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Original Article

Predictors of acute pancreatitis in patients treated with GLP-1 receptor agonists for weight management

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ABSTRACT

Introduction: Glucagon-like peptide-1 receptor agonists (GLP-1RA) are widely prescribed for treating obesity and Type 2 Diabetes (T2D). There have been concerns that GLP-1RA is associated with acute pancreatitis (AP), although the data are controversial. We aimed to identify factors that impact the risk of AP after initiation of GLP-1RA treatment for obesity.

Methods: We performed a retrospective case-control study including adults patients initiated on a GLP-1RA for obesity management to determine risk factors associated with AP in obese patients taking GLP-1RA. We identified patients initiated on GLP-1RA. A multivariable logistic regression model was used to identify predictors of AP with GLP-1RA use.

Results: There were 2245 patients, of which 49 (2.2 %) developed AP after starting a GLP-1RA. A history of gallstone disease (adjusted odds ratio (aOR), 2.9 [95 % CI, 1.6 to 5.3]), history of AP (aOR 4.8 [CI, 1.8 to 13.2]), CAD/PVD (aOR 2 [CI, 1.01 to 3.9]) and tobacco use (aOR 2.4 [CI, 1.2 to 4.7]) were associated with a higher risk of AP with GLP-1RA use. Compared to a BMI $\leq$30 kg/m^2, BMI categories 36–40 and > 40 were associated with a lower risk of AP with GLP-1RA therapy, (aOR 0.2 [CI, 0.06 to 0.6]) and (aOR 0.25 [CI, 0.09 to 0.68]), respectively.

Conclusion: A history of gallstone disease, history of AP, CAD/PVD, and tobacco use were associated with AP after initiation of GLP-1RA for obesity treatment. A higher BMI appears to be protective against AP. Recognizing factors associated AP after GLP-1RA initiation can inform clinicians on risk stratification.

1. Introduction

Glucagon-like-peptide-1 receptor agonists (GLP-1RA) are increasingly being prescribed to people with Type 2 Diabetes (T2D) and obesity. Acute pancreatitis (AP) can be a serious and life-threatening condition [1,2]. GLP-1RA work through multiple mechanisms to promote euglycemia and reduce energy intake [3]. They enhance glucose-dependent insulin secretion, slow gastric emptying, and reduce postprandial glucagon secretion [4]. While delayed gastric emptying contributes to some of the weight reduction seen in GLP-1RA therapy, these agents also have an inhibitory effect on the appetite centers of the brain that can decrease appetite and lead to early satiation [5]. Several large and well publicized studies have demonstrated clinically significant and sustained weight reduction with GP-1RA medications, which have an acceptable safety profile [6,7]. Treatment with GLP-1RA is also associated with favorable cardiovascular risk factor outcomes as studies have shown a decreased risk for cardiovascular events in people with T2D treated with GLP-1RA [8,9]. Based on this evidence, these medications have been adopted as safe and effective treatments for obesity in patients with and without T2D [10].

In many cases, GLP-1RA are preferred first line agents for these chronic diseases because of their impact on decreasing body weight, glycemia, and cardiovascular risk. An increased risk of AP has been observed since drugs impacting the GLP-1 pathway were

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Abbreviations: AP, Acute pancreatitis; BMI, body mass index; CKD, chronic kidney disease; GLP-1RA, Glucagon-like-peptide-1 receptor agonists; T2D, Type 2 Diabetes; CAD, coronary artery disease; PVD, peripheral vascular disease.

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developed. The use of exenatide (a GLP-1RA) or the DPP-4 inhibitor sitagliptin, which decreases breakdown of native GLP-1, was reported to increase the odds ratio of AP 6-fold compared with other T2D therapies [11]. This study was performed using the Food and Drug Administration (FDA) adverse event reporting database and showed an association without establishing a causation. Another population-based case-control study reported that the use of GLP-1-based therapies within 2 years was associated with a significantly increased odds ratio of being hospitalized with AP relative to non-GLP-1RA users [12]. However, not all studies have demonstrated the same association. In a meta-analysis of 41 trials, the overall risk of AP was not different between GLP-1RA and comparators (placebo, oral hypoglycemic agents, and/or insulin) [13]. This discrepancy could be due to heterogeneity in patient risk factors. In patients using GLP-1RA for the sole treatment of obesity, there is a paucity of data to identify which patients are most at-risk of developing AP. This study aims to identify risk factors for AP in obese patients taking GLP-1RA.

