

# Correlation Between the Controlled Attenuation Parameter and the Prevalence of T2DM in Southern China: A Retrospective Cross-Sectional Study

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**Objective:** To explore the relationship between controlled attenuation parameters (CAP) of the liver and the risk of type 2 diabetes mellitus (T2DM), and to determine the optimal cutoff value of CAP for predicting T2DM.

**Methods:** This study employed a cross-sectional study design, enrolling 7035 participants. CAP values were measured using hepatic transient elastography, and participants were categorized into low ( $\leq 231$  dB/m), middle (232–269 dB/m), and high ( $\geq 270$  dB/m) groups. Multivariate logistic regression analysis was used to assess the association between CAP and T2DM, and the optimal cutoff value was determined using receiver operating characteristic (ROC) curves.

**Results:** After adjusting for potential confounders, participants in the high-CAP group had a significantly greater risk of developing T2DM than did those in the low-CAP group (OR=1.545; 95% CI=1.263–1.890;  $p < 0.001$ ). Stratified analyses revealed that compared with a low CAP, a 1.0-increased CAP was associated with a 0.852-fold increased T2DM risk in people aged  $< 55$  years (OR=1.852; 95% CI=1.405–2.441;  $p < 0.001$ ) and a 0.6-fold increased T2DM risk in overweight patients (BMI  $\geq 24$  kg/m<sup>2</sup>) (OR=1.600; 95% CI=1.127–2.271;  $p = 0.009$ ). ROC analysis revealed an optimal CAP cutoff value of 246.5 dB/m for T2DM prediction, with an area under the curve of 0.569 (specificity=48.2%, sensitivity=64.2%).

**Conclusion:** CAP levels are associated with a dose-response relationship with the risk of T2DM, particularly in younger and overweight populations. Although CAP has limited predictive value on its own, it can be used as an auxiliary indicator for T2DM risk screening.

**Keywords:** type 2 diabetes mellitus, controlled attenuation parameter, transient elastography

## Introduction

Type 2 diabetes mellitus (T2DM) has become a major public health issue worldwide. According to data from the International Diabetes Federation (IDF), there were 537 million adults with diabetes globally in 2021, and this number is projected to surge to 783 million by 2045.<sup>1</sup> China, one of the countries with the heaviest diabetes burden, reportedly had 230 million people with diabetes in 2023, with over 90% being T2DM.<sup>2</sup> Asian populations exhibit distinct characteristics in T2DM onset compared to other regions, with a lower body mass index (BMI) threshold for disease onset and earlier onset of beta cell dysfunction, presenting unique challenges for disease prevention and control.

Although current management strategies focus on glycemic control after diagnosis of T2DM, new evidence suggests that intervention during the prediabetic stage (such as impaired glucose tolerance) can delay disease progression.<sup>3</sup> This highlights

the need for reliable early risk stratification tools. Several studies, including the Daqing Study in China,<sup>4–6</sup> the Diabetes Prevention Program (DPP) in the United States,<sup>7</sup> and the Diabetes Prevention Study (DPS) in Finland,<sup>8</sup> have confirmed that intensive lifestyle interventions, particularly active management in the early stages, can significantly reduce the risk of developing diabetes and even achieve disease remission. Scholar Andreas L Birkenfeld further proposed the concept of “pre-diabetes remission”,<sup>9</sup> providing a new theoretical foundation for early intervention in diabetes. Against this backdrop, the role of hepatic steatosis in the onset and progression of type 2 diabetes has garnered increasing attention. Research indicates that the liver, as a key regulatory organ in glucose and lipid metabolism, exhibits a close association between fat accumulation and insulin resistance. In T2DM patients, the prevalence of non-alcoholic fatty liver disease (NAFLD) reaches as high as 70%,<sup>10</sup> significantly higher than in the general population. For every 5% increase in liver fat content, the risk of developing diabetes increases by 27%.<sup>11</sup> Systematic reviews indicate that preventive measures targeting high-risk populations can reduce the incidence of T2DM by 58% through lifestyle interventions.<sup>6,7</sup> Therefore, exploring the underlying mechanisms linking hepatic steatosis to carbohydrate metabolism disorders and developing targeted intervention strategies will be a key direction for future diabetes prevention and treatment research.

A study showed that hepatic steatosis is an early and sensitive indicator of metabolic diseases.<sup>12</sup> Quantitative fat analysis is clinically important for the early identification of hepatic steatosis and metabolic diseases. Ultrasonography is a low-cost and widely used technique for the assessment of steatosis and has the advantages of being noninvasive, safe and radiation free. However, ultrasonography is only qualitatively diagnostic, not quantitatively assessable, and is highly operator dependent. In addition, its sensitivity is reduced in patients with a BMI >40 kg/m<sup>2</sup> and liver fat content <20%,<sup>13</sup> and it is also susceptible to the patient’s own factors, such as gas interference and abdominal obesity.<sup>14</sup> When the quantitative diagnosis of hepatic steatosis by CT reaches 30%, the sensitivity and specificity are 82% and 100%, respectively.<sup>15</sup> However, the diagnostic value of CT for mild fatty liver is low and is limited by its radiologic properties. The traditional MRI technique mainly determines the presence of hepatic steatosis by distinguishing the height of liver tissue signals, but it cannot quantitatively assess the hepatic fat content.<sup>16</sup> MRI-PDFF has a relatively high diagnostic value and can accurately reflect the degree of fatty liver,<sup>17</sup> but its application is limited by its high cost and the relative complexity of information processing.

