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# Prevalence and Risk factors of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) in Patients with Type 2 Diabetes

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## ABSTRACT

**Background:** The coexistence of type 2 diabetes (T2D) and metabolic dysfunction-associated steatotic liver disease (MASLD) accelerates the progression of cardiovascular and metabolic disorders. To mitigate the medical and economic burden associated with these comorbidities, identifying their risk factors is essential.

**Aim:** to assess the prevalence of MASLD and identify its associated risk factors in T2D patients in Tunisia.

**Methods:** This descriptive and analytical study included 202 T2D patients followed over a 12-year period at the Endocrinology Department in Sfax, Tunisia. Patients were divided into two groups: 101 patients with confirmed MASLD (G1) and 101 patients without MASLD (G2).

**Results:** The prevalence of MASLD was 17.03%. A family history of autoimmunity was significantly associated with MASLD. Glycemic control indicators, including HbA1C levels (p=0.943) and blood glucose levels (p=0.627), did not differ between groups. Insulin therapy was significantly less common in G1 (p=0.027). Abdominal obesity and elevated triglyceride levels were strongly associated with MASLD. Additionally, carbohydrate intake was higher in G1 compared to G2 (p=0.041). Multivariate analysis identified non-HDL cholesterol as the most significant independent risk factor for MASLD (odds ratio [OR]: $4.07 \times 10^5$), followed by uric acid levels (OR: 1.03, p=0.024) in a 95% confidence interval of [1.004–1.058].

**Conclusion:** The findings reveal a notable prevalence of MASLD among T2D patients in Tunisia, with non-HDL cholesterol, uric acid levels, abdominal obesity, and dietary patterns emerging as key contributors. These results emphasize the need for targeted screening and management strategies of MASLD.

**KEYWORDS:** Type 2 diabetes, Metabolic Dysfunction-Associated Steatotic Liver Disease, Risk factors, Metabolic disorders.

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## INTRODUCTION:

Metabolic dysfunction-associated steatotic liver disease (MASLD), previously termed non-alcoholic fatty liver disease (NAFLD), represents the most common chronic liver disorder globally, impacting more than 30% of the adult population (1). It is characterized by an abnormal accumulation of triglycerides within liver cells, identified through imaging techniques or histological examination, and linked to at least one cardiometabolic risk factor, while excluding significant alcohol intake and other known causes of hepatic steatosis (2). Type 2 diabetes mellitus (T2DM) poses a significant global public health challenge. In the last four decades, the prevalence of T2DM among adults has increased fourfold (3).

The concurrence of T2DM and MASLD presents a major clinical challenge, as it hastens the development of cardiovascular and metabolic complications (4). In addition, MASLD complicates the treatment of T2DM, hindering the attainment of consistent glycemic control. This coexistence elevates the risk of advanced liver disease, such as cirrhosis and hepatocellular carcinoma, ultimately resulting in worse health outcomes and shortened lifespan for affected individuals (5).

Although T2DM is widely acknowledged as a significant risk factor for MASLD, the available data on the specific factors that contribute to the onset of MASLD in individuals with T2DM remains limited. This study aims to assess the prevalence of MASLD and identify its associated risk factors in T2DM patients in Tunisia.

## METHODS:

### *Study design*

This descriptive and analytical study included T2DM patients followed between 2012 to 2024 at the Endocrinology and Diabetology Department of Hedi Chaker University Hospital in Sfax, Tunisia.

### *Study population*

T2DM was diagnosed based on the 2025 ADA criteria (6): fasting plasma glucose ≥ 1.26 g/L, a 2-hour post-glucose challenge of 75 g ≥ 2 g/L, glycated hemoglobin (HbA1C) ≥ 6.5%, or the presence of classic hyperglycemia symptoms with a random plasma glucose ≥ 2 g/L. MASLD was diagnosed according to the 2024 consensus guidelines from AASLD, EASL, EASD, and EASO (2), which define it as excessive triglyceride storage in the liver, detected by imaging or biopsy, in conjunction with at least one of the following cardiometabolic risk factors:

