ISSN 2307-8960 (online)

# World Journal of *Clinical Cases*

*World J Clin Cases* 2022 October 26; 10(30): 10823-11213

Published by Baishideng Publishing Group Inc

World Journal of Clinical Cases

Contents

Thrice Monthly Volume 10 Number 30 October 26, 2022

## REVIEW



## MINIREVIEWS



## ORIGINAL ARTICLE

### Retrospective Cohort Study



### Retrospective Study



### Observational Study

*World Journal of Clinical Cases*

**Thrice Monthly Volume 10 Number 30 October 26, 2022**

## Contents

*World Journal of Clinical Cases*

**Thrice Monthly Volume 10 Number 30 October 26, 2022**

## Contents

*World Journal of Clinical Cases*

Thrice Monthly Volume 10 Number 30 October 26, 2022

## Contents

## ABOUT COVER

Editorial Board Member of *World Journal of Clinical Cases*, Rajeev Gurunath Redkar, FRCS, FRCS (Ed), FRCS (Gen Surg), MBBS, MCh, MS, Dean, Professor, Surgeon, Department of Pediatric Surgery, Lilavati Hospital and Research Centre, Mumbai 400050, Maharashtra, India. rajeev.redkar@gmail.com

## AIMS AND SCOPE

The primary aim of *World Journal of Clinical Cases* (*WJCC*, *World J Clin Cases*) is to provide scholars and readers from various fields of clinical medicine with a platform to publish high-quality clinical research articles and communicate their research findings online.

*WJCC* mainly publishes articles reporting research results and findings obtained in the field of clinical medicine and covering a wide range of topics, including case control studies, retrospective cohort studies, retrospective studies, clinical trials studies, observational studies, prospective studies, randomized controlled trials, randomized clinical trials, systematic reviews, meta-analysis, and case reports.

## INDEXING/ABSTRACTING

The *WJCC* is now abstracted and indexed in Science Citation Index Expanded (SCIE, also known as SciSearch®), Journal Citation Reports/Science Edition, Current Contents®/Clinical Medicine, PubMed, PubMed Central, Scopus, Reference Citation Analysis, China National Knowledge Infrastructure, China Science and Technology Journal Database, and Superstar Journals Database. The 2022 Edition of Journal Citation Reports® cites the 2021 impact factor (IF) for *WJCC* as 1.534; IF without journal self cites: 1.491; 5-year IF: 1.599; Journal Citation Indicator: 0.28; Ranking: 135 among 172 journals in medicine, general and internal; and Quartile category: Q4. The *WJCC*'s CiteScore for 2021 is 1.2 and Scopus CiteScore rank 2021: General Medicine is 443/826.

## RESPONSIBLE EDITORS FOR THIS ISSUE

**Production Editor:** *Ying-Yi Yuan*; **Production Department Director:** *Xu Guo*; **Editorial Office Director:** *Jin-Lei Wang*.

**NAME OF JOURNAL**
*World Journal of Clinical Cases*

**ISSN**
ISSN 2307-8960 (online)

**LAUNCH DATE**
April 16, 2013

**FREQUENCY**
Thrice Monthly



**PUBLICATION DATE**
October 26, 2022

World Journal of Clinical Cases

Submit a Manuscript: https://www.f6publishing.com

*World J Clin Cases* 2022 October 26; 10(30): 10862-10866

DOI: 10.12998/wjcc.v10.i30.10862

ISSN 2307-8960 (online)

MINIREVIEWS

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

Xian-Qiang Yu, Qian Zhu

**Specialty type:** Endocrinology and metabolism

**Provenance and peer review:** Invited article; Externally peer reviewed.

**Peer-review model:** Single blind

**Peer-review report's scientific quality classification**

Grade A (Excellent): 0
Grade B (Very good): B, B
Grade C (Good): C
Grade D (Fair): 0
Grade E (Poor): E

**P-Reviewer:** Balbaa ME, Egypt; Hegyi P, Hungary; Lee KS, South Korea; Petrov MS, Netherlands

**Received:** July 15, 2022
**Peer-review started:** July 15, 2022
**First decision:** August 20, 2022
**Revised:** August 27, 2022
**Accepted:** September 16, 2022
**Article in press:** September 16, 2022
**Published online:** October 26, 2022

**Xian-Qiang Yu,** Department of General Surgery, Women's and Children's Hospital Affiliated to Qingdao University, Qingdao 266034, Shandong Province, China

**Qian Zhu,** Department of Hepatobiliary and Pancreatic Surgery, Zhongnan Hospital of Wuhan University, Wuhan 430071, Hubei Province, China

