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Introduction

Crossed cerebellar diaschisis (CCD) is frequently observed after supratentorial stroke, yet its relevance to long term activities of daily living (ADL) recovery remains unclear when CCD is quantified rather than dichotomized. This study aimed to evaluate whether quantitative SPECT-defined CCD severity is associated with late-phase ADL recovery from 3 to 12 months using the Korean modified Barthel index (K-MBI), and to explore mechanistic and predictive perfusion features beyond binary CCD status.

Methods

In this retrospective single center study of adults with first ever unilateral middle cerebral artery (MCA) infarction (n=53), the overall study workflow is summarized (Fig. 1). Brain perfusion SPECT was performed within 60 days of stroke onset (median 20; interquartile range (IQR) 15–26; range 6–57). CCD severity was quantified using the absolute asymmetry index (absAI). Late-phase ADL recovery was defined as $\Delta K\text{-MBI} = K\text{-MBI}(12\text{M}) - K\text{-MBI}(3\text{M})$ among participants with both 3 months and 12 months K-MBI available. Group differences and severity effects were assessed using nonparametric tests and Pearson/Spearman correlations; sensitivity analyses examined baseline-adjusted recovery. Exploratory analyses evaluated: (1) determinants of CCD positivity (logistic regression), (2) non-linear relationships between baseline national institutes of health stroke scale (NIHSS) and absAI (quadratic model), (3) the association between mean cerebellar perfusion and 12 months K-MBI, (4) data driven perfusion phenotypes (unsupervised clustering).

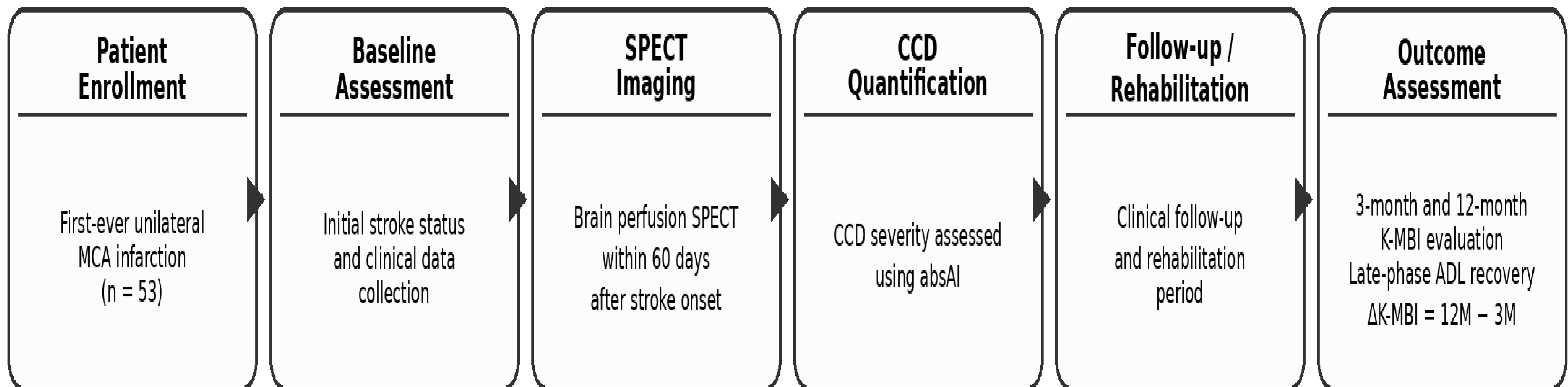


Figure 1. Patients with first-ever unilateral MCA infarction underwent brain perfusion SPECT within 60 days after stroke onset. CCD severity was quantified using absAI, and late-phase ADL recovery was assessed using the change in K-MBI between 3 and 12 months.

Results

CCD was present in 38/53 patients (71.7%). In the subgroup with follow-up data (n=33), late K-MBI gain (12M–3M) averaged 7.00 ± 12.45 in CCD-positive patients (n=27) and -7.33 ± 17.60 in CCD-negative patients (n=6) (Mann-Whitney U $p=0.049$). absAI correlated with $\Delta K\text{-MBI}$ (Pearson $r=0.488$, $p=0.004$, Spearman $\rho=0.463$, $p=0.006$) (Fig. 2). absAI was not associated with the absolute 12 months K-MBI score ($r=-0.013$, $p=0.941$), and the association with recovery magnitude remained after baseline adjustment (residualized $\Delta K\text{-MBI}$: Pearson $r=0.442$, $p=0.010$, Spearman $\rho=0.460$, $p=0.007$). Mechanistic/predictive analyses showed that baseline NIHSS was inversely associated with CCD positivity (odds ratio (OR) 0.89 per 1-point increase, $p=0.044$) and had a U-shaped association with absAI (quadratic term $p=0.006$, nadir at $AI \approx 13.5\%$). Mean cerebellar perfusion correlated with 12 months K-MBI ($r=0.36$, $p=0.037$), and three perfusion phenotypes differed in 12 months K-MBI ($p=0.004$).