2. Methods

2.1. Study population

We performed an IRB-approved, retrospective, case-control study of patients seen at our institution's obesity medicine clinic between January 1, 2015 and 12/31/2021. We used the clinical research database in the electronic medical record (EMR) to identify participants who met our inclusion criteria. The inclusion criteria for this study were any adult (age ≥18 years) who was seen in the weight wellness clinic and started on a GLP-1RA for weight reduction. We divided the cohort into two groups: the acute pancreatitis group and the control group, depending on whether they developed AP after starting GLP-1RA therapy. Participants who developed AP after GLP-1RA initiation that was due to an obvious alternate etiology, such as gallstones, were excluded from the AP group. We then performed a comprehensive chart review to extract relevant factors including baseline characteristics (age, sex, weight, body mass index BMI), past medical history, surgical history, social history, laboratory results, and imaging. BMI was divided into the following categories: ≤30, 31–35, 36–40 and > 40.

2.2. Statistical analysis

Baseline characteristics were compared between the two groups using chi-square χ^2 for categorical variables and Student's *t*-test for continuous ones. A logistic regression model was fitted using a predetermined selection of clinically relevant risk factors for AP. To avoid over-fitting the model and given the limited number of events (AP after initiation of GLP-1RA), a propensity score was created. We first ran a multivariate logistic regression model using age, sex, gallstone disease, alcohol use, and history of AP. Subsequently, a propensity score was created using these factors and another model was then fitted using factors that were statistically significant in the univariate analysis in addition to the propensity score. A two-sided *P*-value of <0.05 was considered statistically significant. All statistical analyses were performed using STATA 14.2 (College Station, Texas, USA).

3. Results

3.1. Baseline Characteristics

The study sample included 2245 participants, 80.5 % women, mean age 49.5 (12.8) years, and BMI 39.7 (7.9) kg/m^2 at the time of a GLP-1RA initiation. Out of the total sample, 52 developed AP after initiation of GLP-1RA treatment. Three of the 52 participants with AP had gallstone pancreatitis and were excluded from the pancreatitis group. Therefore, a total of 49 subjects (2.2 %) developed AP after GLP-1RA initiation. There was no significant difference between the AP group and non-AP group in terms of age (51.3 (12.3) vs 49.2 (12.6), P = 0.81), sex (24.5 % men vs 19.4 % men, P = 0.36) and initial BMI (37.9 (9.1) vs 39.8 (7.9), P = 0.11). Baseline Characteristics are summarized in Table 1. The median time to pancreatitis after initiating GLP-1RA was 208 days (IQR: 105–338 days).

3.2. Factors associated with acute pancreatitis after GLP-1RA initiation

Cases with AP were more likely than controls to have T2D (67.3 % vs 45.6 %, $P = 0.003$), tobacco use (30.6 % vs 10.2 %, $P < 0.001$), and advanced (≥ stage 3) CKD (30.6 % vs 14.7 %, $P = 0.004$). On the univariate analysis, multiple factors were found to be associated with AP after initiation of GLP-1RA including T2D with an odds ratio (OR) of 2.5 (95 % CI, 1.3 to 4.5, $P = 0.003$), tobacco use (OR, 3.9 [95 % CI, 2.1 to 7.2], $P < 0.001$), advanced CKD (OR, 2.6 [95 % CI, 1.4 to 4.8], $P = 0.003$), coronary artery disease/peripheral vascular disease (CAD/PVD) (OR, 3.2 [95 % CI, 1.8 to 5.6], $P < 0.001$), gallstone disease (OR, 3.1 [95 % CI, 1.74 to 5.5], $P < 0.001$), past history of acute pancreatitis (OR, 6.6 [95 % CI, 2.5 to 17.7], $P < 0.001$) and depression (OR, 2.1 [95 % CI, 1.2 to 3.7], $P = 0.02$) (Table 2). BMI 36–40 kg/m^2 and BMI >40 kg/m^2 were associated with a lower risk of acute pancreatitis after initiation of GLP-1RA compared to BMI ≤30 (OR, 0.24 [95 % CI, 0.08 to 0.7], $P = 0.009$) and (OR, 0.34 [95 % CI, 0.13 to 0.9], $P = 0.020$), respectively.

