Abstract 187 Table 1 Characteristics of GP referral documentation vs RACPC review

Characteristic	GP Referral (%)	RACPC (%)
Demographics	100	n/a
CAD history documented	93	100
Risk factors documented	92	100
Presence/absence of murmur commented on	100	100
Chest pain category:		
<i>Typical</i>	35	24
<i>Atypical</i>	35	45
<i>Non-anginal</i>	30	25
ECG present	100	n/a

27 days [IQR 16.5]). Actual median time from referral to diagnosis combining all investigation pathways was 77 days (IQR 28), or 81 days (IQR 25) for obstructive CAD. This reduced by a median 22 days with the hypothetical pathway. 23 (21%) patients with actual RACPC management had no CAD identified, representing pathways where 1 encounter (CTCA) may have been sufficient, which extrapolates to 138 less RACPC appointments annually (£21,666 in current NHS tariffs). 32 (30%) patients had non-obstructive CAD and were discharged via letter.

Conclusion A novel pathway with a referral-based triage to upfront CTCA ± subsequent RACPC would have been safe and efficient. The modelled reduction in hospital encounters would be cost effective and, importantly, enable the RACPC to review risk factors after identification of non-obstructive CAD. This improves treatment for a key group at risk of future events, particularly in those with aggressive CAD phenotypes on CTCA, whilst streamlining patients with obstructive CAD.

Conflict of Interest Nil

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IDENTIFICATION OF FAMILIAL HYPERCHOLESTEROLAEMIA IN PATIENTS PRESENTING WITH PREMATURE ACUTE CORONARY SYNDROME

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10.1136/heartjnl-2021-BCS.185

Introduction Familial hypercholesterolaemia (FH) is an under-diagnosed inherited disorder which confers substantial risk of premature cardiovascular disease. Over 90% of those with FH in the UK remain undiagnosed. Its importance was acknowledged in the 2019 NHS long term plan with an aim to identify 25% of FH patients by 2025. Patients with FH may present with an early acute coronary syndrome (ACS) which may represent the first opportunity for diagnosis. Identification of possible FH in this cohort enables specialist follow up, mitigation of risk and investigation of family members via cascade testing. We evaluated local disease burden, assessment of dyslipidaemia and identification of possible FH in a retrospective single centre review.

Methods A retrospective review was conducted of patients presenting with premature ACS to Royal United Hospitals Bath over a 1-year period.

Results We identified 96/573 (17%) patients who presented with ACS under age 60. Although 77/96 (80%) patients had cholesterol measured within 24 hours, only 13/96 (14%) patients had a full lipid profile checked. 14/77 (18%) had a total cholesterol >6.0, of whom 4/14 (29%), 4/14 (29%) and 7/14 (50%) had investigation for hypothyroidism, diabetes or alcohol excess respectively, as secondary causes for hypercholesterolaemia. 5/14 (36%) patients with total cholesterol >6.0 had a family history taken with ages of relatives at onset of CVD; 4/5 (80%) of those asked had a family history of premature CVD. Upon review of high risk cases, 8 were deemed appropriate for referral to lipid clinic, while a further 5 patients may have warranted referral in the presence of a significant family history, but this information was absent in the medical notes. A decision to refer to lipid clinic was documented in 4 patients' notes, although only 2 referrals were received.

Conclusion We identified missed opportunities in the identification of genetic dyslipidaemia (including FH) within a high-risk population. Further investigation through referral to lipid clinic was limited by a number of factors. Limited details of family history, particularly in patients with significant dyslipidaemia, is likely to contribute to under-identification of FH. We identified variable risk stratification and reduced clarity on the accepted threshold for referral to lipid clinic as key barriers to appropriate diagnosis. We propose the introduction of a degree of protocolisation through an 'ACS proforma' and 'lipid clinic referral decision aid' to facilitate appropriate identification, assessment and management of patients with possible FH.