1. Body mass index (BMI) ≥ 25 kg/m² or waist circumference > 94 cm (men) or > 80 cm (women)
2. Fasting glucose ≥ 5.6 mmol/L, a 2-hour glucose after a 75 g glucose load ≥ 7.8 mmol/L, HbA1c ≥ 5.7%, T2DM, or treatment for T2DM
3. Blood pressure ≥ 130/85 mmHg or treatment with antihypertensive medications
4. Plasma triglycerides ≥ 1.70 mmol/L or treatment with lipid-lowering medications
5. HDL-cholesterol ≤ 1.0 mmol/L (men) or ≤ 1.3 mmol/L (women) or treatment with lipid-lowering medications

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We excluded patients with other types of diabetes, including type 1, monogenic, secondary, and gestational diabetes. Patients with known liver diseases (e.g., chronic viral hepatitis, autoimmune hepatitis, drug-induced liver injury) or conditions contributing to hepatic steatosis (e.g., Cushing's syndrome, acromegaly, uncontrolled hypothyroidism) were also excluded, as well as those using steatogenic medications (e.g., methotrexate, antiretrovirals, amiodarone, corticosteroids). Additionally, we excluded individuals with significant alcohol consumption (≥20 g/day for women and ≥30 g/day for men) or incomplete medical records.

Patients were divided into two groups according to the presence or absence of MASLD.

***Data collection***

We collected demographic information, including age, sex, personal history of dyslipidemia and hypertension, and patient habits such as smoking and occasional alcohol use. Physical examination data included BMI, calculated as body weight (kg) / height ($m^2$)), waist circumference, systolic and diastolic blood pressure, heart rate, acanthosis nigricans, and the presence of metabolic syndrome. Diabetes-related data included disease duration in years and chronic complications, both microvascular and macrovascular. Biological data included HbA1C, fasting blood glucose, lipid profile (LDL-cholesterol, triglycerides, HDL-cholesterol, and non-HDL cholesterol), liver function tests (aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP)), C-reactive protein (CRP), uric acid levels, and glomerular filtration rate. Imaging data included ultrasound, CT scans, and MRIs. Additionally, we conducted a dietary survey to assess macronutrient intake, including daily caloric intake, lipids, proteins, and carbohydrates as percentages of total intake, along with saturated fatty acids, monounsaturated fatty acids, and polyunsaturated fatty acids as percentages of daily intake. We also evaluated micronutrient intake, including fiber, iron, calcium, and cholesterol. For therapeutic data, we assessed treatments related to diabetes (oral hypoglycemic agents and insulin), hypertension, and dyslipidemia.

***Outcomes***

In the first part of our study, we described the prevalence of MASLD in T2DM patients. Then, we compared the parameters of the two groups, G1 and G2, to identify the risk factors of MASLD in patients with T2DM.

***Statistical analysis***

Data was analyzed using statistical software SPSS version 26.0 (SPSS Inc., Chicago, IL). Categorical variables were presented as frequencies (n) and percentages (%). The Shapiro-Wilk test was employed to assess the normality of the continuous variables' distribution. The univariate analysis was conducted using the Chi-square test when applicable. In cases with small sample sizes, the Fisher exact test was used. For the comparison of quantitative variables, the Mann-Whitney U test was applied. Multivariate logistic regression analysis was performed to analyze the independent variables associated with the development of MASLD. The odds ratios and 95% confidence intervals were calculated. $P < 0,05$ was considered statistically significant.

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**RESULTS:**

From an initial pool of 6,149 patients with T2DM, we excluded 1,068 individuals diagnosed with other forms of diabetes. Among the remaining 5,081 patients, abdominal imaging was available for only 734. Further exclusions were applied for patients with known liver diseases, conditions contributing to hepatic steatosis, use of steatogenic medications, significant alcohol consumption, and incomplete records.

After applying these criteria, 202 patients were included in the study. They were divided into two groups based on the presence or absence of MASLD: Group 1 (G1) comprised 101 T2DM patients with confirmed MASLD, while Group 2 (G2) included 101 T2DM patients without MASLD.

***Prevalence of MASLD in Type 2 Diabetic Patients***

The prevalence of MASLD in our population of patients with T2DM was 17.03%.

