Case Report

Hypertriglyceridemia revealing acute pancreatitis: A case report

El Aidouni Ghizlane, a, b, d, Merbouh Manal, a, b, d, Taouihar Salma, a, b, d,
El Kaouini Abderrahim, a, b, d, Maarad Mohammed, a, b, d, Zaid Ikram, a, b, d, Aftiss Fatem-Zahra, a, b, d,
El Mezzioui Sanae, a, b, d, Bkiyar Houssam, a, b, d, Housni Brahim, a, b, c, d

a International Care Unit, Mohammed VI University Hospital Center, Oujda, Morocco
b Mohammed First University Oujda, Faculty of Medicine and Pharmacy, Oujda, Morocco
c Mohammed First University Oujda, FMP Oujda, LAMCESM, Oujda, Morocco
d Anesthesiology and Intensive Care Unit Department, Mohammed First University, Faculty of Medicine and Pharmacy, Oujda, Morocco

A R T I C L E   I N F O

Keywords:
Acute pancreatitis
Hypertriglyceridemia
Lipid metabolism
Insulin injection
Lipase
Plasmapheresis

A B S T R A C T

Introduction: Acute pancreatitis (AP) is considered one of the potentially rare complications of severe hypertriglyceridemia (HTG). Multiple treatment modalities have been suggested for patients with HTG-AP, such as permanent removal of TG by plasmapheresis, the use of insulin and heparin to enhance lipoprotein lipase activity and fibrate therapy, but the data remains limited.

Case management: we reported a case of 33-year-old women admitted for HTG-induced PA (HTG-AP). The patient had hypertriglyceridemia for 7 years under fibrate therapy as a medical history. On admission to our intensive care unit, his triglyceride level was 1060 mg/dl and the lipase level was 298 IU/L. An abdominal CT scan revealed stage E AP. The patient was treated with a low dose insulin infusion (0.05 unit/kg/h) with heparin and 5-day course of plasmapheresis, Fibrate therapy was maintained. His triglycerides went down to 130.9 mg/dl and she was discharged.

Conclusion: Early recognition of severe HTG can prevent progression to multiples diseases such as acute pancreatitis, can facilitate appropriate or even aggressive treatment to minimize complications of this.

1. Introduction

Hypertriglyceridemia is the third most common cause of acute pancreatitis. it is relatively rare and the diagnosis can be very difficult. However, this requires a high level of clinical suspicion with a good history as well as the presence of high levels of triglycerides (TG) [1].

Treatment of this acute pancreatitis due to fibrer hypertriglyceridemia involves lowering lipid levels through the use of plasmapheresis, hepafarin and insulin infusions to enhance lipoprotein lipase activity, and/or fibrate therapy [2].

This paper presented a case of 33-year-old women admitted with HTG-induced PA (HTG-AP).

2. Case management

A 33-year-old women admitted to our intensive care unit for HTG-induced PA (HTG-AP). The patient had hypertriglyceridemia for 7 years under fibrate therapy as a medical history. She was referred after 7 days of abdominal pain (epigastric pain) radiating posteriorly with notions of vomiting and deterioration of general condition. The patient reports the notion of having eaten a hearty meal rich in fat and carbohydrate (fatty food) while forgetting her diet. Clinical examination showed an abdominal tenderness elicited on palpation, normal hemodynamic state with tachycardia 109 pulse/minute, normal respiratory state with SpO2 98% on room air.

The complete blood count showed: high level of triglyceride with 1060 mg/dl and lipase level was 298 IU/L, high C-reactive protein with 320 mg/L, normal white blood cells 6390/mm3 and normal kidney
function. Aspartate aminotransferase (AST) and alanine aminotransf erase (ALT) were normal. Total cholesterol was 372.9 mg/dl, high-density lipoprotein (HDL), 13.8 mg/dl and Low-density lipoprotein (LDL) was not recorded. Hba1c was 7.9% and glycaemia level was 2.1 g/L. Her venous blood gas showed a pH of 7.51 and HCO3 of 22 mmol/L.

An abdominal CT scan revealed stage E of acute pancreatitis. Then she was diagnosed with HTG-AP.

On day 1 of stay, the patient was kept nil per mouth and started on a continuous infusion of normal saline at 135 ml/hr for 24 hours, she was treated with a low dose insulin infusion (0.05 unit/kg/h) by the endocrinologist with heparin. Besides, atorvastatin (40 mg once daily (OD)), fenofibrate (145 mg OD), and aspirin (100 mg OD) were given. On day 3, the patient showed no improvement leading to initiating plasma exchange (PLEX) therapy: for 5 sessions with exchange of 3 L of plasma each session.

On day 7, her lipase level reduced to normal (40 IU/L), and TG level came down to 130.9 mg/dl. Throughout her stay in the intensive care unit, oral nutrition was maintained, as was social interaction face-to-face with loved ones. Indeed, she was informed daily of his health state and she was discharged after 8 days of her stay in intensive care unit.

This case reports follows scarce guidelines [3].

3. Discussion

While AP has multiple etiologies, hypertriglyceridemia is considered the third most common cause after gallstones and alcohol. It is the underlying cause of pancreatitis in 7% of the population [4,5]. Hypertriglyceridemia can lead to acute pancreatitis as a precipitating agent of pancreatitis or as an epiphenomenon [6].

