

# World Journal of *Clinical Cases*

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## New-onset diabetes secondary to acute pancreatitis: An update

Xian-Qiang Yu, Qian Zhu

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### Abstract

Diabetes is a condition of persistent hyperglycemia caused by the endocrine disorder of the pancreas. Therefore, all pancreatic diseases have the risk of diabetes. In particular, increasing attention has been paid recently to new-onset diabetes secondary to acute pancreatitis (AP). The complications of secondary diabetes have caused a lot of trouble for patients and have garnered increasing attention. At present, the pathophysiological mechanism of new-onset diabetes caused by AP is not clear. This review summarizes the current understanding of new-onset diabetes secondary to AP.

**Key Words:** Acute pancreatitis; New-onset diabetes;  $\beta$ -cell; Hyperglycemia

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**Core Tip:** Increasing attention has been paid recently to new-onset diabetes secondary to acute pancreatitis (AP). The complications of secondary diabetes have caused a lot of trouble to patients and have garnered increasing attention. This review summarizes the current understanding of new-onset diabetes secondary to AP.

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## INTRODUCTION

At present, new-onset diabetes secondary to acute pancreatitis (AP) is considered to be the most common type of pancreatogenic diabetes[1-3]. Structural or functional disorders of blood glucose caused by pancreatogenic factors are the main pathophysiological mechanisms, including AP and chronic pancreatitis, pancreatic trauma, and surgery. The pancreas is the largest exocrine gland of the digestive tract. Although the volume of pancreatic islet B cells is very limited, the insulin secreted by the pancreas plays a key role in maintaining the stability of endocrine blood glucose[4]. In short, any cause of pancreatic damage can lead to diabetes. In recent years, reports on pancreatogenic diabetes have garnered increasing attention.

AP is a common acute abdomen and the number one cause of acute digestive system hospitalizations in the United States[5,6]. Most patients have mild AP and can recover and be discharged after 3 to 5 d of conservative treatment. However, about 20% of patients still develop severe AP, which leads to systemic inflammatory response syndrome and multiple organ dysfunction syndromes, leading to poor prognosis[7,8]. The risk factors for triggering pancreatic endocrine insufficiency in AP include age (> 45 years), obesity, hypertriglyceridemia, family history of diabetes, and recurrent pancreatitis[9]. But these factors do not affect the severity of endocrine function. In addition, some studies have shown that the severity of AP is not associated with the incidence of new-onset diabetes[10-13]. However, Chinese scholars suggest that pancreatic necrosis (PN) and persistent organ failure are risk factors for a high incidence of new-onset diabetes secondary to AP[14]. These results suggested that further studies should be conducted to determine the impact of PN on secondary diabetes. There have been few reports on the incidence of new-onset diabetes after pancreatitis, with one meta-analysis showing a prevalence of 23%[15]. Therefore, the current clinical understanding of the characteristics of new-onset diabetes secondary to AP is not exact.

## DIAGNOSTIC CRITERIA AND DEFINITIONS

The diagnostic criteria for pancreatogenic diabetes include: no previous history of diabetes, definite abnormalities of glucose metabolism caused by benign and malignant diseases of the pancreas, and the criteria for diabetes diagnosis. Diabetes including impaired glucose tolerance was defined according to the 1999 World Health Organization standard. The American Diabetes Association classifies it as type 3 diabetes[2]. Its main causes include AP, pancreatic cancer, and cystic fibrosis[16,17]. Among them, new-onset diabetes secondary to AP is caused by AP, which occurs based on impaired pancreatic exocrine function. In addition to the similar clinical manifestations, complications, and prognosis of type 2 diabetes, glucose fragility is an obvious clinical characteristic of this disease. Multiple episodes of hypoglycemia can further deteriorate pancreatic islet function and greatly increase the risk of pancreatic cancer[18-21]. Therefore, standardized and individualized treatment and management of pancreatic diabetes are more necessary.

Diabetes is diagnosed by typical diabetes symptoms with any of the following parameters (Tables 1 and 2).

