

LETTERS TO THE EDITOR

Insulin and heparin in treatment of hypertriglyceridemia-induced pancreatitis

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Received: 2007-03-07 Accepted: 2007-03-29

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Jain P, Rai RR, Udawat H, Nijhawan S, Mathur A. Insulin and heparin in treatment of hypertriglyceridemia-induced pancreatitis. *World J Gastroenterol* 2007; 13(18): 2642-2643

<http://www.wjgnet.com/1007-9327/13/2642.asp>

TO THE EDITOR

We read with great interest the case report, "Hypertriglyceridemia-induced pancreatitis: A case-based review" by Gan *et al*^[1] in the November 2006 issue of *World Journal of Gastroenterology*. We agree that in acute setting, pancreatitis due to hypertriglyceridemia (HTG) should be ruled out as it is a treatable and preventable condition. It needs to be treated conservatively along with measures to lower the triglyceride level. The various modalities to treat hypertriglyceridemia are plasmapheresis, insulin and heparin, purified apo C II, and fibrin acid derivatives^[2-5]. Plasmapheresis and purified apo C II infusion are not easily available. There is limited literature about the efficacy of intravenous insulin and heparin, both of which can enhance lipoprotein lipase activity.

We treated two patients with hypertriglyceridemia-induced pancreatitis with insulin and heparin. The details of the two patients are given in Table 1. Both of the patients had recurrent acute pancreatitis, one of them was a diabetic patient. Other etiologies such as gallstones, alcohol, drugs, hypercalcemia and trauma were ruled out.

Investigations revealed lipemic serum in both patients. The liver function tests and calcium were normal. The serum triglycerides were more than 1000 mg/dL in both the patients at admission. The chest roentgenogram and fundus examination were normal.

Both of the patients were treated with regular insulin in 5% dextrose infusion to maintain blood sugar levels between 150-200 mg/dL, and 5000 U heparin subcutaneously twice a day to lower the triglyceride level in addition to conservative

Table 1 Clinical and laboratory parameters of the patients with hypertriglyceridemia-induced acute pancreatitis

	Patients	
	1	2
Age (yr)/gender	55/Male	46/Male
Time to diagnosis (d) ¹	3	2
Random plasma glucose (mg/dL)	186	335
Total leucocyte count (cells/mm ³)	13700	18220
AST (U/L) (0-40 U/L)	41	82
ALT (U/L) (5-36 U/L)	26	87
Serum alkaline phosphatase (U/L) (< 310 U/L)	288	290
Serum amylase (< 125 U/L)	540	377
Serum lipase (< 195 U/L)	850	376
Serum calcium ² (9-11 mg/dL)	8.2	8
Serum albumin (gm/dL)	4	3.6
HbA _{1c}	5.90%	8.20%
Serum triglycerides (mg/dL)		
d 1	1808	3743
d 2	867	1804
d 3	570	1015
d 4	480	470
d 5	325	350
CT severity index	5	2

¹Time to diagnosis between symptoms and presentation. ²Corrected calcium levels.

treatment (nil per mouth, intravenous fluids, proton pump inhibitors, analgesics, antiemetics and antibiotics) followed by fenofibrate (160 mg OD) once the pain subsided.

Both the patients responded to the treatment, and the pain and vomiting subsided by d 3. Serum triglyceride decreased to less than 500 mg/dL by d 4 and normalized within 8 d. They remained asymptomatic at 12 and 24 mo of follow-up. The serum triglycerides level returned to normal and no further episodes of pancreatitis occurred after a follow-up of 12 and 24 mo with fenofibrate. The triglyceride levels of their parents and siblings were all normal.

In conclusion, enhancing LPL activity with insulin and heparin may be an effective alternative treatment modality for patients with HTG-induced acute pancreatitis. Long-term use of fenofibrate may normalize triglyceride levels and prevent the recurrence of pancreatitis.

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