

Gliptin induced acute pancreatitis: a literature review

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Abstract

The association between dipeptidyl peptidase-4 (DPP-4) inhibitors, known as gliptins, and acute pancreatitis raises growing concern. A systematic search of PubMed, Scopus and Web of Science was conducted for studies published between January 2005 and July 2024. Studies focusing on gliptin-induced pancreatitis were selected based on predefined criteria. While gliptins are effective in managing type 2 diabetes mellitus, several case reports and observational studies suggest a potential risk for inducing acute pancreatitis. Proposed mechanisms include increased pancreatic activity and cellular stress. Clinical presentation often includes abdominal pain and elevated pancreatic enzymes, necessitating prompt diagnosis and discontinuation of the offending agent. Early recognition and management, including discontinuation of the drug and supportive care, are crucial. Balancing benefits and risks of gliptin therapy is essential for ensuring patient safety and optimal outcomes. This review underscores the importance of vigilance among healthcare providers and the need for further research to develop safer therapeutic strategies. (*Acta gastroenterol belg.*, 2025, 88, 151-155).

Keywords: Vildagliptin, acute pancreatitis, drug-induced pancreatitis, DPP-4 inhibitors, type 2 diabetes mellitus.

Introduction

The pancreas is a vital organ located in the abdomen, responsible for converting food into energy through its exocrine and endocrine functions (1). Acute pancreatitis (AP) is a common clinical condition that arises from an acute injury to the pancreas, typically leading to self-limiting inflammation of the pancreas (2). In recent years, the incidence of acute pancreatitis has increased in Europe and North America, while remaining consistent in Asia. This trend coincides with the global rise in diabetes, which affects 10.5% of adults aged 20 to 79 and is projected to impact 530 million people worldwide, leading to significant damage to the heart, blood vessels, eyes, kidneys, and nerves (3,4). Diabetes is a chronic metabolic disease marked by elevated blood glucose levels, leading to serious complications over time. Currently, 10.5% of adults globally are affected by diabetes, a number expected to rise to 530 million (5). With increasing diabetes prevalence, dipeptidyl peptidase-4 (DPP-4) inhibitors, or gliptins, have become popular due to their efficacy and safety.

Gliptins for type 2 diabetes inhibit DPP-4, preserving incretins and enhancing GLP-1's blood sugar-lowering effect (6,7). By inhibiting DPP-4, gliptins also enhance GLP-1's glucose-lowering effects. Clinical trials have shown that gliptins

improve HbA1c levels, β -cell function, and glucose levels when used alone or with other treatments (8). For individuals with type 2 diabetes, DPP-4 inhibitors can be prescribed as an alternative to thiazolidinedione medicine and as a second or third line of treatment following metformin and sulphonylureas (9).

The review will focus on the association between gliptins (dipeptidyl peptidase-4 inhibitors) and acute pancreatitis.

Methods

Search Strategy

A comprehensive literature search was conducted across multiple databases, including PubMed, Embase, Scopus and Web of Science, to identify relevant studies on gliptin-induced pancreatitis. The search terms included combinations of keywords such as “gliptin,” “DPP-4 inhibitors,” “pancreatitis,” “adverse effects,” and “drug-induced pancreatitis.” The search covered articles published between January 2005 and July 2024. Reference lists of relevant articles were also screened for additional studies.

Selection Criteria

Studies were included if they focused on gliptin-induced pancreatitis in adults and provided detailed clinical data. Case reports, case series, observational studies, and randomized controlled trials were considered. Reviews, editorials, and studies lacking specific diagnostic criteria for pancreatitis were excluded. Two independent reviewers conducted the initial screening of titles and abstracts, followed by a full-text review for eligibility. Disagreements were resolved through consensus or consultation with a third reviewer. A narrative synthesis was employed to summarize the findings, highlighting common patterns in clinical presentation and risk factors associated with gliptin-induced pancreatitis.