Hepatic transient elastography is an emerging examination method with many advantages, such as being noninvasive, painless, accurate, intuitive, fast, easy and economical. The controlled attenuation parameter (CAP) measured by transient elastography is mainly used to quantitatively detect the degree of hepatic steatosis.<sup>18</sup> As fat content increases, the CAP index rises, enabling non-invasive, quantitative evaluation of the severity of hepatic steatosis. This technique compensates for the limitations of traditional testing methods and extends the clinical applications of ultrasound images.<sup>19</sup> It is now well established and widely used in clinical practice. The CAP measured by transient elastography is based on the principle that ultrasound propagates through a medium with significant attenuation. It is used to quantify the extent of hepatic steatosis by measuring the degree of attenuation of the ultrasound signal.<sup>18</sup> Data from established studies have shown that the CAP is a good and objective method for assessing steatosis,<sup>20</sup> with a sensitivity of 80–85% or better.<sup>21</sup> There is evidence that NAFLD status is a risk factor for T2DM.<sup>22</sup> Fatty liver not only precedes the development of T2DM but also predicts the development of T2DM and cardiovascular disease.<sup>12</sup> Markov’s modeling of comparative strategies for screening for NAFLD in patients with T2DM suggests that liver blood tests and transient elastography screening are the most cost-effective (ie, a better cost–benefit ratio) methods.<sup>23</sup> Therefore, measurement of the CAP by transient elastography may be a tool for early screening of people at risk for T2DM.

Previous studies have shown that CAP values are positively correlated with the insulin resistance index (HOMA-IR), and that patients with NAFLD have a significantly increased risk of developing T2DM.<sup>24</sup> However, most existing studies have focused on the relationship between CAP and insulin resistance, while few have directly explored the association between CAP and T2DM prevalence. Additionally, there is no consensus on the optimal cutoff value for CAP in predicting T2DM. Furthermore, the predictive value of CAP may vary across different populations, such as those stratified by age, gender, or BMI.

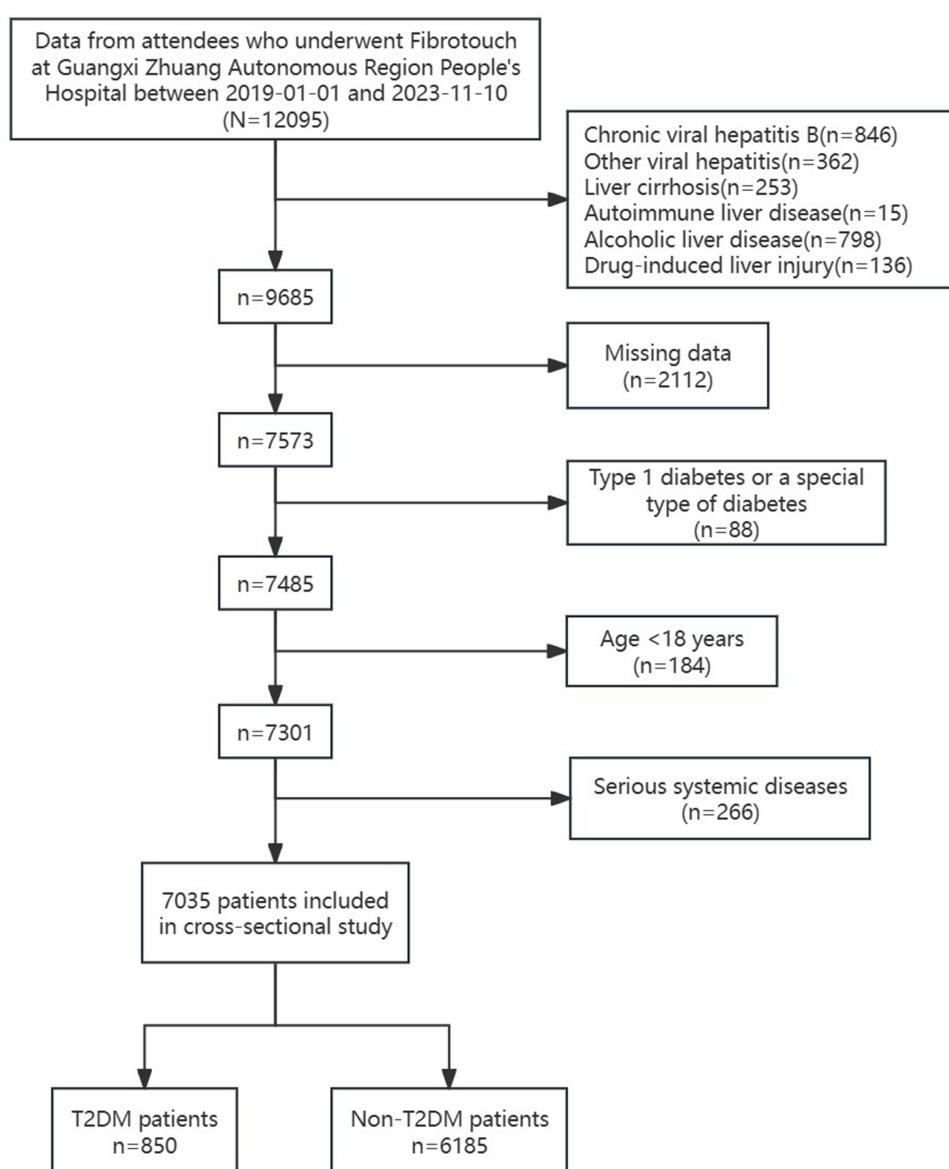
This study employs a cross-sectional design to investigate the association between CAP and the prevalence of T2DM and its predictive value for T2DM, aiming to provide new insights for early identification of high-risk populations for T2DM in clinical practice and to offer epidemiological evidence for the role of hepatic fat accumulation in the pathogenesis of T2DM. This will contribute to the development of an early warning system for T2DM and provide clinical guidance for metabolic risk management in patients with NAFLD.

## Research Design and Methods

### Study Population

This study used a cross-sectional study design to screen 12,095 patients who underwent Fibrotouch testing for the first time between 2019-01-01 and 2023-11-10. Ultimately, 7035 patients were included in the analysis. The data for this study were obtained from the People's Hospital of Guangxi Zhuang Autonomous Region, the largest tertiary-level hospital in southwest China, with an annual outpatient volume of approximately 2 million visits, including approximately 20,000 T2DM patients treated annually by the Endocrinology and Metabolism Department. The exclusion criteria were as follows: (1) chronic viral hepatitis B, other viral hepatitis, liver cirrhosis, autoimmune liver disease, alcoholic liver disease or drug-induced liver injury; (2) missing data; (3) type 1 diabetes or a special type of diabetes; (4) age <18 years; and (5) serious systemic diseases (including heart, lung, liver or kidney diseases; infectious diseases; mental diseases)(Figure 1).

