ISSN 2307-8960 (online)

World Journal of *Clinical Cases*

World J Clin Cases 2022 October 6; 10(28): 9970-10390

Published by Baishideng Publishing Group Inc

World Journal of Clinical Cases

Contents

Thrice Monthly Volume 10 Number 28 October 6, 2022

REVIEW

MINIREVIEWS

ORIGINAL ARTICLE

Clinical and Translational Research

Retrospective Study

World Journal of Clinical Cases

Thrice Monthly Volume 10 Number 28 October 6, 2022

Contents

World Journal of Clinical Cases

Thrice Monthly Volume 10 Number 28 October 6, 2022

Contents

World Journal of Clinical Cases

Thrice Monthly Volume 10 Number 28 October 6, 2022

Contents

LETTER TO THE EDITOR

World Journal of Clinical Cases

Thrice Monthly Volume 10 Number 28 October 6, 2022

Contents

ABOUT COVER

Editorial Board Member of *World Journal of Clinical Cases*, Kaleem Ullah, FCPS, MBBS, Assistant Professor, Solid Organ Transplantation and Hepatobiliary Surgery, Pir Abdul Qadir Shah Jeelani Institute of Medical Sciences, Gambat 66070, Sindh, Pakistan. drkaleempk@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: *Xu Guo*; **Production Department Director:** *Xiang Li*; **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

EDITORS-IN-CHIEF
Bao-Gan Peng, Jerzy Tadeusz Chudek, George Kontogeorgos, Maurizio Serati, Ja Hyeon Ku

EDITORIAL BOARD MEMBERS
https://www.wjgnet.com/2307-8960/editorialboard.htm

PUBLICATION DATE
October 6, 2022

INSTRUCTIONS TO AUTHORS
https://www.wjgnet.com/bpg/gerinfo/204

GUIDELINES FOR ETHICS DOCUMENTS
https://www.wjgnet.com/bpg/GerInfo/287

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH
https://www.wjgnet.com/bpg/gerinfo/240

PUBLICATION ETHICS
https://www.wjgnet.com/bpg/GerInfo/288

PUBLICATION MISCONDUCT
https://www.wjgnet.com/bpg/gerinfo/208

ARTICLE PROCESSING CHARGE
https://www.wjgnet.com/bpg/gerinfo/242

STEPS FOR SUBMITTING MANUSCRIPTS
https://www.wjgnet.com/bpg/GerInfo/239

ONLINE SUBMISSION
https://www.f6publishing.com

World Journal of Clinical Cases

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

World J Clin Cases 2022 October 6; 10(28): 10273-10278

DOI: 10.12998/wjcc.v10.i28.10273

ISSN 2307-8960 (online)

CASE REPORT

Double filtration plasmapheresis for pregnancy with hyperlipidemia in glycogen storage disease type Ia: A case report

Jie Wang, Yi Zhao, Pan Chang, Bin Liu, Rong Yao

Specialty type: Emergency medicine

Provenance and peer review: Unsolicited 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
Grade C (Good): C, C
Grade D (Fair): 0
Grade E (Poor): 0

P-Reviewer: Cavoretto PI, Italy; Rotondo JC, Italy; Tolunay HE, Turkey

Received: May 13, 2022
Peer-review started: May 13, 2022
First decision: June 16, 2022
Revised: July 4, 2022
Accepted: August 23, 2022
Article in press: August 23, 2022
Published online: October 6, 2022

Jie Wang, Yi Zhao, Pan Chang, Bin Liu, Department of Anesthesiology, West China Hospital of Sichuan University, Chengdu 610041, Sichuan Province, China

Rong Yao, Department of Emergency, West China Hospital of Sichuan University, Chengdu 610041, Sichuan Province, China

Corresponding author: Rong Yao, MD, Doctor, Department of Emergency, West China Hospital of Sichuan University, No. 37 Guoxue Road, Chengdu 610041, Sichuan Province, China. yaorong_hx@163.com

Abstract

BACKGROUND

Glycogen storage disease type Ia (GSDIa) is an autosomal recessive inborn error of carbohydrate metabolism that is caused by deficiency of the enzyme glucose-6-phosphatase (G6Pase), leading to disturbed glycogenolysis and gluconeogenesis. Patients with GSDIa show severe fasting hypoglycemia, hyperlipidemia, hyperlactacidemia, and hyperuricemia, which are associated with fatal outcomes in pregnant women and fetuses.