**Corresponding author:** Xian-Qiang Yu, PhD, Doctor, Department of General Surgery, Women's and Children's Hospital affiliated to Qingdao University, No. 6 Tongfu Road, Shibei District, Qingdao 266034, Shandong Province, China. yuxianqiang302@126.com

## 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; β-cell; Hyperglycemia



**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.

**Citation:** Yu XQ, Zhu Q. New-onset diabetes secondary to acute pancreatitis: An update. *World J Clin Cases* 2022; 10(30): 10862-10866
**URL:** https://www.wjgnet.com/2307-8960/full/v10/i30/10862.htm
**DOI:** https://dx.doi.org/10.12998/wjcc.v10.i30.10862

## 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 APand 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 garneredincreasing 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 isdiagnosed 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 | ≥ 7.0 mmol/L |
| Random blood glucose | ≥ 11.1 mmol/L |
| OGTT | 2hPG > 11.1 mmol/L after a 75-g OGTT |
| HbA1c | ≥ 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 ≥ 11.1 mmol/L for 2 times |
| IGT | FPG < 7.0 mmol/L and 7.8 mmol/L < 2hPG < 11.1 mmol/L after a 75-g OGTT |
| HbA1c | ≥ 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

**Author contributions:** Yu XQ and Zhu Q completed the design and writing of the paper; Yu XQ participated in the overall design and revision of the paper.

**Conflict-of-interest statement:** All authors have no conflicts ofinterest to declare.



**Country/Territory of origin:** China

**ORCID number:** Xian-Qiang Yu 0000-0001-7879-9826; Qian Zhu 0000-0002-5656-8475.

**S-Editor:** Liu JH
**L-Editor:** Filipodia
**P-Editor:** Liu JH

## REFERENCES

1 **Hart PA**, Bellin MD, Andersen DK, Bradley D, Cruz-Monserrate Z, Forsmark CE, Goodarzi MO, Habtezion A, Korc M, Kudva YC, Pandol SJ, Yadav D, Chari ST; Consortium for the Study of Chronic Pancreatitis, Diabetes, and Pancreatic Cancer(CPDPC). Type 3c (pancreatogenic) diabetes mellitus secondary to chronic pancreatitis and pancreatic cancer. *Lancet Gastroenterol Hepatol* 2016; **1**: 226-237 [PMID: 28404095 DOI: 10.1016/S2468-1253(16)30106-6]

2 **American Diabetes Association**. 2. Classification and Diagnosis of Diabetes: *Standards of Medical Care in Diabetes-2020 . Diabetes Care* 2020; **43**: S14-S31 [PMID: 31862745 DOI: 10.2337/dc20-S002]

3 **Woodmansey C**, McGovern AP, McCullough KA, Whyte MB, Munro NM, Correa AC, Gatenby PAC, Jones SA, de Lusignan S. Incidence, Demographics, and Clinical Characteristics of Diabetes of the Exocrine Pancreas (Type 3c): A Retrospective Cohort Study. *Diabetes Care* 2017; **40**: 1486-1493 [PMID: 28860126 DOI: 10.2337/dc17-0542]

4 **Chen N**, Unnikrishnan I R, Anjana RM, Mohan V, Pitchumoni CS. The complex exocrine-endocrine relationship and secondary diabetes in exocrine pancreatic disorders. *J Clin Gastroenterol* 2011; **45**: 850-861 [PMID: 21897283 DOI: 10.1097/MCG.0b013e31822a2ae5]

5 **Peery AF**, Dellon ES, Lund J, Crockett SD, McGowan CE, Bulsiewicz WJ, Gangarosa LM, Thiny MT, Stizenberg K, Morgan DR, Ringel Y, Kim HP, DiBonaventura MD, Carroll CF, Allen JK, Cook SF, Sandler RS, Kappelman MD, Shaheen NJ. Burden of gastrointestinal disease in the United States: 2012 update. *Gastroenterology* 2012; **143**: 1179-1187.e3 [PMID: 22885331 DOI: 10.1053/j.gastro.2012.08.002]

6 **Fagenholz PJ**, Fernández-del Castillo C, Harris NS, Pelletier AJ, Camargo CA Jr. Direct medical costs of acute pancreatitis hospitalizations in the United States. *Pancreas* 2007; **35**: 302-307 [PMID: 18090234 DOI: 10.1097/MPA.0b013e3180cac24b]

7 **Forsmark CE**, Vege SS, Wilcox CM. Acute Pancreatitis. *N Engl J Med* 2016; **375**: 1972-1981 [PMID: 27959604 DOI: 10.1056/NEJMra1505202]