The study used a formula  $n = Z^2_{1-\alpha/2} P(1 - P)/d^2$  to estimate the sample size, assuming a T2DM prevalence of 11.2% in China from literature.<sup>25</sup> The allowable error was set at 10% of the prevalence, or  $d = 0.1p = 0.011$ , with



**Figure 1** Flowchart of study participants.

a significance level of  $\alpha = 0.05$ . The calculation resulted in a required sample size of  $n = 3158$ . To ensure statistical significance and reliability, we aimed for at least 3158 participants. After rigorous screening, a total of 7035 patients were ultimately included in this study.

## Data Collection

The study population's sex, age and history of diabetes were obtained from outpatient medical records or admission records, and the patients' height and weight were obtained from nursing records. The laboratory biochemical parameters included alanine aminotransferase (ALT), aspartate aminotransferase (AST), total bilirubin (TBIL), direct bilirubin (DBIL), indirect bilirubin (IBIL), alkaline phosphatase (ALP), creatinine (Cr), uric acid (UA), urinary nitrogen (UREA), total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) and fasting blood glucose (FBG) concentrations. Body mass index (BMI) was calculated according to the formula  $BMI = \text{weight}/\text{height}^2$  ( $\text{kg}/\text{m}^2$ ). All subjects fasted for at least 8 h after dinner, and morning fasting venous blood was collected and sent for examination.

The patients were examined with hepatic transient elastography (Fibrotouch, pro5000, Wuxi Hessel Medical Technology Co., Ltd), and they were asked to abstain from alcohol for 1 week before the examination and fast on the day of the measurement. Then, the patients were asked to assume a lying position, and an examination point was selected. The test was conducted more than 10 times on average for each subject, and the median values of the valid measurements of the CAP and the liver stiffness measurement (LSM) were used for evaluating the degree of hepatic steatosis and the extent of hepatic fibrosis, respectively. All demographic data, anthropometric data, laboratory biochemical indicator data, and inpatient medical record information were collected anonymously. The data were sourced from the database of the People's Hospital of Guangxi Zhuang Autonomous Region and extracted in a structured manner from the hospital's electronic medical record (EMR) system by research members of the hospital's information department who had undergone standardized training.

## Related Definitions

According to the 1999 World Health Organization (WHO) guidelines,<sup>26</sup> the diagnosis of type 2 diabetes requires meeting one of the following conditions: typical diabetes symptoms plus random blood glucose test  $\geq 11.1$  mmol/L; fasting blood glucose test  $\geq 7.0$  mmol/L; 2-hour blood glucose test after OGTT glucose loading  $\geq 11.1$  mmol/L. Individuals without diabetes symptoms should repeat the test on another day. Participants were divided into three groups based on the CAP value tertiles: low-level ( $\leq 231$ ,  $n = 2361$ ), moderate-level (232~269,  $n = 2330$ ) and high-level ( $\geq 270$ ,  $n = 2344$ ). Patients aged  $<55$  years were defined as young or middle-aged people, and those aged  $\geq 55$  years were defined as elderly people. A  $BMI \geq 24$   $\text{kg}/\text{m}^2$  was considered to indicate overweight status.<sup>27</sup>

## Statistical Analyses

The Kolmogorov–Smirnov method was used to test for a normal distribution, and the chi-square test was used to test for between-group differences. Normally distributed variables are expressed as  $\bar{x} \pm s$ , and nonnormally distributed variables are expressed as medians (interquartile ranges). When quantitative information was normally distributed and variance was homogeneous, analysis of variance (ANOVA) was used for between-group comparisons. When the data were not normally distributed, the Kruskal–Wallis  $H$ -test was used for comparisons between multiple groups. Categorical variables are expressed as a percentage (%). The chi-square test was used for comparison of categorical variables among the three groups. Using binary logistic regression analysis, three regression models were established: Model I: adjust for none. Model II: adjust for gender, age, BMI. Model III: adjust for Model II, LSM, ALP, UA, UREA, TG, HDL-C, LDL-C. Gradually adjust for potential confounding factors affecting T2DM to explore the relationship between CAP and the risk of developing T2DM. The predictive value of the CAP in patients with T2DM was assessed using receiver operating characteristic (ROC) curves. The data were analyzed using SPSS 26.0 (IBM Corp, Armonk, NY, USA) and R language 4.2.2 (R Foundation for Statistical Computing, Vienna, Austria) statistical software. A two-tailed value of  $P < 0.05$  was considered to indicate statistical significance.

## Results

### Baseline Characteristics of Participants

A total of 7035 patients were included in this study, including 4896 (69.6%) males and 2139 (30.4%) females, with a mean age of 45.12±12.03 years. In the overall population, 12.1% of the participants had T2DM, and the mean age of the patients with T2DM (53.18±11.87 years) was significantly greater than that of the non-T2DM patients (44.01±11.62 years) ( $P<0.001$ ). The enrolled patients were classified into CAP-tertile-based groups: low ( $\leq 231$ ,  $n = 2361$ ), moderate (232~269,  $n = 2330$ ) and high ( $\geq 270$ ,  $n = 2344$ ) (Table 1). There were significant between-group differences in sex, age, LSM, BMI, ALT concentrations, AST concentrations, DBIL concentrations, ALP concentrations, Cr concentrations, UA concentrations, UREA concentrations, TC concentrations, TG concentrations, HDL-C concentrations, LDL-C concentrations and FBG concentrations (all  $p < 0.05$ ), while there were no significant between-group differences in TBIL and IBIL concentrations (all  $p > 0.05$ ). The prevalence of T2DM in patients in the three groups was 9.7%, 11.2% and 15.4%, for the low-high CAP groups, respectively, and these differences were statistically significant ( $P<0.001$ ). Clinical and laboratory characteristics grouped by T2DM status are listed in Table 2. Compared with the non-T2DM group, T2DM patients were older and had higher CAP, LSM, BMI, ALT, ALP, TG, LDL-C, and FBG, and lower Cr, UA, and HDL-C (all  $P < 0.05$ ). The Mantel–Haenszel chi-square test showed a linear relationship between the CAP and the risk of developing T2DM ( $\chi^2 = 38.796$ ,  $p < 0.001$ ). Spearman correlation analysis revealed that as the CAP increased, the prevalence of T2DM tended to increase ( $r = 0.078$ ,  $P = 0.012$ ) (Figure 2).