On the adjusted multivariate logistic regression model (controlling for T2D, CAD/PVD, advanced CKD, depression, tobacco use, BMI category, history of acute pancreatitis and propensity score), the adjusted odds ratio (aOR) of gallstone disease was 2.9 ([95 % CI, 1.6 to 5.3], $P < 0.001$), history of pancreatitis (aOR, 4.8 [95 % CI, 1.8 to 13.2], $P = 0.002$), CAD/PVD (aOR, 2 [95 % CI, 1.01 to 3.9], $P = 0.048$) and tobacco use (aOR, 2.4 [95 % CI, 1.2 to 4.7], $P = 0.010$) (Table 3). Compared with a BMI category ≤30 kg/m^2, BMI categories 36–40 kg/m^2 and BMI >40 kg/m^2 were associated with a lower risk of AP with GLP-1RA therapy, (aOR, 0.2 [95 % CI, 0.06 to 0.6], $P = 0.004$) and (aOR, 0.25 [95 % CI, 0.09 to 0.68], $P = 0.007$), respectively. Individuals with BMI of 31–35 demonstrated a lower association with AP (aOR: 0.56; [95 % CI, 0.20–1.50], P = 0.25), although the association did not reach statistical significance. Other factors were also not significantly associated with the development of AP with GLP-1RA use.

4. Discussion

The use of GLP-1RA medications has witnessed an exponential surge in recent years due to the remarkable profile these drugs present in addressing the endemic problem of excess body weight, along with their effectiveness in managing a range of cardiometabolic and adiposity-associated complications. In this case-control, single-center study of 2245 patients who were treated with GLP-1RA for obesity, 49 people developed AP, of which 36 occurred within 1 year of starting therapy. The presence of gallstone disease, history of acute pancreatitis, history of CAD/PVD, and tobacco use were associated with developing AP after initiation of GLP-1RA therapy. In addition, a higher BMI category (BMI 36–40 and ≥ 40 kg/m^2) was associated with a lower risk of developing AP after GLP-1RA initiation, compared to a BMI category ≤30 kg/m^2, suggesting that higher BMI at the start of GLP-1RA therapy may be a protective factor. Notably, alcohol use and a

Table 1
Baseline characteristics comparing patients who developed acute pancreatitis to those who did not after initiating GLP-1RA.

Demographics	Pancreatitis After GLP-1RA, n = 49	No Pancreatitis after GLP-1RA (Control), n = 2196	*P* Value
Age at GLP-1RA initiation (years), mean (SD)	51.3 ± 12.3	49.2 ± 12.6	0.81
Weight at GLP-1RA initiation (lbs), mean (SD)	235.6 ± 69.6	243 ± 54.4	0.35
BMI at medication initiation (kg/m^2), mean (SD)	37.9 ± 9.1	39.8 ± 7.9	0.11
Sex, n (%)			0.36
Male	12 (24.5)	426 (19.4)	
Female	37 (75.5)	1770 (80.6)	
Ethnicity, n (%)			
Non-Hispanic	32 (65.3)	1737 (79.1)	0.50
Hispanic	17 (34.7)	368 (16.8)	
Declined	0 (0.0)	18 (0.8)	
Unknown	0 (0.0)	73 (3.3)	
Race (%)			0.68
White	27 (55.1)	1282 (58.3)	
Black	16 (32.7)	589 (26.8)	
Asian	1 (2.0)	43 (2.0)	
American Indian	1 (2.0)	7 (0.3)	
Other/unknown	4 (8.2)	254 (11.6)	
Hawaiian/Pacific Islander	0 (0.0)	1 (0.05)	
More than one race identified	0 (0.0)	20 (1.0)	

Table 2
Univariate analysis comparing risk factors for acute pancreatitis with GLP-1RA treatment.

