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Prevalence, morbidity and mortality of hypertriglyceridemic acute pancreatitis in the General Hospital of Playa del Carmen, México. Analysis of five years

Argel de Jesús Concha May[1] and Guillermo Padrón Arredondo[2]

[1]Internal medical undergraduate, Surgery Service of Hospital General Playa del Carmen, México
[2]General Surgeon, Surgery Service of Hospital General Playa del Carmen, México

***Correspondig Author:** Guillermo Padrón Arredondo General Surgeon, Surgery Service of Hospital General Playa del Carmen, Constituyentes Avenue with Avenue 135 Ejido Suburb, Playa del Carmen, Solidaridad, Quintana Roo, CP. 77712, México, Tel: 01-984-206-1691; Email: gpadronarredondo@hotmail.com; hospitalplay@hotmail.com

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Abstract

Introduction: Hypertriglyceridemia (HTG) is the underlying cause of pancreatitis in 7% of the general population and is the third cause after gallstones and alcohol. HTG may be associated with acute pancreatitis as an epiphenomenon or as a precipitant thereof. Generally, more than 75% of pancreatitis induced by hypertriglyceridemia is due to secondary causes and although these are not sufficient to elevate triglycerides to cause pancreatitis, a preexisting defect is required to obtain a TG>1000 mg/dL to induce acute pancreatitis.

Material and Method: To identify the prevalence morbidity and mortality of acute pancreatitis due to hypertriglyceridemia, a retrospective and cross-sectional observational clinical study was performed for a period of five years.

Results: During the study period, 100 cases of acute pancreatitis of various etiologies were collected, 29 (29%) of which corresponded to acute pancreatitis of hypertriglyceridemic origin; history of risk: type 1 Diabetes mellitus one case (3.4%); Type 2 Diabetes mellitus 27 cases (24%); history of alcoholism nine cases (31%); positive smoking 4 cases (13.8%); hypertriglyceridemia 27 cases (94%); obesity 17 cases (59%); lipemic serum 19 cases (65.5%), and In-hospital stays average six days. Mortality in one case.

Discussion: In the Mexican national survey of ENASUT 2012, it was found by age group that hypercholesterolemia is highest in the age groups of 50-69 years of age. (Table 1) The frequency of hyperlipidemia in patients with pancreatitis ranges from 12 to 38%, and of hypertriglyceridemia, between 4 and 53%; what is important is to define whether its presence is primary or causal, or secondary or consequence of other clinical conditions such as Diabetes mellitus, alcohol abuse, pregnancy or use of medications. The triglycerides were obtained on routine laboratory tests in our hospital and their values were steadily elevated and the hipertrigliceridemic acute pancreatitis is the principal cause instead the alcoholic acute pancreatitis.

Keywords: Pancreatitis acute; Hypertriglyceridemic; Prevalence; Morbidity; Mortality

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Introduction

An association between lipid and pancreatic metabolism was observed many years ago. The appearance of milky serum during an acute pancreatitis (AP) attack was first recognized by Speck in 1846 [1]. Hypertriglyceridemia (HTG) is the underlying cause of pancreatitis in 7% of the general population and is the third cause after gallstones and alcohol. HTG may be associated with acute pancreatitis as an epiphenomenon or as a precipitant thereof. Generally, more than 75% of pancreatitis induced by hypertriglyceridemia is due to secondary causes and although these are not sufficient to elevate triglycerides (TG) to cause pancreatitis, a preexisting defect is required to obtain a TG>1000 mg / DL to induce acute pancreatitis [2].

Material and Method

In order to identify the incidence of acute pancreatitis due to hypertriglyceridemia, a retrospective and cross-sectional observational clinical study was performed at Playa del Carmen Solidaridad Hospital, Quintana Roo. Mexico, for a period of 5 years (2012-2016). Risk factors for lipemic pancreatitis are diabetes mellitus, hypertriglyceridemia and systemic arterial hypertension. Admission criteria: acute inflammatory process of the pancreas accompanied by triglyceride levels between 500 and 999 m/dL and values>1000 mg/dL. (TG values<500 mg/dL were labeled as epiphenomena). The ultrasound is very deficient to assess the pancreatitis and, in our cases, showed it. As the study of image, we preferred the CTS because we allow staging the degree of pancreatitis. In the classification of the acute pancreatitis we include the Ranson, Acute Physiology and Chronic Health Evaluation II (APACHE II) and Glasgow scales and The CT severity index (CTSI) derived by Balthazar was used for description of CT findings in lipemic acute pancreatitis. The variables observed were: independent variables: diagnosis of pancreatitis, lipemic serum, serum amylase, lipase, triglycerides and total cholesterol. Dependent variables: age, sex, clinical history, severity ratings used, hospitalization days, morbidity and mortality. Statistic analysis Descriptive statistics were used; the qualitative variables were expressed in percentages; and the quantitative variables in means, medians and standard deviation.