**Table 1** Baseline Clinical Characteristics of Participants

| Variables               | Total<br>(n=7035) | Q1 ( $\leq 231$ )<br>(n=2361) | Q2 (232–269)<br>(n=2330) | Q3 ( $\geq 270$ )<br>(n=2344) | P-value |
|-------------------------|-------------------|-------------------------------|--------------------------|-------------------------------|---------|
| Sex, n(%)               |                   |                               |                          |                               | <0.001* |
| Female                  | 2139 (30.4)       | 1135 (48.1)                   | 621 (26.7)               | 383 (16.3)                    |         |
| Male                    | 4896 (69.6)       | 1226 (51.9)                   | 1709 (73.37)             | 1961 (83.7)                   |         |
| T2DM, n(%)              |                   |                               |                          |                               | <0.001* |
| NO                      | 6185 (87.9)       | 2132 (90.3)                   | 2070 (88.8)              | 1983 (84.6)                   |         |
| YES                     | 850 (12.1)        | 229 (9.7)                     | 260 (11.2)               | 361 (15.4)                    |         |
| Age, years              | 45.12±12.03       | 42.65±12.24                   | 46.39±11.75              | 46.36±11.71                   | <0.001* |
| LSM, kPa                | 5.3 (4.5,6.3)     | 4.8 (4.1,5.7)                 | 5.3 (4.5,6.1)            | 5.9 (5.1,6.9)                 | <0.001* |
| BMI, Kg/m <sup>2</sup>  | 24.6 (22.4,26.8)  | 22.1 (20.4,23.6)              | 24.5 (23.2,26.0)         | 27.2 (25.5,29.1)              | <0.001* |
| ALT, U/L                | 21 (15,31)        | 16 (12,23)                    | 21 (15,29)               | 28 (20,40)                    | <0.001* |
| AST, U/L                | 23 (20,28)        | 22 (19,26)                    | 23 (20,27)               | 25 (21,30)                    | <0.001* |
| TBIL, $\mu\text{mol/L}$ | 13.0 (10.4,16.4)  | 12.8 (10.4,16.5)              | 13.0 (10.3,16.3)         | 13.1 (10.5,16.4)              | 0.425   |
| DBIL, $\mu\text{mol/L}$ | 2.3 (1.8,2.9)     | 2.4 (1.9,3.0)                 | 2.3 (1.8,2.9)            | 2.3 (1.8,2.9)                 | 0.001*  |
| IBIL, $\mu\text{mol/L}$ | 10.6 (8.5,13.5)   | 10.4 (8.4,13.5)               | 10.7 (8.4,13.5)          | 10.8 (8.7,13.6)               | 0.116   |
| ALP, U/L                | 69 (58,83)        | 65 (54,78)                    | 70 (59,84)               | 73 (61,86)                    | <0.001* |
| Cr, $\mu\text{mol/L}$   | 81 (68,91)        | 75 (63,88)                    | 82 (70,92)               | 83 (72,92)                    | <0.001* |
| UA, $\mu\text{mol/L}$   | 387 (324,457)     | 342 (286,402)                 | 392 (333,454)            | 429 (367,498)                 | <0.001* |
| UREA, mmol/L            | 4.85 (4.14,5.69)  | 4.68 (3.96,5.52)              | 4.93 (4.21,5.80)         | 4.93 (4.26,5.73)              | <0.001* |
| TC, mmol/L              | 5.23 (4.61,5.94)  | 5.07 (4.48,5.72)              | 5.28 (4.642,5.96)        | 5.39 (4.73,6.11)              | <0.001* |
| TG, mmol/L              | 1.35 (0.94,2.03)  | 1.00 (0.76,1.42)              | 1.39 (0.98,1.96)         | 1.85 (1.29,2.66)              | <0.001* |
| HDL-C, mmol/L           | 1.31 (1.15,1.52)  | 1.44 (1.26,1.65)              | 1.31 (1.15,1.50)         | 1.21 (1.07,1.38)              | <0.001* |
| LDL-C, mmol/L           | 3.42 (2.92,3.95)  | 3.25 (2.74,3.76)              | 3.47 (2.98,3.99)         | 3.55 (3.06,4.07)              | <0.001* |
| FBG, mmol/L             | 5.08 (4.78, 5.47) | 4.92 (4.66, 5.22)             | 5.1 (4.79, 5.45)         | 5.26 (4.92,5.78)              | <0.001* |

**Notes:** Mean  $\pm$  standard deviation (SD) and Median (Inter Quartile Range) for continuous variables. Percentage (%) for categorical variables, \* $P<0.05$ .

**Abbreviations:** LSM, liver stiffness measurement; BMI, body mass index; ALT, alanine transaminase; AST, aspartate transaminase; TBIL; total bilirubin; DBIL, direct bilirubin; IBIL, indirect bilirubin; ALP, alkaline phosphatase; Cr, creatinine; UA, uric acid; UREA, urea nitrogen; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; FBG, fasting blood glucose.

