National Inpatient Sample	Retrospective analysis of weighted discharge data	189,440 women with NAFLD; 9,415 with PCOS; 1,390 NASH among PCOS	Reproductive-age women with NAFLD	Median 33 years PCOS vs 40 years non-PCOS	Obesity 83.0% vs 58.0%	NR (administrative codes)	Reproductive-age women with NAFLD without PCOS	NAFLD/NASH discharge codes	Administrative database study
Recuero 2022 [23]	NR; tertiary center	Cross-sectional study	163	Patients with PCOS at tertiary center	NR; age >32 years was a risk factor	NR	NR	None	NAFLD prevalence; fibrosis by transient elastography; PNPLA3 genotyping	No non-PCOS control
Shi 2025 [27]	China	Secondary analysis of randomized clinical trial (PCOS Act)	998 (of 1000)	Chinese infertile women with PCOS	NR	NR	NR in abstract	None	NAFLD; criteria not specified	Overlaps PCOSAct with Feng 2025 [26]
Wang D 2023 [19]	NR	Case-control study	61 (41 obese PCOS, 20 controls)	Obese PCOS patients and age- and BMI-	NR (matched)	Obese; matched; mean NR	NR	20 non-PCOS controls	Liver CAP by transient elastography	Potential overlap with Wang 2022 (excluded)

				matched non-PCOS controls						
Wen 2024 [24]	NR	Cross-sectional study	442	Reproductive-aged women with PCOS; phenotypes OD-HA n=138, OD-PCOM n=161, HA-PCOM n=143	NR	OD-HA and HA-PCOM higher than OD-PCOM ($p<0.05$); mean NR	NR	None	NAFLD prevalence; criteria NR	Phenotype comparison
Vine 2024 [14]	Alberta, Canada	Retrospective observational case-control study	16,531 PCOS cases; 49,335 age-matched controls	PCOS vs age-matched controls; Alberta Health Services database 2002–2018	NR (age-matched)	Obesity 2–3 fold higher; mean NR	NR	49,335 age-matched controls	NAFLD prevalence from database/ICD; criteria NR	Administrative database study

NR: Not reported.

Table 2: Objectives, variables, outcomes, and main findings of included studies.

Study [Ref]	**Objective/aim**	**Main metabolic/hormonal variables**	**Liver/NAFLD outcome**	**Main findings/effect estimates**	**Notes/limitations**	**Overall Risk of Bias**
Gulumsek 2021 [15]	Determine LS and related parameters in PCOS vs healthy controls	BMI, WC, SBP, glucose, ALT, hs-CRP, DHEAS, testosterone, HOMA-IR, LH/FSH, CTRP3	LS by point shear wave elastography	PCOS had higher BMI, WC, SBP, glucose, ALT, hs-CRP, DHEAS, testosterone, HOMA-IR, LH/FSH; lower CTRP3. LS was higher in PCOS ($P<0.001$). LS positively correlated with WC, calcium, DHEAS, testosterone, HOMA-IR; negatively with CTRP3. In linear regression, only CTRP3 was related to LS ($P<0.001$; $\beta=0.734$)	LS is not a confirmed NAFLD diagnosis; small sample	Low
Kara 2023 [20]	Investigate NAFLD presence in adolescents with PCOS and related metabolic/hormonal risk factors	BMI z score, WC, total cholesterol, TG, ALT, androgens; hyperandrogenemia vs non-	USG hepatic steatosis; LSM and CAP by VCTE	WC, total cholesterol, TG, and ALT were higher in PCOS. Hepatic steatosis on USG was similar overall; rate was higher in hyperandrogenic PCOS ($P=0.01$). LSM and CAP	Adolescent population; no overall increase in	Moderate

