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Case Report

Use of insulin infusion in management of hypertriglyceridemia-induced acute pancreatitis in a non-diabetic patient

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Abstract: It is estimated that 1-4 out of every 100 cases of acute pancreatitis are precipitated by hypertriglyceridemia. Insulin, heparin and plasmapheresis are amongst a few modalities utilized in management of these patients. Limiting dangerous complications and extended length of hospital stay in patients with hypertriglyceridemia induced pancreatitis is an important endeavor with a dearth of data for guideline directed therapy. We present the case of a patient without a history of diabetes mellitus that presented with hypertriglyceridemia induced pancreatitis and was treated with continuous insulin infusion at a rate of 0.1 units/kg/hour, which is higher than the previously reported insulin infusion dose of 0.05 – 2 units/kg/day. In doing so we were able to safely and efficiently reduce the patient's triglyceride level as well symptomology in less than 48 hours and show the potential of using this method in future patients. Further studies with increased number of subjects may further verify this higher dose of insulin as effective for treatment of HTG-induced pancreatitis.
Keywords: hypertriglyceridemia, Insulin, pancreatitis

INTRODUCTION:

In recent climate of patient centered care, efficient use of resources including minimizing hospital stay remains at heart of advancements in medicine. It is estimated that 1 to 4 out of every 100 cases of acute pancreatitis are precipitated by hypertriglyceridemia (HTG), defined as serum triglycerides (TG) > 1000mg/Dl [1]. Insulin, heparin and plasmapheresis are amongst a few modalities utilized in management of HTG-induced pancreatitis (HTGP). In continuance with Tamez-Perez *et al.;* who report reduction in TG to levels below 400 mg/dL through intravenous insulin infusion dosed 0.05 – 2 units/kg/day [2]. We present the use of continuous insulin infusion at a rate of 0.1 units/kg/hour for safe reduction of TG in a non- diabetic patient with HTG induced-acute or chronic pancreatitis.

CASE PRESENTATION:

44 year-old male with history of chronic pancreatitis suspect secondary to alcohol abuse, and hyperlipidemia (treated with omega-3 fatty acids and diet modification) presented to emergency department with chief complaint of abdominal pain x 1 day. Abdominal pain associated with nausea, non-bloody vomits and exacerbated with oral intake. Last alcohol use five days prior to presentation with consumption of 10 cans of beer, family history of uncle with recurrent pancreatitis and review of systems unremarkable for signs and symptoms of diabetes mellitus or pancreatic insufficiency. Physical examination remarkable for epigastric tenderness without rebound. Bedside blood draw grossly lipemic serum sample with white blood cell count of 16,100 cells/uL, hemoglobin/hematocrit indeterminate, glycosylated hemoglobin of 5.2%, lipase 582 U/L, triglyceride 4391mg/dL and serum chemistry as illustrated temporally in figure 1. Computed Tomography of abdomen and pelvis with intravenous contrast demonstrated "diffuse fat stranding" consistent with acute or chronic pancreatitis involving pancreas body and tail. Initially nothing per mouth diet instated, intravenous analgesics and resuscitation through normal saline boluses for total of two liters with transition to continuous rate of normal saline at 250mL/hr. At hour 10 of hospital admission, regular insulin drip started at 0.1units/kilogram/hour with goal to continue infusion till serum triglycerides less than 500 mg/dL; and concomitant administration of 300mL/hr Dextrose 10% in ½ normal saline plus 20mEq/L potassium chloride titrated to blood glucose 80 – 180 mg/dL, which in our patient ranged from 64 – 181 mg/dL. Electrolytes monitored with point of care blood glucose checks every hour, basic metabolic panel every 4 hours, serum triglyceride levels every 12 hours and complete blood count with differentials, magnesium and phosphorous every morning. Within 24 hours of admission, patient reported resolution of abdominal pain thus enteral

nutrition started with fat free diet. At 48 hour triglyceride check, patient's triglyceride level had decreased to 446 mg/dL thus regular insulin discontinued and patient started on gemfibrozil 600mg per oral twice daily. Fifty-four hours post admission, nutrition counseling including resources for alcohol cessation provided prior to discharge home with 30 day supply of gemfibrozil and instructions to follow up with primary care physician.

Table-1: Temporal trend of serum chemistry and triglycerides with continuous insulin infusion

Serum Chemistry	**At presentation**	**Onset of insulin drip**	**24**	**48**	**Discharge**
Sodium (135 - 145	131	135	136	139	137
Potassium (3.6-5.2	4.3	3.9	3.8	3.8	4.2
Chloride (101-111	101	103	108	108	109
Bicarbonate (22-29	20	23	22	22	20
BUN (9-21 mg/dL)	13	9	5	4	4
Creatinine (0.6 – 1.3	0.6	0.7	0.8	0.8	0.8
Glucose (70-110 mg/dL)	131	115	133	125	95
Anion Gap	10	9	5	9	8
Triglycerides (<150	**4391**		**919**	**446**	**437**
Total Cholesterol (0-200	522				243
LDL cholesterol (70-200	Not Calculated				Not Calculated
HDL cholesterol (30-70	21				31

DISCUSSION

HTG remains the third most common cause of acute pancreatitis after gallstones and alcohol in the United States. Mechanism of injury postulated secondary to hydrolysis of triglycerides to free fatty acids within the pancreatic mass leading to free radical generation in combination with hyper viscosity leading to ischemia. Literature supports use of high intensity insulin infusion in combination with heparin subcutaneous or continuous infusion for HTGP in patients with uncontrolled diabetes mellitus however we affirm the ability of continuous insulin infusion in reducing duration of hospital stay and TG levels in a non-diabetic patient.

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