Results

During the study period, 100 cases of acute pancreatitis of various etiologies were collected, 29 (29%) of which corresponded to acute pancreatitis of hypertriglyceridemic origin, of which 10 were female (34%) and 19 were male (66%), mean age 38 with age minimum of 18 and maximum age 98 years; History of risk: Diabetes mellitus type 1 one case (3.4%); Type 2 diabetes mellitus 27 cases (24%); History of alcoholism 9 cases (31%), positive smoking 4 cases (13.8%); Hypertriglyceridemia 27 cases (94%) and obesity 17 cases (59%). Gasometries performed only 15 cases (53%); lipemic serum 19 cases (65.5%). Laboratory test with high glucose, triglycerides, amylase. lipase, lactic dehydrogenase and gamaglutamil-transpepidase (Table 1). Ultrasound diagnoses in a poor method for the diagnostic of acute pancreatitis in our hospital (Table 2). CTS in 15

cases (54%) the Balthazar classification was revised [A=1, B=2, C=2 D=3, E=8] cases and 23 without tomography. Risk classification in five cases (17%): (APACHE 2, Bisap 2, and Marshall 1). Morbidity and mortality. (Table 3) Admission to the intensive care unit 4 cases (14%). They received medical treatment 27 cases (96%) and surgical treatment in one case (4%); in-hospital stays average six days; morbidity 15 cases (51%) and mortality one case (4%). The hypertriglyceridemic pancreatitis acute obtained second place after of the pancreatitis acute biliary in this population.

Discusión

It is known that Mexico is suffering from an epidemic of overweight occupying the first places in the world it due to the change in the nutritional habits of an autochthonous feeding based on vegetal abundant in vegetal fiber towards a diet based on fast food rich in fats and sugars. It is well known that dyslipidemia is related to acute pancreatitis as a precipitating factor and as an epiphenomenon and according to Frederickson's classification, it is hyperlipoproteinemia type I or familial chylomicronemia syndrome, which is related to this entity and characterized by Deficiency of lipoprotein lipase (LPL) and apoC-II, which results in alterations in lipolysis and deep elevations in plasma chylomicrons. Under normal conditions chylomicrons are disintegrated and withdrawn from circulation within 12 hours and in patients with LPL deficiency the chylomicrons rich in triglycerides persist in the circulation for several days. In our cases, no diagnosis was included such as Frederickson type I hyperlipoproteinemia [3,4].

The frequency of hyperlipidemia in patients with pancreatitis ranges from 12 to 38%, and of hypertriglyceridemia, between 4 and 53%; what is important is to define whether its presence is primary or causal, or secondary or consequence of other clinical conditions such as Diabetes mellitus, alcohol abuse, pregnancy or use of medications. Thus, in our patients, no distinction was made between hypertriglyceridemia and hyperlipidemia. However, triglycerides were obtained on routine laboratory tests in our hospital and their values were steadily elevated [5,6].

Chengen, *et al*., [7] in their study on biliary pancreatitis and hypertriglyceridemia, found that when the latter is elevated, the prognosis is poor. In our patients, this association was not observed because when triglycerides were elevated and lipemic sera with negative ultrasound were seen for bile duct lithiasis, we classified them as triglyceride pancreatitis. Nawaz H, *et al*., [8] report that hypertriglyceridemia is independently associated with multiorgan failure, regardless of the etiology of pancreatitis. However, hypertriglyceridemia itself is an etiology of acute pancreatitis, which does not prevent triglycerides from elevating in cases of pancreatitis of other etiologies as a systemic response to pancreas aggression since normally diabetes mellitus and obesity are strongly associated with pancreatitis of lipemic origin. Regarding the severity of pancreatitis, Xu C, *et al*., [9] reports that fatty liver affects the severity of pancreatitis, however, in our series we found eight cases (28%) of hepatic steatosis diagnosed by ultrasound without important influence on the development of complications of acute pancreatitis, although it was related to the diagnosis of acute triglyceride pancreatitis. High levels of triglycerides as well as total cholesterol were key for the diagnosis of acute pancreatitis of lipid etiology, however, Tariq H, *et al*., [10] recommend this laboratory test as a predictor of complications; In our series 15 complicated cases (52%) in this series and one case of mortality, which indicates that this etiology is more serious than those of biliary or alcohol etiology [11].

Table 1: Statistical analysis of laboratory tests in 29 cases of hypertriglyceridemic acute pancreatitis.