**Table 2** Baseline Characteristics of Individuals with or Without T2DM

| Variables              | Total (n=7035)   | Non-T2DM (N=6185) | T2DM (N=850)      | P-value |
|------------------------|------------------|-------------------|-------------------|---------|
| Gender, n(%)           |                  |                   |                   | 0.777   |
| Female                 | 2139 (30.4)      | 1877 (30.3)       | 262 (30.8)        |         |
| Male                   | 4896 (69.6)      | 4308 (69.7)       | 588 (69.2)        |         |
| Age, years             | 45.12±12.03      | 44.01±11.62       | 53.18±11.87       | <0.001* |
| CAP, dB/m              | 250 (222,278)    | 249 (221, 277)    | 263 (229, 284)    | <0.001* |
| LSM, kPa               | 5.3 (4.5,6.3)    | 5.3 (4.5, 6.3)    | 5.6 (4.8, 6.6)    | <0.001* |
| BMI, Kg/m <sup>2</sup> | 24.6 (22.4,26.8) | 24.5 (22.3, 26.8) | 25.0 (23.0, 27.1) | <0.001* |
| ALT, U/L               | 21 (15,31)       | 21 (15, 31)       | 22 (16, 32)       | 0.013*  |
| AST, U/L               | 23 (20,28)       | 23 (20, 28)       | 23 (20, 28)       | 0.88    |
| TBIL, μmol/L           | 13.0 (10.4,16.4) | 12.9 (10.4, 16.4) | 13.0 (10.5, 16.5) | 0.487   |
| DBIL, μmol/L           | 2.3 (1.8,2.9)    | 2.3 (1.8, 2.9)    | 2.3 (1.9, 3.0)    | 0.104   |
| IBIL, μmol/L           | 10.6 (8.5,13.5)  | 10.6 (8.5, 13.5)  | 10.7 (8.6, 13.4)  | 0.527   |
| ALP, U/L               | 69 (58,83)       | 69 (57, 83)       | 71 (59, 86)       | 0.002*  |
| Cr, μmol/L             | 81 (68,91)       | 81 (68, 91)       | 78 (65, 90)       | <0.001* |
| UA, μmol/L             | 387 (324,457)    | 389 (325, 458)    | 376.50 (315, 449) | 0.013*  |
| UREA, mmol/L           | 4.85 (4.14,5.69) | 4.83 (4.11, 5.66) | 5.00 (4.32, 6.01) | <0.001* |
| TC, mmol/L             | 5.23 (4.61,5.94) | 5.23 (4.62, 5.93) | 5.25 (4.51, 6.01) | 0.922   |
| TG, mmol/L             | 1.35 (0.94,2.03) | 1.34 (0.93, 1.99) | 1.52 (1.01, 2.38) | <0.001* |
| HDL-C, mmol/L          | 1.31 (1.15,1.52) | 1.32 (1.15, 1.52) | 1.28 (1.10, 1.50) | <0.001* |
| LDL-C, mmol/L          | 3.42 (2.92,3.95) | 3.43 (2.94, 3.95) | 3.38 (2.78, 3.89) | 0.013*  |
| FBG, mmol/L            | 5.08 (4.78,5.47) | 5.04 (4.75, 5.38) | 5.81 (5.03, 7.56) | <0.001* |

**Notes:** Mean ± standard deviation (SD) and Median (Inter Quartile Range) for continuous variables. Percentage (%) for categorical variables.\*P<0.05.

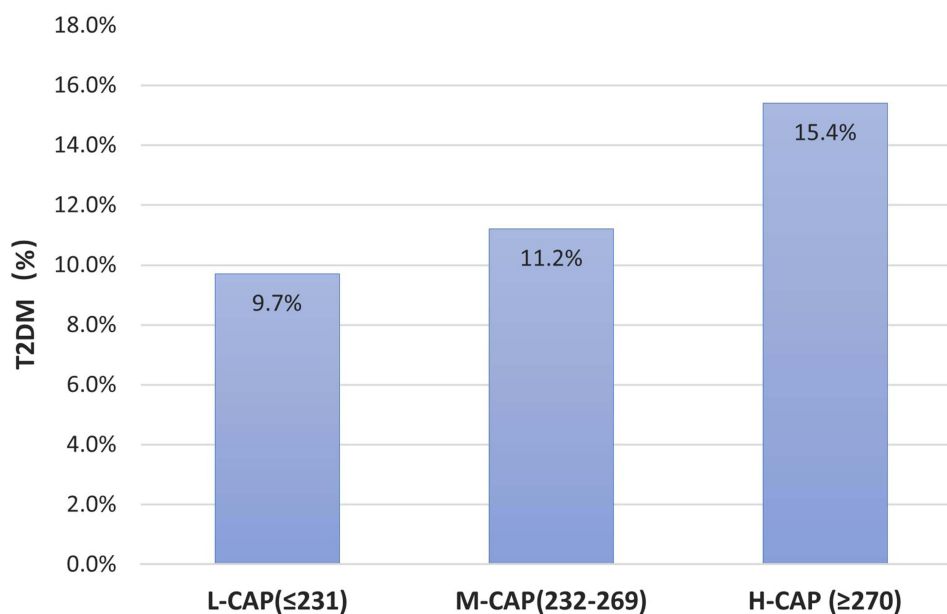
**Abbreviations:** CAP, controlled attenuation parameter; LSM, liver stiffness measurement; BMI, body mass index; ALT, alanine transaminase; AST, aspartate transaminase; TBIL, total bilirubin; DBIL, direct bilirubin; IBIL, indirect bilirubin; ALP, alkaline phosphatase; Cr, creatinine; UA, uric acid; UREA, urea nitrogen; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; FBG, fasting blood glucose.

## Analysis of Risk Factors Associated with the Occurrence of T2DM

To assess the relationship between the CAP and the risk of developing T2DM, logistic regression analysis was performed, and variables with  $P < 0.1$  in the one-way logistic regression analysis were included in the multifactorial logistic regression analysis to calculate the odds ratio (OR) and 95% confidence interval (CI) (Table 3). According to Model I (unadjusted), the risk of developing T2DM in the high-CAP group was 1.695 times greater than that in the low-CAP group (OR = 1.695; 95% CI = 1.421–2.022;  $p < 0.001$ ). Model II (Model I adjusted for sex, age and BMI) (OR = 1.630; 95% CI = 1.352–1.965;  $p = < 0.001$ ) and Model III (Model II adjusted for the LSM and ALP, UA, UREA, TG, HDL-C, LDL-C concentrations) (OR = 1.545; 95% CI = 1.263–1.890;  $p < 0.001$ ) also showed that the risk of developing T2DM in the high-CAP group was 1.630 and 1.545 times greater than that in the low-CAP group, respectively. The trend test was used to assess the risk of developing T2DM. Similarly, in Model I, the CAP was independently and positively associated with the risk of developing T2DM (OR = 1.311; 95% CI = 1.199–1.433;  $P$  for trend  $< 0.001$ ). Model II (OR = 1.293; 95% CI = 1.176–1.421;  $P$  for trend  $< 0.001$ ) and Model III (OR = 1.256; 95% CI = 1.135–1.391;  $P$  for trend  $< 0.001$ ) also indicated that the greater the CAP was, the greater the risk of developing T2DM. In addition, as the CAP level increased, regardless of the model used, a  $p$  value for trend  $< 0.05$  indicated that the higher the CAP level was, the better the prediction of the risk of developing T2DM. Therefore, the CAP is an important predictor of T2DM.

