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Conventional High Volume Hemofiltration for Hypertriglyceridemic Pancreatitis

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Abstract

Several therapies have been proposed for the treatment of Hypertriglyceridaemic Pancreatitis; an uncommon cause (1-4%) of acute pancreatitis in which physio-pathology is unclear. Different combinations of medical drugs have been used to reduce hypertriglyceridaemia. Plasmapheresis is an invasive but effective alternative for severe cases but it is an expensive and not very accessible option in most of the centres. Other therapies have been considered like hemofiltration plus hemoperfusion but a standardised technique has not been established. Here, we report a 45-year-old female patient with recurrent episodes of pancreatitis related to hypertriglyceridaemia who was successfully managed in her last episode with conventional high-volume hemofiltration.

Introduction

There have been proposed several therapies for hypertriglyceridaemic pancreatitis (HTGP). All of them are based in lowering triglycerides in blood which seems to be the cause of the pancreas damage. Hypertriglyceridaemia is cause of acute pancreatitis in (1-4%) [1]. The mechanism for HTGP is postulated to involve hydrolysis of TG by pancreatic lipase and release of free fatty acids that induce free radical damage [2]. It is believed, as well, that large lipoproteins could block capillaries causing ischaemia and oedema of pancreatic cells. A genetic element could be present like a mutation of lipoprotein lipase. Serum triglycerides levels of more than 11.3 mmol/L (1000 mg/dl) are the range described that precipitate clinical signs of pancreatitis [3]. Minimal invasive methods have been used to decrease hypertriglyceridaemia like insulin and heparin [4] or insulin infusion with gem fibrozil and niacin [5]. Plasma triglycerides can also be lowered by plasmapheresis but is a very expensive and not very accessible treatment [6]. Other therapies have been considered like hemofiltration plus hemoperfusion [7], sequential blood purification therapy [8] or hemodialfiltration combined with resin-mediated absorption [9].

Objectives

To evaluate a simple an accessible treatment for patients with HTGP.

Case Report

A 45-year-old female presented to Accident and Emergency Department with progressive epigastric pain radiated to the flanks in the last week associated with 20 episodes of vomiting in the last 24 hours. The patient had similar episodes of pancreatitis in the past that required admission in the Intensive Care Unit. She has been treated with hypolipidemic drugs (fibrates and statins) in the last two years but despite having a good lipemic control, she was having about one episode of acute pancreatitis each year. No family history of dyslipidaemia or genetic disorder was found and there were no other precipitants for pancreatitis episodes. On examination, she was lethargic and dehydrated tachycardic 130/minute, tachypnoeic 30/minute, blood pressure 130/70 mmHg and temperature 37 ˚C; abdomen was distended and markedly tender without bowel sounds. Initial laboratory findings were a total leukocyte count of 8.4 10^{*}9/L, neutrophils 5.7 10^{*}9/L, platelets 149 10^{*}9/L, Amylase 470 IU/L and c-reactive protein 252 mg/L. Lipid profile was: Triglycerides 21.81 mmol/L, High density lipoprotein (HDL) 0.86 mmol/L, Total cholesterol 7.8 mmol/L and non HDL cholesterol 6.94 mmol/L. Coagulation tests were normal. CT abdomen revealed diffuse pancreatic parenchymal enlargement with surrounding retroperitoneal fat stranding and large amount of fluid consistent with acute pancreatitis without evidence of parenchymal necrosis. Due to severe metabolic acidosis (pH 7.0), oliguria and tachycardia she was admitted in Intensive Care Unit. Standard treatment was stablished with pantoprazole 40 mg, thromboprophylaxis with Tinzaparin 4.500 units and fluid replacement with sodium chloride 0.9% while nil by mouth.

Methods

Adsorption of triglyceride and hemofiltration

High volume veno - venous hemofiltration was performed by Aquarius System in which blood was driven through a highly permeable hemofilter type Aquamax HF 12 poly-ether-sulfone from Baxter (cut-off point of 30.000 Dalton); systemic anticoagulation was added in the hemofilter. Extracorporeal blood flow ranged from 250 to 360 ml/min (60 ml/Kg). Pre dilution mode was used. The principle of clearance in this model of continuous dialysis was convection. No fluid was taken from the patient to maintain her haemodynamically stable. The procedure was performed during 24 h. Albumin 20%, 100 ml, was administered three times during this procedure expecting that some fatty acids may bind to albumin [10], which has a higher molecular weight, and could be "trapped" better by the filter. One filter clotted and had to be changed in the first 12 h of the procedure.

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Table 1: Lipid profile before, during and after hemofiltration.

