I. INTRODUCTION

Hypertriglycemia is defined as the presence of high plasma levels of triglycerides (usually > 5.6 Mmol/l). At such high levels, the risk of complications which include acute pancreatitis is high. Standard management of SHTG-induced pancreatitis include dietary modifications, lipid-lowering therapy, intravenous fluids, pain management and other supportive measures. However, these measures can take a while to control or reduce TG in severe situations, this prompts the need of the use of plasmapheresis as treatment option. We initiated early plasmapheresis in 2 cases who presented with SHTG-induced acute pancreatitis and observed a significant reduction in TG and eventual recovery of the patients.

II. CASE 1

38-year-old patient presented with acute onset abdominal pain for one day associated with vomiting. Had background history of fatty liver disease, T2DM, Alcohol misuse, recurrent depressive disorder, chronic pancreatitis and hypertriglycemia. She had problems with diet adherence at home because of social difficulties. On admission BP 142/90 mmHg, PR 108 bpm, T 37, RR 21, Sats 96% On RA, Weight 94.2 kg, BM 16.7. Was clearly in pain, Chest was clear, Normal heart sounds. Abdomen was distended, tender but no guarding. CT-Abdomen was reported as moderate acute pancreatitis, no evidence of gall stone or peptic ulcer perforation. No retroperitoneal collection. Blood samples were taken 4 times but could not be analyzed because samples were too lipemic to analyze. On the fourth sample, only TG were reported as >64.2 mmol/L. Patient was kept nil per mouth, was given Iv fluids and insulin and lipid lowering agents. Pain was managed with PCA. A decision was made to consider patient for urgent plasmapheresis. After one session of plasmapheresis, TG was measured and came down to 11.90. Patient made a remarkable recovery and was discharged 3 days later with further advise.

III. CASE 2

34-year-old patient who presented with abdominal pain. Had a background history of T2DM on insulin, alcohol misuse and Hypertriglycemia. On examination; BP PR T Sats, RR. Had reduced breath sounds in the lung bases on chest examination. Normal heart sounds, distended abdomen with generalized tenderness and guarding. CT scan abdomen was reported as appearances in keeping with necrotizing pancreatitis with splenic vein thrombosis. Initial blood samples were too lipemic to analyze hence no baseline values to use for monitoring. Patient was admitted in ITU with antibiotics, Insulin, Lipid-lowering agents, anticoagulation and pain management with PCA. Patient was considered for urgent plasmapheresis and was done a day after admission. After the plasmapheresis, there was an enormous drop in TG and patient made a remarkable recovery to discharge.
IV. DISCUSSION

Acute pancreatitis due to severe HTG can be difficult to manage and monitor medically as often there is no baseline results as samples are too lipemic to analyze. The complications if not managed well are serious. In light of this, an intervention that will safely reduce the levels of serum TG is essential for proper monitoring thereafter. Medical therapy for lowering serum TG take time to lower such high levels to acceptable levels meanwhile increasing the risk of complications in the patient.

Although there is limited evidence that the use of plasmapheresis reduces HTGP-related morbidity and mortality, we observed a very profound acute reduction in the serum TG in these 2 cases. Besides, the recovery after plasmapheresis of the patients was remarkable. Our patients made quick recoveries and probably had reduced length of stay in hospital. In both cases, with only one session, there was a TG level reduction of > 70% with an average hospital stay of 3 days after the plasmapheresis.

V. CONCLUSION

In as much as there is lack of evidence on long term benefits on using plasmapheresis, its use as acute intervention in managing SHTG-induced pancreatitis has demonstrated a good outcome in patients.

REFERENCES