***Risk factors of MASLD in T2DM patients***

The present study included 202 T2DM patients, with each group consisting of 101 patients. The characteristics of the studied groups are presented in Table 1. Demographic factors did not influence the development of steatotic lesions, as both groups (G1 and G2) had similar characteristics regarding age (55 (45-66) years vs 59 (49-68) years, respectively; p=0.087) and sex (p=0.773). The family history of cardiometabolic diseases was not significantly associated with MASLD (p=0.949), but a family history of autoimmune diseases showed a significant association with MASLD (p=0.020). Smoking and occasional alcohol consumption were not lifestyle factors contributing to steatotic disease in this sample (p=0.525, p=0.485, respectively). Both groups had a similar median duration of T2DM (3 years; p=0.441). HbA1c (p=0.943) and blood glucose levels (p=0.627) were similar between the groups, suggesting no impact of glycemic control on MASLD occurrence. Oral antidiabetic treatments (metformin: p=0.131, sulfonylureas: p=0.557) were not associated with MASLD, while insulin therapy was significantly linked to the absence of MASLD lesions (p=0.027). Chronic micro- and macrovascular complications of T2DM were associated with MASLD, particularly retinopathy (p=0.020) and peripheral arterial disease (p=0.024). The prevalence of metabolic syndrome was high in both groups (81.2% vs 79.8%, respectively; p=0.805), without significant differences. Abdominal obesity was significantly associated with MASLD development. Acanthosis nigricans was more prevalent in diabetics with MASLD (40% vs 18.3%, p=0.001).

Clinical and pharmacological characteristics of hypertension and dyslipidemia were not significantly associated with MASLD (hypertension: p=0.480, dyslipidemia: p=0.819), but lipid parameters such as higher triglycerides (p=0.022) were significantly associated with MASLD. Liver function parameters were significantly elevated,

reflecting liver cytolysis (AST (p=0.009), ALT ($p<10^{-3}$)) and cholestasis (GGT (p=0.016)). Purine metabolism disorders were more pronounced in G1, with significantly higher uric acid levels compared to G2 (366 [293-421] vs 318.5 [235-396] µmol/L, p=0.026).

The dietary factors associated with MASLD are listed in Table 2. Total energy intake was similar between groups 1 and 2 (2125 [1900-2585] vs 2100 [1700-2400] kcal/day,

