Objective To investigate the associations between triglyceride (TG) level and its variability with lung related infections, cancer, and mortality outcomes, a retrospective cohort-based was conducted.
Methods This was a retrospective cohort study of patients attending the Hospital Authority from 1st January 2000 to 31st December 2003 who were followed up until 31st December 2019 with at least three TG measurements in Hong Kong. Standard deviation (SD), root mean square (RMS), coefficient of variation (CV) were used as measures of variability. The primary outcome was Lung infection related mortality, and the secondary outcomes included lung cancer development, lung infections (bacterial, virus, and influenza infection), and all-cause mortality. Univariate and multivariate Cox regression models were conducted to identify the associations of TG level and its variabilities with the primary and secondary outcomes.
Results 47871 patients were included in the study (Median age 65.35 years old, 39.75% male). We found a high triglyceride baseline level is significantly associated with increased risk of all-cause mortality (HR: 1.11, 95% CI [1.104-1.116], p value < 0.0001), respiratory infection (HR: 1.14, 95% CI [1.13–1.15], p value < 0.0001), lung-infection associated mortality (HR: 1.14, 95% CI [1.13–1.15], p value < 0.0001) and lung cancer (HR: 1.10, 95% CI [1.07–1.12], p value < 0.0001). A further sub-analysis on specific respiratory infections revealed that high baseline TG has a similar risk incretion in bacterial, influenza and other viral infection (HR > 1, p value < 0.0001).
Conclusion A high serum triglyceride level is associated with increased all-cause mortality, lung cancer, respiratory infections and its associated mortality. Clinicians should be aware of such correlations and offer appropriate lipid control management to minimise these risks. Further studies should be conducted to investigate this relationship in other ethnical groups and whether TG-lowering medications may reduce the aforementioned adverse outcomes.
Conflict of Interest None

201 SUITABILITY FOR LOW-DOSE RIVAROXABAN BASED ON COMPASS TRIAL: A DISTRICT GENERAL HOSPITAL PERSPECTIVE

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Introduction COMPASS trial has recommended that low-dose rivaroxaban reduces major adverse cardiac and limb events among patients with stable atherosclerotic vascular disease. In the real-world practice, the recommendations from COMPASS trial can be used as a standard to recognise potentially suitable patients. The objective of our study was to establish the cohort of patients identified as COMPASS-eligible for low dose rivaroxaban.
Methods A health service evaluation of Cardiology Outpatients from Shrewsbury and Telford Hospital NHS Trust (SaTH) was carried out. The specific characteristics of the selected cohort included known stable atherosclerotic vascular disease while the inclusion and exclusion criteria incorporated in the COMPASS trial was used as a standard. The SaTH clinical databases from January 2021 were utilized to conduct a retrospective analysis to identify patients who could prospectively benefit from low-dose rivaroxaban.
Results Among the 99 patients who were found to have stable atherosclerotic vascular disease, 34 patients were deemed eligible for low dose rivaroxaban. Patients in our COMPASS-eligible group included 26 patients who were ≥65years of age while 8 patients were noted to be <65 years of age. Further analysis revealed that 94% of the patients had coronary artery disease as compared with only 6% found to have peripheral artery disease. In this cohort of patients, 79 % of the non-eligible patients were excluded due to underlying atrial fibrillation.
Conclusion About one-third of our cohort of patients met the COMPASS criteria and could potentially benefit from low dose rivaroxaban therapy. There is certainly a strong mandate for introduction of rivaroxaban following the COMPASS trial recommendations. Local protocols should be established to ensure that this window of opportunity to prevent major adverse cardiovascular and limb events is not missed in the clinical practice.
Conflict of Interest None

202 DEVELOPING STEM CELL-BASED MODELS OF FOAM CELL FORMATION TO STUDY NEW PATHWAYS OF LIPID CLEARANCE THAT REGULATE INFLAMMATION

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Foam cells play a critical role in driving atherosclerotic plaque formation and contribute to chronic inflammatory events within the arterial wall. These monocyte-derived cells are characterised by the excessive accumulation of oxidised low-density lipoproteins (ox-LDL) that are stored within lipid droplets. We have generated an in vitro model of foam cell formation from primary human monocytes and are able to detect lipid assimilation via flow cytometry and immunofluorescence imaging techniques. We have previously shown that foam cell formation can be inhibited in the presence of omega-3 polyunsaturated fatty acid (n-3 PUFA), eicosapentaenoic acid (EPA). This inhibitory action of EPA was linked to autophagy, with similar inhibition observed in the presence of triciribine, a known autophagy inducer. We aim to further investigate this by generating a genetically tractable stem cell-based model of foam cell formation that will enable genetic modifications of key autophagic pathway genes. We will also explore the role of endocannabinoids (derived from PUFAs) with the indication that EPA and its endocannabinoid derivatives can drive autophagy-mediated lipid efflux from foam cells in order to reverse their pro-inflammatory phenotype.
Conflict of Interest none