Conflict of Interest None

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PREVALENCE OF COMORBIDITIES AND THE INCIDENCE OF CARDIOVASCULAR DISEASE IN THOSE WITH NON-CARDIAC CHEST PAIN

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10.1136/heartjnl-2021-BCS.186

Introduction Chest pain is a common presentation in primary care with many patients recorded with non-cardiac chest pain (NCCP), i.e. chest pain either unattributed to a cause or recorded with a non-cardiac reason. The relative prevalence of potential causes of NCCP in these patients and how these are related to risk of future cardiovascular disease (CVD) remains unclear. The aims of this study were to: (i) identify the

recorded prevalence of comorbidities potentially linked to chest pain in patients with NCCP in primary care; and (ii) determine the incidence of future CVD in those with NCCP.

Methods A cohort study was conducted between 2002-2015 using the Consultations in Primary Care Archive (CiPCA) database of pseudonymised primary care data. Patients aged >18 years with no prior CVD presenting with NCCP (index date) were included. Recorded comorbidities (osteoarthritis [OA]; lower back pain [LBP]; depression/anxiety; respiratory; reflux; and cancer) were identified between 24 months prior to and 6 months after the index date of NCCP. NCCP was categorised into those recorded with a non-cardiac cause at index date, unattributed chest pain with no comorbidity, and unattributed chest pain with comorbidity. Variation in patient characteristics were investigated. Incidence rates (IRs) of CVD events per 1,000 person years were calculated by NCCP category and stratified by time windows from the index date to CVD.

Results In total, 20,996 patients presented with NCCP with 9,000 (43%) recorded with a non-cardiac cause at index date, 5,993 (28%) unattributed no comorbidity, and 6,003 (29%) unattributed with comorbidity. The non-cardiac cause NCCP group were younger (44.1 versus 44.3 and 49.3 years) with a lower proportion of men (41.6% versus 42.4% and 54.1%) compared to the unattributed no comorbidity and unattributed with comorbidity groups, respectively. The most common comorbidity was respiratory (35%) followed by depression/anxiety (33%), reflux (15%), LBP (8%), OA (6%) and cancer (3%) in the unattributed with comorbidity group. The unattributed with comorbidity group had the highest incidence of CVD (IR 16.3, 95% confidence interval [CI] 15.0-17.6) vs. non-cardiac cause NCCP (IR 8.8, 95% CI 8.1-9.6) and unattributed no comorbidity groups (IR 11.7, 95% CI 10.7-12.8). The highest incidence of CVD occurred between 6-12 months after the index date (IR 45.0, 95% CI 32.7-61.8). Patients in the unattributed with comorbidity group with OA (IR 29.2, 95% CI 23.4-36.4) and cancer (IR 29.2, 95% CI 20.0-42.6) had the highest incidence of CVD, reflecting their older age.

Conclusions Depression and respiratory symptoms may be the most common reasons for NCCP in primary care. A higher incidence of CVD was observed for patients with unattributed NCCP and comorbidity, and in the first 6-12 months after presentation to primary care. This study highlights the importance of following up patients with unattributed chest pain symptoms.

Conflict of Interest N/A

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ASSOCIATION BETWEEN CAROTID DISTENSIBILITY AND HEART RATE VARIABILITY IN OLDER AGE

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10.1136/heartjnl-2021-BCS.187

Background Heart rate variability (HRV) is a prognostically relevant biomarker of autonomic regulation. Carotid stiffening that may precede hypertension, could theoretically inhibit the baroreceptor reflex, resulting in cardiac autonomic dysfunction and abnormal HRV indices. We investigated the association between carotid artery distensibility (the inverse of carotid arterial stiffness) and HRV in an older age population-based cohort.

Method Participants were from the 1946 Medical Research Council (MRC) National Survey of Health and Development (NSHD) British birth cohort. Carotid intima media thickness (cIMT) and carotid artery distensibility (exposures) were measured by ultrasound. Calibrated images and dedicated software automatically measured carotid distensibility, while semi-automatic edge detection software was used to measure cIMT. Time- and frequency-domain analysis of each participant's 6-minute resting 3-lead tachogram at age 60-64 years yielded the following HRV biomarkers (outcomes): standard deviation of normal-to-normal beats (SDNN); root mean square of successive differences (RMSDD); HRV triangular index; high-frequency (HF) power; low-frequency (LF) power; total power spectral density (PSD); power spectral density squared; normalised LF and HF power and HF:LF ratio. Generalized linear models (glm) were used, and adjustment was made for relevant demographic and health-related confounders including BMI, sex socio-economic position and plasma triglyceride levels.