CASE SUMMARY

Herein, we report the case of a 24-year-old female who on her first visit to the hospital, presented with pregnancy combined with extremely high hyperlipidemia and hyperlactic acidosis with anemia, and frequent hypoglycemia occurred during the treatment. Genetic tests revealed a mutation in the G6Pase gene (*G6PC*) at 17q21, the patient was finally diagnosed with glycogen storage disease type Ia for the first time after 22 years of inaccurate treatment. She has been treated with a continuous double filtration plasmapheresis (DFPP) strategy to remove blood lipids, and a cornstarch diet therapy. The patient did not develop pancreatitis during the course of the disease and a healthy baby girl weighing 3 kg was delivered.

CONCLUSION

Patients with GSDIa may be misdiagnosed as epilepsy. DFPP can be used to control hyperlipidemia in GSDIa patients during pregnancy.

Key Words: Glycogen storage disease type Ia; Pregnancy; Hyperlipidemia; Double filtration plasmapheresis; Case report

Core Tip: Glycogen storage disease type Ia (GSDIa) is a glucose metabolic disorder caused by the deficiency of the enzyme glucose-6-phosphatase. The gold standard for diagnosis is genetic testing, and the main treatment is a corn starch diet. Specific risks are associated with GSDIa patients during pregnancy. Herein, we described a woman who suffered from GSDIa but had been misdiagnosed with recurrent seizures for 22 years. She developed anemia and extreme hyperlipidemia during pregnancy. After genetic testing and double filtration plasmapheresis lipid-lowering treatment, the patient was accurately diagnosed and eventually gave birth to a healthy baby.

Citation: Wang J, Zhao Y, Chang P, Liu B, Yao R. Double filtration plasmapheresis for pregnancy with hyperlipidemia in glycogen storage disease type Ia: A case report. *World J Clin Cases* 2022; 10(28): 10273-10278
URL: https://www.wjgnet.com/2307-8960/full/v10/i28/10273.htm
DOI: https://dx.doi.org/10.12998/wjcc.v10.i28.10273

INTRODUCTION

Glycogen storage disease type I (GSDI) is a glucose metabolic disorder caused by the deficiency of the enzyme G6Pase. The incidence of GSDI is approximately 1/100000, of which glycogen storage disease type Ia (GSDIa) accounts for about 80% of cases[1]. GSDIa patients usually experience severe hypoglycemia, hyperlactatemia, hyperlipidemia, hyperuricemia as well as growth and development retardation mainly because the last step of glycogenolysis and gluconeogenesis is affected. A rounded "doll-like" face is a typical feature of this disease due to the deposition of fat. Also in rare cases, repeated hypoglycemic seizures affect neurological development. If it remains untreated, complications from GSDIa can lead to a failure to thrive, an enlarged liver, abdominal swelling, and delayed motor development[2].

The chances of a successful pregnancy are still possible in patients with GSDIa, but specific risks are associated with GSDIa which mainly include prematurity, retarded growth, macrosomia, hypoglycemia, and fetal death[3-5]. These pregnancies need to be monitored closely because they are associated with a risk of worsening kidney problems and increased risk of bleeding, thereby requiring satisfactory metabolic control for the fetus. Among GSDIa mothers, most newborns are delivered by a cesarean section[2]. As more and more patients reach adult age, pregnancy is becoming an important problem among female patients with GSDIa.

In recent years, double filtration plasmapheresis (DFPP) has emerged as a new type of plasma exchange technology accompanied by the development of membrane separation technology[6]. DFPP is a semi-selective method where a filter separates the whole blood into cells and plasma. Then the plasma is allowed to pass through a second filter, which does not allow the passage of high-molecular-weight molecules including lipid-binding proteins. Thus using this technique, smaller proteins can be reinfused into the patient's bloodstream together with blood cells that were previously separated from the plasma.