The secondary causes of hypertriglyceridemia are generally not sufficient to cause an elevated level of TG to cause pancreatitis, thus necessitating the need for a pre-existing defect. Indeed, it is necessary to have a TG level greater than 1000 mg/dl (or 20 mmol/l) to induce acute pancreatitis [6,7].

Yoshifumi and all showed in his paper that the ingestion of fatty foods in the two cases studied had an acute abdomen with a high level of inflammation parameters accompanied by hypertriglyceridemia and lipasemia [8]. As in our case, our patient had an acute abdomen following the ingestion of fatty foods with a high rate of hypertriglyceridemia, lipasemia and C-reactive protein.

On imaging, abdominal ultrasound could not detect acute pancreatitis, abdominal CT remains the exam of choice for confirmation of this pathology. As indicated in our case.

Treatment of hypertriglyceridemia-induced pancreatitis involves early management of acute pancreatitis and prevention of any of his complications. In the study by Tsuang and al; plasmapheresis has been used to facilitate the removal of chylomicrons from the bloodstream and to improve insulin sensitivity and glycemic control [9]. As our case report shows, our patient had benefited from 5 plasma exchange sessions with a clear improvement. However, several studies showed conflicting results regarding the effectiveness of the use of plasmapheresis, hence different studies are needed in this context [9]. Another effective therapy has been used to reduce TG in Jennifer study is the use of insulin and heparin infusions, either in combination or in monotherapy, as well as aggressive rehydration [10]. our patient was treated in the same way with a marked improvement in her clinical condition.

Our patient was satisfied from our medical care. The early measurement of serum triglyceride levels and pancreatic enzymes such as lipasemia, as well as the identification of chylomicrons could aid in the development of the treatment regimen and the prevention of the progression of complications due to this pathology.

4. Conclusion

HTG-AP is a serious disease requiring prompt diagnosis and treatment. In our case, HTG-AP was managed with an infusion of low dose insulin, heparin and the use of a 5-day course of plasmapheresis which prevented the progression of complications from pancreatitis. Several investigations are necessary to understand the efficacy of low dose insulin in the management of HTG-AP as well as the indication of plasmapheresis.

Ethical approval

The ethical committee approval was not required give the article type (case report). However, the written consent to publish the clinical data of the patients was given and is available to check by the handling editor if needed.

Sources of funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contribution

EL AIDOUNI Ghizlane: Corresponding author, study concept, Data collection, data analysis, writing review & editing. MERBOUH Manal: Contributor. Taouihar Salma: Contributor. El Kaouini Abderrahim: Contributor. Maarad Mohammed: Contributor. Zaid Ikram: Contributor. Aftiss Fatem-Zahra: Contributor. El Mezzioni Sanae: Contributor. BKIYAR Houssam: Supervision and data validation. HOUSNI Brahim: Supervision and data validation.

Registration of research studies

This is not an original research project involving human participants in an interventional or an observational study but a case report. This registration is was not required.

Guarantor

EL AIDOUNI Ghizlane.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Provenance and peer review

Not commissioned, externally peer-reviewed.

Declaration of competing interest

The authors state that they have no conflicts of interest for this report.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jamsu.2021.102914.

References

[1] Natasha Weston, Upul Fernando, Varadarajan Baskar, Hypertriglyceridaemia-induced pancreatitis, BMJ Case Rep. 2013 (2013), https://doi.org/10.1136/bcr.2013-008722.

[2] R. Garg, T. Rustagi, Management of hypertriglyceridaemia induced acute pancreatitis, BioMed Res. Int. 2018 (2018) 4721357.
[3] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, for the SCARE Group, The SCARE 2020 guideline: updating consensus surgical Case REport (SCARE) guidelines, Int. J. Surg. 84 (2020) 226–230.

[4] S.I. Gan, A.L. Edwards, C.J. Symonds, et al., Hypertriglyceridemia-induced pancreaticitc: a case-based review, World J. Gastroenterol. 12 (2006) 7197–7202.

[5] S.K. Kota, S.K. Kota, S. Jammula, et al., Hypertriglyceridemia-induced recurrent acute pancreaticitc: a case-based review, Indian J Endocrinol Metab 16 (2012) 141–143.

[6] D. Yadav, C.S. Pitchumoni, Issues in hyperlipidemic pancreatitis, J. Clin. Gastroenterol. 36 (2003) 54–62.

[7] S. Sandhu, A. Al-Sarraf, C. Taraboanta, et al., Incidence of pancreatitis, secondary causes, and treatment of patients referred to a speciality lipid clinic with severe hypertriglyceridemia: a retrospective cohort study, Lipids Health Dis. 10 (2011) 157.

[8] Yoshihumi Okura, Kozo Hayashi, Tetsuji Shingu, Goro Kajiyama, Yoshiyuki Nakashima, Keijiro Saku, Diagnostic evaluation of acute pancreatitis in two patients with hypertriglyceridemia, World J. Gastroenterol. 10 (24) (2004 Dec 15) 3691–3695, https://doi.org/10.3748/wjg.v10.i24.3691.

[9] W. Tsuang, U. Navaneethan, L. Ruiz, et al., Hypertriglyceridemic pancreatitis: presentation and management, Am. J. Gastroenterol. 104 (2009) 984–991.

[10] D. Jennifer, T. Mancell, J. Mancell, Hypertriglyceridemia-induced acute pancreatitis treated with insulin and heparin, Am. J. Health Syst. Pharm. 69 (2012) 213–216.