Results A total of 896 participants were included (46.5% men, figure 1). On univariate analysis carotid distensibility, but not cIMT, correlated with the following HRV markers: SDNN, total PSD and HRV triangular index (markers of overall variability), RMSDD (marker of parasympathetic activity), HF power and LF power (markers of parasympathetic and sympathetic activity, all $p < 0.05$). These associations persisted in fully adjusted multivariable models (table 1): SDNN beta 0.63; 95% confidence interval (CI) 0.4,0.9; $p < 0.001$; RMSDD beta 0.60; CI 0.3,0.9; $p < 0.001$; HRV triangular index; beta 0.13;

Abstract 190 Table 1 Multivariable regression analysis. Showing results of multivariable regression analysis separately for each of the HRV biomarkers with exposure variables (including carotid distensibility) that were significant at univariate analysis ($p < 0.05$). Some boxes, for example in hypertension, do not have data because they were not significant at univariate analysis for the relevant HRV outcome. SDNN = standard deviation of normal-to-normal beats. RMSDD = root mean square of successive differences. HRV = heart rate variability. HF = high-frequency. LF = low-frequency. CI = confidence interval. BMI = body mass index. MI = myocardial infarction. SEP=socio-economic position

Variable	SDNN		RMSDD		HRV Triangular index		HF power		LF power		Total power spectral density	
	β Coefficient (95% CI)	p-value	β Coefficient (95% CI)	p-value	β Coefficient (95% CI)	p-value	β Coefficient (95% CI)	p-value	β Coefficient (95% CI)	p-value	β Coefficient (95% CI)	p-value
Carotid distensibility	0.63 (0.4,0.9)	<0.001*	0.60 (0.3,0.9)	<0.001*	0.13 (0.1,0.2)	<0.001*	8.04 (2.3,13.8)	0.006*	9.86 (4.7,17.0)	0.012*	25.40 (8.8,43.4)	0.008*
Sex	-2.97 (-5.1,-0.9)	0.005*	-0.04 (-1.9,1.9)	0.966	-0.31 (-0.8,0.1)	0.165	21.45 (-22.3,65.2)	0.336	-23.04 (-84.7,48.3)	0.474	-135.00 (-273.0,-2.5)	0.045*
BMI	-0.02 (-0.3,0.2)	0.857	0.11 (-0.1,0.4)	0.338	-0.02 (-0.1,0.03)	0.419	4.81 (-0.5,10.1)	0.076	0.15 (-5.8,7.0)	0.962	3.43 (-11.7,19.9)	0.671
SEP 1989	-0.08 (-2.6,2.6)	0.950	-0.56 (-2.8,1.8)	0.631	-0.25 (-0.8,0.3)	0.372	-21.10 (-76.1,33.9)	0.452	-23.04 (-84.7,48.3)	0.474	2.49 (-153.8,179.0)	0.976
Triglyceride	-1.32 (-2.8,0.3)	0.078	-1.46 (-2.6,-0.2)	0.017*	-0.26 (-0.6,0.1)	0.101	-38.26 (-69.6,-6.9)	0.017*	-36.83 (-61.8,4.7)	0.023*	-86.01 (-162.9,13.3)	0.043*
HbA1c	-0.12 (-0.2,0.01)	0.053			-0.03 (-0.1,-0.004)	0.019*			-1.88 (-3.7,1.1)	0.135	-5.70 (-10.7,2.2)	0.079
MI or angina	-3.62 (-7.5,0.9)	0.085										
Stroke			-1.12 (-1.6,-0.1)	0.001*	-0.22 (-0.4,0.03)	0.033*	-15.49 (-43.2,12.2)	0.272	-18.15 (-29.6,9.9)	<0.001*	-56.78 (-86.9,-0.4)	<0.001*
Hypertension			-1.27 (-3.2,0.7)	0.191								