## Stratified Analyses via Subgroups

Stratified analyses of age, sex and BMI in the subgroups are shown in Table 4. Adjusted variables included sex, age, BMI, LSM, ALP, UA, UREA, TG, HDL-C and LDL-C. The results showed that in the sex stratification, the trend test was significant for both females and males. However, in the stratified analysis of age and BMI, the trend of developing T2DM was significant only for younger patients ( $< 55$  years) and patients with overweight (BMI  $\geq 24$  kg/m<sup>2</sup>). The greater the CAP was, the greater the risk of developing T2DM (OR  $> 1$ ,  $P$  for trend  $< 0.05$ ). In addition, for every 1.0 increase in



**Figure 2** Comparison of the prevalence of T2DM among the three groups. The prevalence of type 2 diabetes mellitus showed a dose-response relationship with the level of CAP, and the prevalence of T2DM with low, moderate and high levels of CAP was 9.7%, 11.2% and 15.4%, respectively ( $P$  for trend < 0.01). Correlation analysis showed that the prevalence of T2DM tended to increase with increasing levels of CAP ( $r = 0.078$ ,  $P = 0.012$ ).

CAP, there was a 0.852-fold increase in the risk of developing T2DM in people aged <55 years (OR = 1.852; 95% CI = 1.405–2.441;  $p < 0.001$ ) and a 0.6-fold increase in the risk of developing T2DM in patients with overweight (BMI  $\geq 24$  kg/m<sup>2</sup>) (OR = 1.600; 95% CI = 1.127–2.271;  $p = 0.009$ ) compared with the low-CAP group. Thus, stratified analyses clearly indicate that an elevated CAP is strongly associated with the risk of developing T2DM in young and middle-aged people with overweight.

### Predictive Value of the CAP in T2DM Patients

To further explore the predictive value of the CAP in patients with T2DM, ROC curve analysis was performed, as shown in Figure 3. The optimal CAP cutoff value for the prediction of developing T2DM patients was 246.5 dB/m, with an area under the curve of 0.569 (specificity of 48.2% and sensitivity of 64.2%). This indicates that CAP alone has moderate discriminatory ability for identifying high-risk populations for T2DM. In addition, as exhibited in Table 5, the study subjects were divided into two groups in accordance with CAP below or equal to and above the optimal cutoff value

**Table 3** Logistic Regression Analysis of the Relationship Between CAP and T2DM

|                      | Model I<br>OR (95% CI) | Model II<br>OR (95% CI) | Model III<br>OR (95% CI) |
|----------------------|------------------------|-------------------------|--------------------------|
| CAP ( $\leq 231$ )   | Reference              | Reference               | Reference                |
| CAP (232–269)        | 1.169 (0.969, 1.411)   | 1.071 (0.882, 1.301)    | 1.049 (0.861, 1.279)     |
| p-value              | 0.102                  | 0.486                   | 0.634                    |
| H-CAP ( $\geq 270$ ) | 1.695 (1.421, 2.022)   | 1.630 (1.352, 1.965)    | 1.545 (1.263, 1.890)     |
| p-value              | <0.001*                | <0.001*                 | <0.001*                  |
| CAP value            | 1.311 (1.199, 1.433)   | 1.293 (1.176, 1.421)    | 1.256 (1.135, 1.391)     |
| P for trend          | <0.001*                | <0.001*                 | <0.001*                  |

**Notes:** Model I: adjust for none. Model II: adjust for sex, age, BMI. Model III: adjust for Model II, LSM, ALP, UA, UREA, TG, HDL-C, LDL-C. \* $P < 0.05$ .

**Abbreviations:** BMI, body mass index; LSM, liver stiffness measurement; ALP, alkaline phosphatase; UA, uric acid; UREA, urea nitrogen; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

**Table 4** Stratification of CAP and T2DM Prevalence Risk by Sex, Age and Body Mass Index

| Variables                | Case/Total | Serum CAP Value                     |                                |                                     | CAP Value P for Trend |
|--------------------------|------------|-------------------------------------|--------------------------------|-------------------------------------|-----------------------|
|                          |            | L-CAP ( $\leq 231$ )<br>OR (95% CI) | M-CAP (232–269)<br>OR (95% CI) | H-CAP ( $\geq 270$ )<br>OR (95% CI) |                       |
| Sex                      |            |                                     |                                |                                     |                       |
| Female                   | 262/2139   | Reference                           | 1.247 (0.904,1.720)            | 1.677 (1.143,2.459)                 | 1.290 (1.067,1.560)   |
| p-value                  |            |                                     | 0.179                          | 0.008*                              | 0.009*                |
| Male                     | 588/4896   | Reference                           | 1.070 (0.832,1.377)            | 1.575 (1.228,2.020)                 | 1.284 (1.134,1.453)   |
| p-value                  |            |                                     | 0.598                          | <0.001*                             | <0.001*               |
| Age(years)               |            |                                     |                                |                                     |                       |
| <55                      | 460/5404   | Reference                           | 1.115 (0.853,1.458)            | 1.852 (1.405,2.441)                 | 1.378 (1.199,1.585)   |
| p-value                  |            |                                     | 0.424                          | <0.001*                             | <0.001*               |
| $\geq 55$                | 390/1631   | Reference                           | 0.913 (0.672,1.241)            | 1.122 (0.817,1.542)                 | 1.072 (0.913,1.257)   |
| p-value                  |            |                                     | 0.561                          | 0.477                               | 0.396                 |
| BMI (Kg/m <sup>2</sup> ) |            |                                     |                                |                                     |                       |
| <24                      | 308/2970   | Reference                           | 1.201 (0.908,1.590)            | 1.195 (0.695,2.054)                 | 1.142 (0.919,1.418)   |
| p-value                  |            |                                     | 0.200                          | 0.519                               | 0.230                 |
| $\geq 24$                | 542/4065   | Reference                           | 1.094 (0.760,1.574)            | 1.600 (1.127,2.271)                 | 1.330 (1.135,1.558)   |
| p-value                  |            |                                     | 0.629                          | 0.009*                              | <0.001*               |

