CASE REPORT

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Severe hypertriglyceridemia-induced pancreatitis in a young female managed with plasmapharesis: A case report

Ahmed A. Khalifa[1], Sheref A. Elseidy[1*], Ahmed M. Khalifa[2]

[1]Department of Rheumatology, Ain shams university
[2]Department of Forensic Medicine and Toxicology, Ain Shams University

ABSTRACT

Hypertriglyceridemia (HTG) is postulated to be a rare cause of acute pancreatitis accounting for approximately 4% of causes. A serum triglyceride (TG) level above 1,000 mg/dl in the setting of pancreatitis and absent of other major causes is necessary to describe hypertriglyceridemia as a cause of acute pancreatitis. The mechanism involves the degradation of triglycerides by pancreatic lipase to release free fatty acids that induces free radical damage to the tissues. The major cornerstone in management is by reducing the triglyceride level to below 1000 mg/dL to achieve both the subsidial of the ongoing attack and the effective prevention of further episodes of pancreatitis. This can be achieved by a spectrum of treatment modalities ranging from dietary restriction of fat, administration of lipid-lowering agents to experiences with plasmapheresis and effective lipid pharesis however, the latter modality haven't been sufficiently discussed regarding therapeutic effects.

This case reports a young female, 22 years old female patient who is non alcoholic, non diabetic, non obese with negative history for gall stones or significant drug use, diagnosed with hypertriglyceridemic acute pancreatitis associated with acute kidney injury, and received one session of hemodialysis then plasmapharesis was initiated and she received 8 sessions with full dose of hypolipidemics, and responded only to plasmapharesis.

Key Words: Acute pancreatitis, hyperlipidemia, hypertriglyceridemia, plasmapheresis

Introduction

Hypertriglyceridemia (HTG) is the third most common cause of acute pancreatitis (AP) episodes (1-3). Workup of AP caused by hyperlipidemia is the same as in all types of pancreatitis. In addition to exclusion of heavy alcohol consumption, gallstone disease and secondary causes as drugs and iatrogenic causes as post ERCP pancreatitis, a positive family history of signs and symptoms of hypertriglyceridemia is necessary to steer the diagnosis. Current guidelines of management must include full dose hypolipidemic mainly fibrates, minimization of dietary fat consumption, insulin and/or heparin, administration of plasmapheresis and/or lipid pheresis have be found to be the most useful in reduction of TG levels, improving clinical signs and symptoms during the active phase of pancreatitis also effectively preventing further episodes of pancreatitis.(4,5).

In this case report a female patient with positive family history for hyperlipedemia, no past significant history of diabetes or history of alcoholism or gall stones or significant drug history, was diagnosed with hyperlipidemia induced pancreatitis and received one session of hemodialysis then plasmapharesis was initiated and she received 8 sessions with full dose of hypolipidemics. She responded only to plasmapharesis which reversed the acute attack and exponentially reduced her TGs levels, but recurrence occurred although multiple sessions of plasmapharesis were done.

Case Report

Young 22 year old female patient presented to our ER by epigastric pain, vomiting and fever.

O/E: she was fully conscious, feverish, with epigastric tenderness.

Pelvi-abdomenal ultrasound showed bulky heterogeneous pancreas with surrounding rim of free peripancreatic fluid collection and was diagnosed with pancreatitis. While withdrawing

***Corresponding Author:** Sheref A. Elseidy MBBCH, Department of rheumatology, faculty of medicine, Ain shams university, Abbassyia 11566, Cairo, Egypt, Phone: +2001001571545, Email: drsherif.elseidy@gmail.com
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her blood for laboratory tests, her blood looked milky white.

Laboratory results showed: elevated kidney functions (creatinine 3.4 mg/dL) and serum amylase 209 IU. She had hyperlipidemia, her TGs level was (1335 mg/dL) and serum cholesterol was (750 mg/dL). She did not improve on full dose of hypolipidemics, then developed acute kidney injury and her serum blood urea nitrogen was 73 mg/dL and serum creatinine was 6.2 mg/dL and received one session of hemodialysis followed by 8 sessions of plasmapharesis with full dose of hypolipidemics. She responded only to plasmapharesis. Her TGs and cholesterols started to decrease and reached 545 mg/dL and 194 mg/ dL respectively and she clinically started to improve and her serum amylase was 11 IU. Plasmapharesis was stopped. Unfortunately she deteriorated again with more severe symptoms and re-elevation of TGs and cholesterol. Computerized tomography abdomen revealed bulky heterogeneous pancreas with high density fluid collection and multiple air foci suggestive of necrotizing pancreatitis with moderate ascites and bilateral pleural effusion. The patient then had 2 attacks of generalized tonic clonic fits followed by deterioration of conscious level. She developed disseminated intravascular coagulopathy (DIC) and acute respiratory distress syndrome (ARDS) and unfortunately she passed away.

Discussion

Hyperlipidemia as a cause of acute pancreatitis was first discussed in 1865 by Speck, the relationship then has been thoroughly studied in numerous papers and reports. The mechanism behind which hyperlipedemia leads to pancreatitis is thought to be that serum chylomicrons or triglycerides are degraded by the pancreatic lipase to generate free fatty acids that causes pancreatic capillary inflammation and thrombosis within the pancreatic capillary bed leading to pancreatitis. Free fatty acids also either exerts a toxic effect on pancreatic acinar cells or damages the capillaries directly via free radical damage. Elevated concentrations of free fatty acids in the pancreatic capillaries causes capillary inflammation and thrombosis leading to ischemia, infarction and acidosis, also free fatty acids in this highly acidic environment activate pancreatic enzymes and thus initiating acute pancreatitis (6-9).

The two major signs of hyperlipidemic induced pancreatitis are high serum triglyceride level above 1,000 mg/dL and hyperchylomicronemia at the early stage of pancreatitis in a non obese non diabetic, non alcoholic patient with no other secondary cause for pancreatitis.

Current management guidelines include: hypolipidemic agents, fat restriction, heparin infusion, insulin and plasmapharesis. However, with heparin the effect on triglyceride reductions is transient and the patient experience reaccumulation of TGS with long term infusion (10,11), on the other hand insulin use in non-diabetics has no clear evidence (11).

Patients with HTG_AP are more susceptible to systemic inflammatory response syndrome and end organ damage than in other types of pancreatitis (8). Making therapeutic plasma exchange most beneficial to reduce serum triglyceride levels, as it was proposed that its efficacy is superior especially when there is evidence of shock or end-organ failure due to systemic inflammatory response syndrome (5).

In our case report the patient had a positive family history of hyperlipedemia and presented with severe type V hyperlipedemia in the acute phase at first of (1335 mg/dL), management with a trial of hypolipidemics was unsatisfactory and only plasma exchange lowered the lipid level and TGs with a value reaching (934mg/dl) after the first session and also improved the clinical status of the patient. But even though eight sessions were sufficient to control active disease they did not prevent recurrence in this patient with a more severe picture and superadded ARDS and DIC.

Plasmapharesis is the best treatment modality to reverse the acute phase of hyperlipidemic induced pancreatitis. However the role of plasmapharesis administration to prevent recurrence of acute pancreatitis remains unclear and further research is indicated.

Competing interests: The authors declare that they have no competing interests

Ethical considerations: Ethical approval for this case report was obtained from the Ethics Review Committee of Ain Shams faculty of medicine, informed consent was obtained following a full explanation of the procedure from first degree relative.

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