Letter to Editor



# Failure of heparin associated to insulin in treatment of a hypertriglyceridemia-induced acute pancreatitis

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## Introduction

We report a case of death three days after diagnosing a hypertriglyceridemia-induced acute pancreatitis despite of heparin-insulin association therapy. A 44 years old female with newly diagnosed but untreated diabetes mellitus type 2, accusing epigastric abdominal pain associated to non-bilious and non-bloody emesis and thirstiness lasting for three days. On admission, patient was hypothermic at 35.9°C, confused, breath rate of 32/min, saturating 94% on room air, heart rate of 126/min and blood pressure of 87/68 mmHg. The patient family denied any history of alcohol consumption, jaundice, cholelithiasis or ulcer disease. The abdomen examination was significant for sensibility in the epigastric region without palpable spleen or any mass. Capillary dextrose was 4.47g/l and urines ketones were positives. Intensive management was started immediately. Physiologic saline serum 0.9% was administered by two peripheral venous catheters (16G). A tracheal tube after a crush induction by Etomidat and succinylcholine allowed invasive ventilation under 60% of oxygen to reach 98% of saturation. Sedation was realized with fentanyl only at first. Then, femoral venous catheter allows adrenalin administration at progressively dose to 0.5µg/kg/min for obtaining stabilized hemodynamic status. Her first labs were significant for blood lipase of 609 UI/L. White blood cells count was 17100/mm3 and serum triglyceride level was elevated at 11g/l. Serum anion gap was normal. Chest X-ray was normal. Abdominal CT scan showed a necrosis of the pancreatic parenchyma and peri pancreatic tissues without fluid collection.

These findings were consistent with a hypertriglyceridemia-induced acute pancreatitis. In addition to sedation and intravenous fluids, we started the patient on an intravenous insulin infusion with 5% dextrose titrated for keep blood glucose between 1.5 and 2 g/l after urines ketones being negatives. Furthermore, we started the patient on intravenous heparin infusion at 600 units/h. lipid apheresis was not available. One day after admission, unlike decreasing serum triglycerides level to 6 g/l, a multiple organ failure appears rapidly with anuria and increased oxygen requirements. Creatinine level firstly at 11 mg/l increased to 43 mg/l. Theses organs failures was associated to increased requirements of adrenalin. The patient died two days after admission in refractory hemodynamic shock and multiple organ failure. Actually, there are no concrete guidelines about treatment of hypertriglyceridemia induced pancreatitis. However, despite of dietetic approach or plasmapheresis, insulin infusion is proposed as a therapeutic alternative.[1] In fact, some cases of successful treatment of hypertriglyceridemia induced pancreatitis with insulin and heparin are published.[2–5] But the literature still limited about the efficacy of this association which may enhance lipoprotein lipase activity. Despite of being retrospective, Meng Jin et al.,[6] study showed that insulin-heparin therapy is effective and non-inferior to plasma exchange in lowering triglyceride levels.[6] In this case, the multiple organ failure was faster than insulin and heparin effects. More cases and studies still required for determining the place of this association in treatment of hypertriglyceridemia induced acute pancreatitis.

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## Conflicts of interest

The authors declared that there are no conflicts of interest.

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