		hyperandrogenemia		were similar between groups. Hyperandrogenemia was a risk factor for NAFLD	NAFLD prevalence	
Feng 2025 [26]	Assess associations of TyG with anthropometric, metabolic/endocrine profiles, and pregnancy outcomes in PCOS	TyG, age, BMI, WC, HC, WHR, SBP, DBP, FPG, fasting insulin, HOMA-IR, TC, TG, LDL, ApoB, FAI, ALT, AST, IR, MS, NAFLD, QUICKI, HDL, ApoA1, SHBG, ovulation, conception, pregnancy, live birth	NAFLD prevalence/prediction	TyG positively associated with age, BMI, WC, HC, WHR, SBP, DBP, FPG, fasting insulin, HOMA-IR, TC, TG, LDL, ApoB, FAI, ALT, AST, and prevalence of IR, MS, NAFLD. Negatively correlated with QUICKI, HDL, ApoA1, SHBG, ovulation, conception, pregnancy, live birth. AUC for NAFLD: 0.705 (95% CI 0.644–0.766), cutoff 8.665, sensitivity 76.8%, specificity 59.3%. Q4 vs Q1 TyG: NAFLD OR 2.56 (95% CI 1.00–6.55, P-trend=0.069)	Secondary analysis; NAFLD association marginal after adjustment	Moderate
Devas 2026 [25]	Compare metabolic profiles and liver disease severity in PCOS+MASLD vs MASLD alone	Age, BMI, diabetes, metformin use, fibrosis stage	MASLD fibrosis by NAFLD Fibrosis Score, FIB-4, TE, MRE, and biopsy when available	PCOS diagnosed MASLD younger (median 32.5 vs 54.0 vs 36.0 years; P<0.001) and had higher median BMI (37.3 kg/m²). PCOS was not independently associated with advanced fibrosis after age/BMI adjustment. Metformin associated with lower odds of advanced fibrosis: OR 0.59 (95% CI 0.36–0.98; P=0.039). Diabetes associated with increased odds: OR 2.34 (95% CI 1.23–4.46; P=0.010). Propensity-matched PCOS had lower fibrosis stage than MASLD alone (W=22,160; P=4.35×10^−16)	Retrospective; PCOS not associated with worse fibrosis	Low
Ganie 2024 [12]	Estimate nationwide PCOS prevalence, phenotypes, and comorbidities in India	Age, menarche, obesity, dyslipidemia, NAFLD, MS, IGT, diabetes, hypertension	NAFLD prevalence	PCOS prevalence: 7.2% by NIH 1990, 19.6% by Rotterdam 2003, 13.6% by AE-PCOS. Phenotype C 40.8%, D 24.6%. Among PCOS: obesity 43.2%, dyslipidemia 91.9%, NAFLD 32.9%, MS 24.9%, IGT 9.1%, diabetes 3.3%, hypertension 8.3%. Pre-	Cross-sectional; NAFLD criteria NM	Moderate