| Parameters | Group 1 With MASLD (N=101) | Group 2 Without MASLD (N=101) | p |
|---|---|---|---|
| Age (years) | 55 (45-66) | 59 (49-68) | *0,087* † |
| Male gender | 37,6% | 39,6% | *0,773* # |
| Family history of cardiometabolic diseases | 42,0% | 41,6% | *0,949* # |
| Family history of autoimmunity | 33,3% | 18,6% | **0,020** # |
| Smoking | 22,7% | 19,0% | *0,525* # |
| Occasional alcohol concumption | 6,2% | 4,0% | *0,485* F |
| BMI (kg/m²) | 32,5 [27,9-36,1] | 29,2 [25,2-33,4] | ***<10⁻³*** † |
| Waist circumference (cm) | 104,0 [99-112] | 101,0 [90-110] | ***0,05*** † |
| Obesity | 63,0% | 45,8% | **0,014** # |
| Acanthosis nigricans | 40,0% | 18,3% | ***0,001*** # |
| Metabolic syndrome | 81,2% | 79,8% | *0,805* # |
| SBP (mmHg) | 130 [120-150] | 130 [120-150] | *0,791* † |
| DBP (mmHg) | 80,0 [70,0-80,0] | 80,0 [70,0-90,0] | *0,631* † |
| Heart rate (Beats/min) | 76,0 [71,0-89,0] | 78,0 [74,0-90,0] | *0,290* † |
| Hepatomegaly | 12,9% | 0,0% | **<10⁻³** F |
| Hypertension | 56,4% | 51,5% | *0,480* # |
| Treatment with ACE/ARBs | 80,4% | 69,2% | *0,182* # |
| Treatment with CCB | 46,4% | 61,5% | *0,116* # |
| Treatment with TZD | 16,1% | 17,3% | *0,999* F |
| Dyslipidemia | 41,6% | 40,0% | *0,819* # |
| Lipid-lowering drugs | 62,6% | 59,9% | 0,470 # |
| Diabetes duration (years) | 3,0 [0,7-10,0] | 3,0 [1,0-10,0] | *0,441* † |
| HbA1C (%) | 9,0 [6,8-11,3] | 9,0 [7,0-11,3] | *0,943* † |
| Fasting blood glucose (g/L) | 10,3 [6,7-14,0] | 9,0 [6,7-13,7] | *0,627* † |
| Microvascular complications | 77,5% | 64,9% | *0,095* # |
| Retinopathy | 48,1% | 26,0% | ***0,020*** # |
| Nephropathy | 40,9% | 41,3% | *0,951* # |
| Neuropathy | 86,5 [58,2-114,2] | 82,9 [55,0-113,0] | *0,773* † |
| Erectile dysfunction | 35,6% | 32,6% | *0,398* # |
| Macrovascular complications | 35,4% | 31,6% | *0,577* # |
| Ischemic heart disease | 23,1% | 25,6% | *0,723* # |
| Ischemic stroke | 31,7% | 28,0% | *0,568* # |
| Peripheral Arterial Disease | 26,7% | 26,7% | *1,000* # |
| Treatment with insulin | 5,9% | 3,0% | *0,498* F |
| Treatment with metformin | 13,9% | 4,0% | ***0,024*** F |
| Treatment with sulfonylureas | 27,7% | 31,7% | *0,557* # |
| Total cholesterol (mmol/L) | 4,9 [3,9-5,6] | 4,6 [3,7-5,3] | *0,170* † |
| LDL cholesterol (mmol/L) | 2,9 [2,2-3,6] | 3,1 [2,4-3,8] | ***0,05*** † |
| Triglycerides (mmol/L) | 2,0 [1,3-2,7] | 1,7 [1,2-2,3] | ***0,022*** † |
| HDL cholesterol (mmol/L) | 1,0 [0,8-1,2] | 1,0 [0,8-1,2] | *0,894* † |
| Non HDL cholesterol (mmol/L) | 3,9 [2,8-4,6] | 3,4 [2,9-4,2] | *0,153* † |
| GFR (mL/min/1.73 m²) | 86,5 [58,2-114,2] | 82,9 [55,0-113,0] | *0,773* † |
| CRP (µmol/L) | 4,0 [2,0-9,0] | 4,0 [2,0-23,0] | *0,436* † |
| AST (IU/L) | 20,0 [14,5-33,5] | 17,0 [13,0-23,0] | ***0,009*** † |
| ALT (IU/L) | 23,0 [15,5-37,5] | 16,0 [12,0-23,0] | ***<10⁻³*** † |
| GGT (IU/L) | 45,0 [25,5-65,0] | 36,0 [19,0-50,0] | ***0,016*** † |
| ALP (IU/L) | 87 [62-120] | 82 [65-121] | *0,664* † |
| Uric acid (µmol/L) | 366 [293-421] | 318,5 [235-396] | ***0,026*** † |

respectively; p=0.251). Regarding macronutrients, carbohydrate intake was significantly higher in G1 compared to G2 (53% [50%-57%] vs 52% [48%-54%], respectively; p=0.044). As for micronutrients, daily intakes of iron, calcium, and fiber were similar between the two groups.

**Table 1. Demoraphic and linical features of T2DM patients with or without MASLD**

† : Mann-Whitney U test ; # : Chi-square test ($\chi^2$) ; F : Fisher's exact test ; BMI : Body Mass index ; SBP : systolic blood pressure ; DBP : diastolic blood pressure ; ACE : Angiotensin-converting enzyme inhibitors ; Angiotensin II receptor antagonists ; CCB : Calcium channel blocker ; TZD : thiazide diuretics ; HbA1C : glycated hemoglobin ; GFR : Glomerular Filtration Rate ; CRP : C reactive protein ; AST : Aspartate Aminotransferase ; ALT : Alanine Aminotransferase ; GGT : Gamma-glutamyl transferase ;ALP :Alkaline phosphatase.