In this report, we describe a case of a pregnant woman who presented with extreme hyperlipidemia and seizure and was diagnosed with GSDIa during treatment. She delivered a healthy baby at 37 wk of gestation.

CASE PRESENTATION

Chief complaints

A 24-year-old Chinese pregnant woman presented to the emergency department with complaints of anemia for 3 mo, gingival bleeding for 1 mo, and hematuria for 2 wk.

History of present illness

She was 24 wk pregnant and presented to the emergency department complaining of anemia for 3 mo, gingival bleeding for 1 mo, and hematuria for 2 wk.

History of past illness

Before pregnancy, she had a history of chronic seizures for 22 years that occurred about every 2-3 mo. The patient was on a long-term regimen of levetiracetam tablets (1000 mg per day) but stopped taking antiepileptic drug 1 d before admission.

Personal and family history

The patient had no family medical history of lipid disorders.

Physical examination

Although she had a normal physique (height: 1.58 m, weight: 61 kg; body mass index: 24.4 kg/m^2) with no impaired intelligence, she was anxious with a characteristic round "doll-like" face.

Laboratory examinations

The biochemistry of this patient was found to be abnormal as indicated by various biochemical tests (Table 1): hemoglobin: 67 g/L, extremely elevated triglyceride: 5483.5 mg/dL (normal value < 150 mg/dL), cholesterol: 836.6 mg/dL (normal value < 200 mg/dL), elevated lactic acid: 13.3 mmol/L (normal value 0.5-1.7 mmol/L), slightly elevated uric acid: 412 μmol/L (normal value 89-360 μmol/L), albumin: 33.6 g/L, qualitative urine protein: 0.3 (1+) g/L, urinary erythrocyte++++/haptoglobin, and elevated thyroid-stimulating hormone: 8.080 mU/L (the recommended value for pregnant women is less than 2.5 mmol/L). The patient's enzymes such as amylase and lipase were in the normal range.

Imaging examinations

Ultrasound examination showed abnormal enlargement of the liver where the maximum oblique diameter of the right lobe was found to be about 17.2 cm. Two stones smaller than 0.5 cm were found in the left kidney. The patient had been undergoing regular prenatal check-ups after pregnancy and the nuchal translucency thickness, as well as Down's syndrome screening, were normal. Obstetric ultrasound examination at 24 wk of gestation revealed a live single fetus in the uterus with a thickened and sail-shaped placenta, which needed to be closely monitored during the pregnancy.

Further diagnostic work-up

After eating, the patient's blood glucose returned to normal levels (6-9 mmol/L) after two episodes of intermittent fasting hypoglycemia (minimum 2.66 mmol/L). We highly suspected that she was suffering from GSDI. To confirm this diagnosis, we treated the patient with cornstarch diet therapy and performed genetic testing using samples of her blood and fingernails. The genetic test results showed that there was a gene c.648G>T mutation on chromosome 17q21, indicating that the patient had glucose-6-phosphatase (G6Pase) deficiency.

FINAL DIAGNOSIS

Based on her medical history and examination, the patient was diagnosed with GSDIa (OMIM232200), combined with 24 wk gestation, dyslipidemia, moderate anemia, hypoproteinemia, proteinuria, hematuria, epilepsy, hypothyroidism, and left hydronephrosis with kidney stones. This was the first time she had been diagnosed with GSDIa after 22 years of inaccurate treatment.

TREATMENT

The patient was treated with a DFPP strategy intended to rapidly lower the elevated blood lipid levels (Figure 1), followed by infusion of a red blood cell suspension to correct the anemia. Furthermore, the DFPP therapy was supplemented with thyroid hormone to control hypothyroidism. Levetiracetam tablets (1000 mg per day) were also added to her prescription to control a seizure. No further seizures were observed during the subsequent hospitalization period. Following DFPP therapy for 4 d, her triglycerides were reduced to 1288 mg/dL and then she was switched to oral fenofibrate for lipid-lowering. In Table 1, the biochemical data of the patient during treatment are shown. We treated the patient with cornstarch diet therapy after she developed hypoglycemia during hospitalization.