**Notes:** Models are adjusted for sex, age, BMI, LSM, ALP, UA, UREA, TG, HDL-C, LDL-C.\* $P < 0.05$ .

**Abbreviations:** BMI, body mass index; LSM, liver stiffness measurement; ALP, alkaline phosphatase; UA, uric acid; UREA, urea nitrogen; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

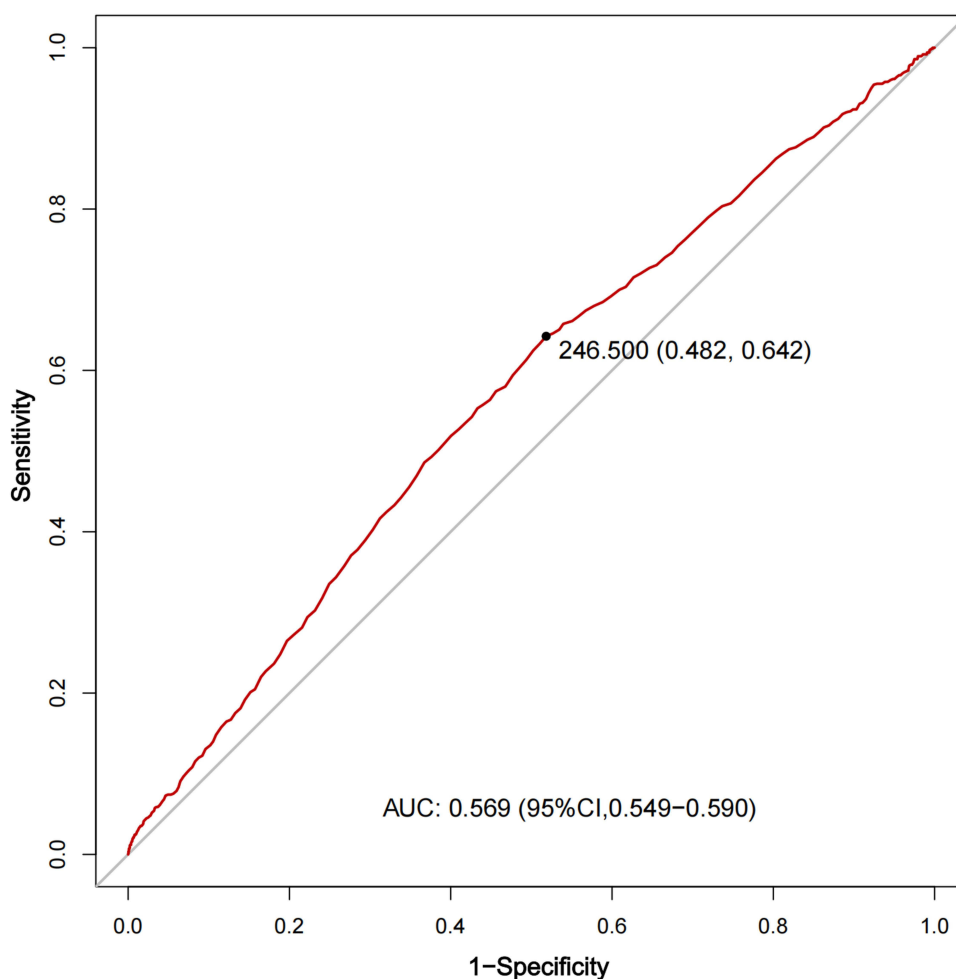
(246.5 dB/m), and after adjusting for sex, age, BMI, LSM, ALP, UA, UREA, TG, HDL-C and LDL-C, the logistic regression model showed that, compared with the low-value group, the high-value group (CAP  $\geq 246.5$  dB/m) had an increased risk of developing T2DM, which was 1.669-fold in Model I (unadjusted) (OR = 1.669; 95% CI = 1.438–1.937;  $p < 0.001$ ) and 1.594-fold in Model II (Model I adjusted for sex, age and BMI) (OR = 1.594; 95% CI = 1.364–1.863;  $p < 0.001$ ), and 1.550-fold in Model III (Model II adjusted for LSM, ALP, UA, UREA, TG, HDL-C and LDL-C) (OR = 1.550; 95% CI = 1.314–1.829;  $p < 0.001$ ).

## Discussion

T2DM is the most common endocrine disorder. Under the impact of population aging, industrialization, and urbanization, the prevalence of diabetes and prediabetes is on the rise worldwide. In addition to traditional risk factors (such as obesity, lack of exercise, and genetic predisposition), the association between NAFLD and T2DM has increasingly drawn attention in recent years. As the central organ for glucose and lipid metabolism, the accumulation of fat in the liver may lead to insulin resistance and impaired glucose metabolism, thereby promoting the onset and progression of T2DM.

The development of quantitative assessment techniques for hepatic steatosis has provided new tools for research in this field. Among these, CAP, as a derivative technology of the hepatic transient elastography device, has been validated for its efficacy in multiple studies.<sup>28,29</sup> Compared to liver biopsy, the gold standard, CAP offers advantages such as non-invasiveness, reproducibility, and ease of use, making it more suitable for large-scale population screening and long-term follow-up. Multiple studies have shown a positive correlation between CAP and the insulin resistance index (HOMA-IR) ( $r = 0.407$ – $0.568$ ),<sup>24,30</sup> but direct investigations into the relationship between CAP and the risk of T2DM remain limited. Therefore, this study aims to clarify the dose-response relationship between CAP and T2DM incidence, fill the epidemiological evidence gap between liver fat quantification indicators and diabetes risk, and explore the predictive efficacy heterogeneity of CAP across different subgroups, providing a basis for precise screening.

