# Management of hypertriglyceridaemia with insulin in traumatic brain injury

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## Introduction

Hypertriglyceridaemia in critically ill patients is considered a therapeutic (medical) emergency and may lead to severe cardiovascular events and acute pancreatitis.

This case report describes the management of hypertriglyceridaemia on an intensive care unit in a 33-year-old man with a traumatic brain injury.

## Discussion

Hypertriglyceridaemia is a common metabolic complication in critical illness. Primary causes should be checked by taking a detailed family history. The identification and elimination

### Case report

A 33-year-old man weighing 95 kg was admitted to a neurological intensive care unit following a traumatic brain injury. His background medical history was insignificant except for a history of smoking. A non-contrast computed tomography scan had shown left frontal, temporal contusions and a left frontoparietal subdural haematoma. He was admitted for intracranial pressure monitoring, with deep sedation to reduce intracranial hypertension. This sedation protocol included an infusion of propofol at 2–4 mg/kg/hr, morphine at 5–10 mg/hr and midazolam at 5–10 mg/hr. As per the traumatic brain injury protocol of the department of anaesthesia at Beaumont Hospital, on day one of admission a lipid profile screening test was conducted, together with other routine blood tests. His triglyceride level was initially 0.9 mmol/litre which had increased to 2.2 mmol/litre by day two. The patient also developed diabetes insipidus which was treated with one dose of desmopressin.

On day three of admission, the patient's triglyceride level climbed to 3.6 mmol/litre leading to the discontinuation of the propofol infusion. A thiopentone infusion was commenced to control the intracranial hypertension, along with a muscle relaxant as per standard guidelines. The patient developed ventilator-associated pneumonia and was commenced on piperacillin with tazobactam. Weaning of thiopentone began on day four, guided by intracranial pressure monitoring. On days five, six and seven, his triglyceride level increased to 4.3, 7.1 and 9.2 mmol/litre, despite no ongoing trigger. The patient's aspartate transaminase, cholesterol and amylase levels, as well as his thyroid function tests, were within normal limits. Owing to the rising triglyceride level, enteral feeding was stopped, and intravenous fluid was commenced to reduce the fat load.

Owing to concerns about the rapidly rising triglyceride level in the absence of an ongoing precipitant, the authors actively managed the patient's hypertriglyceridaemia using an insulin infusion concurrently run with dextrose to prevent hypoglycaemia, which would be exceedingly injurious to a brain recovering from a traumatic injury. The insulin infusion was at a higher dose than what is prescribed for hyperglycaemia treatment. Insulin and dextrose infusions were started in two separate infusions but via the same connector and cannula, with insulin started at a rate of 3 units/hour and 50% dextrose at 9 ml/hour. The insulin infusion rate was adjusted according to the triglyceride level, whereas the dextrose infusion rate was adjusted according to the glucose levels. In the events of hypoglycaemia, the dextrose infusion rate was increased but the rate was decreased if the patient developed hyperglycaemia. The insulin infusion rate did not change according to the patient's glucose level. Following the brain protection protocol of their institute, by keeping the patient's blood sugar levels between 5-8 mmol/litre, the authors were eager to avoid even mild hypoglycaemia. On day eight, the patient's triglyceride level dropped to 6.6 mmol/litre with an insulin infusion rate of 5 units/hour and 50% dextrose at 20 ml/hour. On day nine, the patient's triglyceride level dropped to 2.7 mmol/litre (Figure 1). At this point, the insulin and dextrose infusions were discontinued. Atorvastatin 20 mg twice a day was prescribed, and enteral feeding was recommenced.

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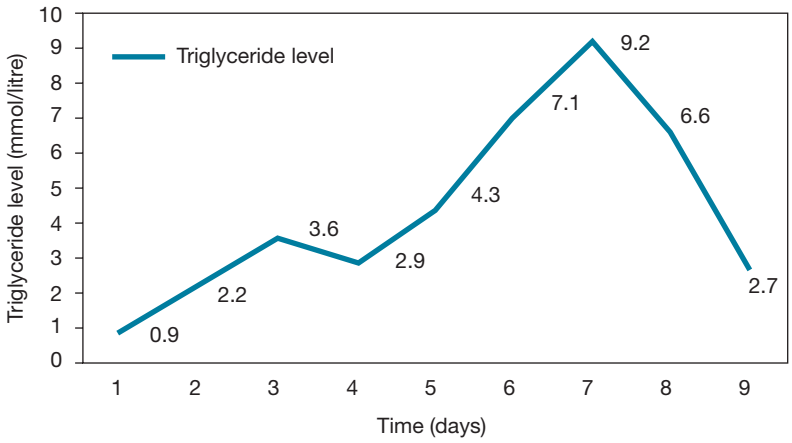


**Figure 1.** The patient's triglyceride level during the stay in the intensive care unit.

of secondary contributing factors are essential as hypertriglyceridaemia commonly occurs during insulin resistance, hypothyroidism and liver disease (Garg and Rustagi, 2018).

A propofol infusion is an emulsified lipid solution and has a known risk of increasing triglyceride levels, ultimately increasing the risk of developing hypertriglyceridaemia (Devaud et al, 2012). Thus, patients on a propofol infusion should undergo regular monitoring of their triglyceride levels. Sepsis leads to an increase in the hepatic production of triglycerides (Golucci et al, 2018).

Hypertriglyceridaemia should be treated to improve the prognosis of patients with coronary artery disease, and acute pancreatitis is significantly improved when treatment lowers the patient's triglyceride levels to the recommended targets (Simha, 2020).

In an intensive care unit, there are many available therapies for hypertriglyceridaemia, including heparin therapy, intravenous insulin and plasmapheresis (Garg and Rustagi, 2018).

Plasmapheresis lowers the lipid levels within hours in comparison to conservative therapy. However, plasmapheresis is associated with a higher risk of infection, an allergic reaction to the donor plasma and central venous access problems (Garg and Rustagi, 2018). In this study, the authors did not consider plasmapheresis because of the concerns of bleeding in patients with a considerable traumatic brain injury.

A heparin infusion can also be used to treat hypertriglyceridaemia as it stimulates the release of lipoprotein lipase from the endothelium into circulation, which subsequently leads to a decrease in serum triglyceride levels (Garg and Rustagi, 2018). Heparin infusion is associated with a higher risk of bleeding, so it is also contraindicated in this case.

Insulin is one of the therapies that can be used to manage hypertriglyceridaemia and can be administered subcutaneously or intravenously as a continuous infusion. In this study, the authors chose an intravenous infusion as it was easy to titrate the dose of insulin. Insulin promotes synthesis and activation of lipoprotein lipase activity (Thuzar et al, 2014). It effectively reduced the level of serum triglycerides as there was a significant drop in serum triglycerides by 30% and 75% over 1 and 2 days respectively. While insulin carries a risk of hypoglycaemia, this can be prevented in the intensive care setting with frequent monitoring and a concomitant dextrose infusion.

## Conclusions

Insulin appears to be a safe, effective, and inexpensive first line therapy for the treatment of hypertriglyceridaemia in a patient with a traumatic brain injury, and should be considered in cases where plasmapheresis is contraindicated.

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## Learning points

- Hypertriglyceridaemia in critically ill patients is considered a therapeutic (medical) emergency as it leads to severe cardiovascular events and acute pancreatitis.
- Propofol infusion is a major risk factor so patients receiving a propofol infusion should undergo regular monitoring of their triglyceride level.
- Insulin is a safe first line therapy for the treatment of hypertriglyceridaemia in a patient with a traumatic brain injury and should be considered in cases where plasmapheresis is contraindicated.

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