

Coronary Artery Bypass Graft, Percutaneous Coronary Intervention, Percutaneous Transluminal Coronary Angioplasty.

Results 8525 patients registered, 487 had T2DM (5.7%) and 68 patients were identified as having T2DM and ASCVD. 5 were excluded from analysis, one patient had left the surgery, three had no coronary atheroma on angiogram and one was mis-coded. Therefore 63 patients were used for analysis (12.9% of those with T2DM). Mean age 77.2 (SD 10.13), 41 male and 22 female. 58 patients had an eGFR >30, 29 patients had an eGFR >60. 45 patients were receiving at least one medication for T2DM (table 1). 18% of patients taking medication were receiving either GLP-1 or SGLT2i which confer CV benefit (figure 1). 75% of patients on dual therapy were taking DPP-4 inhibitors compared with 6.25% patients who were taking SGLT2i. DPP-4 inhibitors have no CV benefit. SGLT2i were typically used as a third line or later therapy (figure 2).

Conclusion SGLT2i were used sparingly and typically as a third line therapy or later in this cohort. DPP-4 inhibitors tended to be used as second line but lack any CV benefits. With a NNT of 39, concerted efforts to increase SGLT2i use in patients with T2DM and ASCVD have the potential to be an effective intervention in primary care that ultimately saves lives.

Conflict of Interest None

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AUTONOMIC FUNCTION IN RESISTANT AND MALIGNANT HYPERTENSION

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Background Enhanced sympathetic activity and reduced parasympathetic activity, assessed by heart rate variability (HRV) indices, have been linked to the pathogenesis of hypertension. Some studies showed that sympathetic and parasympathetic activity tend to restore after long-term hypertension exposure, as a result of cardiac output adaptation. It has not been known whether resistant hypertension (RH) and treated malignant hypertension (MHT) patients experienced similar restoration of autonomic balance.

Purpose To explore the autonomic changes in treated MHT, RH and normotensives subjects.

Methods We studied 23 patients with RH (57±11 y), 18 patients with treated MHT (54±13 y), and 23 normotensives controls (NC) (50±5 y). Time domain and frequency domain HRV indices of 5 minutes recordings were used to evaluate autonomic function. In the time domain, standard deviation of normal-to-normal R-R intervals (SDNN) reflect parasympathetic activity. Reduced SDNN is a marker of lower parasympathetic tone. The ratio between low frequency and high frequency spectrum was assessed in frequency domain. LF/HF increased is a marker of increased sympathetic activity.

Results The groups were matched by age and body surface area (all p>0.05). Time domain and frequency domain variables of HRV were not significantly different between three groups (p>0.05 for all) (table 1). Antihypertensive

Abstract 199 Table 1 Demographic and cardiovascular parameters

	NC(n=23)	MHT(n=18)	RH(n=23)	P-value
Office SBP (mmHg)	120 7	166 32*	163 21†	<0.001
Office DBP (mmHg)	78 8	97 17*	95 16†	<0.001
Duration of hypertension (years)	0[0-0]	7[5-8] *	8[5-10] †	<0.001
SDNN (ms)	48 [30-81]	47 [34-64]	48 [25-68]	0.89
rMSSD (ms)	33 [25-51]	30 [23-50]	27 [16-40]	0.26
pNN50 (%)	9 [3-30]	6 [3-18]	6 [1-17]	0.39
LF (ms ²)	809 [198-1849]	642 [276-955]	398 [186-935]	0.39
HF (ms ²)	314 [185-789]	327 [128-366]	203 [92-481]	0.49
HF (n.u)	34 18.3	33.1 16	36 22	0.37
LF (n.u)	66 18.3	67 15.7	64 22	0.91
LF/HF	1.9 0.4	2.02 0.3	1.77 0.5	0.97
RSA	14 3.2	14 3.2	14 3	0.89

Normally distributed data are expressed as (mean ±SD). Non-normally distributed data are displayed as median with interquartile ranges. *P <0.05 between healthy controls and malignant hypertension. †P <0.05 between healthy controls and resistant hypertension. DBP: diastolic blood pressure; HF: High frequency spectrum; LF: Low frequency spectrum; MHT: Malignant hypertension; NC: Normotensives control; pNN50: Percentage of successive differences between R-R intervals greater than 50 ms; RH: Resistant hypertension; rMSSD: Square root of the mean of the successive differences between adjacent R-R intervals; RSA: respiratory rate; SBP: systolic blood pressure; SDNN: Standard deviation of normal to normal R-R intervals.

RH was defined as office systolic and diastolic blood pressures exceeds 140/90 mmHg despite the use of three or more antihypertensive medications, one of which is a diuretic. MHT was defined as a diastolic blood pressure of 120 mmHg or more, accompanied by bilateral retinal haemorrhages and/or exudates, with or without papilloedema.

medications used were similar, except for the higher use of diuretics in RH group (100% vs. 67%, p<0.05). On linear regression, independent predictors of decreased SDNN were high creatinine level, decreased subendocardial viability ratio and increased central systolic blood pressure(p<0.05). On linear regression, independent predictor of high HF/ LF ratio was presence of left ventricular hypertrophy (β=-2.6, p=0.04).

Conclusion No differences were detected in HRV parameters between groups. These findings support the hypothesis of potential restoration of sympatho-vagal balance after prolonged hypertension exposure. Presence of target organ damage independently predicts decreased parasympathetic and increased sympathetic tone.

Conflict of Interest None

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ASSOCIATIONS OF TRIGLYCERIDE LEVEL AND VARIABILITIES WITH LUNG RELATED INFECTIONS, CANCER, AND MORTALITY OUTCOMES: A TERRITORY-WIDE COHORT STUDY

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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 increment 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

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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 ≥65 years 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

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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