				PCOS had similar metabolic abnormalities		
Guimarães 2023 [16]	Evaluate and compare dietary patterns and nutritional intake in PCOS with and without NAFLD	BMI, WC, physical activity, 24-hour recall, FFQ, Healthy Eating Index	Abdominal ultrasound for liver steatosis	77% had NAFLD. PCOS+NAFLD had higher BMI (34.9 ± 4.5 vs 30.4 ± 4.9 kg/m²; p=0.001), higher WC (103 vs 95 cm; p<0.001), and were less physically active (34.3% vs 60%; p=0.04). No differences in food intake or diet quality	No non-PCOS control; cross-sectional	Low
Eslami 2022 [17]	Determine NAFLD prevalence and predictive value of BMI, WC, and visceral fat in PCOS	BMI, WC, visceral fat	Liver ultrasonography for NAFLD grade	NAFLD in 53.5% (n=38); mild 65.8%, moderate 31.6%, severe 2.6%. BMI best predictor: cutoff 25.5 kg/m², sensitivity 75%, specificity 75%. Visceral fat: cutoff 5.5%, sensitivity 79%, specificity 67%. WC: cutoff 89.5 cm, sensitivity 73%, specificity 64%	Small sample; no non-PCOS control	Moderate
Patel-Sanchez 2023 [21]	Evaluate NAFLD prevalence and risk factors in overweight/obese adolescents by PCOS status	PCOS, T2DM, IR, hypercholesterolemia, TG, ALT	NAFLD definition: ALT ≥44 U/L ×2 and/or ≥80 U/L ×1, imaging steatosis, or biopsy	NAFLD prevalence 19.1% in PCOS vs 16.8% non-PCOS (p=0.6). PCOS more likely to have IR, hypercholesterolemia, and higher TG. PCOS+T2DM increased NAFLD risk: PR 2.5 (p=0.04); PCOS without T2DM: PR 0.9 (p=0.8). PCOS+NAFLD vs PCOS without NAFLD: hypercholesterolemia 77% vs 48%, T2DM 29% vs 8%, hypertriglyceridemia 65% vs 37% (p<0.01)	T2DM was key risk modifier	Moderate
Özgürlük 2025 [18]	Evaluate relationship between spexin and NAFLD in PCOS	Spexin, glucose, urea, ALT, AST, total cholesterol, LDL, HDL, TG, progesterone, FSH, LH, estradiol, total testosterone, free testosterone, DHEAS	Liver steatosis grading by ultrasound-guided attenuation parameter	Spexin lower in PCOS (p<0.001). Glucose, TG, LH, total testosterone, free testosterone, DHEAS higher in PCOS; HDL lower (p<0.001). Liver steatosis prevalence higher in PCOS (p<0.001). Spexin negatively correlated with BMI, glucose, TG, LH, total testosterone, free testosterone, DHEAS, and liver steatosis grading (p<0.001)	Exact prevalence not reported	Low

Hong 2023 [22]	Explore prevalence and risk factors of NAFLD and MAFLD in PCOS	Medical history, physical characteristics, liver function, lipids, glucose/insulin, sex hormones, PCOM	Liver Fat Score > −0.640 for NAFLD; MAFLD by 2020 definition	Both NAFLD (33.6%) and MAFLD (42.8%) was more prevalent in PCOS patients than in controls (4.2%, P < 0.001). Logistic regression results showed that HOMA-IR ≥ 3.54 and ALT ≥ 18.2 were independently associated with NAFLD (P < 0.001) and MAFLD (P ≤ 0.001).	Cross-sectional; MAFLD P value ambiguous	Low
Maldonado 2022 [13]	Evaluate association of PCOS with NASH among reproductive-age women with NAFLD	Demographics, diabetes, obesity, dyslipidemia, hypertension	NAFLD/NASH discharge codes	PCOS women were younger (median 33 vs 40 years; p<0.001) and more likely to have diabetes (37.0% vs 34.0%), obesity (83.0% vs 58.0%), dyslipidemia (26.0% vs 21.0%), and hypertension (38.0% vs 35.0%). Adjusted OR for NASH with PCOS: 1.22 (95% CI 1.05–1.42; p=0.008)	Administrative database	Moderate
Recuero 2022 [23]	Determine frequency of PNPLA3 rs738409 and impact on NAFLD risk/severity in PCOS	PNPLA3 polymorphism, HOMA-IR, age, HDL, metabolic syndrome, AST	NAFLD prevalence; fibrosis by transient elastography	NAFLD prevalence 72.4%. Heterozygous polymorphism 41.7%, homozygous 8%. HOMA-IR ≥2.5 main factor for NAFLD: OR 4.313 (p=0.022); effect amplified by polymorphism: OR 12.198 (p=0.017). Age >32 years increased risk. HDL ≥50 mg/dL protective. Metabolic syndrome OR 13.030 (p=0.020) and AST >32 U/L OR 9.039 (p=0.009) were independent risk factors for advanced fibrosis	No non-PCOS control; cross-sectional	Moderate
Shi 2025 [27]	Evaluate association between MAP and anthropometric, metabolic, endocrine parameters in infertile PCOS	MAP, age, height, weight, BMI, WC, HC, WHR, SBP, DBP, hirsutism, acanthosis nigricans, FBG, fasting insulin, HOMA-IR, LDL, TG, TC, ApoB, ApoB/ApoA1, total testosterone, FAI, IR, MetS, NAFLD, hyperlipidemia,	NAFLD risk/prediction	MAP positively associated with age, height, weight, BMI, WC, HC, WHR, SBP, DBP, hirsutism, acanthosis nigricans, FBG, fasting insulin, HOMA-IR, LDL, TG, TC, ApoB, ApoB/ApoA1, total testosterone, FAI, and prevalence of IR, MetS, NAFLD, hyperlipidemia. Negatively correlated with QUICKI, HDL, SHBG,	Secondary analysis; overlaps PCOS Act with Feng 2025 [26]	Moderate