OUTCOME AND FOLLOW-UP

At 26 wk of gestation, she was discharged, cornstarch diet therapy and oral fenofibrate were continued, and her blood cholesterol levels were regularly monitored. At 37 wk + 5 d gestation, a healthy baby girl weighing 3 kg was delivered *via* cesarean section.

Table 1 Treatment/pregnancy course and biochemical data

	Day 1 (24 wk)	Day 2	Day 3	Day 4 (25 wk)	Day 5	Day 6	Day 8	Day 11	Day 14	Day 17	27 wk	29 wk	30 wk	32 wk	33 wk	34 wk	36 wk
		DFPP															
CHOL in mg/dL	837	645	558	348	339	296	322	324	308	289	293	324	339	433	340	304	251
TG in mg/dL	5484	2932	1352	1256	1269	1069	1727	1297	1297	1111	1263	1432	1476	2080	1532	953	1138
Uric acid in μmol/L	412	393	396	384	361	321	413	387	337	346	326	364	481	508	438	408	448
Creatinine in mg/dL	30	22	21	22	23	22	29	32	27	32	23	22	24	25	27	28	28
Lactate in mmol/L	13.8	10.1	12.8	12.1	11.3	9.3	8.2										
Glucose in mmol/L	6.11	6.71	5.90	5.86	6.0	4.14	2.66	2.83	3.69	3.54							
Hemoglobin in g/L	67	50	65	75	72	74	70	65	72	73	71	72	74	74	70	71	69
TSH in μmol/L		8.1					5.3	3.4	3.6	2.7							
Proteinuria	+	-					+/-		+								

Days 1-17 were the days of hospitalization; weeks 24-37 were the weeks of pregnancy. DFPP: Double filtration plasmapheresis; CHOL: Cholesterol; TG: Triglyceride; TSH: Thyroid-stimulating hormone.

DOI: 10.12998/wjcc.v10.i28.10273

Figure 1 Image of the patient undergoing double filtration plasmapheresis therapy. Red arrow indicates lipemic plasma sample during double filtration plasmapheresis therapy.

DISCUSSION

In this case, the patient presented with typical symptoms of GSDIa including hypoglycemia, hypertriglyceridemia, hyperlactatemia, hyperuricemia, and liver enlargement. This GSDIa patient,

however, was unusual. A large European study found that the median age at which GSDIa was diagnosed is 6 mo, with 80% diagnosed within < 1 year[7]. In this case, it is worth noting that the patient had never been diagnosed with GSDIa in her 24 years and she was largely untreated due to a lack of symptoms as well as poor compliance. Furthermore, she developed extreme hyperlipidemia (triglycerides > 1000 mg/dL) during pregnancy, with a triglyceride level of 5483.5 mg/dL that had never been reported for patients with GSDIa.

Hyperlipidemia among GSDIa patients usually results from both increased synthesis from an excess of acetyl-coenzyme A *via* malonyl-coenzyme A (the first step of fatty acid synthesis) and decreased lipid serum clearance[2]. For pregnant patients, the elevated estrogen levels enhance lipogenesis and suppress hepatic lipase activity, while elevated human placental lactogen causes insulin resistance leading to a decrease in lipoprotein lipase activity and increased lipolysis in adipose tissue[8], and these gestational changes may pose an additional pregnancy-related risk for GSDIa patients who are already predisposed to hyperlipidemia[9].

The metabolism of excessive triglycerides by pancreatic lipase leads to acute pancreatitis in about 15%-20% of women with severe hypertriglyceridemia during pregnancy[2]. During pregnancy, anti-inflammatory factors increase to counteract stresses/inflammation-like states occurring in pregnancy along with favoring maternal immune tolerance toward the fetus to result in a successful pregnancy outcome[10]. Hypertriglyceridemia-induced acute pancreatitis, however, is often accompanied by inflammogenesis during pregnancy and if inflammation persists, it can result in abortive events, preeclampsia, and placental abruption[11]. We carried out DFPP in this patient to rapidly lower the triglyceride levels and significantly reduce the risk of adverse events for maternal and infants.

