different ML classifiers to reduce the risk of data leakage and overfitting. Mean cross validation (CV) accuracy, area under the receiver operating curve (AUC) and 95% confidence intervals (CI) are reported.

Results

The dataset comprised 82 carotid arteries from 41 symptomatic patients (41 culprit; 41 non-culprit) and 50 carotid arteries from 25 asymptomatic patients. CC and NC carotids showed significant differences in both first- and second-order features (IH Median: CC 618 (61); NC 646 (97), p<0.0005) and (GLDM Large Dependence High Grey-Level Emphasis: CC 3147 (1837), NC 4811 (2181), p<0.0001), respectively. Both CC and NC carotids had higher heterogeneity than asymptomatic carotids (GLDM Dependence Entropy: CC 6.59 (0.43), NC 6.57 (0.52), ASX 6.24 (0.26), p<0.0001). All ML classifiers performed better than a randomly guessing classifier (mean accuracy 33.3%) in this multi-class (n=3) classification task (Table 2; Figure 2), with the neural network achieving the highest accuracy of 69%, CI [61%, 77%] with AUC 0.82 CI [0.78, 0.86].

Conclusions

Textural analysis combined with machine learning on carotid CT scans reveals highly significant differences between symptomatic and asymptomatic patients, and between culprit and non-culprit carotid arteries within symptomatic patients. This approach could help identify patients at high-risk of stroke for aggressive medical therapy and surveillance.

Conflict of Interest

None

CORONARY FLOW RESERVE IS REDUCED IN ASYMPTOMATIC LIVING KIDNEY DONORS – RESULTS OF THE CHRONIC RENAL IMPAIRMENT IN BIRMINGHAM CORONARY FLOW RESERVE (CRIB-FLOW) STUDY

Introduction

Coronary microvascular dysfunction (CMD) is prevalent in chronic kidney disease (CKD), and may cause diffuse myocardial ischaemia and fibrosis, thus contributing to the elevated cardiac risk seen in CKD. Coronary flow reserve (CFR) is a marker of coronary microvascular function and can be measured using Doppler transthoracic echocardiography (TTE). Multiple studies have shown a graded inverse relationship between CFR and CKD stage, and this has prognostic significance.
Living kidney donors (LKD) provide a population of subjects with reduced estimated glomerular filtration rate (eGFR), without the confounding cardiovascular risk factors usually associated with CKD. This provides an opportunity to study the effects of an isolated reduction in eGFR (which is usually in the range of CKD stage 2-3) on CFR.

**Methods**

23 LKD and 25 controls of similar age and gender were studied. Individuals with diabetes, uncontrolled hypertension or ischaemic heart disease were excluded. All LKD were >12 months post donation. Subjects underwent adenosine stress TTE, myocardial contrast echocardiography (MCE) and CFR assessment. Peak diastolic coronary flow velocity (CFV) was measured in the distal left anterior descending artery at rest and at maximal hyperaemia (figure 1). CFR was calculated as the ratio of hyperaemic CFV/rest CFV.

**Results**

Controls and LKD were of similar age, gender and ethnicity (table 1). Rates of hypertension, smoking and hypercholesterolaemia were similar between the groups. Serum phosphate was significantly lower and detectable C reactive peptide was significantly higher in LKD.

Left ventricular mass index and indices of systolic and diastolic function were similar between the two groups (table 2). No subjects had wall motion abnormalities on stress TTE or perfusion defects on MCE.

CFR data was available in 22 controls and 23 LKD. CFR was significantly reduced in LKD compared to controls (mean CFR 3.8±0.6 vs 3.4±0.7, mean difference 0.4 95%CI 0.03-0.8, p=0.036) – figure 2. 6/23 (26%) LKD had CFR ≤ 2.7 (the lowest CFR value in controls.)

**Conclusions**

This is the first study of CFR in LKD. Similar to data in CKD, we have shown that a modest drop in eGFR is associated with reduced CFR in asymptomatic LKD. This suggests that isolated reductions in renal function may contribute to altered microvascular function, even in the absence of progressive CKD or cardiovascular risk factors. The higher prevalence of detectable CRP in LKD suggests that chronic inflammation may play a role in this process and this effect appears to persist even beyond the initial peri-operative period (median time from donation in LKD was 30 months). Reassuringly, no LKD had CFR<2, which is known to be a poor prognostic marker.

These early microvascular changes highlight the importance of long term follow up and aggressive risk factor management in LKD, to minimise any future cardiac risk. They also shed light on the pathophysiology of myocardial disease in CKD. Further work is needed to elucidate the mechanisms of microvascular dysfunction in LKD and CKD.

**Conflict of Interest**

None