8 **Párniczky A**, Lantos T, Tóth EM, Szakács Z, Gódi S, Hágendorn R, Illés D, Koncz B, Márta K, Mikó A, Mosztbacher D, Németh BC, Pécsi D, Szabó A, Szücs Á, Varjú P, Szentesi A, Darvasi E, Erőss B, Izbéki F, Gajdán L, Halász A, Vincze Á, Szabó I, Pár G, Bajor J, Sarlós P, Czimmer J, Hamvas J, Takács T, Szepes Z, Czakó L, Varga M, Novák J, Bod B, Szepes A, Sümegi J, Papp M, Góg C, Török I, Huang W, Xia Q, Xue P, Li W, Chen W, Shirinskaya NV, Poluektov VL, Shirinskaya AV, Hegyi PJ, Bátovský M, Rodriguez-Oballe JA, Salas IM, Lopez-Diaz J, Dominguez-Munoz JE, Molero X, Pando E, Ruiz-Rebollo ML, Burgueño-Gómez B, Chang YT, Chang MC, Sud A, Moore D, Sutton R, Gougol A, Papachristou GI, Susak YM, Tiuliukin IO, Gomes AP, Oliveira MJ, Aparício DJ, Tantau M, Kurti F, Kovacheva-Slavova M, Stecher SS, Mayerle J, Poropat G, Das K, Marino MV, Capurso G, Małecka-Panas E, Zatorski H, Gasiorowska A, Fabisiak N, Ceranowicz P, Kuśnierz-Cabala B, Carvalho JR, Fernandes SR, Chang JH, Choi EK, Han J, Bertilsson S, Jumaa H, Sandblom G, Kacar S, Baltatzis M, Varabei AV, Yeshy V, Chooklin S, Kozachenko A, Veligotsky N, Hegyi P; Hungarian Pancreatic Study Group. Antibiotic therapy in acute pancreatitis: From global overuse to evidence based recommendations. *Pancreatology* 2019; **19**: 488-499 [PMID: 31068256 DOI: 10.1016/j.pan.2019.04.003]

9 **Das SL**, Singh PP, Phillips AR, Murphy R, Windsor JA, Petrov MS. Newly diagnosed diabetes mellitus after acute pancreatitis: a systematic review and meta-analysis. *Gut* 2014; **63**: 818-831 [PMID: 23929695 DOI: 10.1136/gutjnl-2013-305062]

10 **Shen HN**, Yang CC, Chang YH, Lu CL, Li CY. Risk of Diabetes Mellitus after First-Attack Acute Pancreatitis: A National