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Clinical and research consequence

It is essential to comprehend the clinical and research implications of pancreatitis resulting from gliptin in order to enhance patient outcomes and shape future investigations. Clinically, this information affects how medical professionals deal with and treat gliptin-prescribed patients, guiding early identification and timely measures of intervention to reduce the risk of severe pancreatitis.(10) From the perspective of science, figuring out the causes and risk factors for medication-induced pancreatitis can help focus future studies on safer diabetic therapies and further our knowledge of medication-related side effects.(11) The consequences of these findings for clinical practice, recommendations, and future directions for research are examined in this section.

Adverse effects and safety considerations of Gliptins (Dipeptidyl peptidase-4 inhibitors)

Gliptins, generally well-tolerated medications for type 2 diabetes, can be associated with various adverse drug reactions (ADRs). Among these, gastrointestinal disturbances such as nausea, abdominal discomfort, and diarrhea are the most commonly reported (12). However, serious but less frequent ADRs, including hypersensitivity reactions (e.g., angioedema and Stevens-Johnson syndrome) and an increased risk of acute pancreatitis, have also been documented (13, 14). Notably, based on an analysis of the FDA's Adverse Event Reporting System (FAERS), reports of pancreatitis are over six times more common in patients taking exenatide or sitagliptin compared to those on alternative type 2 diabetes therapies (15).

In addition to pancreatitis, the FDA has issued

warnings about severe and disabling joint pain associated with DPP-4 inhibitors like sitagliptin, saxagliptin, linagliptin, and alogliptin. While symptoms often resolve within a month of discontinuing the medication, some patients may experience recurrence upon re-initiation or switching to a different DPP-4 inhibitor (16). Other potential ADRs include headaches, musculoskeletal pain, and an elevated risk of heart failure, particularly with saxagliptin and alogliptin (17, 18).

Patients with renal impairment must exercise caution with gliptins, as dose adjustments are frequently necessary (12). Additionally, due to limited safety data, gliptins are not recommended for use in pregnant or breastfeeding women (19). Ultimately, to minimize potential ADRs and contraindications, healthcare professionals must carefully assess the risk-benefit ratio of gliptins for individual patients, taking into account comorbidities and concurrent medications.

A large observational study using FAERS found the reporting rate of pancreatitis in patients treated with DPP-4 inhibitors to be approximately 0.15 per 1,000 patient-years (20). This highlights the importance of post-marketing surveillance to detect and analyze ADRs that may not have been evident in pre-marketing clinical studies (21). Table 1 summarizes the estimated cases of gliptin-induced pancreatitis, incidence rates for individual gliptins, and other notable ADRs, including abdominal pain, enzyme elevation, and the risk of heart failure.

Pancreas and DPP-4 inhibitors: Risk factors and observational evidence

Incidence rate has grown to 34 cases per 100,000 people, although global AP mortality has decreased

Table 1. — Summary of Gliptin-Induced Pancreatitis: Reported Cases, Incidence Rates, and Notable Adverse Effects.

Gliptin Type	Reported Cases of Pancreatitis (Estimated)	Incidence Rate (per 1000 patient-years)	Notable Adverse Effects
Sitagliptin	~50-60 cases reported	0.1-0.2	Severe abdominal pain, enzyme elevation
Saxagliptin	~20-30 cases reported	0.1-0.15	Increased risk of heart failure, abdominal pain
Vildagliptin	~5-10 cases reported	0.03-0.05	Mild abdominal pain, nausea, potential enzyme elevation
Linagliptin	~5-10 cases reported	0.02-0.04	Nausea, abdominal discomfort
Alogliptin	~5 cases reported	0.01-0.03	Musculoskeletal pain, nausea