Compared with traditional plasmapheresis, DFPP requires only 10%-15% of all plasma each time, which can be replaced with 4%-5% of albumin when the plasma supply is insufficient. A study by Lu *et al*[12] found that patients with hypertriglyceridemic pancreatitis who received DFPP early had a rapid and efficient reduction in triglyceride levels compared to those without DFPP. DFPP not only effectively and accurately reduces serum triglyceride levels but also reduces allogenic plasma infusion, thereby minimizing blood transfusion-related complications, making it an optimal method of choice for this patient to achieve lipid removal rapidly.

Seizure is a relatively rare symptom among GSDIa patients. Cerebral tissue is generally found to be highly sensitive to energy metabolism, and severe hypoglycemia can lead to hypoglycemic encephalopathy. Imad *et al*[13] study found that generalized seizures might relate to a glucose level of < 2.0 mmol/L. Severe hypoglycemia that starts during infancy may have been the primary cause of her seizures instead of structural abnormalities of the brain, and her seizures masked her actual diagnosis of GSDIa. This might explain why she continues to have seizures despite taking levetiracetam to stabilize her condition.

GSDIa occurs in an autosomal recessive pattern. Both parents of an affected child are heterozygotes. In each pregnancy, there is a 25% chance of recurrence[2]. It may be possible that her infant is a genetic carrier. Identification of the human G6Pase gene (*G6PC*) mutation in the patient enables the diagnosis of potential heterozygotes in the family.

CONCLUSION

In summary, we described a case of a woman who was having GSDIa but had been misdiagnosed as a patient with a recurrent seizure only for 22 years. During her pregnancy, she developed anemia and extremely high hyperlipidemia. After DFPP lipid-lowering treatment and genetic testing, the patient was accurately diagnosed and eventually gave birth to a healthy baby.

FOOTNOTES

Author contributions: Wang J analyzed the data and wrote the manuscript; Zhao Y and Chang P assisted in the collection of patient's data; Liu B and Yao R supervised the writing of this article; All authors have read and approve the final manuscript.

Informed consent statement: Informed written consent was obtained from the patient for publication of this report and any accompanying images.

Conflict-of-interest statement: The authors have no conflicts of interest to declare.

CARE Checklist (2016) statement: The authors have read the CARE Checklist (2016), and the manuscript was prepared and revised according to the CARE Checklist (2016).

Country/Territory of origin: China

ORCID number: Jie Wang 0000-0002-7141-6973; Yi Zhao 0000-0002-0696-3655; Pan Chang 0000-0003-3333-1861; Bin Liu 0000-0001-5503-0053; Rong Yao 0000-0001-9086-255X.