Test	Median	Average	Mode	SD	Maximum	Minimum
Leukocytes	10880	**11,231**	9300	3590	20300	5800
Glucose	150	**238**	188	186	826	84
CT	**255**	**339**	**NA**	**232**	**1091**	**85**
Triglycerides	**858**	**1447**	**NA**	**2034**	**7960**	**139**
Lipase	322	**1027**	NA	1701	7326	23
Amylase	441	**613**	NA	624	2353	48
AST	33	**59**	37	66	266	4.
ALT	31	**76**	54	134	572	6
LDH	388	**510**	NA	372	1253	138
Hto	41	**41**	47	6.8	53	28
ALP	109	**143**	NA	143	756	54
GGT	49	**353**	NA	845	3432	9
Calcium	8.5	**8.4**	8	1.4	11	6
BUN	10	**13**	8	9.4	38	0.6
Creatinine	0.67	**1**	0.8	1	4.6	0.27
Albumin	3.9	**3.8**	3.9	1	5.24	0.8
DB	0.3	**1.2**	0.7	2.6	11	0.09
IB	0.3	**0.4**	0.27	0.29	1.1	0.06
PT	13	**14**	13	1.8	17.2	11.3
PTT	31	**27**	NA	8.8	38.2	0.1

Abbreviations and normal values: Leukocytes (4000-11,500 x10^3/mm^3); Glucose (74-109 mg /dL); CT (<200 mg/dL); TG (150-199 mg/dlL); Lipase (13-60 U/L); Amylase (60-128 UI/L); AST aspartate aminotransferase (0-32 U/I); ALT, alanine Aminotransferase (0-33 U/I); LDH, Lactic dehydrogenase (103-227 U/l); Ht, hematocrit (male: 40.7 to 50.3%, female: 36.1 to 44.3%); CRP, C-reactive protein (+ or -); ALP, alkaline phosphatase (64-306 U/I); GGT, gamaglutamil-transpepidase (0-51 U/l); Calcium (8.5-10.5 mg/dL); BUN, blood urea nitrogen (10-20 mg/dL); Creatinine (0.4-1.4 mg/dL);

albumin (3.5-5.3 g/dL); DB, direct bilirrubin (0-0.20 mg/dL); IB, indirect bilirrubin (0,075 mg/dL); PT, Prothrombin time, (12.8-15.1/sec); PTT, partial thromboplastin time (24.3-35/sec.)

Table 2: Diagnoses by ultrasonography in 19 patients de 29 cases of hypertriglyceridemic acute pancreatitis.

Diagnostic	Cases	%
Hepatic steatosis	8	42.5
Acute pancreatitis	6	31.5
Colelitiasis	1	5.2
Pancreatic pseudocyst	1	5.2
Colitis	1	5.2
Normal study	2	10.4
Total	**19**	**100.0**

Table 3: Morbidity and mortality in 29 cases of hypertriglyceridemic acute pancreatitis.

Complications	Cases	%
Peripancreatic collections	4	14
Calcificaciones	1	3
Pancreatic pseudocyst	3	10
Pleural effusion	4	14
Multiple Organic Failure	3	10
Mortality	1	4
Without complications	13	45
Total	**29**	**100**

Other authors have apparently found normal laboratory results such as amylase and triglycerides at the beginning of the evaluation and should alert us in this respect not to subdivide this type of cases. [12]. The prevalence of hypertriglyceridemic pancreatitis in a private hospital in Mexico City was 10% much lower than that found by us in our study, which was 29% and ranked second in frequency after biliary pancreatitis [13]. With respect to the management of these patients Insulin-stimulated lipoprotein lipase is known to decrease serum Triglyceride levels. Hence insulin is indicated in these cases [14]. In a report by Tamez-Perez *et al*., [15] they used insulin. The dose used was 0.05 at 2 U/kg of body weight /hour. At an average of 2.5 days until the quantification of triglycerides were <400 mg / dL concluding that Insulin is a resource effective and safe treatment in patients with severe hypertriglyceridemia [16]. Other authors handled insulin plus heparin infusion with similar results [17]. Plasmapheresis is a treatment for pancreatitis due to hypertriglyceridemia that has shown its benefits in several studies. Same that it is not used routinely in our hospital and only in highly selected cases [18-20]. On the other hand, one of the late complications of severe acute pancreatitis is the formation of pancreatic pseudocyst and in our case series we find only one case behaving in the same way as the case of Monib SM., *et al* [21].