during the last ten years (22). Patients with diabetes have a higher chance of developing pancreatitis because of things like using some antidiabetic drugs (such as GLP-1 receptor agonists and DPP-4 inhibitors), (23) being obese, having high blood sugar, drinking alcohol, and having a history of pancreatitis. The occurrence of severe pancreatitis in obese patients can be attributed to the accumulation of excessive fat around the pancreas, which can lead to fat necrosis and the release of virulence factors. This process triggers an excessive inflammatory response, limits respiratory motion, and promotes the formation of infection foci due to necrosis (24). Gallstones, hypercalcemia, infections, and genetic predispositions are additional risk factors (25,26). Obesity increases the risk of cholesterol gallstones, gallstone-related complications, and cholecystectomy. Rapid weight loss through very low-calorie diets or bariatric surgery further elevates the risk of cholelithiasis, necessitating prophylactic treatment with oral ursodeoxycholic acid. Obesity, increasingly prevalent in children and adolescents, contributes to higher rates and severity of acute pancreatitis due to factors like supersaturated bile, crystal formation, rapid weight loss, and visceral obesity (27). Although drugs only make up around 5% of AP cases (28)—the majority of which are mild to moderate—some have been linked to fatalities or major consequences (29). For the early detection and prevention of pancreatitis in this population, it is imperative to identify these risk factors.

Cellular stress and inflammation: a consequence of Gliptin

The exact mechanisms behind gliptin-induced acute pancreatitis are unclear, but several theories exist. Gliptin inhibits DPP-4, increasing incretin levels like GLP-1,

which in turn boosts insulin release and reduces glucagon production, enhancing blood glucose control as shown in Figure 1 (30). This heightened pancreatic activity may cause cellular stress and inflammation, potentially leading to pancreatitis (31). Additionally, increased incretin activity from gliptin may raise ductal pressure and obstruct pancreatic outflow, damaging acinar cells and contributing to pancreatitis (32).

Clinical presentation and diagnosis

Patients with gliptin-induced pancreatitis typically present with acute abdominal pain, predominantly in the epigastric region, often radiating to the back. The symptoms that are characteristic of inflammatory processes include fever, nausea, and vomiting. (33) Elevated serum levels of pancreatic enzymes, such as lipase and amylase, are frequently found in laboratory tests, which indicate inflammation-induced pancreatic damage.(34)

Not all cases of acute pancreatitis will result in high lipase and amylase levels. For instance, in the case reported by Mungan et al., (35) a 61-year-old woman presented with significant abdominal pain but had normal lipase and amylase levels. Imaging confirmed pancreatitis, and her condition improved significantly upon discontinuation of vildagliptin. Another case involved a 62-year-old man with acute abdominal pain and normal pancreatic enzyme levels; imaging again confirmed pancreatitis, and he also improved after stopping vildagliptin. These cases highlight the necessity of considering gliptin-induced pancreatitis even in the absence of elevated enzyme levels.

Furthermore, there may be an increase in inflammatory markers such as white blood cell count (WBC) and

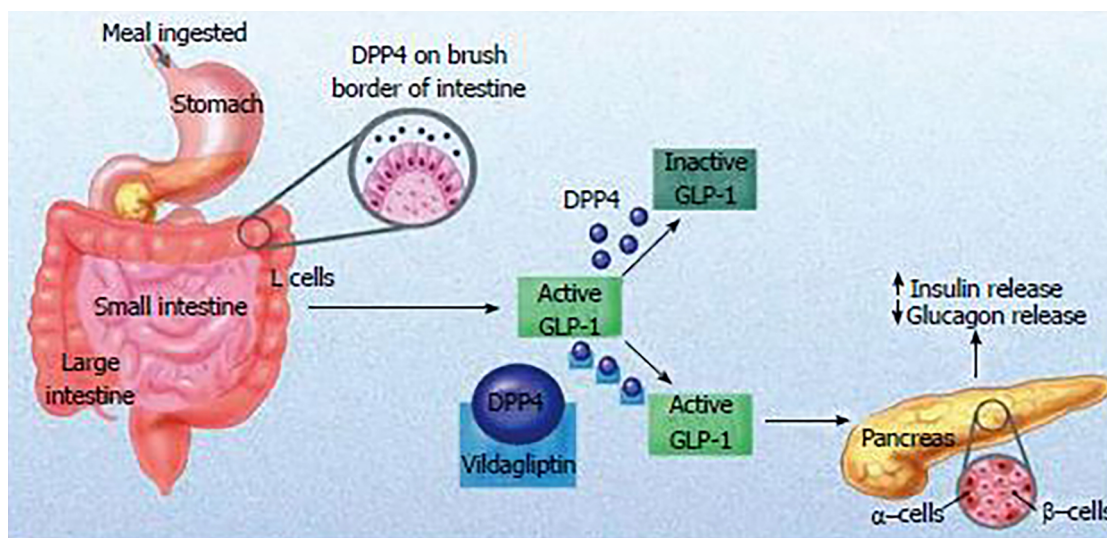


Figure 1.

