Journal of Community Hospital Internal Medicine Perspectives

co-action PUBLISHING

CASE REPORT

Hypertriglyceridemic pancreatitis associated with confounding laboratory abnormalities

Stephen Melnick, DO*, Salik Nazir, MD, David Gish, MD and Madan Raj Aryal, MD

Department of Internal Medicine, Reading Health System, West Reading, PA, USA

We present the case of a 36-year-old woman who presented to our hospital with epigastric abdominal pain and tenderness. Laboratory evaluation identified high lipase, normal amylase, pseudohyponatremia, and relatively falsely low triglyceride levels (initial value of 2,329 mg/dl which on repeat was found to have corrected value of >10,000 mg/dl). The overall clinical picture was consistent with acute pancreatitis due to hypertriglyceridemia. The patient was commenced on IV insulin and eventually required plasmapheresis with good clinical outcome. This case highlights the importance of being cognizant of falsely low amylase and TG levels that can be present in patients with hypertriglycereidemic pancreatitis

Keywords: *hypertriglyceride*; *pancreatitis*; *epigastric pain*

*Correspondence to: Stephen Melnick, Department of Internal Medicine, Reading Health System, 6th Avenue and Spruce Street, West Reading, PA, 19611, USA, Email: Stephen.Melnick2@readinghealth.org

Received: 1 April 2016; Revised: 21 May 2016; Accepted: 31 May 2016; Published: 6 July 2016

Hypertriglyceridemia (HTG) is the third most common cause of acute pancreatitis after alcohol and gallstones. The risk of developing acute pancreatitis is approximately 5% with triglyceride (TG) levels above 1,000 mg/dl and 10–20% with TG levels above 2,000 mg/dl. (1). Hypertriglyceridemia-induced acute pancreatitis (HTGP) is associated with increased severity and complications when compared with pancreatitis caused by other causes (2). Various confounding laboratory abnormalities can be present in patients with HTGP including falsely low TG levels and normal amylase levels. This case highlights the importance of a clinically driven decision in the diagnosis and treatment of HTGP, as early clinical recognition is important to provide appropriate treatment and to prevent further complications and recurrences.

Case presentation

A 36-year-old female smoker presented to the emergency department with epigastric abdominal pain, radiating to her back, of 1 week duration. Her past medical history was significant for type 2 diabetes for which she was on oral hypoglycemic agents and insulin; presumed secondary HTG for which she was on atorvastatin and gemfibrozil; furthermore, she used medroxyprogesterone acetate for contraception. She had stopped all these medications except the contraceptive 12 months prior to her presentation. The patient has no history of alcohol abuse, thyroid dysfunction, or family history of HTG. On admission, all vital signs were within normal limits. Physical examination was normal except for epigastric tenderness and a BMI of 31.4 kg/m^2.

The initial relevant laboratory testing revealed sodium of 129 meq/l, potassium 3.7 meq/l, chloride 97 meq/l, BUN 10 mg/dl, creatinine 0.77 mg/dl (at baseline), bicarbonate 13.7 meq/l, anion gap 18 meq/l, glucose of 324 mg/dl, amylase 31 IU/l, lipase 206 IU/l, and TG 2,329 mg/dl with a previous baseline of around 2,000 mg/dl, serum calcium 9.0 mg/dl, TSH 1.182 μIU/ml, serum lactate 1.8 meq/l, negative urine ketones with serum beta-hydroxybutyrate 0.16 mmol/l, and negative urine pregnancy test. The abdominal computerized tomography scan showed no definite evidence of pancreatitis or any other pathology.

Despite unremarkable imaging, normal amylase, mild elevation of lipase, and a TG level that was mildly elevated from previous baseline, she was treated for possible HTGP with IV insulin based on the overall clinical picture. The differential diagnosis includes diabetic ketoacidosis (DKA); however, the patient lacked serum or urinary ketones. Regardless, IV insulin would treat both conditions of HTGP and DKA. The clinical impression of HTGP was further supported when her lipase increased to 370 IU/L and the TG levels on the initial sample was re-assayed resulting in a corrected reading of >10,000 mg/dl. The TG levels decreased to 6,069 mg/dl with IV insulin on day 5. However, given the worsening abdominal pain, plan for plasmapheresis to rapidly reduce the TG levels was taken.

Citation: Journal of Community Hospital Internal Medicine Perspectives 2016, **6**: 31808 - http://dx.doi.org/10.3402/jchimp.v6.31808

The TG levels decreased to 2,055 mg/dl after the first exchange and to 642 mg/dl after the second session, and her symptoms improved significantly.