This study investigated the relationship between CAP and the risk of developing T2DM through a cross-sectional analysis. The results showed a significant dose-response relationship between CAP levels and the risk of developing T2DM. As CAP levels increased, the prevalence of T2DM showed a gradual upward trend (9.7%, 11.2%, and 15.4%, respectively;  $P$  for trend  $< 0.01$ ). This finding is consistent with previous studies and supports the notion that increased



**Figure 3** ROC curve analysis for predicting the optimal cutoff value of CAP for risk of T2DM. The area under the ROC curve was 0.569, the optimal cutoff value was 246.5 dB/m, the sensitivity was 64.2% and the specificity was 48.2%.

liver fat content is an independent risk factor for T2DM.<sup>31</sup> Stratified analysis results showed that the association between the high CAP group ( $\geq 270$  dB/m) and T2DM risk was significant in both women and men (ORs of 1.677 and 1.575, respectively), but the trend test P-value (0.009) was slightly higher in women than in men ( $<0.001$ ). The risk was significantly increased in the high CAP group among those aged  $<55$  years (OR=1.852,  $P<0.001$ ), while no significant association was observed in those aged  $\geq 55$  years (OR=1.122,  $P=0.477$ ), suggesting that the impact of hepatic fat accumulation on T2DM may be more pronounced in younger and middle-aged populations. Additionally, only in the population with a BMI  $\geq 24$  kg/m<sup>2</sup>, the high CAP group was significantly associated with T2DM risk (OR = 1.600,  $P = 0.009$ ), consistent with the synergistic effect of obesity itself as a risk factor for T2DM.<sup>32</sup> ROC curve analysis determined

**Table 5** Logistic Regression Based on the Optimal Cutoff Value of CAP

| CAP level    | T2DM, n  | Prevalence, % | Model I              | Model II             | Model III            |
|--------------|----------|---------------|----------------------|----------------------|----------------------|
|              |          |               | OR (95% CI)          | OR (95% CI)          | OR (95% CI)          |
| <246.5       | 304/3283 | 9.3           | Reference            | Reference            | Reference            |
| $\geq 246.5$ | 546/3752 | 14.6          | 1.669 (1.438, 1.937) | 1.594 (1.364, 1.863) | 1.550 (1.314, 1.829) |
| p-value      |          |               | $<0.001^*$           | $<0.001^*$           | $<0.001^*$           |

**Notes:** Models are adjusted for sex, age, BMI, LSM, ALP, UA, UREA, TG, HDL-C, LDL-C.\* $P<0.05$ .

**Abbreviations:** BMI, body mass index; LSM, liver stiffness measurement; ALP, alkaline phosphatase; UA, uric acid; UREA, urea nitrogen; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

the optimal cutoff value for CAP to be 246.5 dB/m (AUC = 0.569, sensitivity 64.2%). Although the AUC value indicates limited discriminative ability of CAP, the risk of T2DM was significantly increased in individuals with CAP  $\geq$  246.5 dB/m (OR = 1.550,  $P < 0.001$ ), suggesting that this cutoff value may have practical value in clinical screening.

The results of this study are consistent with those of several international studies. Guido et al<sup>33</sup> found in a study conducted in southern Italy that the incidence of hepatic steatosis was significantly higher in diabetic patients than in non-diabetic patients (89.7% vs 52%,  $P < 0.001$ ), and the mean CAP in the diabetic group was significantly higher than that in the control group ( $P < 0.001$ ), which is highly consistent with the results of this study. Additionally, Yoon et al<sup>34</sup> reported a dose-response relationship between CAP and metabolic syndrome similar to that observed in this study, noting that for every 10 dB/m increase in liver fat content, the risk of metabolic syndrome increased by approximately 9.3% (95% CI: 1.009–1.118), consistent with the trend analysis results of this study (OR = 1.256 for each unit increase in CAP). However, this study also identified some differences. For example, age-stratified analysis showed that the association between CAP and T2DM was not significant in individuals aged  $\geq 55$  years, which is inconsistent with the results of some studies<sup>35</sup> and may reflect the greater contribution of other metabolic factors (such as insulin resistance and  $\beta$ -cell dysfunction) to T2DM risk in the elderly population. Additionally, the AUC value in this study (0.569) was lower than those reported in some literature,<sup>36</sup> suggesting that CAP has limited efficacy as a single predictive indicator and may need to be combined with other clinical indicators (such as HbA1c, fasting blood glucose) to improve screening efficiency.

This study supports CAP as an adjunct tool for assessing T2DM risk, particularly in overweight or middle-aged and young populations where it may be of greater value. The non-invasive nature of CAP testing makes it suitable for large-scale screening, and the cutoff value of 246.5 dB/m can provide a reference for clinical decision-making. We adopted a standardized data collection method, and measurement and assessment of participants' indices in a single center while controlling for most of the potential confounders, which can improve the reliability of our findings. However, our study was conducted on a specific regional population, and its results may not be generalizable to other populations or environments. Future studies could address this issue by including a wider range of populations. As a cross-sectional study, this research can only establish associations and not causal relationships. Although the study controlled for several variables, potential confounding factors that were not included (such as diet and exercise) may have influenced the results.

## Conclusion

CAP exhibits a dose-response relationship with the risk of T2DM, particularly in overweight and younger populations. Although CAP has moderate discriminatory ability as a single predictive indicator, its non-invasive and convenient nature supports its role as an adjunct in metabolic risk assessment. Future studies should further validate the predictive value of CAP using longitudinal designs and explore its combined application with other biomarkers. If feasible, comparisons should also be made between the diabetes prevention outcomes of CAP-guided interventions and traditional methods.

## Ethics Approval and Consent to Participate

Our study complies with the Declaration of Helsinki, and the study was approved by the Medical Ethics Committee of the People's Hospital of Guangxi Zhuang Autonomous Region (Ethics-KY-IIT-2023-91). The informed consent requirement was exempted because of the retrospective study.

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## Disclosure

All authors report no conflicts of interest in this work.

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