S-Editor: Zhang H
L-Editor: Filipodia
P-Editor: Zhang H

REFERENCES

1 **Ellingwood SS**, Cheng A. Biochemical and clinical aspects of glycogen storage diseases. *J Endocrinol* 2018; **238**: R131-R141 [PMID: 29875163 DOI: 10.1530/JOE-18-0120]
2 **Froissart R**, Piraud M, Boudjemline AM, Vianey-Saban C, Petit F, Hubert-Buron A, Eberschweiler PT, Gajdos V, Labrune P. Glucose-6-phosphatase deficiency. *Orphanet J Rare Dis* 2011; **6**: 27 [PMID: 21599942 DOI: 10.1186/1750-1172-6-27]
3 **Martens DH**, Rake JP, Schwarz M, Ullrich K, Weinstein DA, Merkel M, Sauer PJ, Smit GP. Pregnancies in glycogen storage disease type Ia. *Am J Obstet Gynecol* 2008; **198**: 646.e1-646.e7 [PMID: 18241814 DOI: 10.1016/j.ajog.2007.11.050]
4 **Ryan IP**, Havel RJ, Laros RK Jr. Three consecutive pregnancies in a patient with glycogen storage disease type IA (von Gierke's disease). *Am J Obstet Gynecol* 1994; **170**: 1687-90; discussion 1690 [PMID: 8203427 DOI: 10.1016/s0002-9378(12)91836-0]
5 **Yamamoto T**, Suzuki Y, Kaneko S, Hattori Y, Obayashi S, Suzumori N, Sugiura M. Glycogen storage disease type Ia (GSD Ia) during pregnancy: report of a case complicated by fetal growth restriction and preeclampsia. *J Obstet Gynaecol Res* 2010; **36**: 1125-1129 [PMID: 21058447 DOI: 10.1111/j.1447-0756.2010.01287.x]
6 **Grupp C**, Beckermann J, Köster E, Zewinger S, Knittel M, Walek T, Hohenstein B, Jaeger B, Spitthöver R, Klingel R, Fassbender CM, Tyczynski B. Relapsing and Progressive Complications of Severe Hypertriglyceridemia: Effective Long-Term Treatment with Double Filtration Plasmapheresis. *Blood Purif* 2020; **49**: 457-467 [PMID: 32191938 DOI: 10.1159/000506506]
7 **Rake JP**, Visser G, Labrune P, Leonard JV, Ullrich K, Smit GP. Glycogen storage disease type I: diagnosis, management, clinical course and outcome. Results of the European Study on Glycogen Storage Disease Type I (ESGSD I). *Eur J Pediatr* 2002; **161** Suppl 1: S20-34 [PMID: 12373567 DOI: 10.1007/s00431-002-0999-4]
8 **Chyzhyk V**, Kozmic S, Brown AS, Hudgins LC, Starc TJ, Davila AD, Blevins TC, Diffenderfer MR, He L, Geller AS, Rush C, Hegele RA, Schaefer EJ. Extreme hypertriglyceridemia: Genetic diversity, pancreatitis, pregnancy, and prevalence. *J Clin Lipidol* 2019; **13**: 89-99 [PMID: 30352774 DOI: 10.1016/j.jacl.2018.09.007]
9 **Carvalho PM**, Silva NJ, Dias PG, Porto JF, Santos LC, Costa JM. Glycogen Storage Disease type 1a - a secondary cause for hyperlipidemia: report of five cases. *J Diabetes Metab Disord* 2013; **12**: 25 [PMID: 23738826 DOI: 10.1186/2251-6581-12-25]
10 **Rotondo JC**, Oton-Gonzalez L, Selvatici R, Rizzo P, Pavasini R, Campo GC, Lanzillotti C, Mazziotta C, De Mattei M, Tognon M, Martini F. SERPINA1 Gene Promoter Is Differentially Methylated in Peripheral Blood Mononuclear Cells of Pregnant Women. *Front Cell Dev Biol* 2020; **8**: 550543 [PMID: 33015055 DOI: 10.3389/fcell.2020.550543]
11 **de Oliveira C**, Khatua B, Noel P, Kostenko S, Bag A, Balakrishnan B, Patel KS, Guerra AA, Martinez MN, Trivedi S, McCullough A, Lam-Himlin DM, Navina S, Faigel DO, Fukami N, Pannala R, Phillips AE, Papachristou GI, Kershaw EE, Lowe ME, Singh VP. Pancreatic triglyceride lipase mediates lipotoxic systemic inflammation. *J Clin Invest* 2020; **130**: 1931-1947 [PMID: 31917686 DOI: 10.1172/JCI132767]
12 **Lu Z**, Chen Y, Wu Y, Lin Y, Yang N, Wang X, Guo F. The role of double filtration plasmapheresis in hypertriglyceridemic pancreatitis: A propensity score matching analysis. *J Clin Apher* 2020; **35**: 388-397 [PMID: 32629547 DOI: 10.1002/jca.21811]
13 **Imad H**, Zelano J, Kumlien E. Hypoglycemia and risk of seizures: a retrospective cross-sectional study. *Seizure* 2015; **25**: 147-149 [PMID: 25455725 DOI: 10.1016/j.seizure.2014.10.005]

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