C-reactive protein (CRP), which would suggest a systemic inflammatory response (33). An abdominal ultrasound can detect gallstones in the ducts, while a CT scan offers more detailed imaging of the pancreas when needed (36). Contrast-enhanced computed tomography (CECT) is the standard imaging tool for diagnosing acute pancreatitis and detecting complications like fluid collections, pseudocysts, abscesses, and thrombosis (37). These findings aid in diagnosis and severity assessment. Together, these results advance the clinical diagnosis and treatment of pancreatitis caused by gliptin.

Management gliptin-induced pancreatitis: effective strategies

Supportive therapy like fluid resuscitation, pain control, and monitoring for consequences such as organ failure are all part of immediate management. It also involves identifying the offending substance and stopping its use. Gliptin-induced pancreatitis must be diagnosed immediately, and stopping DPP-4 inhibitors (gliptins) right away is essential to ceasing more pancreatic damage and speeding up recovery (38). To maintain glycaemic control without aggravating pancreatitis, consideration of alternate antidiabetic medications such as insulin, sulfonylureas, or GLP-1 receptor agonists may be required. Regular evaluation of pancreatic function and imaging tests to identify any recurrence or long-term alterations are part of long-term surveillance. Maintaining communication with healthcare specialists guarantees proper management and prevention of issues (37).

Expert opinions and regulatory views regarding gliptin-induced pancreatitis

Regulatory agencies such as the FDA and EMA have issued warnings regarding the potential risk of pancreatitis associated with DPP-4 inhibitors. However, the exact risk and causative relationship remain debated and are subject to ongoing regulatory scrutiny and updates (38). Experts in the field hold varied opinions on the role of gliptins in pancreatitis. Some emphasize the need for cautious use and monitoring, particularly in patients with pre-existing pancreatic conditions or risk factors, while others argue that the benefits of gliptins outweigh the potential risks in most cases. (40) In a combined analysis of three large randomized trials (SAVOR-TIMI 53, EXAMINE, and TECOS), 18,238 patients receiving gliptin and 18,157 receiving a placebo were included in the study. The findings showed that gliptin-treated patients had a considerably greater incidence of AP when compared to control groups (41).

Prevention and risk mitigation

Preventing and mitigating the risk of pancreatitis in patients treated with gliptins (DPP-4 inhibitors) requires a comprehensive approach that includes patient selection, monitoring, and patient education. Conducting a thorough medical history and assessment helps identify patients at higher risk of pancreatitis, such as those with a history of pancreatitis, gallstones, alcohol use disorder, hypertriglyceridemia, and other relevant conditions (42). Evaluating comorbidities that may increase the risk of pancreatitis, such as obesity, hypercalcemia, and a family history of pancreatic diseases, is also essential (23). For patients at high risk, alternative antidiabetic medications, such as GLP-1 receptor agonists, may be more suitable (42). Educating patients about the potential risk of pancreatitis associated with gliptins and the importance of reporting symptoms such as severe abdominal pain, nausea, vomiting, and back pain immediately is vital (43). Advising patients on lifestyle modifications, including maintaining a healthy diet, avoiding excessive alcohol consumption, managing blood sugar levels effectively, and achieving a healthy weight, can also help reduce the risk (23).

Conclusion

In conclusion, while gliptins are effective agents in the management of type 2 diabetes, their association with acute pancreatitis necessitates vigilance among healthcare providers. The risk, although relatively low, is significant enough to warrant careful patient selection and monitoring. Early detection and management of gliptin-induced pancreatitis are crucial to prevent severe complications and ensure patient safety. Further research is needed to elucidate the precise mechanisms and risk factors involved, which will help in developing safer therapeutic strategies. Healthcare providers should be aware of the potential risks and educate patients accordingly, ensuring that the benefits of gliptin therapy are maximized while minimizing adverse outcomes.

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