Population-Based Study. *Am J Gastroenterol* 2015; **110**: 1698-1706 [PMID: 26526084 DOI: 10.1038/ajg.2015.356]
11 **Vipperla K**, Papachristou GI, Slivka A, Whitcomb DC, Yadav D. Risk of New-Onset Diabetes Is Determined by Severity of Acute Pancreatitis. *Pancreas* 2016; **45**: e14-e15 [PMID: 26658044 DOI: 10.1097/MPA.0000000000000536]
12 **Uomo G**, Gallucci F, Madrid E, Miraglia S, Manes G, Rabitti PG. Pancreatic functional impairment following acute necrotizing pancreatitis: long-term outcome of a non-surgically treated series. *Dig Liver Dis* 2010; **42**: 149-152 [PMID: 19836318 DOI: 10.1016/j.dld.2009.08.007]
13 **Symersky T**, van Hoorn B, Masclee AA. The outcome of a long-term follow-up of pancreatic function after recovery from acute pancreatitis. *JOP* 2006; **7**: 447-453 [PMID: 16998241]
14 **Tu J**, Yang Y, Zhang J, Yang Q, Lu G, Li B, Tong Z, Ke L, Li W, Li J. Effect of the disease severity on the risk of developing new-onset diabetes after acute pancreatitis. *Medicine (Baltimore)* 2018; **97**: e10713 [PMID: 29851776 DOI: 10.1097/MD.0000000000010713]
15 **Xiao AY**, Tan ML, Plana MN, Yadav D, Zamora J, Petrov MS. The Use of International Classification of Diseases Codes to Identify Patients with Pancreatitis: A Systematic Review and Meta-analysis of Diagnostic Accuracy Studies. *Clin Transl Gastroenterol* 2018; **9**: 191 [PMID: 30287807 DOI: 10.1038/s41424-018-0060-1]
16 **Moran A**, Becker D, Casella SJ, Gottlieb PA, Kirkman MS, Marshall BC, Slovis B; CFRD Consensus Conference Committee. Epidemiology, pathophysiology, and prognostic implications of cystic fibrosis-related diabetes: a technical review. *Diabetes Care* 2010; **33**: 2677-2683 [PMID: 21115770 DOI: 10.2337/dc10-1279]
17 **Granados A**, Chan CL, Ode KL, Moheet A, Moran A, Holl R. Cystic fibrosis related diabetes: Pathophysiology, screening and diagnosis. *J Cyst Fibros* 2019; **18** Suppl 2: S3-S9 [PMID: 31679726 DOI: 10.1016/j.jcf.2019.08.016]
18 **Hardt PD**, Brendel MD, Kloer HU, Bretzel RG. Is pancreatic diabetes (type 3c diabetes) underdiagnosed and misdiagnosed? *Diabetes Care* 2008; **31** Suppl 2: S165-S169 [PMID: 18227480 DOI: 10.2337/dc08-s244]
19 **Kirkegård J**, Cronin-Fenton D, Heide-Jørgensen U, Mortensen FV. Acute Pancreatitis and Pancreatic Cancer Risk: A Nationwide Matched-Cohort Study in Denmark. *Gastroenterology* 2018; **154**: 1729-1736 [PMID: 29432727 DOI: 10.1053/j.gastro.2018.02.011]
20 **Cho J**, Scragg R, Petrov MS. Postpancreatitis Diabetes Confers Higher Risk for Pancreatic Cancer Than Type 2 Diabetes: Results From a Nationwide Cancer Registry. *Diabetes Care* 2020; **43**: 2106-2112 [PMID: 32616613 DOI: 10.2337/dc20-0207]
21 **Kirkegård J**, Mortensen FV, Heide-Jørgensen U, Cronin-Fenton D. Predictors of underlying pancreatic cancer in patients with acute pancreatitis: a Danish nationwide cohort study. *HPB (Oxford)* 2020; **22**: 553-562 [PMID: 31521499 DOI: 10.1016/j.hpb.2019.08.013]
22 **Czakó L**, Hegyi P, Rakonczay Z Jr, Wittmann T, Otsuki M. Interactions between the endocrine and exocrine pancreas and their clinical relevance. *Pancreatology* 2009; **9**: 351-359 [PMID: 19454837 DOI: 10.1159/000181169]
23 **Hart PA**, Bradley D, Conwell DL, Dungan K, Krishna SG, Wyne K, Bellin MD, Yadav D, Andersen DK, Serrano J, Papachristou GI. Diabetes following acute pancreatitis. *Lancet Gastroenterol Hepatol* 2021; **6**: 668-675 [PMID: 34089654 DOI: 10.1016/S2468-1253(21)00019-4]
24 **Nagy A**, Juhász MF, Görbe A, Váradi A, Izbéki F, Vincze Á, Sarlós P, Czimmer J, Szepes Z, Takács T, Papp M, Fehér E, Hamvas J, Kárász K, Török I, Stimac D, Poropat G, Ince AT, Erőss B, Márta K, Pécsi D, Illés D, Váncsa S, Földi M, Faluhelyi N, Farkas O, Nagy T, Kanizsai P, Márton Z, Szentesi A, Hegyi P, Párniczky A. Glucose levels show independent and dose-dependent association with worsening acute pancreatitis outcomes: Post-hoc analysis of a prospective, international cohort of 2250 acute pancreatitis cases. *Pancreatology* 2021; **21**: 1237-1246 [PMID: 34332908 DOI: 10.1016/j.pan.2021.06.003]
25 **Szentesi A**, Párniczky A, Vincze Á, Bajor J, Gódi S, Sarlós P, Gede N, Izbéki F, Halász A, Márta K, Dobszai D, Török I, Farkas H, Papp M, Varga M, Hamvas J, Novák J, Mickevicius A, Maldonado ER, Sallinen V, Illés D, Kui B, Erőss B, Czakó L, Takács T, Hegyi P. Multiple Hits in Acute Pancreatitis: Components of Metabolic Syndrome Synergize Each Other's Deteriorating Effects. *Front Physiol* 2019; **10**: 1202 [PMID: 31620021 DOI: 10.3389/fphys.2019.01202]
26 **Mikó A**, Farkas N, Garami A, Szabó I, Vincze Á, Veres G, Bajor J, Alizadeh H, Rakonczay Z Jr, Vigh É, Márta K, Kiss Z, Hegyi P, Czakó L. Preexisting Diabetes Elevates Risk of Local and Systemic Complications in Acute Pancreatitis: Systematic Review and Meta-analysis. *Pancreas* 2018; **47**: 917-923 [PMID: 30113426 DOI: 10.1097/MPA.0000000000001122]

Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
**Telephone:** +1-925-3991568
**E-mail:** bpgoffice@wjgnet.com
**Help Desk:** https://www.f6publishing.com/helpdesk
https://www.wjgnet.com