**Table 2. Dietary factors associated with the development of MASLD**

| Parameter | Group1 With MASLD (N=101) | Group 2 Without MASLD (N=101) | p |
|---|---|---|---|
| Daily caloric intake (Kcal/day) | 2125 [1900-2585] | 2100 [1700-2400] | 0,251 † |
| Proteins (%) | 10,0 [10,0-12,0] | 11,0 [10,0-12,0] | 0,080 † |
| Carbohydrates (%) | 53,0 [50,0-57,0] | 52,0 [48,0-54,0] | **0,041 †** |
| Fats (%) | 2,6 [0,7-5,4] | 2,0 [0,1-3,9] | 0,302 † |
| SFA (%) | 8,0 [7,0-9,0] | 8,0 [7,0-9,0] | 0,403 † |
| MUFA (%) | 15,0 [12,0-20,0] | 16,0 [14,0-21,0] | 0,225 † |
| PUFA (%) | 10,0 [7,0-15,0] | 11,0 [6,0-15,0] | 0,896 † |
| Iron (mg/day) | 7,0 [6,0-10,0] | 7,0 [5,0-10,0] | 0,759 † |
| Calcium (mg/day) | 485 [380-620] | 453 [355-610] | 0,559 † |
| Cholesterol (mg/day) | 180 [120-330] | 168, [92,5-275] | 0,365 † |
| Fibers (g/day) | 15,0 [12,0-20,0] | 15,0 [10,0-21,0] | 0,962 † |

† : Mann-Whitney U test ; SFA : Saturated Fatty Acids ; MUFA : Monounsaturated Fatty Acids ; PUFA : Polyunsaturated Fatty Acids.

### *Independent risk factors of MASLD in T2DM patients*

To identify independent factors (table 3) driving the development of MASLD in diabetic individuals, we performed a multivariate logistic regression analysis. This revealed three factors independently influencing the onset of MASLD. Non-HDL-cholesterol levels emerged as the strongest risk factor in our cohort, with a high odds ratio (OR) of 4.07 $x10^5$, suggesting a robust effect on MASLD prediction (p=0.029) in a wide confidence interval (CI) of [3.82-4.35 $x10^{10}$]. Uric acid levels also stood out as a relevant predictor for MASLD, with an OR of 1.03 (p=0.024) in a CI of [1.004-1.058]. Interestingly, LDL-cholesterol was assigned a β coefficient of -3.85. Its OR of 0.02 (CI = [0.001 - 0.433]) indicated that higher LDL-cholesterol levels were associated with a lower probability of developing MASLD. While this may seem counterintuitive, it's important to note that the MASLD group had lower LDL-cholesterol levels due to pharmacological interventions reducing lipid levels.

**Table 3. Independant risk factors of MASLD in T2DM patients**

| Factors | β | p | OR | CI 95% |
|---|---|---|---|---|
| Non-HDL cholesterol (mmol/L) | 12,92 | *0,029* | 4,07 x$10^{5}$ | [4,35 x$10^{10}$] |
| Uric Acid (µmol/L) | 0,03 | *0,024* | 1,03 | [1,004-1,058] |
| LDL cholesterol | -3,85 | *0,012* | 0,02 | [0,001-0,433] |

**OR : Odds Ratio ; CI : Confidence Interval.**

## DISCUSSION :

MASLD stands as the leading cause of chronic liver disease across the globe. At present, it affects approximately one in every four individuals worldwide (7). The relationship between T2DM and MASLD is intricate and reciprocal, fueled by inflammation, fibrosis, and insulin resistance, all of which contribute to an elevated risk of cardiovascular complications (8). Consequently, it is crucial to assess MASLD in T2DM patients.

Various studies report a prevalence of MASLD ranging from 21.5% to 75.1% among patients with T2DM (9–13) . In this cohort of a Tunisian population, the prevalence of MASLD was 17.03%, slightly lower than the values reported in the literature. This disparity seems to stem from two factors: the characteristics of the population under study and the diagnostic methods employed.

The duration of T2DM and glycemic control, as evidenced by HbA1c levels (p=0.943) and blood glucose levels (p=0.627), did not influence the development of MASLD. The same result was reported by Assarrar et al (11). Insulin therapy was significantly associated with the absence of MASLD lesions in diabetic patients. By effectively controlling blood sugar levels and reducing hepatic inflammation, insulin therapy helps lower the risk of MASLD development. This is primarily achieved by mitigating insulin resistance, a crucial factor in liver fat accumulation (14).