		QUICKI, HDL, SHBG, LH, LH/FSH, AMH		LH, LH/FSH, AMH. Q4 MAP had no significantly higher OR for IR, NAFLD, or hyperlipidemia after adjustment. AUC for NAFLD: 0.621 (95% CI 0.554–0.687), sensitivity 69.4%, specificity 53.5%, cutoff 87.17		
Wang D 2023 [19]	Investigate CAP changes/factors and NAFLD prevalence/risk in obese PCOS	General data, body composition, biochemical parameters, sex hormones, liver CAP	Liver CAP by transient elastography	NAFLD more common in obese PCOS vs controls: 75.61% vs 45.00% (P=0.018). PCOS had higher ALT, AST, CAP, TG, TC, LDL-C, total testosterone, FAI, fasting insulin, HOMA-IR; lower HDL-C and SHBG. CAP positively correlated with ALT, AST, TG, total testosterone, FAI, fasting insulin, HOMA-IR; negatively with SHBG. TG, total testosterone, fasting insulin, HOMA-IR were risk factors; total testosterone was independent risk factor for NAFLD	Small sample; potential overlap with Wang 2022 (excluded)	Low
Wen 2024 [24]	Investigate metabolic characteristics of different PCOS phenotypes	BMI, WC, WHR, insulin, HOMA-IR, TC, TG, LDL-C, IGT, T2DM, IR, MS, NAFLD, dyslipidemia, testosterone, Ferriman-Gallwey score	NAFLD prevalence	Prevalence: IGT 17.9%, T2DM 3.6%, IR 58.4%, MS 29.4%, NAFLD 46.6%, dyslipidemia 43.4%. OD-HA and HA-PCOM groups had higher prevalence of IGT, IR, MS, NAFLD, and dyslipidemia than OD-PCOM ($p<0.05$). Testosterone >1.67 nmol/L and F-G score >3 significantly increased risk of metabolic disorders ($p<0.05$)	No non-PCOS control	Moderate
Vine 2024 [14]	Determine prevalence of adverse health outcomes in PCOS vs age-matched controls	Hypertension, renal disease, GI disease, eating disorders, mental illness, depression-anxiety, rheumatoid arthritis, respiratory infections, malignancies,	NAFLD prevalence	Hypertension, renal disease, GI disease, eating disorders, mental illness, depression-anxiety, rheumatoid arthritis, respiratory infections, and all malignancies were 20–40% higher in PCOS ($P<0.0001$). Obesity, dyslipidemia, NAFLD, and T2DM were 2–3 fold	Administrative database	Moderate

		obesity, dyslipidemia, NAFLD, T2DM, cardiovascular, cerebrovascular, peripheral vascular disease, dementia		higher (P<0.001). Cardiovascular, cerebrovascular, and peripheral vascular disease were 30–50% higher and occurred 3–4 years earlier. Dementia was 2-fold higher		