	Pancreatitis After GLP-1RA, n = 49	No Pancreatitis after GLP-1RA (Control), n = 2196	OR (95 % CI)	*P* Value
Type 2 Diabetes, n (%)	33 (67.3)	1001 (45.6)	2.5 (1.3–4.5)	0.003
Hypertension, n (%)	37 (75.5)	1587 (72.3)	1.2 (0.6–2.3)	0.75
Hyperlipidemia, n (%)	44 (89.8)	1867 (85.0)	1.6 (0.6–3.9)	0.36
CAD/PVD, n (%)	24 (49.0)	510 (23.2)	3.2 (1.8–5.6)	<0.001
Advanced CKD (stage ≥3), n (%)	15 (30.6)	322 (14.7)	2.6 (1.4–4.8)	0.003
Autoimmune Disease, n (%)	10 (20.4)	379 (17.3)	1.2 (0.6–2.5)	0.57
Depression, n (%)	20 (40.8)	545 (24.8)	2.1 (1.2–3.7)	0.02
Eating Disorder, n (%)	12 (24.5)	532 (24.2)	1.0 (0.5–2.0)	0.96
Gallstone Disease, n (%)	27 (55.1)	625 (28.5)	3.1 (1.7–5.5)	<0.001
Thyroid Disease, n (%)	18 (36.7)	757 (34.5)	1.1 (0.6–2.0)	0.74
Prior AP, n (%)	5 (10.2)	37 (1.7)	6.6 (2.5–17.7)	<0.001
Liver Transplant Recipient, n (%)	0 (0.0)	19 (0.9)	–	>0.99
Alcohol Use Disorder, n (%)	3 (6.1)	118 (5.4)	1.2 (0.4–3.7)	0.82
Tobacco Use Disorder, n (%)	15 (30.6)	224 (10.2)	3.9 (2.1–7.2)	<0.001
Bariatric Surgery, n (%)	9 (18.4)	295 (13.4)	1.5 (0.7–3.0)	0.32
Sleeve Gastrectomy, n (%)	5 (10.2)	116 (5.3)	2 (0.8–5.2)	0.14
Roux-En-Y Gastric Bypass, n (%)	0 (0.0)	97 (4.4)	–	0.27
Cholecystectomy, n (%)	18 (36.7)	553 (25.2)	1.7 (1.0–3.1)	0.07

history of T2D were not associated with an increased risk of AP after GLP-1RA initiation in the multivariate analysis.

Several mechanisms have been proposed to explain the association between GLP-1RA use and AP, but none have been confirmed. GLP-1RA therapy is associated with hyperplasia of pancreatic acinar cells, which leads to a constitutive increase in circulating lipase levels, at or above the upper limit of normal, in around 44 % of people treated with GLP-1RA for weight management [14]. GLP-1RA may also cause a universal decrease in gut motility leading to sluggish flow of exocrine pancreatic fluid that increases the risk of pro-enzyme activation and autodigestion [15]. Another proposed explanation is that decreased biliary tract motility leads to biliary sludge formation and functional biliary pancreatitis [16]. Smoking is an established independent risk factor for AP and is known to increase exocrine pancreatic secretions and premature activation of zymogens [17], which likely compounded the risk of AP caused by the proposed changes in small duct architecture that GLP-1RA therapies may cause [18].

A population case-control study, which included patients treated with GLP-1RA for T2D reported an association between AP and hypertriglyceridemia, alcohol use, gallstones, tobacco use, obesity, biliary and pancreatic cancer, and any neoplasm in the unadjusted analysis [12]. Our findings align with the results from this study in terms of association with T2D, tobacco use and gallstone disease. However, our study found an interesting inverse association between BMI and GLP-1RA. Compared with a BMI category ≤30 kg/m^2,

Table 3
Multivariate analysis for risk factors for acute pancreatitis with GLP-1RA treatment.

	aOR (95 % CI)	*P* Value
Sex		
Male	Reference	
Female	0.7 (0.35–1.4)	0.30
Age	0.99 (0.97–1.02)	0.69
Gallstone disease	2.9 (1.6–5.3)	<0.001
Alcohol use disorder	1 (0.3–3.3)	0.99
History of pancreatitis	4.8 (1.8–13.2)	0.002
Type 2 Diabetes, n (%)	1.5 (0.8–2.9)	0.20
CAD/PVD, n (%)	2 (1.01–3.9)	0.048
Advanced CKD (stage ≥3), n (%)	1.2 (0.6–2.5)	0.56
Depression, n (%)	1.8 (0.97–3.3)	0.06
Tobacco use disorder, n (%)	2.4 (1.2–4.7)	0.01
BMI Categories (kg/m^2)		
≤30	Reference	–
31–35	0.56 (0.2–1.5)	0.25
36–40	0.2 (0.06–0.6)	0.004
>40	0.25 (0.09–0.68)	0.007