References

1. Speck L. 1949. **Fall von Lipämie.** *Arch des Verein J. wissensch Heilkunde*. 1865; 1:232. (Cited by THANNHAUSER, S. J.) In: Lipidoses: diseases of cellular lipid metabolism. The Oxford Medicine. Vol. IV. Oxford University Press, New York. 214(3)-214 (595) (part II, chap. VII-A). Ref.: https://bit.ly/2HtXelM

2. Weston N, Upul F, Varadarajan B. 2013. Hypertriglyceridaemia-induced

pancreatitis BMJ Case Rep. Ref.: https://bit.ly/2sGrcsv
3. Viruez Soto JL, Vera Carrasco O, Torrez Cruz KM. 2009. Pancreatitis aguda asociada a hipertrigliceridemia. Rev Med La Paz. 15: 32-35. Ref.: https://bit.ly/2sK9KDo
4. Gutiérrez-Grobe Y. 2012. Pancreatitis aguda por hipertrigliceridemia. Rev Invest Med Sur Mex. 19: 170-175. Ref.: https://bit.ly/2Uf1XcC
5. Pérez-Rada F de JM, Morales-Garza LA, Sánchez-Ávila MT, et al. 2004. Pancreatitis por hipertrigliceridemia: experiencia en el Hospital San José Tec de Monterrey. Revista AVANCES. 2: 9-11. Ref.: https://bit.ly/2Dvnw2K
6. Senosiain-Lalastra C, Tavío-Hernández E, Moreira-Vicente V, et al. 2013. Pancreatitis aguda por hipertrigliceridemia. Gastroenterol Hepatol. 36: 274-279.
8. Cheng L, Luo Z, Xiang K, et al. 2015. Clinical significance of serum triglyceride elevation at early stage of acute biliary pancreatitis. BMC Gastroenterology. 15: 19. Ref.: https://bit.ly/2DtPDzl
9. Nawaz H, Koutroumpakis E, Easler J, et al. 2015. Elevated serum triglycerides are independently associated with persistent organ failure in acute pancreatitis. Am J Gastroenterol. 110: 1497-1503. Ref.: https://bit.ly/2FL0qrm
10. Xu C, Qiao Z, Lu Y, et al. 2015. Influence of Fatty Liver on the Severity and Clinical Outcome in Acute Pancreatitis. PLoS ONE.10. Ref.: https://bit.ly/2MtvjkF
11. Tariq H, Gaduputi V, Peralta R, et al. 2016. Serum Triglyceride Level: A Predictor of Complications and Outcomes in Acute Pancreatitis? Canadian J Gastroenterol Hepatol. 2016. Ref.: https://bit.ly/2Udtf2S
12. He WH, Zhu Y, Zhu Y, et al. 2016. Comparison of severity and clinical outcomes between hypertriglyceridemic pancreatitis and acutepancreatitis due to other causes. Zhonghua Yi Xue Za Zhi. 96: 2569-2572. Ref.: https://bit.ly/2FWfHoD
13. Melnick S, Nazir S, Gish D, et al. 2016. Hypertriglyceridemic pancreatitis associated with confounding laboratory abnormalities. Citation: J Community Hosp Internal Med Perspect. 6. Ref.: https://bit.ly/2sJT9zr
14. Brizuela Alcántara DC, Pérez Gutiérrez Ome, Uribe Esquivel M, et al. 2011. Pancreatitis aguda por hipertrigliceridemia. Rev Invest Med Sur Mex. 18: 11-13.
15. Coskun A, Erkan N, Yakan S, et al. Treatment of hypertriglyceridemia-induced acute pancreatitis with insulin. Prz Gastroenterol. 10: 18-22. Ref.: https://bit.ly/2CIlaw9
16. Tamez-Pérez HE, Sáenz-Gallegos R, Hernández-Rodríguez K, et al. 2016. Terapia con insulina en pacientes con hipertrigliceridemia severa. Rev Med Inst Mex Seguro Soc. 44: 235-237. Ref.: https://bit.ly/2Ht4lKZ
17. Aryal MR, Mainali NR, Gupta S, et al. 2013. Acute pancreatitis owing to very high triglyceride levels treated with insulin and heparin infusion. BMJ Case Rep.
18. Gutiérrez Restrepo J, Muñoz Ortiz É, Arango Toro CM, et al. Pancreatitis aguda inducida por hipertrigliceridemia y tratamiento con plasmaféresis: reporte de un caso. Iatreia. 25: 391-397.
19. Galán Carrillo I, Demelo-Rodriguez P, Rodríguez Ferrero ML, ET AL. 2015. Double filtration plasmapheresis in the treatment of pancreatitis due to severe hypertriglyceridemia. J Clin Lipidol. 9: 698-702. Ref.: https://bit.ly/2MtF822
20. Nasa P, Alexander G, Kulkarni A, ET AL. 2015. Early plasmapheresis in patients with severe hypertriglyceridemia induced acute pancreatitis. Indian J Crit Care Med. 19: 487-489. Ref.: https://bit.ly/2RLGQBT
21. Sherif M. Monib Hany M. El-Barbary. 2013. Acute Relapsing Pancreatitis with Pseudocyst Formation due to Sporadic Hypertriglyceridemic Pancreatitis: A Case Report. Indian J Surg. 75: 340-344. Ref.: https://bit.ly/2TbcNQz