according to a number of included studies [15, 18, 19, 22]. Total testosterone was identified as an independent risk factor for NAFLD in obese PCOS patients (OR = 6.339, 95% CI 1.834–21.906, P = 0.004) [19], and there was a positive association between total testosterone and FAI and the liver-controlled attenuation parameter (CAP) [19]. The emerging evidence that androgen increases promote hepatic steatosis due to increased lipid activity (lipotoxicity and dysregulated de novo lipogenesis) through androgenic effects on VAT supports these observations mechanistically [30]. Furthermore, the pathophysiology of PCOS-associated NAFLD has been discovered to be significantly influenced by mitochondrial dysfunction, and hyperandrogenism has been connected to the regulation of hepatic lipid metabolism, including modifications in the expression of genes involved in lipid synthesis and oxidation [31]. In 2025, a systematic review of literature specifically focused on lean women with PCOS suggested that hyperandrogenism is a strong independent risk factor for NAFLD, as those women had a 40% rate of NAFLD, and that high levels of androgens were strongly associated with hepatic fat accumulation independent of BMI or insulin resistance [32]. Another common and strong factor in the studies included was insulin resistance (IR) [15, 19, 22, 23, 26]. HOMA-IR was found to be significantly increased in PCOS women with MASLD (P = 0.02) and was also included with waist-to-hip ratio and testosterone as an important predictor for screening in the early stage of PCOS in a meta-analysis of East Asian studies [33]. This was supported by the included studies; for instance, in PCOS patients, HOMA-IR ≥ 3.54 was found to be an independent risk factor for NAFLD and MAFLD [22] and HOMA-IR ≥ 2.5 was the primary factor associated with the risk of NAFLD, and its effect was enhanced by the PNPLA3 polymorphism (OR = 12.198, p = 0.017) [23]. It seems that the relationship between IR and NAFLD in PCOS is bidirectional, leading to a vicious circle of metabolic worsening. PCOS-associated IR stimulates de novo lipogenesis in the liver and reduces fatty acid oxidation, whereas MASLD increases insulin resistance and metabolic dysfunction in the body, which will further drive disease progression [34]. In a large cohort study, lean women with PCOS had nearly double the odds of NAFLD compared with lean women without PCOS, which may be explained by this vicious cycle [35]. An interesting consequence is the interaction between IR and hyperandrogenism, as hyperinsulinemia directly stimulates ovarian androgen production and androgens have direct effects on skeletal muscle and adipose tissue that lead to increased IR, resulting in a self-perpetuating endocrine-metabolic loop that culminates in hepatic steatosis [36]. The association between PCOS and NAFLD appears to be influenced by a number of factors, including type 2 diabetes mellitus (T2DM) and obesity. Obese women with PCOS had a higher NAFLD prevalence than age- and BMI-matched obese controls without PCOS (75.61% vs. 45.00%, P = 0.018) [19]. However, PCOS may also be a risk factor for NAFLD in non-obese women. Another 2025 study of lean women with PCOS reported an overall MASLD prevalence of 13.4% based on the Fatty Liver Index, with a higher prevalence in phenotype A (hyperandrogenic) [35]. T2DM turned out to be a substantial effect modifier: concurrent T2DM was a significant risk factor for the development of NAFLD in overweight/obese adolescents with PCOS (PR 2.5, p = 0.04), but not for PCOS alone (PR 0.9, p = 0.8) [21]. According to the evidence presented here, obesity and type 2 diabetes are associated with a higher risk of NAFLD in PCOS patients. However, PCOS itself may confer additional risk of liver disease beyond traditional risk factors, although causality cannot be inferred from these predominantly cross-sectional data. Our study's findings paint a more nuanced picture of the severity of liver disease. After controlling for age and BMI, a single-centre retrospective study of 885 patients with MASLD