	Laboratory parameters			
	Before hemofiltration	During hemofiltration		After hemofiltration
	Admission	12 h	24 h	36 h
Triglycerides (mmol/l)	21.81	6.12	4.34	4.89
HDL cholesterol (mmol/l)	0.86	0.5	0.51	0.43
Total cholesterol (mmol/l)	7.8	3.1	2.5	2.4
Non HDL cholesterol (mmol/l)	6.94	2.6	1.99	1.97

Therapy criteria for initiation and cessation

Informed consent was obtained from the patient to perform this treatment and laboratory analysis before, during and after the procedure. It was previously explained to the patient that this therapy would be performed during 24 h but it could be stopped if not improvement was seen in 12 hours or any complication was observed.

Laboratory parameters studied

Lipid profile was measured just before starting the procedure, 12 h after starting hemofiltration, 24 h after starting hemofiltration and 12 h after hemofiltration cessation. Triglycerides, HDL, total cholesterol, non HDL cholesterol were the tests measured. Lipoprotein A and full blood count were also measured before and after hemofiltration.

Results

Therapy was stopped in the first 24 h. Abdominal symptoms had disappeared and vital signs were normalised: heart rate 88/min, respiratory rate 15/min. The most relevant benefit for the patient was that in 12 hours the abdominal pain had disappeared. Admission in Intensive Care was 48 hours compared with a 5 days admission in Intensive Care in the previous episode when treated with insulin, heparin and hypolipidemic drugs. No further complications were observed after filtration when discharged to the ward. Clinical signs and symptoms disappeared in 12 hours when triglycerides were reduced 71.93% from 21.81 mmol/L to 6.12 mmol/L. 80% reduction in triglycerides was achieved in 24h. No modification was observed in Lipoprotein A levels.

Discussion

The exact mechanisms involved in hypertriglyceridaemia - induced pancreatitis are unclear. Our patient was previously studied for genetic dyslipemia but not family or genetic disorder was identified. From the clinical history, only a passed episode of Hepatitis B was a possible etiology that triggered these episodes 20 years before, because no other conditions like pregnancy or previous hyperlipidaemia were present with the first episode. Lower triglyceride values shown in the table 1 could not have been due to another process because the patient did not have any other medical treatment for that purpose.

Wen-hua He, et al. with a similar management by early high-volume hemofiltration observed a significant improvement in triglycerides reduction and symptoms in about 9 hours, when triglycerides decreased to 500 mg/dl (5.65 mmol/L) [11]. In this case symptoms disappeared in 12 h with triglyceride levels at 530 mg/dl (6.12 mmol/L).

Our patient was treated successfully in previous episodes with heparin and insulin but clinical symptoms lasted several days and hospital admission was longer. There are not too many studies that have tried high-volume hemofiltration but it seems to be effective and safe in terms of triglycerides reduction and clinical improvement as we demonstrated in our patient.

Conclusions

High-volume hemofiltration is a good alternative for acute pancreatitis when hypertriglyceridaemia is the cause of these episodes. Reduction in triglycerides and clinical outcome resolved faster with better comfort for our patient. This therapy is cheaper than plasmapheresis and available in most of Intensive Care Units.

References

1. Khan AS, Latif SU, Eloubeidi MA (2010) Controversies in the etiologies of acute pancreatitis. JOP 11: 545-552.
2. Tsuang W, Navaneethan U, Ruiz L, Palascak JB, Gelrud A, et al. (2009) Hypertriglyceridemic pancreatitis Presentation and management. Am J Gastroenterol 104: 984-991.
3. DA Leaf (2008) Chylomicronemia and the chylomicronemia syndrome: a practical approach to management. The American Journal of Medicine 12: 10-12.
4. Jain D, Zimmerschied J (2009) Heparin and insulin for hypertriglyceridemia-induced pancreatitis: case report. Scientific World Journal 9: 1230-1232.
5. Sujani Poonuru, Sumedha R Pathak, Hemender S Vats, Ram D Pathak (2011) Rapid Reduction of Severely Elevated Serum Triglycerides with Insulin Infusion, Gemfibrozil and Niacin. Clin Med Res 9: 38-41.
6. Berglund L, Brunzell JD, Goldberg AC, Goldberg IJ, Sacks F, et al. (2012) Evaluation and treatment of hypertriglyceridemia: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab 97: 2969-2989.
7. Sun S, He L, Bai M, Liu H, Li Y, et al. (2015) High-volume hemofiltration plus hemoperfusion for hyperlipidemic severe acute pancreatitis: a controlled pilot study. Ann Saudi Med 35: 352-358.
8. Wang HL, Yu KJ (2015) Sequential blood purification therapy for critical patients with hyperlipidemic severe acute pancreatitis. World J Gastroenterol 2: 6304-6309.
9. Li MQ, Shi ZX, Xu JY, Lu B, Li JQ, et al. (2014) Hemodiafiltration combined with resin-mediated absorption as a therapy for hyperlipidemic acute pancreatitis. Cell Biochem Biophys 69: 699-702.
10. Van der Vusse GJ (2009) Albumin as fatty acid transporter. Drug Metab Pharmacokinet 24: 300-307.
11. He Wen-hua, Yu Min, Zhu Yin (2016) Journal of Clinical Gastroenterology 50: 772-778.