Retinopathy and peripheral artery disease have been linked to hepatic lesions. Elevated levels of mediators like advanced glycation end products, C-reactive protein, interleukin-6, and tumor necrosis factor-alpha can cause vascular injury, especially in the retinal vessels. These markers are notably increased in diabetic individuals with MASLD (15).

In our study, obesity (p=0.014), was associated with an increased risk of developing MASLD. The development of MASLD is closely linked to insulin resistance in white adipose tissue (16). In obesity, adipocytes expand in number and size, leading to localized hypoxia and infiltration by immune cells. This dysfunctional white adipose tissue releases abnormal adipokines with pro-inflammatory and pro-atherogenic properties, which promote insulin resistance. Consequently, uncontrolled triglyceride breakdown occurs, increasing the delivery of free fatty acids to the liver. This process facilitates hepatic fat accumulation and the development of MASLD (17).

In our study, triglyceride levels (p=0.022) were significantly associated with the development of MASLD. Disruptions in lipid metabolism are frequently seen in individuals with hepatic steatosis, typically manifesting as increased triglycerides and LDL cholesterol, decreased HDL cholesterol, and excessive total cholesterol accumulation (18). The accumulation of fatty acids in hepatocytes is lipotoxic (19). It stimulates the lysosomal apoptosis pathway, induces stress in the endoplasmic reticulum, and initiates oxidative stress. Around 60% of the triglycerides found in the livers of MASLD patients are believed to derive from dysfunctional white adipose tissue. Another 25% of hepatic triglycerides result from enhanced de novo lipogenesis, which is driven by compensatory hyperinsulinemia linked to insulin resistance (17).

In this study, significantly higher purine metabolism was observed in T2DM patients with MASLD (p=0.026). Several studies indicate that uric acid is a significant risk factor for the development of MASLD (20). Hepatic fat accumulation and insulin resistance are driven by multiple pathways, including the activation of the NLRP3 inflammasome, increased mitochondrial oxidative stress, and enhanced fructose-stimulated lipogenesis. Additionally, insulin resistance linked to MASLD can raise uric acid levels by reducing its renal clearance in the proximal tubules. This reciprocal relationship highlights the intricate connection between uric acid and the development of MASLD (20).

Dietary carbohydrates are the third major contributor to hepatic triglyceride accumulation (21). Dietary carbohydrates play a central role in stimulating hepatic de novo lipogenesis and have a more direct impact on MASLD development compared to dietary fats. The phosphorylation of fructose in the liver depletes ATP, causing an accumulation of ADP, which then acts as a precursor for uric acid synthesis. This process contributes to oxidative stress in the liver, further advancing MASLD progression (21). A high-calorie diet, excessive intake of saturated fats and refined carbohydrates are well-established risk factors for MASLD in individuals with T2DM (22). Conversely, studies have demonstrated that adopting a Mediterranean diet can significantly reduce liver fat content, even in the absence of weight loss (22).

## CONCLUSION:

Our data suggest that MASLD is prevalent among patients with T2DM, with lipid and uric acid parameters, particularly elevated non-HDL cholesterol levels, being independently associated with an increased risk of developing MASLD. These findings underscore the importance of early diagnosis and effective management of MASLD, which can ultimately reduce liver-related morbidity and mortality in the diabetic population. Further studies, particularly those conducted with prospective and randomized cohorts, are needed to explore the relationship between MASLD and metabolic control, as well as the impact of MASLD on microvascular and macrovascular complications. It remains crucial to determine whether better metabolic control and improved dietary habits can alter the course of MASLD and its associated complications.

## ETHICS APPROVAL

The medical ethical comity of Sfax approved this research: number 0486/24.

## DECLARATION OF CONFLICTING INTERESTS

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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## AUTHORS CONTRIBUTIONS

Yesmine Elloumi, Mouna Elleuch: Conceptualization, Data curation, Investigation, Formal analysis, Methodology, Writing – original draft, Supervision, Validation.

Wissal Abbes, Khouloud Boujelben, Dhoha Ben Salah, Nabila Rekik Mejdoub: Supervision, Validation.

## PATIENT CONSENT STATEMENT

Not applicable because this was a retrospective study, and there was no contact with the patients. All the data for the study were obtained from the case notes of the patients.

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