## PATHOPHYSIOLOGY OF NEW-ONSET DIABETES SECONDARY TO AP

The main functions of the pancreas include exocrine and endocrine parts. The exocrine part consists of the acinar and duct, secreting pancreatic juice containing a large amount of bicarbonate and a variety of digestive enzymes, involved in the digestion of food. The endocrine part of the pancreas, namely the islet, is composed of A, B, D, and PP cells, which can secrete insulin, glucagon, somatostatin, and pancreatic polypeptide, respectively. The pancreas as a whole cannot be separated from its exocrine and endocrine functions. The exocrine and endocrine secretory parts of the pancreas interact and influence each other in pancreatic physiology and disease. AP is often accompanied by elevated blood glucose [22], which may be related to the following factors: (1) Under stress, insulin can reverse regulate the secretion of the hormone, while insulin secretion is relatively reduced, which leads to the enhancement of lipolysis and proteolysis, and the increase of liver glucose production; (2) Acute inflammation of the pancreas, pancreatic tissue swelling, ischemia, and microcirculation disorders affect the secretion and excretion of insulin, when a large number of pancreatic cells undergo necrosis in a short period time, which can lead to a serious shortage of endogenous insulin secretion; (3) Sympathetic nervous system excitatory catecholamine secretion increases, accelerates liver glycogen decomposition and inhibits pancreatic B cell secretion, increases blood glucose, and further aggravates endogenous insulin secretion deficiency; and (4) AP may be accompanied by insulin resistance. This high blood glucose state is AP glands, exocrine function in the performance of the different degree of damage, AP early hyperglycemia, and the correlation between the severity of AP has been recognized and valued. However, in the past, blood glucose metabolism disorder was considered a transient manifestation of the disease, so the monitoring and management of blood glucose after discharge did not receive enough attention.



**Table 1 Diagnosis of diabetes: Typical diabetes symptoms and any of the following**

Parameter	Value of number
FPG	$\geq 7.0$ mmol/L
Random blood glucose	$\geq 11.1$ mmol/L
OGTT	2hPG $> 11.1$ mmol/L after a 75-g OGTT
HbA1c	$\geq 6.5\%$ mmol/L

FPG: Fasting plasma glucose; HbA1c: Glycosylated hemoglobin; OGTT: Oral glucose tolerance test.

**Table 2 Diabetes can be diagnosed by any of the following parameters if without classical diabetes symptoms**

Parameter	Value of number
FPG	$> 7.0$ mmol/L for 2 times
OGTT	2hPG $\geq 11.1$ mmol/L for 2 times
IGT	FPG $< 7.0$ mmol/L and $7.8$ mmol/L $< 2$ hPG $< 11.1$ mmol/L after a 75-g OGTT
HbA1c	$\geq 6.5\%$ mmol/L

FPG: Fasting plasma glucose; HbA1c: Glycosylated hemoglobin; IGT: Impaired glucose tolerance; OGTT: Oral glucose tolerance test.

In AP, there is usually simultaneous pancreatic and exocrine dysfunction, and the disorder of blood glucose metabolism, as a common clinical manifestation of AP in the early stage, has gradually attracted attention. However, the pathophysiology of onset diabetes secondary to AP remains unclear[23]. But its occurrence may be related to some factors of AP, including islet cell damage associated with AP, pancreatic autoimmunity induced by AP, and insulin secretion disorder induced by the inflammatory response, *etc.* At present, basic and clinical studies on the pathogenesis of diabetes are still insufficient. Defining mechanisms is essential to guide clinical interventions.

It is important to emphasize that diabetes and hyperglycemia levels themselves can increase the severity of AP, mortality, and complications, and in turn increase the severity of diabetes[24,25]. However, higher body mass index and other factors are often closely associated with the development of diabetes[26]. Therefore, attention should be paid to the risk of new-onset diabetes in patients with AP caused by weight and other related indicators.

## MANAGEMENT OF NEW-ONSET DIABETES SECONDARY TO AP

Currently, there is no detailed standard for the management of new-onset diabetes secondary to AP. However, as a special type of diabetes, in addition to its general clinical manifestations, complications, and prognosis, blood glucose fragility is a significant clinical feature of new-onset diabetes secondary to AP. Such fluctuations in blood glucose can lead to dysfunction in the pancreas, further increasing the risk of pancreatic cancer[19,20]. Therefore, it is necessary to pay attention to the changes in blood glucose in time and select an individualized treatment plan.

Based on the current clinical data, the management of new-onset diabetes secondary to AP mainly includes prevention, screening, and treatment. From a prevention perspective, it is important to guide the population to avoid risk factors or lifestyles that contribute to AP and diabetes, such as timely control of obesity and hyperlipidemia. In terms of treatment, despite the lack of clinical trial evidence and relevant evidence-based guidelines, type 2 diabetes-based control strategies can still be used for new-onset diabetes secondary to AP. It is important to clarify the pathogenesis and inducement of diabetes secondary to AP for precise treatment. Regarding follow-up screening, given the potential risk of pancreatic cancer after new-onset diabetes secondary to AP, regular follow-up is necessary for standard assessment of pancreatic endocrine function.

## CONCLUSION

New-onset diabetes secondary to AP is increasingly recognized as a sequela of AP. The few studies to date show that the severity of AP does not indicate the risk of developing secondary diabetes. Further

elucidation of the risk factors and pathogenesis of new-onset diabetes secondary to AP will facilitate more effective early treatment. Early warning, screening, and follow-up findings will benefit new-onset diabetes secondary to AP patients. At the same time, worldwide evidence-based studies will help to enrich the in-depth understanding of the disease.

## FOOTNOTES

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