

Role of Plasma exchange in hypertriglyceridemia-induced acute pancreatitis

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Description: Hypertriglyceridemia accounts for up to 10% of all acute pancreatitis episodes¹. The role of plasma exchange (PEX) in reducing triglycerides (TG) was first reported in 1978 by Betteridge et al². There is sparse evidence in the role of PEX in hypertriglyceridemia-induced pancreatitis (HTG-AP).

It has been reported that a single session of PEX can reduce TG levels by up to 70%, with clinical and laboratory improvement². However, Chen et al. found no statistically significant improvement in mortality or morbidity with PEX³. He et al found that high volume filtration can reduce TG more efficiently than LMWH and insulin therapy, although not superior in terms of clinical outcomes and costs⁴.

There is minimal data regarding choice of replacement fluid but historically fresh frozen plasma (FFP) and Human Albumin solution (HAS) have been used⁵. Gubensak et al found no difference in mortality in patients who received PEX early (<36 hours after onset of pain) vs. late⁶.

We present two cases of severe HTG-AP requiring PEX.

Case one: A 34 years old obese Romanian male, with no known co-morbidities, presented with acute epigastric pain. He was diagnosed with radiologically confirmed acute severe pancreatitis secondary to hypertriglyceridemia (normal range amylase) leading to diabetic ketoacidosis. Initial blood tests done were reported as 'lipaemic'. An admission serum triglyceride done was 63.6mmol/L (normal range 0.4–1.9mmol/L) and had risen to 81.1mmol/L within 24 hours (with a total cholesterol level 24.2mmol/L; normal range <5.1mmol/L).

He was initially admitted to the intensive care unit (ITU) and managed supportively with analgesia, fluid resuscitation and diabetic ketoacidosis protocol. He was started on bezafibrate and atorvastatin. His renal function remained stable throughout.

He was given a single session of PEX with FFP (4L exchanges) on Day 3 of his admission. TG fell to 9mmol/L and subsequently to 3.0mmol/L. Cholesterol improved to 2.3mmol/L. He clinically improved after a 10-days hospitalisation.

Case two: A 34 years old Asian man, with multiple co-morbidities, presented with abdominal pain and vomiting. His background includes long-standing hypertriglyceridemia, dyslipidaemia, hypertension, type 2 diabetes mellitus, obesity and two previous episodes of pancreatitis (presumed hypertriglyceridemia induced). His initial TG and cholesterol were 74 mmol/L (normal range 0.4 – 1.9mmol/L) and 14.5 mmol/L (normal range <5.1mmol/L) respectively. He was already on fenofibrate and atorvastatin.

He was diagnosed with HTG-AP, with associated sepsis, paralytic ileus and acute kidney injury Stage 3. He was started on Omacor (Omega-3). He required 2 ITU admissions for continuous venovenous hemofiltration during his 14 days hospital stay. He received 2 sessions of PEX with HAS 4.5% (4L exchanges) on Day 5 and

Day 8. His TG levels improved to 13.8mmol/L after the first session and to 5.8mmol/L after the subsequent PEX. He clinically improved, with recovery of his renal function.

Both patients were followed up in lipid clinic.

Conclusion: These 2 cases demonstrate that PEX was effective in lowering TG and cholesterol in HTG-AP, with favourable clinical outcome, using two different replacement fluid. The number of PEX sessions was guided by their clinical condition and laboratory improvement.