BMI categories 36–40 kg/m^2 and BMI >40 kg/m^2 were associated with a lower risk of AP with OR, 0.2 and 0.25, respectively. This intriguing finding challenges conventional expectations that higher BMI is associated with pancreatitis [19]. One might argue that patients with higher BMI may have had fewer conventional risk factors for AP compared to those with lower BMI. However, our multivariable model still showed a protective effect for low BMI after adjusting for these factors such as tobacco use and a propensity score, which included age, sex, gallstone disease, alcohol use, and prior history of pancreatitis. This unexpected observation suggests that GLP-1RA medications might have a unique influence on individuals with higher BMI. These medications enhance insulin sensitivity, promote weight loss, and have anti-inflammatory properties [20]. In individuals with higher BMIs, who may have more pronounced metabolic disturbances and inflammation, GLP-1RAs could exert a more substantial positive impact. By improving metabolic parameters and dampening inflammation, GLP-1RAs might counteract some of the risk factors associated with pancreatitis in this specific subset of patients.Further research is essential to unravel the complex interplay between GLP-1RA therapy, obesity, and pancreatitis risk.

Another noteworthy finding was the association between CAD and AP in patients initiating GLP-1RA. This relation likely reflects both inherent cardiovascular-pancreatic pathophysiology and medication specific mechanisms. CAD may independently increase the risk of AP through shared pathways like systemic inflammation, endothelial dysfunction and metabolic disturbances. Chronic low-grade inflammation in CAD may sensitize pancreatic tissue to injury. A Swedish population study found CAD increased AP risk (aOR 1.10) [21]. Our study found that CAD is an independent predictor of AP in GLP-1RA users (aOR 2.0), even after adjusting for traditional AP risk factors. This may represent synergistic effects: ie: CAD predisposes to chronic vascular inflammation which may be exacerbated by acinar cell hyperplasia with GLP-1RA use.

It is important to acknowledge some limitations of our study. An important limitation is the potential for residual confounders for possible risk factors inherently associated with AP despite our use of propensity score matching to mitigate this issue. While our analysis identified CAD, prior history of pancreatitis and tobacco use as potential risk factors for AP after initiating GLP-1RA for wight loss, we acknowledge that these conditions independently could increase the risk of AP. Therefore, it remains possible that the observed associations reflect amplification of pre-existing susceptibility rather than true effect modification. In fact, history of pancreatitis was the strongest risk for AP in our cohort which is well established risk factor for recurrent pancreatitis. In addition, our study utilized real-world clinical data that were extracted from the EMR, which may have included incomplete or variable documentation given the large number of patients included in the analysis. Diagnosis codes and other EMR sources were used to determine and classify certain variables (e.g., tobacco use and alcohol use), which did not provide quantitative data for a more detailed analysis. These diagnoses were present in the patient's electronic chart, however we do not know if they were actively using tobacco or alcohol at the time of GLP-1RA therapy or development of AP. Therefore, it is not possible to determine, for example, if adults with a greater cumulative exposure to tobacco use have a higher risk of developing AP with GLP-1RA therapy, which is a potential future area of research. Additionally, there was a low number of patients who developed AP after taking a GLP-1RA relative to the total number of participants (49 out of 2245). Given that lower sample size can lead to low statistical power and thus an inflated false discovery rate, future research should analyze data from large or national healthcare systems to provide a greater number of GLP-1 RA associated AP events. Researchers should also assess the risk of AP with different incretin therapies, especially as a new generation of combined incretin receptor analogs are being studied to tackle the obesity epidemic. Finally, our study did not include a comparison group of obese patients not treated with GLP-1RA, which limits our ability to determine whether GLP-1RA therapy itself increases the risk of pancreatitis. While we identified several risk factors associated with AP among GLP-1RA users, the absence of a non-GLP-1RA control group prevents any definite conclusion regarding the actual risk of AP or any direct attribution of increased risk to the medication itself.

In conclusion, our data showed that history gallstone disease, history of AP, CAD/PVD and tobacco use were associated with lower risk for AP in patients taking GLP-1RA for weight management. There was a lower association with AP after initiation of GLP-1RA with higher BMI. Most cases of AP occurred within one year of GLP-1RA initiation. Recognizing factors associated with AP after initiation of GLP-1RA for weight management can inform clinicians on risk stratification and symptom monitoring.

Author contributions

Study concept and design: AMA, JA and TS Acquisition of data: RP and AMA Statistical analysis: TS Data interpretation: RP, AMA, JA and TS, Drafting of the manuscript: RP, AMA, RA, JA and TS Critical revision of the manuscript for important intellectual content AMA, JA and TS.

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Robert Postlethwaite, Amin M Amin, Rand Alsawas and Tarek Sawas: There is no conflict of interest to declare.

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Declaration of competing interest

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