Discussion

HTG is defined as fasting TG levels greater than 150 mg/dl and levels exceeding 1,000 mg/dl is defined as severe HTG (1). Use of injectable and oral contraceptives containing estrogen have been associated with HTG, but use of medroxyprogesterone, as in our patient, has not been associated with HTG (3). The exact mechanism by which severe HTG precipitates acute pancreatitis remains unknown, and various theories have been postulated (2).

HTGP should be suspected with severe HTG and clinical picture consistent with acute pancreatitis, which is established when two of the following three findings are present: epigastric abdominal pain; elevated serum enzyme, three or more times the upper limit of normal; or radiologic imaging consistent with acute pancreatitis (4). Our patient had various spurious laboratory abnormalities including normal amylase levels, pseudohyponatremia, and falsely low TG levels compared with the repeat testing on the same initial sample which was consistent with few previously reported cases (5, 6). Normal amylase levels can be present in patients with TG levels above 500 mg/dl due to interference with the calorimetric reading. Pseudohyponatremia occurs due to excess TGs in serum sample displacing water containing sodium. TG interference is due to anaerobic conditions associated with grossly lipemic plasma disrupting the enzymatic reactions associated with TG assays, which considerably underestimate the final value of TG concentrations. The solution for samples with surprisingly low results is to re-assay after diluting or using a smaller volume of sample (6). Although our patient had high lipase levels, there is literature on cases of HTGP patients with normal lipase levels(5), the reason being unknown (4). To the best of our knowledge, this is the second case in literature where falsely low TG levels were present on admission requiring repeated laboratory testing on the same initial sample for accurate results. The previously described case by Markota et al. (7) presented with falsely low TG levels and was treated with plasma exchange due to worsening clinical status and evidently lipemic serum.

Treatment of HTGP aims at reducing the TG levels with the use of IV insulin and plasmapheresis (8). Although a number of falsely low lab values (amylase, TG) were noted in our patient, the overall clinical impression of pancreatitis with history of elevated TG levels, IV insulin was started. Although, the TG levels decreased by 30%, at 72 hours of treatment, due to worsening abdominal pain, rapid reduction in TG levels by therapeutic plasma exchange was undertaken.

In summary, our case highlights the importance of being cognizant of falsely low amylase and TG levels that can be present in patients with HTGP. Hence, it is suggested that the diagnosis of HTGP be considered in patients with history of elevated TG and clinical picture consistent with pancreatitis, despite the presence of confounding laboratory abnormalities (falsely low amylase and TG).

Conflict of interest and funding

The authors have not received any funding or benefits from industry or elsewhere to conduct this study.

References

1. Berglund L, Brunzell JD, Goldberg AC, Goldberg IJ, Sacks F, Murad MH, et al. Evaluation and treatment of hypertriglyceridemia: An Endocrine Society clinical practice guideline. J Clin Endocrinol Metab 2012; 97: 2969–89.
2. Tsuang W, Navaneethan U, Ruiz L, Palascak JB, Gelrud A. Hypertriglyceridemic pancreatitis: Presentation and management. Am J Gastroenterol 2009; 2013: 984–91.
3. Berenson AB, Rahman M, Wilkinson G. Effect of injectable and oral contraceptives on serum lipids. Obstet Gynecol 2009; 114: 786–94.
4. Banks PA, Freeman ML. Practice guidelines in acute pancreatitis. Am J Gastroenterol 2006; 101: 2379–400.
5. Singh A, Shrestha M, Anand C. Acute pancreatitis with normal amylase and lipase – An ED dilemma. Am J Emerg Med 2016; 34: 940.e5–7.
6. Shephard MD, Whiting MJ. Falsely low estimation of triglycerides in lipemic plasma by the enzymatic triglyceride method with modified Trinder's chromogen. Clin Chem 1990; 36: 325–9.
7. Markota A, Knehtl M, Sinkovic A, Ekart R, Hojs R, Bevc S. Plasma exchange treatment for acute hyperlipidemic pancreatitis with falsely low levels of serum triglycerides – A case report. Transfus Apher Sci 2014; 51: 178–80.
8. Aryal MR, Mainali NR, Gupta S, Singla M. Acute pancreatitis owing to very high triglyceride levels treated with insulin and heparin infusion. BMJ Case Rep 2013; 2013.