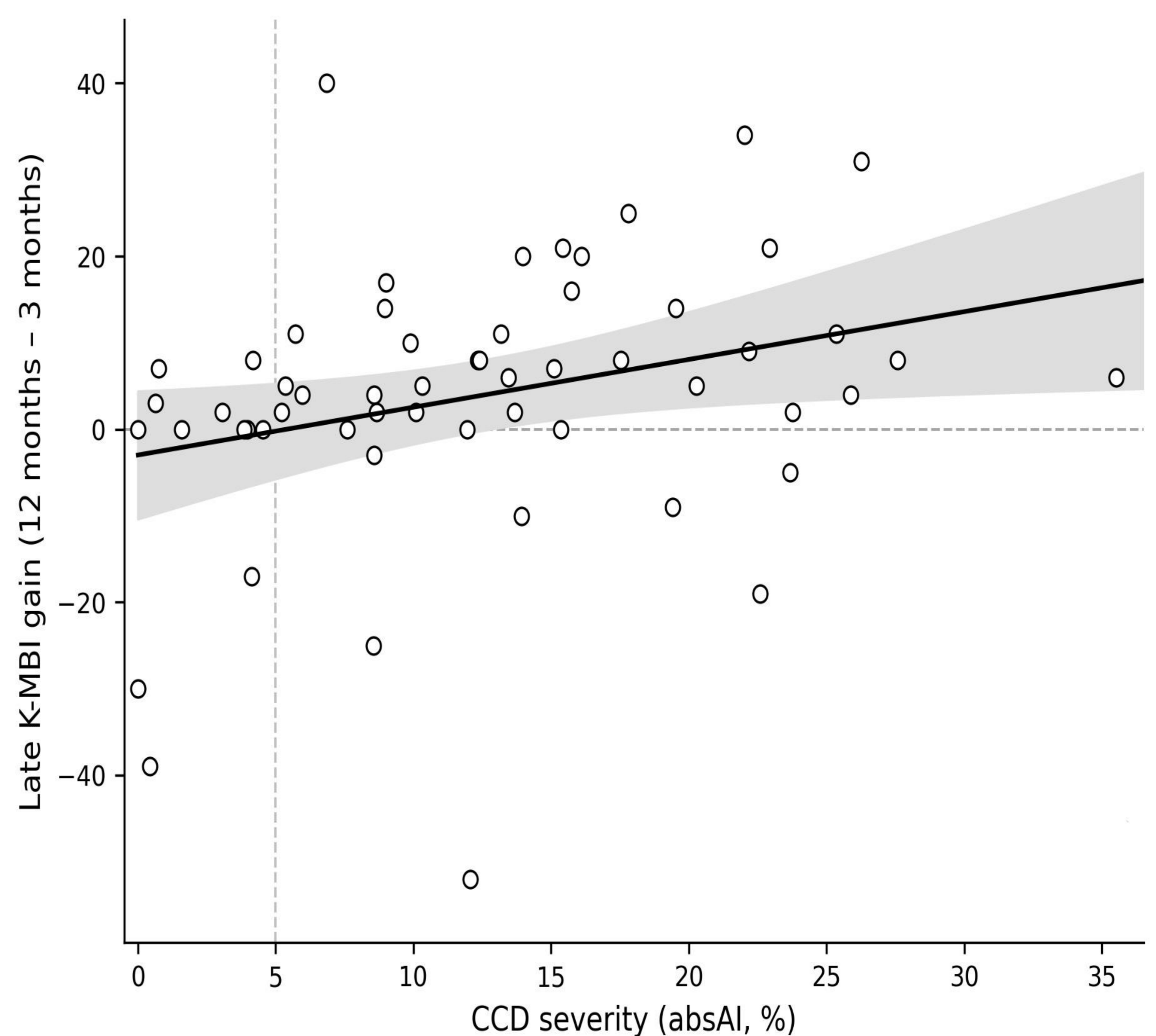


Figure 2. Association between CCD severity (absAI, %) and late-phase ADL gain ($\Delta K\text{-MBI} = K\text{-MBI}(12\text{M}) - K\text{-MBI}(3\text{M})$) in the study (n=33). The line indicates a least-squares linear fit. Pearson and Spearman correlations are reported.

Conclusion

Quantitative CCD severity was associated with the magnitude of late ADL gains rather than final 12 months ADL level. Exploratory perfusion metrics and phenotypes may better capture late phase ADL recovery sustainability, supporting prospective validation with standardized imaging timing and strategies to address missing follow-up.

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