revealed that whereas women with PCOS were diagnosed at a considerably younger age (median 32.5 years vs. 54.0 years for non-PCOS females and 36.0 years for males, $P < 0.001$) and had a higher median BMI (37.3 kg/m2), PCOS was not independently linked with more severe liver fibrosis [25]. It's interesting to note that metformin use was associated with lower odds of advanced fibrosis (OR = 0.59, 95% CI 0.36–0.98) [25]. The results are in accordance with the negative correlation of FIB-4 to the serum concentrations of testosterone, DHEAS and FAI found in another study, suggesting a potential protective role of hyperandrogenemia for liver fibrosis despite its association with steatosis [37]. It should be noted that a recent review reported more severe fibrosis and cirrhosis in some PCOS cohorts [29]; these findings require confirmation in prospective studies. The apparent paradox of higher steatosis and potential preservation of fibrosis in PCOS patients will need to be further explored and may depend on the younger age at diagnosis in women with PCOS, but also the protective effect that hyperandrogenism may exert on the progression of fibrosis, and/or the possibility of early intervention to prevent the progression of fibrosis before it is a permanent consequence of the disease. These findings have significant clinical implications. At present there is uncertainty about the utility of screening for NAFLD in women with PCOS. Liver screening thresholds have not been defined by the 2023 International PCOS Guideline and there is no prospective trial evidence to support routine FIB-4 testing in PCOS [38]. Awareness is recommended in the clinical practice guidelines from the Endocrine Society with no recommendations for routine screening in women with PCOS, and there are currently no specific recommendations from the American Association of Clinical Endocrinologists and the International PCOS Network [7, 39]. The high prevalence of NAFLD in PCOS populations (up to 50% in many cohorts) and the risk for progression to steatohepatitis and fibrosis in higher risk subgroups, however, support greater clinical awareness. No included study evaluated screening strategies; therefore, pending prospective evidence, targeted assessment with non-invasive tools (e.g., Fatty Liver Index, HOMA-IR, and liver ultrasound) may be considered in women with PCOS who have hyperandrogenism, central obesity, dyslipidemia, and/or elevated HOMA-IR. The TyG index showed only modest discriminative ability for NAFLD in one included study (AUC 0.705, 95% CI 0.644–0.766), and its adjusted association was marginal (Q4 vs Q1 OR 2.56, 95% CI 1.00–6.55; P-trend = 0.069) [26]; the mean arterial pressure index performed worse (AUC 0.621) [27]. Strengths of this review include a PRISMA-guided process with duplicate screening, data extraction, and risk-of-bias assessment using validated tools, and a structured synthesis of prevalence data, risk factors, and outcomes across 16 studies. However, several constraints are noted: significant heterogeneity in NAFLD assessment methods (different serum markers, imaging techniques, and diagnostic codes) complicates the accurate determination of prevalence. Many studies lack non-PCOS control groups, inhibiting the identification of PCOS's independent risk on NAFLD relative to obesity and metabolic syndrome. The need for longitudinal research is highlighted by the prevalence of cross-sectional studies, which limit causal inference. Furthermore, the literature on PCOS-NAFLD may contain publication bias and misclassification due to dependence on administrative databases. Finally, the results may have been constrained by the omission of non-English studies. In addition, publication was restricted to 2021–2026; 32 of 66 reports could not be retrieved; data for several studies appear to have been extracted from abstract-level information, so many characteristics are not reported; two included studies analyse the same trial cohort; several studies are small or single-centre; protocol registration is not reported; and neither the certainty of evidence nor publication bias could be formally assessed.

Conclusion

The findings suggest that women with PCOS have a high prevalence of NAFLD, with hyperandrogenism and insulin resistance as the main associated factors. Evidence in adolescents and lean women is limited and inconsistent, and no included study evaluated screening strategies; targeted evaluation of women with PCOS and metabolic risk factors is reasonable pending prospective evidence. The connection between liver fibrosis severity and PCOS is still not fully understood, possibly affected by factors such as age, hyperandrogenism, and treatments like metformin. Future studies should emphasize longitudinal designs with standardized diagnostic criteria, investigate the links between hyperandrogenism, hepatic steatosis, and fibrosis, and evaluate targeted medication effectiveness for treating NAFLD in this population.

Conflict of Interest

None

Funding

None

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