Aims Triglycerides (TG) levels of >1000mg/dl (56mmol/l) are considered severe HTG. It is important to recognise the growing problem of HTG in children and the acute management. Methods We report a case of a teenager with initially well controlled DM. His control state has been deteriorated followed by subsequent development of severe hypertriglyceridaemia. We described the acute management with intravenous (IV) insulin and fluids, long term management with dietary changes and a lipid regulating medications. Results Following intensive dietetic regimen, the BMI reduced to 36 with significant control for the blood pressure in June 2021. However, the TG level raised to 7.1mmol/L with further reduction in HDL 0.9mmol/L and significant elevation in HbA1c 77. On assessment in July 2021, he complained of polydipsia. Random TG level was 79mmol/L (confirmed on a fasting sample), HDL 0.2mmol/L and serum glucose 18mmol/L. Serum amylase was documented as normal. Further laboratory tests were not suitable for analysis due to hyperlipidaemia. He was admitted for monitoring and urgent treatment of this weight gain, which may be linked to hypothalamic damage. One of the aims of this pilot, feasibility study was to investigate the downstream effect of CP on brain perfusion by using arterial spin labelling (ASL) as an indirect measure of neural metabolic activity in young people with CP. Methods Nine participants with CP (mean age= 14.6 ± 3.8y; mean BMI SDS= -0.60 ± 0.88) underwent two fasted, functional magnetic resonance imaging scans: one high resolution structural T1-weighted scan and one pseudo-Continuous ASL scan (pCASL) with multiple postlabelling delays. Data was processed with BASIL and analysed with RANDOMISE. Correlations between BMI SDS and brain perfusion were processed using Kendall’s tau analysis. Results Preliminary evidence shows greater perfusion in six of the seven priori regions of interest (ROI) (hypothalamus, insula, amygdala, nucleus accumbens, putamen and ventrofrontal cortex) in the control group compared to the CP participants (p<0.01). No difference was found in the temporal occipital fusiform cortex: the only ROI not associated with food reward processing. No significant correlations (p<0.01) were found between BMI SDS and brain perfusion in the above regions within the two groups. Conclusion These preliminary findings suggest there is reduced neural metabolic activity within and beyond the hypothalamus in those with CP. This is a shift from current literature which focuses primarily on the role of the hypothalamus. The findings have implications for the role of food reward processing within the pathophysiology behind CP-related obesity. There is need for further analysis to contextualise these results to determine this relationship with eating behaviour and obesity.